Background Papers

Burden of Disease in India

National Commission on Macroeconomics and Health

EQUITABLE DEVELOPMENT HEALTHY FUTURE

NCMH Background Papers

Burden of Disease in India

NCMH Background Papers

Burden of Disease in India

National Commission on Macroeconomics and Health
Ministry of Health & Family Welfare, Government of India, New Delhi
September 2005

NCMH Background Papers—Burden of Disease in India (New Delhi, India), September 2005
Ministry of Health & Family Welfare, Nirman Bhawan, Maulana Azad Road
New Delhi 110011, India

Dosage schedules are being constantly revised and new side-effects recognized. The reader is thus strongly urged to consult the printed instructions of drug companies before administering any of the drugs recommended in this book. It is possible that errors might have crept in despite our best efforts to check drug dosages.

© 2005 National Commission on Macroeconomics and Health, Government of India

The report has been technically edited by
BYWORD EDITORIAL CONSULTANTS
New Delhi, India
e-mail: editbyword@yahoo.com

Printed at Shree Om Enterprises Pvt. Ltd., A-98/3 Okhla Industrial Area, Phase II, New Delhi 110020

Contents

Preface ix

Contributors xi

INTRODUCTION

Disease burden in India: Estimations and causal analysis 1

SECTION I: NATIONAL HEALTH PROGRAMMES: COMMUNICABLE DISEASES, AND REPRODUCTIVE AND CHILD HEALTH

National Health Programmes

Programmes for the control of leprosy, tuberculosis and malaria 9
AVTAR SINGH DUA

Burden of tuberculosis in India for the year 2000 24
TUBERCULOSIS RESEARCH CENTRE, CHENNAI

Causal analysis and treatment protocols for tuberculosis 26
TUBERCULOSIS RESEARCH CENTRE, CHENNAI

Causal analysis and treatment protocols for vector-borne diseases: Malaria, dengue, Japanese encephalitis, kala-azar, lymphatic filariasis 30
VECTOR CONTROL RESEARCH CENTRE, PONDICHERRY

HIV/AIDS

Controlling the HIV/AIDS epidemic in India 44
YING-RU J. LO, PADMAJA SHETTY, D.S.C. REDDY, SALIM HABAYEB

HIV-1 trends, risk factors and growth in India 58
RAJESH KUMAR, PRABHAT JHA, PAUL ARORA, NEERAJ DHINGRA, INDIAN STUDIES OF HIV/AIDS WORKING GROUP

Causal analysis and treatment protocols for sexually transmitted infections, HIV/AIDS and opportunistic infections 74
NATIONAL AIDS RESEARCH INSTITUTE, PUNE

Maternal and Child Health

Review of women and children's health in India: Focus on safe motherhood 85
RAJANI R. VED, AVTAR SINGH DUA

Causal analysis and treatment protocols for maternal complications 112
SARALA GOPALAN, SHALINI GAINDER

Components of under-five mortality trends, current stagnation and future forecasting levels 152
ARVIND PANDEY, B.N. BHATTACHARYA, D. SAHU, REHENA SULTANA

Newborn and child health in India: Problems and interventions 179
SIDDARTH RAMJI

Estimation of the burden of diarrhoeal diseases in India 182
NATIONAL INSTITUTE OF CHOLERA AND ENTERIC DISEASES, KOLKATA

Causal analysis and treatment protocols for childhood diseases 188
SIDDARTH RAMJI

SECTION II: NON-COMMUNICABLE DISEASES

Vascular Diseases

Forecasting vascular disease cases and associated mortality in India 197
A. INDRAYAN

Suggested treatment for selected cardiac disorders at secondary-level health care facilities in low-resource countries 216
CENTRE FOR CHRONIC DISEASE CONTROL, NEW DELHI

Cancers

Cancer: Current scenario, intervention strategies and projections for 2015 219
M. KRISHNAN NAIR, CHERIAN VARGHESE, R. SWAMINATHAN

Mental Health

Mental, neurological and substance abuse disorders: Strategies towards a systems approach 226
GURURAJ G., GIRISH N., ISAAC M.K.

Asthma and COPD

Economic burden of asthma 251
K.J.R. MURTHY, J.G. SASTRY

Economic burden of chronic obstructive pulmonary disease 264
K.J.R. MURTHY, J.G. SASTRY

Oral and Dental Diseases

Oral and dental diseases: Causes, prevention and treatment strategies 275
NASEEM SHAH

Blindness

National Programme for Control of Blindness 299
AVTAR SINGH DUA

Blindness estimations, projections and service delivery 305
MURALIKRISHNAN R., PRAVEEN KRISHNA R., THULASIRAJ R.D., DAMODAR BACHANI, SANJEEV GUPTA, G.V.S. MURTHY

Appendix to Section II

Services, equipment, personnel and drugs required at different levels of health care for non-communicable diseases 318
BARIDALYNE NONGKYNRIH, CHERIAN VARGHESE

SECTION III: INJURY

Injuries in India: A national perspective 325
GURURAJ G.

ANNEXURE

Annexure A: Causes of various diseases/health conditions 351
NATIONAL COMMISSION ON MACROECONOMICS AND HEALTH, NEW DELHI

Annexure B: Interventions for the management of diseases/health conditions at different levels of care 362
NATIONAL COMMISSION ON MACROECONOMICS AND HEALTH, NEW DELHI

Preface

In pursuance of the recommendations made by the Commission on Macroeconomics and Health, India established the National Commission on Macroeconomics and Health (NCMH) in March 2004. The main objective of the NCMH was to establish the centrality of health to development and make an evidence-based argument to increase investment in health. The Terms of Reference of the NMCH were mainly centred on identifying a package of essential health interventions that ought to be made available to all citizens and also list systemic constraints that need to be addressed for ensuring universal access to this package of services. The NCMH was also to indicate the resources required and targets that ought to be achieved by 2015.

One of the Terms of Reference of the NCMH was to come up with a baseline of the estimated prevalence of diseases in India, particularly those that disproportionately affect the poor now or have the potential to do so in the future. Based on such estimations, the Commission was to indicate targets that could be achieved within a specific time-frame. Accordingly, the NCMH invited leading experts in the country to assist in identifying those diseases/conditions that were responsible for high levels of mortality and morbidity and, if unchecked, could have ruinous implications for a majority of households in India. The experts were also given three other tasks: (i) to project the disease burden in a decade from now, assuming the current status quo in terms of policy attention and investment levels; (ii) identify the proximate, direct and indirect causal factors that, if tackled adequately, could substantially reduce disease incidence and thereby enhance welfare; and (iii) provide a minimal standard treatment protocol listing the interventions that ought to be undertaken at different levels of care to avert death and reduce progression of disease. This information was to enable us to cost the interventions and arrive at the quantum of investment required to achieve the aspiration of universal access to essential health interventions.

The experts identified 17 diseases /conditions that public policy needed to take note of on priority. The list included both the set of pretransition diseases or diseases of underdevelopment as they disproportionately affected the poor more and post-transition diseases or diseases of affluence, normally referred to as lifestyle diseases, which are believed to affect the rich more. Based on an exhaustive literature review, the experts attempted to provide a baseline of disease prevalence today and causal analysis indicating the various direct and indirect factors that contributed to the persistence of these diseases. Surprisingly, we found it difficult to provide even current-level estimates for diseases/conditions that are being implemented with substantial donor funding under the National Health Programmes. We, therefore, could not come up with any estimates for malaria or other vector-borne diseases, reproductive health, several childhood diseases such as respiratory infections, etc. Even for tuberculosis (TB), arriving at any projections for 2015 under the emerging scenario of the rising HIV/AIDS epidemic was impossible. For these reasons it was difficult to come up with any specific targets to be achieved within a limited time-frame.

Likewise, we were unable to find any studies or research which provided evidence demonstrating the efficacy of specific interventions under a programme in Indian conditions and among different population groups. This inhibited us from being able to identify those sets of interventions that ought to be accorded high priority to achieving an end goal, for example, reduction in infant mortality, or maternal mortality or malaria incidence, etc.

The second lesson that this exercise threw up was the high levels of prevalence of non-communicable diseases such as asthma, chronic obstructive pulmonary disease (COPD), hypertension, diabetes, mental problems, injuries, etc. Some household surveys also conclusively suggested that these diseases affected the poor as well as the not-so-poor segments of the population. These conditions and diseases have received no policy attention as they have been believed to be of little consequence to the poor. That is no longer true. In fact, there is an urgent need to pay attention to formulate appropriate policy responses to these diseases—their prevention as well as treatment—as these diseases are very expensive to treat, well beyond the means of the poor and most lower middle classes and occupational groups in the informal sector. Non-attention carries the implication that in the absence of any social insurance or risk protection, the poor either die for want of care or get impoverished when they do attempt to seek care. Since these diseases affect the younger age groups, the stress to such families could be immense, necessitating a public policy response—either by improving its public provisioning by providing free treatment and/or by having social health insurance policies in place.

A more cost-effective approach to disease containment is to prevent its occurrence in the first instance. Prevention can be achieved by well-organized health education and health information dissemination campaigns at the local level and

through effective use of the mass media; by enforcing regulations and using financial incentives to modify behaviour towards health-enhancing habits such as a healthy diet, exercise, use of seat belts or helmets, reducing dependence on alcohol or tobacco, etc. Several diseases such as diarrhoea can also be drastically reduced by enhancing access to safe water or promoting the habit of washing hands with soap, diseases like polio or measles can be altogether eliminated by immunization campaigns which are equally inexpensive. Clearly then, it is in this area of prevention of diseases and promotion of good health values that public policy should pay attention to.

The experts gave us their unstinted support. We would like to thank all authors and reviewers for having taken time from their busy schedules to help us. We would also like to thank several experts who attended our consultation meetings and gave us the benefit of their advice. To each of them we owe a debt of gratitude. We do hope that the work done will stimulate further research and enable evidence-based policy formulation. If that is indeed done, the efforts put in will not have been in vain.

K. SUJATHA RAO
Secretary
NCMH
New Delhi

Contributors

Authors

ABHAYA INDRAYAN
Professor of Biostatistics
Division of Biostatistics and Medical Informatics
University College of Medical Sciences
Dilshad Garden, Delhi 110095

ARVIND PANDEY
Director
Institute for Research in Medical Statistics (IRMS)
Indian Council of Medical Research (ICMR)
Medical Enclave, Ansari Nagar
New Delhi 110029

AVTAR SINGH DUA
Assistant Professor, Department of PSM
SMS Medical College
Jaipur

B.N. BHATTACHARYA
Professor (PSU), Indian Statistical Institute
203, B.T. Road
Kolkata 700108

BARIDALYNE NONGKYNRIH
Assistant Professor
Centre for Community Medicine
All India Institute of Medical Sciences (AIIMS)
Ansari Nagar, New Delhi 110029

CHERIAN VARGHESE
Coordinator (Non communicable diseases and mental health)
World Health Organization (WHO) India Office
New Delhi

D. SAHU
Research Officer
IRMS, ICMR
Ansari Nagar, New Delhi 110029

D.S.C. REDDY
National Professional Officer
WHO, Nirman Bhawan, New Delhi

DAMODAR BACHANI
Assistant Director General (Ophthalmology)
Directorate General of Health Services
Ministry of Health & Family Welfare, New Delhi

G. GURURAJ
Professor and Head, Department of Epidemiology
National Institute of Mental Health & Neuro Sciences (NIMHANS), Bangalore 560029

G.V.S. MURTHY
Associate Professor
RP Centre for Ophthalmic Sciences, AIIMS
Ansari Nagar, New Delhi 110029

ISAAC M.K.
Professor, Department of Psychiatry
NIMHANS, Bangalore 560029

J.G. SASTRY
Research Associate
Mahavir Hospital and Research Centre
10/1/1, Mahavir Marg
A.C. Guards, Hyderabad 500004

K.J.R. MURTHY
Coordinator HUD, Director PPM DOTS, Consultant Chest Physician, Research Coordinator
Mahavir Hospital and Research Centre
10/1/1, Mahavir Marg
A.C. Guards, Hyderabad 500004

M. KRISHNAN NAIR
Consultant
Department of Clinical Oncology
Amrita Institute of Medical Sciences
Kochi 695026, Kerala

MURALIKRISHNAN R.
Senior Faculty
Lions Aravind Institute of Community Ophthalmology
72, Kuruvikaran Salai, Gandhinagar
Madurai 625020, Tamil Nadu

N. GIRISH
Assistant Professor, Department of Epidemiology
NIMHANS, Bangalore 560029

NASEEM SHAH
Professor
Department of Dental Surgery
AIIMS, Ansari Nagar, New Delhi 110029

NEERAJ DHINGRA
Resident Director
Centre for Global Health Research
University of Toronto, Canada

PADMAJA SHETTY
Public Health Officer
Coordination of Macroeconomics and Health Unit
WHO, 20, Avenue Appia, CH-1211 Geneva 27
Switzerland

PAUL ARORA
Research Fellow
Centre for Global Health Research, St Michael's Hospital
70 Richmond Street East, 2nd Floor, Toronto
Ontario, M5C IN8, Canada

PRABHAT JHA
Canada Research Chair in Health and Development
Public Health Sciences, University of Toronto
Director, Centre for Global Health Research, St Michael's Hospital
Research Scholar, McLaughlin Centre for Molecular Medicine
70 Richmond Street East, 2nd Floor, Toronto, Ontario
M5C IN8, Canada

PRAVEEN KRISHNA R.
Consultant Ophthalmologist
Lions Aravind Institute of Community Ophthalmology
72, Kuruvikaran Salai, Gandhinagar
Madurai 625020, Tamil Nadu

R. SWAMINATHAN
Senior Biostatistician
Division of Epidemiology and Cancer Registry
Cancer Institute (WIA), Chennai 600020

RAJANI R. VED
Public Health Consultant
120, Anupam Apartments, Mehrauli Badarpur Road
Saidullajaib, New Delhi 110068

RAJESH KUMAR
HOD, Community Medicine
PGIMER, Chandigarh 160012

REHENA SULTANA
Senior Research Fellow
IRMS, ICMR, Medical Enclave
Ansari Nagar, New Delhi 110029

SANJEEV GUPTA
RP Centre for Ophthalmic Sciences
AIIMS, Ansari Nagar, New Delhi 110029

SARALA GOPALAN
Professor and Head
Department of Obstetrics and Gynaecology
PGIMER, Chandigarh 160012

SHALINI GAINDER
Research Associate, ICMR project
Department of Obstetrics and Gynaecology
PGIMER, Chandigarh 160012

SIDDARTH RAMJI
Professor, Department of Paediatrics
Maulana Azad Medical College, New Delhi 110002

S.J. HABAYEB
WHO Representative in India
WHO, Nirman Bhawan, New Delhi

THULASIRAJ R.D.
Executive Director
Lions Aravind Institute of Community Ophthalmology
72, Kuruvikaran Salai, Gandhinagar
Madurai 625020, Tamil Nadu

YING-RU J. LO
Medical Officer (HIV/AIDS)
WHO-SEARO, Department of Communicable Diseases
WHO House, I.P. Estate, Ring Road, New Delhi 110002

Institutions as authors

CENTRE FOR CHRONIC DISEASE CONTROL
T-7, Green Park Extension, New Delhi 110016

NATIONAL AIDS RESEARCH INSTITUTE
73, G-Block, MIDC, Bhosari, Pune 411026

NATIONAL INSTITUTE OF CHOLERA AND ENTERIC DISEASES
Indian Council of Medical Research, Kolkata 700010

TUBERCULOSIS RESEARCH CENTRE
Mayor Ramanathan Road
Spur Tank Road, Chetput, Chennai 600031, Tamil Nadu

VECTOR CONTROL RESEARCH CENTRE
Medical Complex, Indira Nagar, Pondicherry 605006

Reviewers

AJAY MAHAL
Assistant Professor
Harvard School of Public Health
Boston, USA

ANOOP MISRA
Professor
Department of Medicine
AIIMS, Ansari Nagar, New Delhi 110029

B.D. KHANDELWAL
Medical Officer
State TB Control Society, Jaipur

DILEEP MAVLANKAR
Associate Professor
Indian Institute of Management
Ahmedabad, Gujarat

DINESH MOHAN
Professor
Transportation Research and Injury Prevention Programme
Indian Institute of Technology, Delhi

H. PARAMESH
Medical and Managing Director
Lakeside Medical Centre & Hospital
Bangalore, Karnataka

H.P.S. SACHDEV
Professor, Department of Paediatrics
Maulana Azad Medical College
New Delhi 110002

J. MULIYAL
Principal, Christian Medical College
Vellore

K. ANAND
Associate Professor
Centre for Community Medicine
AIIMS, New Delhi 110029

K. SRINATH REDDY
Head, Department of Cardiology, AIIMS and
Director, Centre for Chronic Disease Control
T-7, Green Park Extension
New Delhi 110016

KAMALA GANESH
Consultant
D-1, Gulmohar Park, New Delhi

KRISHNAM RAJU
Chairman, Care Foundation
Care Hospital
Road No. 1, Banjara Hills, Hyderabad

L. LAXMINARAYANAN
Vice Principal
Professor and Head
Department of Conservative Dentistry and Endodontics
Saveetha Dental College and Hospital
No. 112, Poonamelle High Road
Velappanchvadi, Chennai

M. BHATTACHARYA
Deputy Director
National Institute of Health and Family Welfare
Munirka, New Delhi

M. GOURIE DEVI
Emeritus Professor
Institute of Human Behaviour and Allied Sciences
Dilshad Garden, Delhi

M.K.C. NAIR
President, Indian Academy of Paediatricians
Director, Child Development Centre
SAT Hospital and Medical College
Thiruvananthapuram

M.V. PADMA
Additional Professor
Department of Neurology
AIIMS, Ansari Nagar
New Delhi 110029

MANJUL JOSHIPURA
Director
Academy of Traumatology
Ahmedabad, Gujarat

MATHEW VERGHESE
Director
St Stephens Hospital
Tis Hazari, Delhi

N.B. MATHUR
Professor, Department of Paediatrics
Maulana Azad Medical College
New Delhi 110002

N.L. KALRA
Retd Deputy Director
National Malaria Eradication Programme, Delhi

NEERA AGARWAL
Vice President
Federation of Gynaecologists Society of India
New Delhi

NIKHIL TANDON
Additional Professor
Department of Endocrinology & Metabolism
AIIMS, New Delhi

P. JAMBULINGAM
Deputy Director
Vector Control Research Centre
Pondicherry

P.N. MARI BHAT
Professor
Institute of Economic Growth
University Enclave
Delhi 110007

PRATHAP THARYAN
Professor and Head
Department of Psychiatry
Christian Medical College
Vellore

P.S. PRABHAKARAN
Director
Kidwai Memorial Institute of Oncology
Bangalore, Karnataka

R. TANDON
Consultant
B-16, Mehrauli Institutional Area
Sitaram Bhartiya Institute
New Delhi

RAJEEV GUPTA
Head
Department of Medicine/Cardiology
Monilek Hospital and Research Centre
Jawahar Nagar
Jaipur 302004

RAZIA PENDSE
Medical Officer
Reproductive Health and Research Unit
FCH Department, WHO-SEARO
New Delhi

REVA TRIPATHI
Professor
Department of Obstetrics and Gynaecology
Maulana Azad Medical College, Delhi 110002

R.C. DHIMAN
Deputy Director
Malaria Research Centre (ICMR)
2, Nanak Enclave
Radio Colony, Delhi 110009

SANTOSH BHARGAVA
Paediatrician
D-7, Gulmohar Park
New Delhi

S.B. TARNEKAR
Assistant Director General (Leprosy)
Ministry of Health & Family Welfare
Nirman Bhawan, New Delhi

SUDANSHU MALHOTRA
WHO-SEARO, IP Estate
Ring Road, New Delhi

SUDHA SALHAN
Head
Department of Obstetrics and Gynaecology
VMMC and SJ Hospital
New Delhi

SUNITA MITTAL
Professor and Head
Department of Obstetrics and Gynaecology
AIIMS, New Delhi

SURINDER K. JINDAL
Professor and Head
Department of Pulmonary Medicine
PGIMER, Chandigarh 160012

USHA SHARMA
Principal
Meerut Medical College, Meerut

V. MOHAN
Director
Madras Diabetes Research Foundation
6B, Conran Smith Road
Gopalapuram, Chennai 600086

VIJAYA CHANDRA
Regional Advisor
WHO-SEARO, WHO House
Indraprastha Estate, Ring Road, New Delhi

VIKRAM PATEL
Reader in International Mental Health
London School of Hygiene and Tropical Medicine
London, UK

VINOD KOCHUPILLAI
Head
Institute Rotary Cancer Hospital
AIIMS, New Delhi 110029

V.K. VIJAYAN
Director
Vallabhbhai Patel Chest Institute
University of Delhi, Delhi

YOGESH JAIN
Coordinator
Community Programme
Jan Swasthya Sahyog
Bilaspur, Chhattisgarh

Disease burden in India

Estimations and causal analysis*

Disease burden estimations based on sound epidemiological research provide the foundation for public policy. Which diseases and what interventions does public policy need to focus upon are normally derived from such evidence. Well researched, longitudinal data can enable judicious targeting and help decide what needs to be done where, for whom, and when. Conversely, the absence of such good quality empirical data can affect programme designing and consequently outcomes. India has ample evidence of such impacts, often due to the mismatch between disease burden and its causal factors, and the interventions adopted and priorities in resource allocation.

Besides the need to avert disease for enhancing the quality of life, neglect can have adverse consequences on the well-being of affected families—social, psychological as well as economic. Diseases that are heavily concentrated among working age adults or the poor, as is the case with HIV/AIDS, cardiovascular disease (CVD), tuberculosis (TB), etc., can have a ruinous impact as such diseases are extremely expensive to treat, especially due to lack of insurance mechanisms. For example, in the case of HIV/AIDS, the out-of-pocket expenditure on treatment and services was reportedly Rs 6000 per HIV-positive person over a six-month reference period, while for clients on antiretroviral treatment (ART), the expenditures were markedly higher, nearly Rs 18,150 per person over a six-month period. Roughly 40%–70% of these expenditures are financed by borrowing. The devastating impact of TB, asthma, chronic obstructive pulmonary disease (COPD), heart diseases, etc. on individual household is similar, with children having to discontinue schooling and/or take up employment to provide an additional source of income. Analysis of data from the 1995–96 survey round of the National Sample Survey (NSS) undertaken by the National Commission on Macroeconomics and Health (NCMH) suggests that the out-of-pocket expenditure by individuals hospitalized on account of heart disease was roughly Rs 11,000 per person, or 120% of the average annual per capita expenditure of the households they belonged to. Likewise, roughly Rs 32,000 is the annual cost of treatment for acute cases of COPD that involve hospitalization. Therefore, it is clear that the onset of disease needs to be averted and when it occurs it should be treated quickly. For policies to ensure this, it is necessary that we have an evidence-based understanding of the extent of disease burden, the population groups that are the most vulnerable, and what interventions are needed to avert premature death or needless suffering.

With the above objectives in mind, the NCMH undertook an exercise to (i) identify major health conditions in terms of their contribution to India's disease burden; (ii) estimate the incidence and prevalence levels of the diseases/conditions at present and in 2015; (iii) list the causal factors underlying the spread of the diseases/conditions; (iv) suggest, based on the available evidence, the most cost-effective and low-cost solutions/strategies, both preventive and curative, for reducing the disease burden, particularly among the poor; and (v) indicate what interventions should be provided where and by whom. For assisting us in this onerous task, the help of leading experts was taken.

The experts identified 17 priority health conditions (Table 1) which they felt to be significant public health problems, affecting all segments of the population. Identification of these conditions was based on three criteria: first, the likelihood of the burden of a specific health condition falling on the poor, such as infectious and vector-borne conditions, TB and many maternal and child health conditions; second, in the absence of interventions, the probability of a listed health condition continuing to impose a serious health burden on the Indian population in the future, say by 2015, such as cancers, cardiovascular conditions and diabetes, or new infections such as HIV/AIDS; and third, the possibility of a health condition driving a sufficiently large number of people into financial hardship, including their falling below the poverty line.

*This overview is based on a paper entitled 'Choosing Investments in Health' prepared by Dr Ajay Mahal, Assistant Professor, Harvard School of Public Health, USA, for the National Commission on Macroeconomics and Health.

Table 1. Health conditions and disability-adjusted life-years (DALYs) lost in India, 1998

Disease/health condition	DALYs lost (x 1000)	Share in the total burden of disease (%)
Communicable diseases, maternal and perinatal conditions		
Tuberculosis	7,577	2.8
HIV/AIDS	5,611	2.1
Diarrhoeal diseases	22,005	8.2
Malaria and other vector-borne conditions	4,200	1.6
Leprosy	208	0.1
Childhood diseases	14,463	5.4
Otitis media	475	0.1
Maternal and perinatal conditions	31,207	11.6
Others	49,517	18.4
Non-communicable conditions		
Cancers	8,992	3.4
Diabetes	1,981	0.7
Mental illness	22,944	8.5
Blindness	3,699	1.4
Cardiovascular diseases	26,932	10.0
COPD and asthma	4,061	1.5
Oral diseases	1,247	0.5
Others	18,801	7.0
Injuries	45,032	16.7
All listed conditions	200,634	74.6
Others	68,319	25.4

COPD: chronic obstructive pulmonary disease

Source: Peters *et al.* 2001

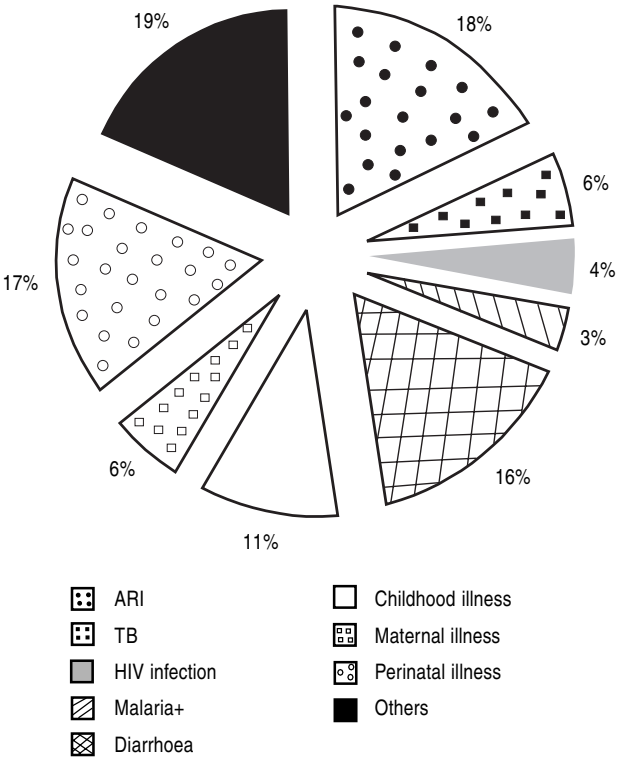

Fig. 1 Priority communicable maternal and child health conditions in India, by share in the burden of disease, 1998

ARI: acute respiratory infection; TB: tuberculosis; HIV: human immunodeficiency virus

Source: Peters *et al.* 2001

Baseline estimates and projections of priority health conditions

Exhaustive review of the available literature brought forth two factors of critical importance to public policy: (a) for almost all diseases/conditions identified, and more particularly the National Health Programmes in which government investment was substantial, namely, malaria and other vector-borne diseases, TB, leprosy, reproductive health and childhood conditions, there is a paucity of high-quality epidemiological information and validated data for arriving at any baseline estimations on prevalence or incidence. In the absence of operational research there was also weak evidence regarding the type of interventions that would be most cost-effective in the different settings in the country; and (b) a literature review threw up evidence of a large number of diseases which were considered to be lifestyle-related and affecting the rich were seen to be affecting the poor as well, and increasingly so. The non-availability of good quality data has been a major handicap in arriving at reliable estimations of the disease burden, affecting our ability to formulate appropriate policies and provide adequate budgets.

Category I: Communicable diseases, maternal and child health conditions

Category I health conditions include HIV, TB, malaria, diarrhoea, acute respiratory infections, maternal and perinatal conditions (Fig. 1). These accounted for nearly half of India's disease burden in 1998. It is expected that the burden on account of most of these pre-transition diseases, and deaths on account of malaria, TB, diarrhoea and other infectious diseases will reduce and leprosy be eliminated. However, HIV/AIDS and opportunistic infections such as TB and drug-resistant malaria are likely to increase. It is estimated that currently there are 51 lakh adults with HIV (adults being defined as the age group of 15–49 years for this purpose), a little less than 1% of the total population in this age group (Kumar *et al.*, unpublished). A conservative set of projections suggests that an estimated 3% of people in the age group of 15–49 years, i.e. about 5 crore people, are likely to be HIV-positive by the year 2025; and around 1.5–1.8 crore by 2015 (Kumar *et al.*, unpublished). These huge numbers of people with HIV at any given point in time do not, of course, include people who may have previously died of AIDS-related causes, and thus only a partial picture is available of the

cumulative future disease burden from HIV/AIDS. Due to lack of data and information, projection of the incidence of TB on account of a rising number of HIV/AIDS cases has not been possible.

Nearly 40% of the Indian population of all ages has *Mycobacterium tuberculosis* infection; and there are about 85 lakh people with TB at any given time. With more than 400,000 dying each year (Yajnik *et al.* 2002; Tuberculosis Research Centre [TRC] 2004), TB is the single most important cause of death in India at present (Yajnik *et al.* 2002).

Maternal, perinatal and childhood conditions account for another significant percentage of the disease burden, of particular importance for the poor. Although no direct estimates of the prevalence/incidence of these health conditions are available, we can indirectly assess their importance by looking at the neonatal, infant, under-5 and maternal mortality rates, which continue to be unacceptably high. While IMR was estimated to be about 66 per 1000 live-births, the under-5 mortality rate was estimated at 95 per 1000 live-births in 1998–99 as per the National Family Health Survey. The maternal mortality ratio (MMR) was estimated at 440 per 100,000 live-births in 1992–96. While no projections are available for MMR, a simplistic set of projections assume that rates of decline in the infant and under-5 mortality would be 46 and 62, respectively by the year 2015, lesser than the goals laid down under the Millennium Declaration. These forecasts are, however, not reliable as these ignore trends in and interplay with factors that underlie changes in the rates of infant, under-5 and maternal mortality (Deolalikar, forthcoming).

Malaria, dengue and other vector-borne conditions were estimated to account for 1.6% of India's total disease burden (WHO 1998). Unfortunately, these estimations lack credibility as reliable population-based data on these conditions do not exist in India. With most information 'reported' by officials, there are strong reasons to suspect underreporting and incomplete reporting of data. Underreporting occurs when a large number of patients visit private health care providers who are under no obligation to report cases to the public health authorities, and when record-keeping and case-finding are done by poorly monitored employees who may receive incentives for underreporting to demonstrate the success of a programme. Household survey methods are also not very useful to fill any gaps in this regard since many of these diseases, especially malaria, are likely to get recorded as 'unspecified fevers'.

Category II: Non-communicable conditions

This category of health conditions accounts for the second-largest share, after communicable health conditions, of the disease burden in India and includes cancers, CVD, diabetes, respiratory conditions such as asthma and COPD, and mental health disorders (Fig. 2). Available data suggest that these conditions will account for a fairly sharp increase in India's disease burden in the future.

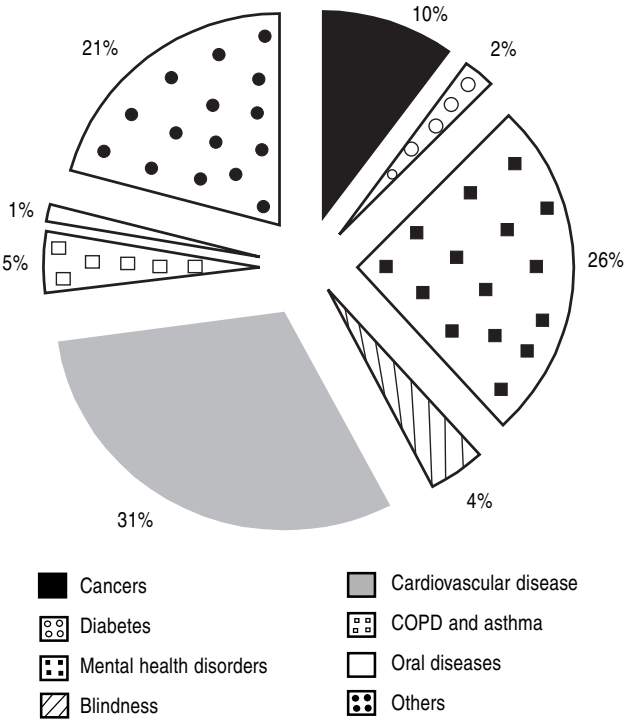

Fig. 2 Priority non-communicable health conditions in India, by share in the burden of disease, 1998

COPD: chronic obstructive pulmonary disease
Source: Peters *et al.* 2001

Cardiovascular disease

According to recent estimates, cases of CVD may increase from about 2.9 crore in 2000 to as many as 6.4 crore in 2015, and the number of deaths from CVD will also more than double. Most of this increase will occur on account of coronary heart disease—a mix of conditions that includes acute myocardial infarction, angina pectoris, congestive heart failure and inflammatory heart disease, although these are not necessarily mutually exclusive terms. Data also suggest that although the prevalence rates of CVD in rural populations will remain lower than that of urban populations, they will continue to increase, reaching around 13.5% of the rural population in the age group of 60–69 years by 2015. The prevalence rates among younger adults (age group of 40 years and above) are also likely to increase; and the prevalence rates among women will keep pace with those of men across all age groups.

Diabetes

Diabetes is also associated with an increased risk for CVD, and is emerging as a serious health challenge in India. Even though it accounted for only about 0.7% of India's disease burden in 1998, data suggest a significant load of diabetes cases in India—rising from 2.6 crore in 2000 to approximately 4.6 crore by 2015, and particularly concentrated in the urban population. The data also reveal that

the prevalence of diabetes is significant even among the 30–39 years' age group (6%), rising sharply to 13% in the 40–49 years' age group, and to nearly one-fifth of the population of those 70 years and above. Moreover, its prevalence among women above the age of 40 years is high.

Cancers

Cancers are a third area of concern. They refer to a group of diseases associated with uncontrolled cell growth that can affect normal body functions, often with fatal outcomes. Worldwide, cancers account for about 5.1% of the disease burden and 12.5% of all deaths. In India, cancers account for about 3.3% of the disease burden and about 9% of all deaths. These estimates will, however, change as many of the common risk factors for cancers, such as tobacco and alcohol consumption, continue to become more prevalent in India. Fairly conservative assumptions show that the number of people living with cancers will rise by nearly one-quarter from 2001 to 2016. Nearly 10 lakh new cases of cancer will be diagnosed in 2016, compared to about 800,000 in 2001. The incidence of cancers common to both men and women will also see a sharp increase during this period; nearly 670,000 people are expected to die of cancer in India in 2016.

Mental health disorders

Mental health was a much neglected field until recently. There is, however, increasing realization that conditions such as schizophrenia, mood disorders (bipolar, manic, depressive and persistent mood disorders) and mental retardation can impose a marked disease burden on Indians. This was confirmed by a study conducted for the NCMH which stated that at least 6.5% of the Indian population had some form of serious mental disorder, with no discernible rural–urban differences; women had slightly higher rates of mental disorder than men. If one were to include some other 'common' mental disorders and alcohol and drug dependency, the estimates would be substantially higher. With the increasing size of the population, these numbers are expected to grow substantially by 2015; the population with serious disorders is expected to grow to more than 8 crore in that year, and even higher if the category of 'common mental disorders' in the population was included in the projections.

Asthma and COPD

Estimates and projections for COPD and asthma show an equally alarming picture. COPD refers to a group of disorders that are persistent and largely irreversible, such as chronic bronchitis and emphysema. It is associated with an abnormal inflammatory response of the lungs to noxious particles or gases, especially tobacco smoke and air pollution—both indoor and outdoor. Asthma is a chronic disease of the airways, characterized by sudden attacks of laboured breathing, chest constriction and coughing. Although asthma can occur at all ages, in about half of the cases it occurs before the age of 10 years. It is estimated that there are around 1.49 crore chronic cases of COPD in India in the age group of 30 years and above, and these are projected to increase by nearly 50% by the year 2016, including 'severe' cases, some of whom may require greater levels of care, including hospitalization. It is also estimated that there were roughly 2.5 crore cases of asthma in 2001 which may increase by nearly 50% by 2016.

Blindness

Data on the current prevalence and future projections for blindness show that the number of blindness cases is expected to remain more or less the same during the next two decades. The projection, however, is based on extremely optimistic projections on cataract treatment that may not be realized.

Oral and dental diseases

Available data on the current prevalence and future projections for oral health conditions suggest an increase by 25% over the next decade.

These data, together with other evidence presented previously on non-communicable diseases, suggest a major future health policy challenge for India. With the continuing burden of communicable conditions, India is in the classic bind of facing a 'dual' burden of disease.

Category III: Accidents and injuries

The third category of health conditions has a significant impact on the overall disease burden. It is estimated that around 9% of the global mortality and 12% of the global disease burden is due to injuries, intentional or unintentional (WHO 2004). Unintentional injuries include road traffic injuries, poisoning, drowning, falls, etc.; whereas intentional injuries include suicide, homicide and war-related violence. Analysis suggests that the share of injuries and accidents in India's disease burden may be even greater, at about 16.7%.

It is estimated that the number of deaths from accidents and injuries in 2005 would range from 730,000 to 985,000, with projections that deaths from injuries will increase by as much as 25% over the next decade. The injury mortality estimates for the year 2000 suggest that about 9% of all deaths in India were accounted for by injuries, a share similar to the global share of deaths due to injuries (WHO 2004). These estimates do not include the health impact of injuries with non-fatal outcomes (including permanent disability), which tend to be heavily underreported in India and could well be in the region of about 5 crore cases per year. Available evidence from India also shows that much of the mortality from injuries due to road traffic accidents, occupational accidents and suicide is concentrated among

adults in their peak work ages, i.e. 15–44 years, and among children.

Identifying cost-effective interventions

A summary of causal analyses of different diseases/health conditions carried out by experts engaged by the NCMH is given in Annexure A. In most cases, disease occurrence and progression can be avoided or significantly reduced/ contained *if access to right information and/or early treatment is assured*.

In countries such as India where there are limited resources and competing demands, not all conditions can be treated and not every intervention provided at public expense. At some point prioritization of interventions or population groups that need to be supported with public funding becomes inevitable. The issue then arises as to the criteria that ought to be used for identifying such publicly supported interventions. There could be two:

- those that are technically effective in substantially ameliorating a major health problem; and
- those that are financially inexpensive (i.e. cost-effective) relative to the outcome gains achieved.

The first ensures that the intervention markedly reduces the burden of disease, and does not simply result in a token improvement in the health status. The second ensures that the intervention is good value for money. Thus, policy-makers can focus on several extremely cost-beneficial and cost-effective interventions that simultaneously yield large gains in outcomes for several major health conditions.

While the probability of death beyond a certain age, say 70 years, tends to be high and is not very dissimilar across developed and developing nations, the largest gains in mortality reduction are likely to be achieved at younger ages. Jha and Nguyen (2001) show that whereas 18% of all Indians can expect to die before the age of 40 years, only 2% of residents of the UK expect to do so. A less marked difference exists in 'middle age', with 51% of all Indians expecting to die before the age of 70 years compared to 23% for residents of the UK. An understanding of why these differences exist at younger ages offers the possibility of identifying cost-effective interventions, particularly among children and younger adults.

In the case of childhood diseases, India presents wide regional disparities—while in Kerala, the IMR is 14 per 1000 live-births, it is 96 in Orissa and 5 more States have an IMR of more than 75 (Deolalikar, forthcoming). Given Bangladesh's rapid advances in recent years, large reductions in the IMR appear feasible even within resource-poor settings as in India. If India could achieve an IMR of about 26 per 1000 live-births as implied by the MDGs (double the rate achieved by Kerala), it could enable India to avoid nearly 10 lakh infant deaths per year, with huge reductions in the overall disease burden.

Achieving such declines requires looking at a range of key interventions that address the major causal factors—tetanus injections during pregnancy, professional attendance with appropriate access to referrals during childbirth, improvement in the mother's nutrition to avoid low birth weight infants, etc. Similarly, malnutrition makes a child susceptible to diarrhoeal diseases and respiratory infections which, when untreated, can be fatal. Beyond the phase of infancy, immunization becomes critical in warding off potentially fatal conditions. The enormous cross-state variations in immunization rates and the low rates of immunization in several States suggest great potential for reducing the mortality from vaccine-preventable conditions.

Apart from these medical and nutritional interventions, there are other non-health interventions that also need to be considered in policy design, such as reducing discriminatory practices towards the girl child, enhanced schooling of females, better roads, access to clean drinking water, electricity and other infrastructure, as these are known to have a beneficial impact on the IMR, widening access to timely care, etc.

Overall, the potential gains from these interventions can be massive. According to NCMH estimates, a reduction in childhood mortality may raise the life expectancy at birth of an Indian by as much as 3.1 years, and India's Gross Domestic Product (GDP) from 4% to 12%.

Similarly, with the likelihood of 18% of all Indians dying before the age of 40 years (Jha and Nguyen 2001; Deolalikar, forthcoming), about 8.5% of a cohort born in any given year can expect to die between the ages of 5 and 40, in contrast to the UK's 1.5% (Jha and Nguyen 2001; World Bank 2004).

Several factors contribute to this difference. First, the MMR in India is substantial. The mortality from HIV/ AIDS and associated infections such as TB, injuries, especially road traffic accidents, and cancers is high and expected to increase given risky heterosexual activity and the factors that promote it—mobile populations, rising incomes and income inequality, the low status of women and the presence of high-risk vulnerable groups; current tobacco consumption patterns among young adults; and increase in traffic and lax enforcement of traffic regulations, etc.

To address the above conditions a combination of interventions will be needed. Most can be effectively countered by implementing a range of low-cost solutions; for example, peer education, access to condoms, a climate of destigmatization, use of antiretroviral drugs to reduce the risk of mother-to-child transmission of HIV infection, and treatment of sexually transmitted diseases (STDs) appear to be extremely cost-effective options for tackling HIV/AIDS. Vaccination against TB infection, effective identification of smear-positive cases of TB before they can infect others, and strict implementation of an appropriately designed Directly Observed Treatment, Short-course (DOTS) are effective methods for reducing the mortality rate from smear-positive TB as well as the rates of transmission.

According to the Commission on Macroeconomics and Health (CMH) estimates, properly administered DOTS can reduce case-fatality rates resulting from smear-positive TB from 60%–70% to 5%. Rough calculations undertaken by the NCMH suggest that a reduction in mortality due to TB in India by one-half would raise the life expectancy of an Indian by 0.12 years and India's overall GDP by as much as 0.5%.

Huge gains in mortality reduction among young adults are likely by reducing smoking and tobacco use. Analysis of the National Sample Survey data by the NCMH showed that nearly 40% of Indian males smoke. Tobacco consumption and smoking have been linked to lung and oral cancers, and TB. Shah (unpublished) suggests that India's current patterns of tobacco use and smoking is likely to sharply increase the incidence of oral cancer in the future. Cost-effective interventions to address smoking include: ending advertising for cigarettes, *beedis* and other tobacco products, enhanced taxes on cigarette sales and production, and dissemination of health messages. The NCMH estimates that a 50% reduction in mortality rates due to CVD can raise the life expectancy at birth of an average Indian by 1.3 years and India's GDP by 2%–5%.

The issue of road traffic accidents is gaining attention as these are major killers of young, and often poor, adults in India. Many of the measures to address accidents and their impact lie outside the realm of the health sector, and may often have to do with urban planning, road designs (including pedestrian and bicyclist access), vehicle quality and design features, driving skills, lack of helmets and control of speed. These require improved regulatory design as well as better enforcement of the law against traffic rule violators. Besides, addressing alcohol consumption, which may impair response time and the overall ability to drive safely, needs far greater attention than has been the case so far. Estimates from Bangalore, Haryana and Punjab suggest that nearly 40% of truck-related accidents and 60% of those involving cars are alcohol-related. Interventions to address alcohol consumption may include tax increases since price elasticity of demand for alcohol in India appears to be high, accompanied by health messages. Another factor that ought to worry policy-makers is the huge burden of disease on account of mental health disorders. These require a range of skills that India does not have in adequate number as well as access to drugs, which are expensive.

The older age groups are typically more vulnerable to chronic diseases and are also at high risk for CVD. As obesity, hypertension and diabetes are linked to the onset of CVD, health education programmes that promote exercise and weight reduction; screening for hypertension as another pathway to both influence exercise and dietary behaviour; early treatment; reduced smoking; selective taxation of foods, etc. need to be accorded high priority. The actual treatment of cancers and procedures for CVD (angioplasties and coronary artery bypass graft are considerably less likely to be cost-effective while their adverse financial implications to affected households could be extremely large.

An important element of the intervention strategy must be to identify the mechanism through which such services are to be delivered. Annexure B presents a schematic framework on how some of the preventive and curative interventions are to be provided at different levels of care—at the community level, subcentre level, primary health centre, community health centre, and ultimately, at the district hospital. The framework of Annexure B is intended to be suggestive rather than prescriptive in that it does not imply that the concerned services have to be provided only by the public sector, or that they ought to be free for everyone.

A key lesson that has emerged from this effort is the acute paucity of good data and the absence of community-based studies, which have made it impossible to come up with any credible estimates of the disease burden in India. This has, in no small measure, been further worsened by the wide diversity and disparities that characterize this country, making it difficult to extrapolate the data of small, localized studies to the entire country. India should urgently undertake operational research, establish good surveillance systems and develop validated data banks. The data gaps need to be bridged, high priority accorded to operational research and adequate resources allocated.

References

Deolalikar A. *Attaining the Millennium Development Goals in India: Role of public policy and service delivery*. Washington, DC: The World Bank, South Asia, Human Development Unit; forthcoming.

Jha P, Nguyen S. *Avoidable mortality in India.* CMH Working Paper series, WG5:1. Geneva, Switzerland: WHO, Commission on Macroeconomics and Health; 2001.

Kumar R, Jha P, Arora P, Dhingra N, India Studies of HIV/AIDS Working Group. HIV-1 trends, risk factors and growth in India. *NCMH background papers—burden of disease in India*. New Delhi: Ministry of Health, Government of India; unpublished.

Peters D, Yazbeck A, Ramana G, Sharma R, Pritchett L, Wagstaff A. *Raising the sights: Better health systems for India's poor*. Washington, DC: The World Bank; 2001.

Shah N. Oral and dental diseases: Causes, prevention and treatment strategies. *NCMH background papers—burden of disease in India*. New Delhi: Ministry of Health, Government of India; unpublished.

Tuberculosis Research Centre (TRC). *Estimation of the burden of pulmonary tuberculosis in India for the year 2000*. Chennai: TRC; 2004.

WHO. *World Health Report 1998*. Geneva, Switzerland: WHO; 1998.

WHO. *World Health Report 2004*. Geneva, Switzerland: WHO; 2004.

World Bank. *World Development Indicators Database 2004*. Washington, DC: The World Bank; 2004.

Yajnik K, Chakraborty A, Jochem K. A mathematical model for determining the effect of tuberculosis control programmes on its prevalence in India (draft report). New Delhi: The World Bank; 2002.

Section I

National health programmes: Communicable diseases, and reproductive and child health

Programmes for the control of leprosy, tuberculosis and malaria

AVTAR SINGH DUA

The objective of this paper is to give a brief overview of the major National Health Programmes (NHPs) that are under implementation throughout the country. Under the NHPs for the control of vector-borne diseases (VBDs), tuberculosis (TB) and leprosy, the Government of India (GOI) provides financial and commodity assistance to State Governments. As these diseases/conditions disproportionately affect the poor and entail wide externalities, the Government has from time to time set targets to eliminate or control them. Thus, while malaria and TB were to be contained, leprosy was to have been eliminated. Yet due to various systemic problems, achievement of these goals continues to elude us. Besides, India is committed to the global community as a signatory of the Agreement to achieve the Millennium Development Goals by 2015 which specifically lay down reductions in the incidence of TB, malaria and HIV/AIDS.

For the reasons stated above, the NCMH accorded a high priority to the achievement of these targets and goals. With this in mind, the NCMH sought to obtain a baseline of the prevalence of these diseases/conditions and identify the casual factors—direct/indirect and, based on a minimum standard of treatment, assess the cost implications of achieving the goals. A detailed review brought out three important points:

1. That on account of inadequate time–series data and sound, validated data, arriving at a baseline estimation of disease prevalence was not possible for vector-borne diseases and leprosy;
2. Identification/prioritization of interventions based on cost-effectiveness and related evidence was not possible for want of any operational research undertaken on a significant scale that could be extrapolated for addressing the diversity and disparities that characterize this country; and
3. Given the huge commitment of resources to these programmes, highest priority should be accorded to undertaking operational research and community-based epidemiological studies to arrive at baseline estimations and evidence on the effectiveness of the various interventions being implemented, so as to redesign programmes to suit community preferences if called for, rather than a one-size-fits-all approach.

Accordingly, this paper contains a broad overview of the NHPs related to malaria, TB and leprosy. This is followed by treatment protocols for vector-borne diseases and also estimations of the prevalence levels of TB undertaken by TRC, Chennai for the NCMH. Standard treatment protocols for malaria, TB and leprosy have already been adopted by the Government for treating patients suffering from these diseases. Based on these estimations and standard treatment protocols (STPs), the budgetary deficits and fund requirement have been worked out in the main Commission Report.

Leprosy

Leprosy has been known through the ages, primarily because of the deformity it causes. Leprosy is closely linked with poverty—poverty is both a cause and a result of leprosy, and leprosy patients are forced to impose significant economic and social burdens on their families (Neira 2001; Gokhale 2001). Eliminating leprosy, therefore, has wider implications than merely resolving a public health problem. It will have a dramatic impact on the overall economic empowerment of the people affected by it (Neira 2001). Twenty-five per cent or more of leprosy patients have some degree of disability (WHO 1960; Reddy and Bansal 1984; Saha and Das 1993; Kalla *et al.* 2000). Prevention or correction of deformity in patients with leprosy would increase the probability of gainful employment, increase annual earnings per patient and raise overall earnings for all patients (Max and Shepherd 1989).

In view of the substantial progress achieved with multi-drug therapy (MDT), in May 1991, the World Health Assembly adopted resolution WHA44.9 on the elimination of leprosy to reach the global target prevalence of less than 1 case per 10,000 population by 2000. To date, out of 122 countries where leprosy was considered a public health

Member, National Commission on Macroeconomics and Health, Government of India, New Delhi
e-mail: avtarsinghdua@yahoo.co.in

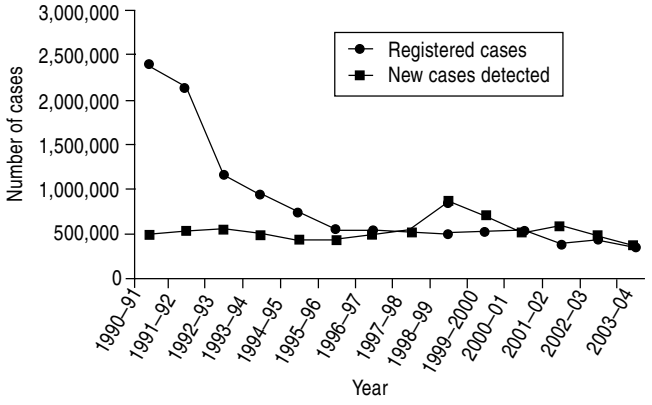

Fig. 1 Number of leprosy cases from 1990–91 to 2003–04

problem, only 10 still have to reach the elimination goal—Angola, Brazil, Central African Republic, Congo, India, Liberia, Madagascar, Mozambique, Nepal and Tanzania. Most countries of the world have eliminated leprosy primarily because of MDT which came into wide use in 1982. Because of MDT, many countries that had a very high prevalence of leprosy could manage to eliminate it as a public health problem within a short span of time. Thailand reduced the prevalence of leprosy from more than 50/10,000 population in 1953 to less than 1/10,000 population by 1995 by dismantling the vertical programme and integrating leprosy control activities into the provincial primary health care services (WHO 2001). Sri Lanka eliminated leprosy by 1995 by using the mass media to change the attitude of the masses and increase case detection. Even though many countries could eliminate leprosy, as on 31 March 2004, India had a prevalence of 2.44 cases per 10,000 population and 265,781 leprosy cases on record, having detected 367,143 new cases during 2003–04 (Fig. 1).

Leprosy control in India

The Government started the National Leprosy Control Programme (NLCP) in 1955 based on domiciliary treatment with dapsone through vertical units implementing survey, education and treatment activities. Following the advent of MDT in 1982, the National Leprosy Eradication Programme (NLEP) was launched in 1983 with the objective of arresting the disease in all known cases of leprosy. However, coverage with MDT remained low due to a range of organizational issues, and fear of the disease and associated stigma. The first World Bank-financed NLEP Project was implemented in India from 1993 to September 2000 wherein the Project supported the vertical programme structure for high-endemic districts, while Mobile Leprosy Treatment Units (MLTUs) were established in moderate- and low-endemic districts. During the Project period, MDT coverage of registered leprosy cases increased to 99.5% (from 62% in 1992), due to which the prevalence rate reduced from over 24/10,000 population in 1991 to 3.7/10,000 by March 2001.

The second phase of the World Bank-financed NLEP (2001–02 to 2003–04) was significant for two reasons: (i) an attempt was made to decentralize the Programme and integrate it with the general health care system so as to develop the required capacity at decentralized levels of implementation for sustaining Programme gains, and (ii) it adopted a more focused approach of intensive campaigns in select areas—Leprosy Elimination Campaign (LEC) Projects in urban areas, Special Action Projects for Elimination of Leprosy (SAPELs) in rural areas, and Modified Leprosy Elimination Campaigns (MLECs) for conducting intensive awareness/detection campaigns. For the first four years, MLECs were conducted throughout the country, but in 2003–04, they were restricted to the eight identified high-endemic States of Bihar, Jharkhand, Chhattisgarh, Orissa, Andhra Pradesh, Uttar Pradesh, Maharashtra and West Bengal. One additional activity undertaken was holding prevention of deformity (POD) camps and providing on-the-job POD training to the general health care staff, and offering self-care learning practices to patients with disabilities and ulcers.

Against the targets of detecting 11 lakh new leprosy cases and curing 11.5 lakh new cases with MDT in the second phase of the Project, 14.36 lakh new cases (130.5%) were detected and 15.54 lakh cases (135.1%) discharged from treatment as cured of leprosy. The prevalence rate of leprosy decreased from 3.74 cases per 10,000 population in 2000–01 to 2.44 cases per 10,000 by March 2004, and the disability rate decreased to 1.44%. As on 1 April 2004, 17 States still had a prevalence rate of more than 1/10,000, 71 districts (12.03%) still have a prevalence rate of more than 5/10,000 population, and the goal of leprosy elimination has not been achieved. Of the 3.67 lakh new leprosy cases detected in 2003–04, 1.44 lakh (39.3%) were multibacillary (MB) cases (an increase from 33.9% in 2000–2001) and over 50,000 (13.8%) were children, thereby indicating that leprosy was being diagnosed at an advanced stage. Thus, there was continuing transmission of the causative organism (*Mycobacterium leprae*) in the environment. This continued transmission is a major hindrance to leprosy control (WHO 1997) and increases the risk of transmission of infection (Beers *et al.* 1999). One reason for the increase in the proportion of MB cases of leprosy from 33.9% to 39.3% is the change in definition of MB cases by the Central Leprosy Division—from the earlier 10 or more to 6 or more skin patches.

The National Health Policy, 2002 now targets the elimination of leprosy by 2005. To achieve this goal, intensive efforts are required in 12 States (Uttar Pradesh, Andhra Pradesh, Bihar, Maharashtra, West Bengal, Orissa, Jharkhand, Tamil Nadu, Chhattisgarh, Gujarat, Karnataka and Madhya Pradesh [MP]), which account for about 93.9% of new cases (Fig. 2).

Funding of the NLEP

The World Bank was the major source of funding for the

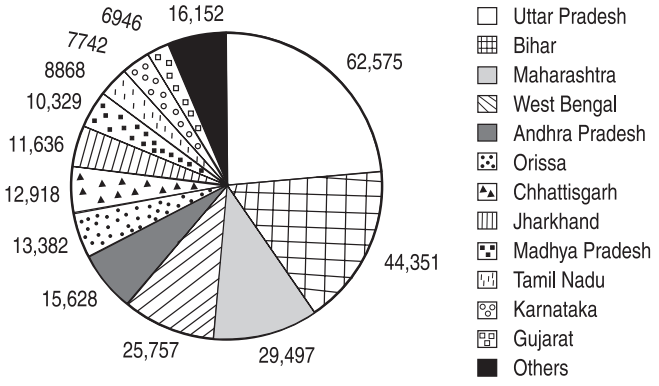

Fig. 2 Leprosy cases in 2003–04

NLEP since 1993 but has stopped financing the project since December 2004. The total allocation under the Programme in 2002–03 was Rs 73.72 crore. In this year, 30% of the expenditure was incurred on information, education and communication (IEC) activities, almost one-fourth on salaries and 16.5% on drugs for leprosy (Table 1). Administration costs were Rs 27.93 crore (42.7%) (consisting of contractual staff involved in administration of the Programme at the Centre, State and district levels, office expenses, purchase of vehicles, furniture), Rs 12.19 crore (18.6%) on patient care (consisting of drugs, salaries of field-level contractual staff involved in leprosy control work, some medical equipment, case detection campaigns, medical/surgical services), Rs 23.78 crore (36.4%) on development costs (comprising IEC activities and training) and Rs 1.49 crore (2.3%) on assessment costs (comprising review, monitoring and research).

Leprosy control: The future and recommendations

Till March 2004, the GOI was providing funds for salaries of staff besides other activities. From 1 April 2004, the Central Government is supporting activities such as training, IEC, vehicle repair and maintenance, TA/DA of staff, etc., and drugs are being supplied with WHO support. However, the States have been directed to reduce 75% of the vertical

Table 1. Expenditure under the National Leprosy Eradication Programme, 2002–03 (Rs in lakh)

Expenditure head	Centre	States	Total	%
01. Salaries				
2 Contractual staff	1.51	1480.14	1481.65	22.7
4 Honorarium	0	59.31	59.31	0.9
02. Maintenance			0	0
03. Medical supplies/consumables				
1 Drugs and medicines	0	1077.46	1077.46	16.5
04. Office expenses				
1 Stationery	0	36.23	36.23	0.6
2 POL	0.31	419.66	419.97	6.4
4 Others	0.44	710.42	710.86	10.9
05. Training	2.45	403.48	405.93	6.2
06. IEC	490.51	1481.13	1971.64	30.2
07. Review and monitoring	16.84	114.66	131.5	2.0
08. Research and studies	10.36	6.7	17.06	0.3
09. Any other				
1 Case detection campaigns	0	99.4	99.4	1.5
2 Medical/surgical services	0	41.85	41.85	0.6
Total	522.41	5930.44	6452.85	98.7
10. Civil works				
1 New construction	0	0	0	0
2 Furniture	0	30.47	30.47	0.5
11. Equipment				
1 Medical equipment	0	22.23	22.23	0.3
2 Surgical equipment	0	0	0	0
12. Vehicle	0	32.08	32.08	0.5
Total	0	84.78	84.78	1.3
Grand total	522.41	6015.22	6537.63	100.0
%	8.0	92.0	100.0	

IEC: information, education and communication; POL: petroleum, oil and lubricants

leprosy staff and utilize them appropriately within the general health care system. The remaining 25% of regular vertical NLEP staff will be retained under the Programme to form State and district NLEP cells/nuclei in leprosy-endemic pockets. The salaries of leprosy officers are now being paid by the States. Some staff have been retained where the prevalence rate is over 1/10,000 while in areas where it is less than 1/10,000, one person has been retained to advise on and provide training for leprosy-related activities. All general health care staff have been imparted 3 days' training in the diagnosis and management of leprosy and related IEC activities in the community.

The process of integration (both structural and functional) for leprosy control activities in the general health care delivery system needs to be accelerated. This could be a more cost-effective and viable strategy that will also help ensure sustainability of the programme based on domestic funding and reduce the dependence on foreign financing/assistance. The vacant posts of multipurpose workers (male) (MPWs) (M) in most States need to be filled as the over-burdened auxiliary nurse–midwife (ANM) alone might not be able to devote enough time towards leprosy control activities in her area.

The fair progress in the control of leprosy at the national level could be deceptive, as areas still remain where the prevalence of leprosy is unacceptably high. There is no time for complacence and focused attention needs to be given, especially to the 11 States that contribute to over 90% of leprosy cases and the 71 districts with a prevalence of over 5/10,000 if we are to achieve the target of leprosy elimination.

Special action needs to be taken for difficult-to-access communities, urban areas and migratory groups as the risk of drop-out and discontinuation of treatment is high in such groups. Although it is now known that leprosy is a curable disease through MDT, there is still a stigma associated with the disease. Messages that should have been imbibed by the community have not reached the masses, especially in rural and disadvantaged urban areas. Innovative approaches, like extensive use of the mass media to change the attitudes of the masses, need to be implemented to improve community awareness and participation so that suspected cases do not fear stigma and report to a health facility at the earliest for timely diagnosis and prompt treatment.

With a decline in the number of cases, priority attention also needs to be given to activities for prevention of deformities, and the Government should continue additional budgetary provisions for this purpose. Elimination of deformity in leprosy cases would also raise productivity enormously in the country.

Tuberculosis

Though the National Tuberculosis Control Programme was launched in 1962, India accounts for nearly one-third of the global burden of TB. About 10 lakh are new smear-positive highly infectious cases and about 5 lakh deaths occur due to tuberculosis (Government of India 2003a). This continued burden of disease is particularly tragic because TB is nearly 100% curable. Besides, control of TB with multidrug regimens has been cited as one of the five highly cost-effective interventions (World Bank 1993). Drug resistance in sputum-positive patients treated with short-course chemotherapy has been shown to be low (2.1%) (TRC, ICMR 2001). Untreated patients can infect 10–15 persons each year; poorly treated patients develop drug-resistant and potentially incurable TB.

> TB imposes an enormous social and economic burden on the people; it affects all age groups but has its greatest impact on productive adults. With TB, there is loss of productivity and increase in debts. The total direct cost of treatment of TB is higher for women than for men. The fear and stigma associated with TB have a greater impact on women than on men.
>
> One single TB case in a family leads to the loss of 2–3 months of income.

Low-income people are at higher risk of getting TB as it spreads in crowded places—households, school, workplace, marketplace and commutes between them (Health and Development Initiative 2004a). TB kills more women in India than any other infectious disease and causes more deaths among women than all causes of maternal mortality combined. Moreover, women with TB are stigmatized—more than 100,000 women are rejected by their families each year because of TB. The disease also has an adverse impact on children—it leads to a large number of children becoming orphans, and every year in India alone, 300,000 children leave school on account of their parents' TB (Government of India 2004b). Because more than three-quarters of people with active TB are in the economically productive age group (15–54 years), the economic and social costs to them and the society are huge (Health and Development Initiative 2004b). On an average, 3–4 months of work time are lost if an adult has TB, resulting in the loss of 20%–30% of annual household income. An average of 15 years of income is lost if an individual dies of the disease (Government of India 2004b). Every year, TB costs India more than Rs 13,000 crore. In addition, every year, TB patients spend more than Rs 645 crore in seeking private care for TB. It has been estimated that if the GOI spent even US$ 200 million (Rs 900 crore) per year on effective implementation of Directly Observed Treatment, Short-course (DOTS), the tangible benefits to the Indian economy would be worth at least US$ 750 million per year (Rs 3375 crore) (WHO 2004).

The World Health Assembly approved two targets in 1991: to cure 85% of newly detected cases of sputum smear-positive TB, and to detect 70% of existing cases of sputum

smear-positive TB. The case-finding target was set at 70% as being the highest level that could reasonably be attained in average developing-country settings (Newell 2002). The WHO emphasizes the need to attain the cure target first, as high numbers of failed treatments can actually lead to an increase in the incidence of TB due to the creation of chronic transmitters. Only when the first target has been met should case finding be increased until the second target is met.

Proxy for the tuberculosis situation: Annual rate of tuberculosis infection

The 1955–58 survey conducted by the Indian Council of Medical Research demonstrated a high prevalence of TB in India at 400/100,000. Annual rate of tuberculosis infection (ARTI) studies, conducted in 2000–03, showed an ARTI of 1.5% at the national level (1.9% in the North Zone, 1.6% in the West Zone, 1.3% in the East Zone and 1.0% in the South Zone), only a marginal decline from the 1.7% reported in earlier studies (Narain *et al.* 1963; Mayurnath *et al.* 1991). There are striking rural–urban differences in ARTI—2.1% in urban areas and 1.2% in rural areas.

Tuberculosis control in India

A 1992 joint review of TB control activities indicated that despite the existence of a national programme, TB patients were not being accurately diagnosed, and most patients did not complete treatment. This review led to the Revised National Tuberculosis Control Programme (RNTCP) being started on a pilot basis in 1993, followed by large-scale implementation in 1997. By December 2004, 545 districts with a population of 94.2 crore had been covered and it is expected that the entire country will be covered under the RNTCP by 2005. With increasing coverage of areas under the RNTCP, the number of deaths due to TB have declined from over 5 lakh to about 4 lakh per year—in registered patients, the death rate has declined seven-fold. It has been further estimated that if a case detection rate of 70% and cure rate of 85% can be maintained, there would be a decline of 6%–12% per annum in the incidence of new cases of TB.

For every 1.0% of ARTI, an estimated 50 new cases of smear-positive pulmonary TB are expected for every lakh population, in addition to sputum-negative pulmonary TB and extrapulmonary TB cases. Thus, there are an estimated 7.95 lakh new cases of smear-positive pulmonary TB in the country (in addition to smear-negative pulmonary TB and extrapulmonary TB cases), and only about 3.59 lakh new sputum-positive cases (45.1%) were put on DOTS treatment in 2003.

Laboratory diagnosis of tuberculosis

Sputum microscopy is the mainstay of the laboratory diagnosis of TB and based on the results of three sputum examinations, patients are categorized as sputum positive or sputum negative and put under treatment as Category I, Category II or Category III patients. Microscopy Centres (MCs) have been established for a population of 100,000, which roughly corresponds to the population of a CHC or Block PHC. For non-RNTCP areas, MCs were set up for every 200,000 population, but now that the major parts of the country have been covered under the RNTCP, it is expected that MCs are being set up for 100,000 population. Patients often have problems in giving three sputum samples at the designated centres, particularly if they live far away from these centres, but it has been observed that strict supervision is required for quality control of sputum examination. The analysis of an External Quality Assurance Scheme in Mumbai showed a false positivity rate of 0.31% between the Senior Tuberculosis Laboratory Supervisor (STLS) and the Laboratory Technician (LT), and 13.8% between the Quality Assurance Centre (QAC) and LT. A false negativity rate of 0.61% between the STLS and LT and 2.7% between the QAC and LT was also found. There is thus a need for quality assurance (QA) and retraining of laboratory personnel under the RNTCP to maintain the quality of sputum examination.

Directly Observed Treatment, Short-course (DOTS)

The implementation of DOTS hinges on five interventions:

- Political commitment for sustained TB control
- Sputum smear microscopy to detect infectious cases among those people attending health care facilities with symptoms of pulmonary TB
- Regular, uninterrupted supply of antituberculosis drugs
- Short-course chemotherapy for all TB cases to be given under direct observation
- Systematic monitoring and accountability for every patient diagnosed (improved monitoring and supervision using the TB Register).

Once a person is diagnosed as having TB by sputum examination, he/she is put on DOTS. A DOTS provider could be any person living in the village or neighbourhood of the patient—a health worker, a teacher, a *sarpanch*, any other local leader, an *anganwadi* worker (AWW), a shop-keeper—who is willing to supervise the administration of drugs to the patient. In Gurgaon, it was observed that about 60% of DOTS providers were community volunteers. In the majority of cases, the DOTS provider is the AWW.

In many instances, TB drugs are not administered under the direct supervision of AWWs; in some cases they give the drugs to the patient on a weekly basis to be taken by himself/herself. Occasionally, they give all the drugs to the patient for the total duration all at once. At some places, the posts of Senior Treatment Supervisor (STS) and STLS are vacant and supervision is lacking. Occasionally, the AWWs, who were the DOTS providers, came across situa-

tions when the patient developed some side-effects due to the antituberculosis drugs, and had to take the patient to the nearest PHC. She does not get anything for taking the patient to the PHC, not even money for transport in such a case. In Bangalore, an NGO had even identified household members as DOTS providers, which was contrary to the guidelines issued under the Programme.

To encourage community involvement in effective implementation of the Programme, provision has been made for an honorarium of Rs 175 to the DOTS provider. However, this scheme was stopped in Orissa on the plea that since many government service providers were also involved in the administration of drugs under DOTS, they might feel demotivated as this honorarium was paid only to non-government DOTS service providers.

DOTS is supposed to be given under supervision but is not strictly supervised in many cases.

Often, the provider gives the drugs to the patient for a week or a month.

In some cases, DOTS providers have been identified from the household itself.

Orissa stopped giving the honorarium of Rs 175 to DOTS providers at the community level.

Thus, although programme strategies are formulated at the level of the Central Government, in effect, deviations from the guidelines are observed when it comes to actual implementation through States or at the local level. Second, as far as supervised treatment is concerned, it has been documented that the need for DOT as a universal requirement is controversial, since there are also other elements that contribute to the success of TB control programmes (Zwarenstein *et al.* 1998; Volmink *et al.* 2000).

A critical activity for the success of DOTS is monitoring and supervision to ensure a new case detection rate of at least 70% and a cure rate of 85%. Besides, monitoring and supervision is also required to ensure that patients who are put on treatment complete the full course. However, the performance under the Programme shows that although as a national average the treatment success rates are over 85%, there are variations between States and some districts have cure rates of less than 40%. At the same time, case detection rates are less than 70% in many States/UTs (barring Andhra Pradesh, Chandigarh, Delhi and Sikkim), and this might be an important cause of India's failure to achieve the target of TB control. This evidence emphasizes the need to strengthen Programme monitoring and supervision of implementation at all levels.

Involvement of private practitioners and NGOs

Many TB cases/chest symptomatics seek treatment from private practitioners (PPs) in the first instance (Newell 2002; Sudha *et al.* 2003; Rangan 1995). Private health care facilities are probably the first and preferred point of contact for both urban and rural chest symptomatics, the major reasons being proximity to the residence and the perception that good quality care would be available. Treatment for TB through PPs is usually of poor quality with very low cure rates. There is a threat that patients who receive poor treatment neither die nor are cured of the disease, but remain sputum-positive and chronic transmitters of TB (Newell 2002; Kalk 2003). India has one of the largest private health care sectors in the world, with an estimated 80 lakh private practitioners (PPs). Despite the rapid expansion of RNTCP, there is delay in the diagnosis of smear-positive TB. Factors associated with this delay were found to be initial consulta-tion with a private provider and patient's residence more than 2 km from a health facility (Rajeswari *et al.* 2002b). Of late, some efforts have been made to involve PPs and about 3000 PPs are officially providing RNTCP services (Government of India 2004a). Some schemes have been designed for the involvement of PPs in the RNTCP. These include the referral of persons suspected of having TB to a designated microscopy centre (DMC) (scheme 1), DOTS treatment by PP or staff for which drugs are provided (scheme 2), private health facility with its own laboratory working as a DMC under the RNTCP and charging fees (scheme 3A), private health facility with its own laboratory working as a DMC and DOTS provider under the RNTCP for which drugs are provided under the Programme and service fees are charged by the PP (scheme 3B), private health facility with its own laboratory working as DMC and providing free diagnostic services for which laboratory materials are provided under the RNTCP (scheme 4A), and private health facility with its own laboratory working as DMC providing free diagnostic services for which the required laboratory materials are provided, and DOTS provider, for which drugs are provided under the RNTCP (scheme 4B).

About 40%–50% of patients, although ultimately diagnosed and treated in a government health facility, were found to have incurred heavy expenditure while seeking a diagnosis in the private sector. This necessitates the active involvement of private practitioners in the management of TB.

More than 750 NGOs are officially providing RNTCP services because of their accessibility and flexibility of services. Various schemes have been formulated for the involvement of NGOs in the RNTCP which include health education and community outreach (scheme 1), provision of directly observed treatment (scheme 2), in-hospital care for TB disease (scheme 3), microscopy and treatment centre (scheme 4) and TB Unit Model (scheme 5).

Private practitioners and NGOs have been suboptimally involved under the RNTCP keeping in mind their large numbers and widespread availability and acceptability. Enormous efforts have to be made for involving them.

Role of pharmacists in the control of TB

In the majority of cases, retail drug outlets dispense the anti-TB drugs prescribed by PPs. In a recent study, the majority of pharmacists were found to be unaware of the existence of the RNTCP, but almost all of them were willing to learn and contribute to TB control (Rajeshwari *et al.* 2002a). No efforts have been made so far to sensitize private pharmacies/pharmacists about the RNTCP.

Management structure at the Central level

A study of the organizational structure of the Central TB Division in the Ministry of Health and Family Welfare shows that there are only five regular government officers managing the programme (DDG [TB], 3 CMOs and 1 TB Specialist), supported by 12 Consultants (Medical), 3 Consultants (Finance), 3 Consultants (LAN and Computer Networking), 5 Statistical Assistants and 2 Data Entry Operators whose services have been hired on contract. Similarly, services of 80 Medical Consultants have been hired at the State and district levels (supported by WHO) to increase their capacity; with rapid expansion of areas covered under the RNTCP, there are plans to hire the services of 40 more Medical Consultants at the State and district levels with WHO support.

Collaboration with the National AIDS Control Organization (NACO)

Tuberculosis is also an opportunistic infection in patients with HIV infection/AIDS. Due to the RNTCP, the incidence of new cases of TB is expected to decline, but due to HIV infection, there is likely to be a five- to six-fold increase in TB cases. Therefore, coordination between NACO and the RNTCP is being strengthened at the Central level. Moreover, there are also interactions between drugs used for the treatment of TB and those for HIV infection/AIDS. NACO and the Central TB Division of the GOI have issued joint guidelines for the treatment of TB in cases with HIV infection/AIDS. In the first two phases, coordination was improved in 14 States (in 6 high-prevalence States—Andhra Pradesh, Karnataka, Maharashtra, Manipur, Nagaland, Tamil Nadu, and in Delhi, Gujarat, Himachal Pradesh, Kerala, Orissa, Punjab, Rajasthan and West Bengal). Sensitization workshops have been conducted and staff of NACO and RNTCP have been trained on TB–HIV-related issues.

Analysis of financial expenditures under the RNTCP

The outlay for the RNTCP has increased from Rs 86 crore in 1997–98 to Rs 130.32 crore in 2003–04, the World Bank financing over 75% of the total and the remaining coming from the GOI, and through grants from other agencies (Table 2).

The Central Government procures microscopes, drugs and X-ray rolls and supplies them to the States for further distribution to the districts. In addition, it provides cash assistance for drugs for smear-negative cases and also releases cash to State and District TB Control Societies: 100% assistance is given for new constructions and minor

Table 2. Abstract summary of funding of the TB Control Programme for the years 2001–02, 2002–03 and 2003–04 (Rs in lakh)

			From external agencies							
Year		Domestic budget	World Bank	WHO	DANIDA	DFID	GFATM	GDF	USAID	Total
2001–02	In cash	450	8111.56	339.03	445	1351.5	0	0	0	10,697.09
	In kind	0	0	0	0	0	0	0	0	0
	Grants-in-aid to societies/NGOs	0	0	0	0	0	0	0	0	0
	Total	450 (4.2%)	8111.56 (75.8%)	339.03 (3.2%)	445 (4.2%)	1351.5 (12.6%)	0	0	0	10,697.09
	Grant/loan		Loan	Grant	Grant	Grant	Grant	Grant		
2002–03	In cash	174.45	7198.9	1241.51	749	1574	0	0	0	10,937.86
	In kind	0	0	0	0	0	0	0	0	
	Grants-in-aid to societies/NGOs	0	0	0	0	0	0	0	0	0
	Total	174.45 (1.6%)	7198.9 (65.7%)	1241.51 (11.4%)	749 (6.8%)	1574 (14.4%)	0	0	0	10,937.86
	Grant/loan		Loan	Grant	Grant	Grant	Grant	Grant		
2003–04 (estimated)	In cash	20	8326.48	1241.51	254.52	610	1550	0	100	12,102.51
	In kind	0	0	0	0	0	0	929	0	929
	Grants-in-aid to societies/NGOs	0	0	0	0	0	0	0	0	0
	Total	20 (0.2%)	8326.48 (63.9%)	1241.51 (9.5%)	254.52 (2.0%)	610 (4.7%)	1550 (11.9%)	929 (7.1%)	100 (0.8%)	13,031.51
	Grant/loan		Loan	Grant	Grant	Grant	Grant	Grant	Grant	

repair of buildings (Microscopy Centres, State Drugs Store, Computer Room, State TB Training and Demonstration Centre), purchase and maintenance/hiring of vehicles, laboratory consumables, training, printing/stationery, IEC activities/publicity, purchase of medical and office equipment and their maintenance, and office expenses. It also provides 100% assistance to the States for the salaries of the STS, STLS, LT, Tuberculosis Health Visitors (TBHV), drivers, part-time accountants and medical officers in 15% of District Tuberculosis Societies, the rest being borne by State Governments. The States provide salaries of one State Tuberculosis Officer along with clerks in his section.

Only 13.3% of expenditure in 2001–02 under the Programme was capital expenditure and 86.7% was of a recurring nature, the majority on drugs (52.3%) and only 2.0% on IEC activities (Table 3). In 2002–03, with expansion of coverage under the RNTCP, the Central TB Division spent Rs 440.96 lakh on IEC activities (compared to only Rs 1.07 lakh in 2001–02). Less amount was spent on IEC in 2001–02 because the RNTCP was not implemented throughout the country at that stage and almost all expenditure on IEC was incurred at the State/district level.

Recommendations

The national ARTI of 1.5% shows an insignificant decline since the 1955–58 survey, thereby indicating that efforts made for TB control have not been fruitful. The immediate priority for the Programme is to cover the entire country under the RNTCP by 2005 as it has been estimated that

Table 3. Expenditure under the Revised National Tuberculosis Control Programme, 2001–02 (Rs in lakh)

Expenditure head	Centre	States	Districts	Total	%
Recurring					
01. Salaries					
1 Regular staff	0	0	520.14	520.14	5.2
2 Contractual staff	16.43	141.25	987.09	1,144.77	11.4
4 Honorarium	0	0.26	18.81	19.07	0.2
02. Maintenance					
1 Of medical equipment	0	0	30.74	30.74	0.3
2 Of office equipment	0	4.76	0	4.76	0
3 Of vehicle	0	12.85	120.09	132.94	1.3
4 Civil works	0	0	0	0	0
03. Medical supplies/consumables					
1 Drugs	0	0	5262.64	5,262.64	52.3
2 Laboratory materials	0	19.23	425.76	444.99	4.4
04. Office expenses					
1 Stationery	0.32	15.37	140.62	156.31	1.6
2 Vehicle hiring	16.0	15.72	20.46	52.18	0.5
3 TA	0.65				
4 Miscellaneous	0	32.27	276.02	308.29	3.1
05. Training	17.32	36.39	326.57	380.28	3.8
06. IEC/publicity	1.07	36.08	168.20	205.35	2.0
07. Review and monitoring	18.21	0	0	18.21	0.2
08. Research and studies	0	0	0	0	0
09. NGO support	0	5.47	36.63	42.10	0.4
Total	70.0	319.65	8333.77	8,723.42	86.7
Capital					
10. Civil works					
1 New construction	0	14.45	263.78	278.23	2.8
11. Equipment					
1 Medical equipment	0		256.38	256.38	2.50
2 Office equipment	0	39.09	275.95	315.04	3.1
12. Vehicles purchase	0	103.36	381.63	484.99	4.8
Total	0	156.90	1177.74	1,334.64	13.3
Grand total	70.0	476.55	9511.51	10,058.06	100.0
%	0.7	4.70	94.60	100.00	

TA: travelling allowance; IEC: information, education and communication; NGO: non-governmental organization

for each year of delay in national expansion beyond 2005, the human and economic costs over the next 20 years will be high: 800,000 preventable cases of TB, 280,000 avoidable deaths and direct and indirect costs of Rs 5700 crore (WHO 2000). With rapid expansion of the RNTCP, the GOI will have to arrange for funds for the maintenance of RNTCP activities in already covered areas either from the domestic budget or from additional external funding, once funding from the World Bank ends. In an exercise undertaken by the NCMH for estimating the costs of providing universal access to the cure of TB, it was estimated that Rs 190.78 crore would be required annually for providing treatment to new cases of TB alone, in addition to administration and programme management costs. Against this, only Rs 130.32 crore were allocated for the RNTCP by the Central Government in 2003–04.

With the fastest expansion of RNTCP in the world, it is critical that the quality of monitoring and supervision be improved at all levels—the Centre, State and district—as the treatment costs of MDR TB will be more than ten times the treatment of a case under the RNTCP. This requires intensive efforts to improve and strengthen the monitoring and supervision capacity at all levels. The RNTCP is being implemented by a few officers from the regular services supported by services of consultants who have been hired on contract and who could be withdrawn any time. While this is beneficial in the short run, in the long run this seriously compromises the quality of management of programmes at all levels, as adequate attention is not given for maintaining the continuity of support to programme managers. To compound the problems, the frequent change of State Tuberculosis Officers (STOs) in many States critically affects the continuity of the Programme. Thus, top priority needs to be accorded to programme management structures and capacity at all levels—the Centre, State and district—instead of simply relying upon the support of consultants.

It has been observed that the DOTS strategy is not universally used for the treatment of TB even in districts that have been covered under DOTS. There are reports that the All India Institute of Medical Sciences (AIIMS), New Delhi and many medical colleges do not use this strategy. There is an urgent need to develop a network and a working relationship between medical colleges and RNTCP implementation. While making all-out efforts to improve monitoring and supervision, intensive efforts are required for maintaining high cure rates among new smear-positive cases. With the rapid expansion of the RNTCP to cover the remaining areas, there is also a need to detect more and more new smear-positive cases of TB so that case detection rates are increased from about 48% to over 70%. This would require intensive IEC efforts to make people aware of the Programme, of the free treatment available and of the success of the treatment.

In Singapore, under the Singapore Tuberculosis Elimination Programme, in addition to DOT, all identified close contacts (including household contacts and contacts in workplaces) receive a tuberculin skin test; chest X-ray is done for those who react positively or who are symptomatic. Thus, active case finding is conducted and infected contacts who would benefit from preventive therapy are identified. In this way, approximately four contacts per infectious case were identified and an average of one contact per index case received preventive therapy (Chee and James 2003). This kind of screening of close contacts would result in a reduction of the burden of TB disease and might be considered under the RNTCP.

Considering the fact that the private sector is growing fast and the majority of chest symptomatics initially consult PPs, there is an urgent need to integrate PPs in TB control programmes to move from individual cure to public health without minimizing the benefit of individual cure (Kalk 2003; Sudha *et al.* 2003). Involvement of just about 3000 PPs in the country is not enough, considering its vast size and the magnitude of the disease burden. So far, no effort has been made to involve pharmacists/private pharmacies. Better compliance and treatment completion rates, and reduced dropout rates would result if pharmacists are made aware of the drug regimens available under the RNTCP. Therefore, along with the involvement of PPs, orientation of private pharmacists about the tuberculosis control programme would be an important step towards complete treatment of TB leading to a reduction in drug-resistant cases.

Numerous NGOs are working at the grassroots level and have the competitive advantage of further reach in remote areas and considerable flexibility of approach. They could be optimally utilized for creating awareness among the masses about TB and its treatment, diagnosing smear-positive cases of TB and providing treatment under supervision. Many schemes have been designed under the RNTCP for involvement of NGOs but so far only about 750 NGOs have been actively involved in the implementation of the RNTCP. These schemes need to be popularized and NGOs have to be involved in a big way if a programme of this magnitude is to succeed.

MPWs (M) should be involved in supervising the administration of DOTS so that the ANM can attend to other duties. Second, since MDT is also administered under the NLEP with good compliance and because there is wide variation in the manner in which drugs are being administered under DOTS, it is possible to reduce supervision to once a week, if not less, especially for Category I and Category III cases where no injection is to be administered.

It is crucial that the cure rates among new sputum-positive cases be maintained above 85%. While at the national level the cure rate is 86%, there are districts with cure rates of less than 80% (as low as 40% in the Nuapada district of Orissa). While making all-out efforts to improve monitoring and supervision, intensive efforts are required in such areas for maintaining high cure rates among new smear-positive cases.

At present, the focus of DOTS is on diagnosing TB in

symptomatic patients who come to the health facilities. Once diagnosed, the patient is put under treatment (DOTS) for which he/she has to come to the health facility or go to another identified DOTS provider for taking the drugs. The involvement of the community has to be increased to supervise the administration of drugs under DOTS, as this has been shown in other countries (including Bangladesh and Africa) to be a more cost-effective strategy than health facility-based treatment. Increasing the involvement of the community in supervising the administration of drugs under DOTS will also have the additional advantage of reducing the stigma associated with TB, improve treatment compliance and completion rates, and result in better health outcomes especially for vulnerable groups such as women, children and the poor.

Vector-borne diseases: Malaria

Malaria situation in India

Malaria, which accounted for about 7.5 crore cases and 8 lakh deaths every year before the launch of the National Malaria Control Programme (NMCP) in 1953, saw a dramatic reduction to one lakh cases and no deaths in 1965 but was followed by a resurgence in 1976 due to resistance of the *Plasmodium* species to drugs and of mosquitoes to insecticides. A modified Plan of Operations launched in 1977 stabilized the incidence of malaria to around 20 lakh cases in the 1980s. During 1994, some epidemics due to malaria were observed in some States. A new control strategy called the Malaria Action Programme was started in 1995.

The reported incidence of malaria during the past decade was between 20 and 30 lakh cases per year, with about 1000 reported deaths annually; this has shown no decline. The number of *Plasmodium falciparum* cases has also remained around 10 lakh per year. Many States had an Annual Parasite Index (API) of over 2/1000 in 2002; these include Arunachal Pradesh, Assam, Chhattisgarh, Goa, Jharkhand, Karnataka, Meghalaya, Mizoram, Orissa, Tripura, West Bengal, and Dadra and Nagar Haveli. The number of malaria cases (total and *Plasmodium falciparum* cases) in the past ten years is shown in Tables 4 and 5 respectively and is depicted in Figure 3.

There is a consensus that malaria as a disease is grossly underreported, largely because information on the number of cases of malaria that is available from the National Vector-borne Disease Control Programme (NVBDCP) is based only on reported cases treated in the public sector, and a large proportion of cases of malaria treated in the private sector go unreported.

National Vector-borne Diseases Control Programme and Enhanced Malaria Control Project

The failure to cap the resurgence of malaria and the stagnant/increased incidence of other VBDs such as kala-azar, dengue, filariasis and Japanese encephalitis resulted in the integration of all these diseases under the NVBDCP with a budgetary outlay of Rs 1370 crore during the Tenth Plan. Of all these, malaria is the most virulent in terms of the number of lives it claims every year, particularly of the poor. The Enhanced Malaria Control Project (EMCP), financed by the World Bank, is being implemented since September 1997 in 1045 PHCs of 100 districts in 8 States and 19 urban areas. At the time of starting the Project, EMCP areas constituted around 7% of the country's population and contributed approximately 40% to the malaria burden. The main components strengthened under the Project are:

- Early detection and prompt treatment: Drug distribution centres (DDCs) and fever treatment depots (FTDs) are now functioning in the vast majority of villages in EMCP areas. Rapid diagnostic test kits have been supplied to EMCP districts for use in remote areas where access to microscopy is limited.
- Selective vector control: Reliance on indoor residual spraying (IRS) has declined by 30% in EMCP areas. This has been achieved by reducing the overall disease burden through expansion of early diagnosis and prompt treatment facilities, use of insecticide-treated bednets and use of larvivorous fish.
- Insecticide-treated bednets: These have been introduced in a big way in the project areas, and their popularity has increased because of partnerships with NGOs for community distribution and re-treatment, and intensive IEC campaigns for increasing the use of these bednets.
- Institutional strengthening: State- and district-level societies have been made functional; 3453 training courses have been organized, and IEC and social mobilization efforts have been intensified.

Central assistance for control of malaria and other vector-borne diseases

For the control of malaria, the Central Government provides assistance to States on the following pattern:

1. 100% central plan assistance for the north-eastern States, which includes material and equipment as well as cash assistance towards operational costs.
2. 100% assistance of material (insecticides, drugs, vehicles, bednets, equipment, diagnostic kits, etc.) and cash assistance through State and District Malaria Control Societies under the EMCP.
3. 50%:50% cost-sharing between the Centre and States as per policy. In the remaining malaria-prone areas the GOI provides material assistance in the form of insecticides (DDT, malathion, larvicides, drugs). The State Governments bear all other expenses of programme implementation.
4. Funds are also allocated to offices of the Regional

Table 4. State-wise distribution of the number of malaria cases from 1994 to 2003

State/UT	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003
Andhra Pradesh	90,301	98,013	127,814	129,577	118,800	129,020	80,557	57,735	38,053	35,995
Arunachal Pradesh	49,703	51,115	48,667	53,196	49,554	58,243	46,165	56,030	46,431	34,810
Assam	161,038	230,702	176,622	123,650	94,645	131,048	84,915	95,142	89,601	76,570
Bihar	71,900	86,722	104,680	74,676	114,958	131,898	9,509	4,108	3,683	2,652
Chhattisgarh							359,155	290,666	235,434	194,419
Goa	3456	3,886	11,632	21,025	25,975	15,380	9,164	12,331	16,818	11,370
Gujarat	242,456	191,028	143,817	159,652	106,825	64,130	36,712	81,347	82,966	130,744
Haryana	29,810	59,621	128,232	69,710	12,115	2,604	1,050	1,202	936	4,374
Himachal Pradesh	3,091	6,695	8,349	5,320	1,433	700	491	349	176	133
Jammu and Kashmir	2,760	9,005	14,289	9,412	5,451	3,574	3,045	912	455	309
Jharkhand							133,453	130,784	126,589	112,740
Karnataka	266,459	285,830	219,198	181,450	118,712	97,274	109,118	197,625	132,584	100,220
Kerala	9,075	11,878	11,653	8,265	7,439	5,141	2,940	2,289	3,360	2,380
Madhya Pradesh	323,628	483,563	500,574	451,552	475,098	527,510	194,689	183,118	108,818	99,708
Maharashtra	330,699	368,796	317,416	204,969	165,985	137,712	81,406	56,043	45,568	62,947
Manipur	7,845	4,652	2,151	1,742	1,306	2,662	1,064	943	1,268	2,589
Meghalaya	11,953	24,920	26,968	22,237	17,618	14,798	13,699	20,630	17,918	18,366
Mizoram	13,998	17,600	10,840	11,021	10,137	14,437	9,059	10,929	7,859	7,293
Nagaland	2,292	4,661	3,091	2,825	1,989	4,396	3,443	4,318	3,945	3,370
Orissa	332,046	369,777	458,554	421,928	478,056	483,095	509,497	454,541	473,223	417,276
Punjab	15,601	28,609	35,742	27,632	5,316	1,113	493	604	250	377
Rajasthan	241,255	250,780	300,547	272,670	76,438	53,154	35,973	129,233	68,627	142,738
Sikkim	58	214	49	38	15	14	16	31	53	278
Tamil Nadu	104,964	92,375	80,586	72,426	63,915	56,366	43,053	31,551	34,523	43,604
Tripura	8,871	12,503	9,843	18,122	12,595	14,408	12,245	18,502	13,319	13,807
Uttar Pradesh	89,617	105,235	169,364	134,362	112,291	99,362	96,971	94,524	90,199	81,853
Uttaranchal							2,008	1,196	1,659	2,350
West Bengal	74,283	91,014	87,686	155,209	132,088	227,480	145,322	145,053	194,421	175,739
Andaman and Nicobar Islands	1,619	1,636	1,165	972	1,247	937	1,002	925	865	753
Chandigarh	7,853	9,875	11,196	4,944	1,675	456	256	298	157	84
Dadra and Nagar Haveli	8,571	15,992	11,968	12,007	6,225	3,303	2,415	848	493	468
Daman and Diu	1,236	1,562	2,052	1,062	625	352	132	87	173	141
Delhi	4,365	7,470	10,562	8,194	4,050	3,996	2,631	1,484	1,484	810
Lakshadweep	2	1	0	2	4	1	5	0	8	6
Pondicherry	548	467	281	210	168	149	137	106	103	63
India	2,511,353	2,926,197	3,035,588	2,660,057	2,222,748	2,284,713	2,031,790	2,085,484	1,842,019	1,781,336

Directors of Health and Family Welfare for IEC/monitoring/supervision of malaria control activities in their respective regions.

5. Funds allocated for the NVBDCP during the years 2001–02, 2002–03 and 2003–04 were Rs 219.78 crore, Rs 206.81 crore and Rs 203.61 crore, respectively (Table 6).
6. Additional funds for activities for the control of kala-azar are released in cash and kind only to four States where kala-azar is endemic (Bihar, Jharkhand, West Bengal and Uttar Pradesh).

Why is malaria persisting? Challenges and constraints in implementation of the Programme

1. The 1994 Expert Committee laid down criteria for identification of 'high-risk' rural as well as urban areas and assigned the major responsibility for case detection and management of malaria cases in rural areas to the MPW (M). However, the large number of vacancies for MPWs (M) in most States has been a major bottleneck in the effective implementation of activities for the control of malaria. Insecticidal spray operations are not performed regularly/periodically as per guidelines.
2. Some vectors of malaria have developed resistance to the insecticides currently in use. Some vectors have developed outdoor biting and outdoor resting habits. Since newer insecticides are very expensive, alternative strategies for vector control need to be implemented.
3. The number of cases of malaria has been stagnant at around 20 lakh cases annually, but the proportion of cases of *Plasmodium falciparum* malaria has increased to over 42% and this species has shown resistance to conventional antimalarial drugs. Moreover, areas reporting resistance of *Plasmodium falciparum* to drugs have been increasing. Second-line drugs for chloroquine-resistant *falciparum* malaria are available to only 1% of the population.

Table 5. State-wise distribution of number of cases of Plasmodium falciparum from 1994 to 2003

State/UT	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003
Andhra Pradesh	32,227	40,803	60,402	57,939	61,611	78,039	46,685	34,387	21,416	20,864
Arunachal Pradesh	9,352	7,512	5,686	6,730	6,935	10,263	6,966	11,025	7,080	5,977
Assam	105,477	145,153	107,742	76,548	54,769	83,064	52,116	58,961	55,825	48,668
Bihar	46,367	55,627	64,859	49,470	75,825	79,881	2,084	1,027	1,705	1,080
Chhattisgarh							246,129	201,569	170,487	144,028
Goa	275	256	1,539	5,768	8,694	5,548	2,598	3,569	3,655	1,503
Gujarat	63,494	44,932	32,091	37,849	18,531	10,617	6,672	18,958	16,244	30,895
Haryana	3,701	11,215	27,868	2,218	306	211	157	143	41	500
Himachal Pradesh	6	13	12	4	1	6	0	0	0	7
Jammu and Kashmir	48	39	69	34	18	37	23	24	10	11
Jharkhand							68,744	63,471	52,892	36,470
Karnataka	37,789	39,287	32,416	43,546	26,333	21,416	28,303	48,008	29,702	23,560
Kerala	272	553	657	659	1,064	568	373	325	375	410
Madhya Pradesh	151,440	223,718	221,080	211,537	247,196	289,187	62,850	61,140	31,545	31,390
Maharashtra	103,616	131,602	83,669	55,230	48,004	33,898	25,694	19,340	14,634	30,340
Manipur	5,314	2,161	927	801	631	1,399	380	371	601	1,168
Meghalaya	7,712	12,174	14,230	10,910	8,510	9,153	9,238	15,890	11,095	12,338
Mizoram	7,327	10,771	6,248	6,990	6,422	9,575	5,358	5,955	3,932	4,167
Nagaland	944	991	663	806	423	202	264	498	234	277
Orissa	284,346	317,204	395,896	364,723	408,509	407,942	428,032	379,432	393,547	346,311
Punjab	185	3,367	1,232	150	52	36	25	41	18	35
Rajasthan	94,020	45,027	72,329	19,554	10,030	5,875	3,425	17,405	5,356	16,481
Sikkim	8	4	2	3	2	2	1	13	7	41
Tamil Nadu	4,935	5,463	4,011	3,049	2,303	2,281	1,738	1,354	2,520	3,758
Tripura	6,975	9,103	7,112	15,491	10,507	11,889	9,480	14,629	10,863	10,800
Uttar Pradesh	7,516	7,367	20,974	11,023	5,407	6,434	6,214	4,546	2,512	1,290
Uttaranchal							424	280	120	265
West Bengal	15,392	17,226	14,725	23,545	25,156	72,755	32,465	42,596	60,726	72,232
Andaman and Nicobar Islands	270	311	215	168	183	182	236	180	158	148
Chandigarh	59	59	84	17	6	18	6	3	6	5
Dadra and Nagar Haveli	1,362	4,310	2,092	2,467	2,694	648	282	59	100	106
Daman and Diu	47	136	47	15	19	35	5	22	32	21
Delhi	22	34	682	122	16	196	249	14	14	25
Lakshadweep	0	0	0	0	0	0	0	0	0	0
Pondicherry	9	5	2	0	2	2	2	1	2	2
India	990,507	1,136,423	1,179,561	1,007,366	1,030,159	1,141,359	1,047,218	1,005,236	897,454	845,173

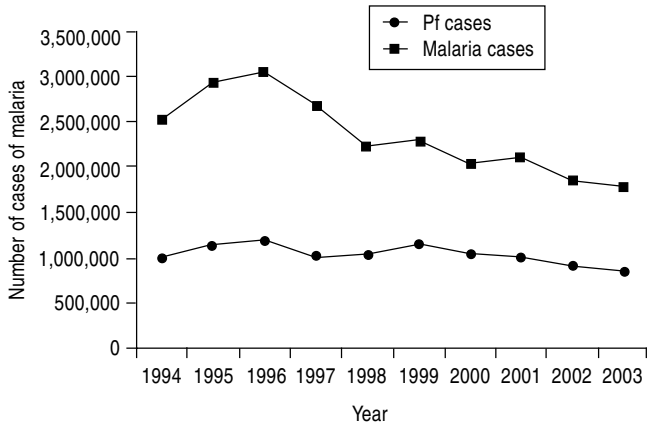

Fig. 3 Number of cases of malaria

Pf: *Plasmodium falciparum*

4. Major ecological imbalances on account of large projects such as irrigation, deforestation and paddy cultivation are continuously taking place, thereby increasing vector breeding and the epidemic potential of malaria in such areas. Involvement of the community is critical in the control of malaria through reduction of breeding sites. However, efforts at mobilizing the community, if at all, have not been successful and communities and NGOs need to be involved in a big way for controlling the disease.
5. The risk of urban malaria has been constantly increasing due to urbanization and industrialization leading to mass migration of people and increase in vector breeding on account of stagnant water. Migration of labourers coming from areas with drug-resistant malaria into such areas further worsens the situation.

Table 6. Abstract summary of funds released to States for the control of vector-borne diseases (Rs in lakh)

	Year											
	2001–02				2002–03				2003–04			
State/Union Territory	In cash	In kind	Grants-in-aid to societies/ NGOs	Total	In cash	In kind	Grants-in-aid to societies/ NGOs	Total	In cash	In kind	Grants-in-aid to societies/ NGOs	Total
Andhra Pradesh	145	810	0	955	93	456	0	549	142	253	0	395
Arunachal Pradesh	203	162	0	365	201	176	0	377	93	187	0	280
Assam	1271	1,106	0	2,377	0	1,935	0	1,935	0	1,404	0	1,404
Bihar	70	804	0	874	0	198	0	198	555	1,884	0	2,439
Chhattisgarh	167	710	0	877	818	2,230	0	3,048	475	1,212	0	1,687
Goa	0	6	0	6	0	8	0	8	0	0	0	0
Gujarat	195	1,158	0	1,353	245	523	0	768	82	247	0	329
Haryana	0	18	0	18	0	67	0	67	0	37	0	37
Himachal Pradesh	0	37	0	37	0	12	0	12	0	5	0	5
Jammu and Kashmir	0	70	0	70	0	382	0	382	0	72	0	72
Jharkhand	89	793	0	882	396	956	0	1,352	54	1,103	0	1,157
Karnataka	0	386	0	386	0	227	0	227	0	205	0	205
Kerala	0	68	0	68	0	13	0	13	63	47	0	110
Madhya Pradesh	362	2,180	0	2,542	681	1,727	0	2,408	142	1,115	0	1,257
Maharashtra	235	2,054	0	2,289	293	655	0	948	244	115	0	359
Manipur	144	131	0	275	72	74	0	146	61	13	0	74
Meghalaya	166	127	0	293	62	240	0	302	99	226	0	325
Mizoram	136	210	0	346	35	156	0	191	148	128	0	276
Nagaland	144	225	0	369	75	297	0	372	146	245	0	391
Orissa	290	1,455	0	1,745	239	2,792	0	3,031	545	1,793	0	2,338
Punjab	0	94	0	94	0	66	0	66	0	38	0	38
Rajasthan	84	841	0	925	41	883	0	924	143	1,272	0	1,415
Sikkim	0	0	0	0	0	4	0	4	2	4	0	6
Tamil Nadu	0	289	0	289	0	187	0	187	0	111	0	111
Tripura	215	291	0	506	0	390	0	390	152	287	0	439
Uttar Pradesh	0	671	0	671	0	607	0	607	27	578	0	605
Uttaranchal	0	40	0	40	0	2	0	2	0	39	0	39
West Bengal	25	1,116	0	1,141	0	519	0	519	126	386	0	512
Andaman and Nicobar Islands	190	30	0	220	185	45	0	230	181	23	0	204
Chandigarh	27	8	0	35	24	14	0	38	25	0	0	25
Dadra and Nagar Haveli	18	23	0	41	16	0	0	16	24	37	0	61
Daman and Diu	9	10	0	19	7	1	0	8	15	4	0	19
Lakshadweep	5	1	0	6	5	0	0	5	12	0	0	12
Pondicherry	1	7	0	8	1	12	0	13	8	3	0	11
Delhi	4	85	0	89	0	60	0	60	0	81	0	81
Total	4195	16,016	0	20,211	3489	15,914	0	19,403	3564	13,154	0	16,718
Estt/Pub/Res	1767			1,767	1278			1,278	3643			3,643
Grand total	5962			21,978	4767			20,681	7207			20,361

Recommendations

Since reliable information on the occurrence of malaria and other VBDs in India is very limited, getting information from the private sector is crucial. In a large number of cases, PPs treat cases of fever for malaria on the basis of symptoms alone rather than basing their diagnosis on a blood smear examination. Even in the public sector, there is a large backlog of peripheral blood smear slides for examination due to the large number of vacancies of LTs at PHCs and CHCs. It is evident that for improving the health outcomes of the people, a health team should be in position and LTs and MPWs (M) are important constituents of the health team. Thus, vacant posts of LTs and MPWs (M) need to be filled, especially in peripheral health institutions so that health improvement in the population can be assured. Filling up the posts of MPWs (M) will reduce the burden on the ANM and she might then be able to deliver maternal and child care services more efficiently and effectively.

Vectors are an important link in the transmission of

malaria and other VBDs, and integrated vector control activities need to be implemented for controlling the burden of VBDs. The success achieved in the control of malaria in the 1950s and 1960s was primarily due to control of the vector. The community should be actively involved in activities aimed at controlling the vector—reduction of vector breeding places (preventing water stagnation, ensuring that overhead tanks are covered with lids, draining the water from coolers and drying them once a week, pouring mineral oil over collections of water in the open), and protection against bites by vectors by using bednets and mosquito repellants. In addition, the community could be given the responsibility of monitoring insecticide spray operations in the area. Integrated activities for vector control are crucial for the containment of malaria as undue reliance on insecticide spraying is bound to lead to resistance of the vector against the insecticide, and already there are areas where the vector has shown resistance against DDT. Synthetic pyrethroids have now been introduced as the insecticide in such areas.

There are reports of drug resistance of *Plasmodium falciparum* against firstline drugs and newer drugs are being introduced under the Programme only now. Since most patients with malaria reportedly consult the private sector in the first instance, involvement of the private sector is crucial for ensuring complete treatment of identified cases of malaria so that the problem of drug resistance of the parasite can be contained.

References

Becx-Bleumink M, Djamaluddin S, Loprang F, De Soldenhoff R, Wibowo H, Aryono M. High cure rates in smear-positive tuberculosis patients using ambulatory treatment with once-weekly supervision during the intensive phase in Sulawesi, Republic of Indonesia. *International Journal of Tuberculosis and Lung Disease* 1999;**3:**1066–72.

Beers Stella M van, Hatta Mohammad, Klatser Paul R. Patient contact is the major determinant in incident leprosy: Implications for future control. *International Journal of Leprosy* 1999;**67:**119–28.

Chee Cynthia BE, James L. The Singapore Tuberculosis Elimination Programme: The first five years. *Bulletin of the World Health Organization* 2003;**81:**217–21.

Gokhale SD. Economic/social rehabilitation. *International Journal of Leprosy* 2001;**69** (Suppl.):S42–S51.

Government of India. *National Health Policy*. Ministry of Health and Family Welfare; 1983.

Government of India. *TB India 2002 RNTCP Status Report*: Ministry of Health and Family Welfare, Central TB Division, Directorate General of Health Services; 2002a:10–11.

Government of India 2002b. Ministry of Health and Family Welfare. National Programme for Control of Blindness. *NPCB-India* 2002:1.

Government of India. Ministry of Health and Family Welfare. *Annual Report 2002–2003*. 2003a:38.

Government of India. *TB India 2003 RNTCP Status Report.* Ministry of Health and Family Welfare, Central TB Division, Directorate General of Health Services; 2003b:33.

Government of India. *TB India 2004 RNTCP Status Report.* Ministry of Health and Family Welfare, Central TB Division, Directorate General of Health Services; 2004a:5, 8, 15, 31.

Government of India. *Annual Report 2003–04*. Ministry of Health and Family Welfare, 2004b.

Health and Development Initiative, 2004a. Available from URL: http://www.healthinitiative.org/html/toolkit/indperspective.htm (accessed on 15 July 2004).

Health and Development Initiative, 2004b. Available from URL: http://www.healthinitiative.org/html/ toolkit/indperspective2.htm (accessed on 15 July 2004).

Kalk A. How private practitioners help to control TB. *Bulletin of the World Health Organization* 2003;**81:**148.

Kalla G, Kachhawa D, Salodkar A. Disabilities in leprosy. *International Journal of Leprosy* 2000;**68:**182–4.

Limburg H, Kumar R, Bachani D. Monitoring and evaluating cataract intervention in India. *British Journal of Ophthalmology* 1996;**80:** 951–5.

Max E, Shepherd DS. Productivity loss due to deformity in India. *International Journal on Leprosy and Other Mycobacterial Diseases* 1989;**57:**476–82.

Mayurnath S, Vallishayee RS, Radhamani MP, Prabhakar R. Prevalence study of tuberculosis infection over fifteen years, in a rural population in Chingelput district (south India). *Indian Journal of Medical Research* 1991;**93:**74–80.

Neira MP. Asian Leprosy Congress—The final push to eliminate leprosy. *International Journal of Leprosy* 2001;**69** (Suppl.):S7–S9.

Newell J. The implications for TB control of the growth in numbers of private practitioners in developing countries. *Bulletin of the World Health Organization* 2002;**80:**836–7.

Narain R, Geser A, Jambunathan MV, Subramanian M. Tuberculosis prevalence survey in Tumkur District. *Indian Journal of Tuberculosis* 1963;**10:**85–116.

Rangan S. User perspective in urban tuberculosis control. In: Chakraborty AK, Rangan S, Uplekar M (eds). *Urban tuberculosis control: Problems and prospects.* Mumbai: The foundation for Research in Community Health; 1995:97–106.

Rajeswari R, Balasubramaniam R, Bose MSC, Sekar L, Rahman F. Private pharmacies in tuberculosis control—a neglected link. *International Journal of Tuberculosis and Lung Disease* 2002a;**6:** 171–3.

Rajeswari R, Chandrasekaran V, Suhadev M, Sivasubramaniam S, Sudha G, Renu G. Factors associated with patient and health system delays in the diagnosis of tuberculosis in South India. *International Journal of Tuberculosis and Lung Disease* 2002b;**6:**789–95.

Reddy BN, Bansal RD. An epidemiological study of leprosy disability in a leprosy endemic rural population of Pondicherry (South India). *Indian Journal of Leprosy* 1984;**56:**191–4.

Saha SP, Das KK. Disability pattern amongst leprosy cases in an urban area (Calcutta). *Indian Journal of Leprosy* 1993;**65:**305–14.

Sudha G, Nirupa C, Rajasakthivel M, Sivasubramanian S, Sundaram V, Bhatt S, *et al*. Factors influencing the care-seeking behaviour of chest symptomatics: A community-based study involving rural and urban population in Tamil Nadu, South India. *Tropical Medicine and International Health* 2003;**8:**336–41.

Tuberculosis Research Centre, Indian Council of Medical Research, Chennai, India. Low rate of emergence of drug resistance in sputum positive patients treated with short course chemotherapy. *International Journal of Tuberculosis and Lung Disease* 2001;**S(1):**40–5.

Volmink J, Matchaba P, Garner P. Directly observed therapy and treatment adherence. *Lancet* 2000;**355:**1345–50.

World Bank. *World Development Report: Investing in health*. Washington, DC: Oxford University Press; 1993.

World Bank. *Implementation Completion Report (IDA-26110)*. World Bank; 2003.

World Health Organization. *Technical Report Series 459*. WHO Expert Committee on Leprosy. Second report; 1960.

World Health Organization. Progress towards leprosy elimination. *Weekly Epidemiological Records* 1997;**72:**165–72.

World Health Organization. *Joint tuberculosis programme review India*. New Delhi: WHO, Regional Office for South-East Asia, SEA-TB-224; 2000:3.

World Health Organization. Leprosy—Learning from success. WHO/CDS/CPE/CEE/2001.20; 2001.

World Health Organization, Dholakia A. *Potential economic benefits of the DOTS strategy against TB in India*. In: Almeida J (ed). Available from URL: http://www.who.int/gtb/publications/pebdots/ (accessed on 18 August 2004).

Zwarenstein M, Schoeman JH, Vundule C, Lombard CJ, Tatley M. Randomised controlled trial of self-supervised and directly observed treatment of tuberculosis. *Lancet* 1998;**352:**1340–3.

Burden of tuberculosis in India for the year 2000

TUBERCULOSIS RESEARCH CENTRE, CHENNAI

Tuberculosis Research Centre (TRC), Chennai proposed to undertake a series of disease and tuberculin surveys from 1999 onwards to estimate various epidemiological indices such as prevalence and incidence of tuberculosis (TB) among those 15 years of age and above, and the annual risk of tuberculosis infection (ARTI) among children below 10 years of age. This is being done to assess the impact of Directly Observed Treatment, Short-course (DOTS) strategy implemented in 1999 in the Tiruvallur district of Tamil Nadu. These specific and unique surveys are meticulously carried out at regular intervals for ten years to measure the trend of TB. Chest symptoms and X-ray examinations were used as screening tools in these surveys unlike in the National Sample Survey (NSS) conducted during 1955–58 where only X-ray was used as a screening tool. The prevalence cases considered for estimation of the burden of disease were adjusted for non-coverage of X-ray and sputum examinations in the survey as done in the NSS.

The prevalence obtained from the first survey was used for estimating the burden of TB in the year 2000. The prevalence of smear-positive cases was estimated to be 333/100,000 population in the age group of ≥15 years. The corresponding figure for smear-negative, culture-positive cases was 332/100,000 population. The ARTI, estimated by the mirror image method using the mode at 15 mm, was 2%. To estimate the prevalence of sputum-negative X-ray abnormality, those who were classified by at least two independent readers, as probably or possibly having TB on mass miniature radiography (MMR) were included. Using this definition, the prevalence of sputum-negative X-ray abnormal cases was estimated to be 2360/100,000 population in the study area.

As there are no data available from the TRC on the prevalence of bacillary or X-ray abnormal cases for children in the age group of 0–14 years, we used the estimate of prevalence obtained from one of the surveys conducted by the National Tuberculosis Institute (NTI).[1] The prevalence of bacillary cases was 150/100,000 in the age group of 5–14 years (17/11,345) and that of X-ray abnormal cases was 304/100,000 (50/16,451). Assuming there were no bacillary cases among those in the age group of 0–4 years, the prevalence of smear-positive cases, smear-negative, culture-positive cases was estimated to be 26/100,000 and 123/100,000, respectively. The prevalence of the ARTI in the south zone obtained from the NSS study on ARTI was 1%.

The recently concluded NSS on ARTI, jointly carried out by TRC and NTI among children in four zones of the country has given reliable estimates of ARTI in rural (north: 1.5%, south: 0.8%, west: 1.4%, east: 1.2%) and urban (north: 3.3%, south: 1.6%, west: 2.1%, east: 1.6%) areas.[2]

The population of India was estimated to be about 100.5 crore using the Census Population of India, 1991 and the decadal growth rate of 21.34%. The burden of disease was estimated at the national level (with its inherent limitations in such exercises and based on different assumptions) using data on adults ≥15 years of age generated by the TRC, data on children 0–14 years of age obtained by the NTI and estimates of ARTI obtained from different parts of the country. This was done by obtaining the prevalence of 1% ARTI from TRC data and proportionately estimating the prevalence for rural and urban areas of the four zones from corresponding ARTI estimates and adding them together to get the overall estimate of burden of the disease in the country.

The number of bacillary cases was estimated to be 0.38 crore and that for sputum-negative X-ray abnormal cases as 1.29 crore. All the X-ray abnormal cases estimated here may not be bacillary cases and most of them may not become active cases in the future. Studies done by NTI[3] and Hong Kong[4] show that about 30% of X-ray abnormal cases are likely to break down to bacillary cases. Thus, 0.39 crore (1.29 crore × 30%) of the X-ray abnormal cases that break down to bacillary cases was alone considered to be abacillary pulmonary cases. The number of extrapulmonary cases was estimated to be 0.08 crore of the 0.39 crore bacillary

Table 1. Estimated burden of tuberculosis in India for the year 2000

Type of case	Number (in crore)
Bacillary	0.38
Abacillary	0.39
Extrapulmonary	0.08
Total	0.85

cases (20% of 0.38 crore). Thus, the burden of TB projected in the country for the year 2000 was 0.85 crore (0.38 + 0.39 + 0.08 crore). This estimate did not consider the possible association of HIV and multidrug resistant (MDR) TB from different parts of India.

References

1. Suryanarayana L, Suryanarayana HV, Jaganatha PS. Prevalence of pulmonary tuberculosis among children in a south Indian community. *Indian Journal of Tuberculosis* 1999;**46:**171–8.
2. Directorate General of Health Services (DGHS), Government of India (GOI), the National Tuberculosis Institute (NTI), Bangalore and the Tuberculosis Research Centre, Chennai. *Annual risk of tuberculosis infection in different zones of India: A national sample survey 2000–2003*. Bangalore: NTI; 2004.
3. Aneja KS, Gothi GD, Rupert Samuel GE. Controlled study of the effect of specific treatment on bacteriological status of 'suspect' cases. *Indian Journal of Tuberculosis* 1999;**46:**50–7.
4. Hong Kong Chest Service, Tuberculosis Research Centre and British Medical Research Council. Sputum smear negative pulmonary tuberculosis: Controlled trial of 3-month 2-month regimens of chemotheraphy. First report; 8131© The Lancet Ltd, 1979.

Causal analysis and treatment protocols for tuberculosis

TUBERCULOSIS RESEARCH CENTRE, CHENNAI

Table 1. Causes of conditions leading to tuberculosis (by significance)

	Direct (normally clinical)	Indirect (diet, exercise, alcohol)	Distant (sociopolitical, economic, empowerment, gender, literacy, etc.)
Main causes	Bacterial infection (*Mycobacterium tuberculosis*)		
Predisposing factors	• Diabetes mellitus • HIV infection • Silicosis • Close contact (especially of young children below 6 years of age) with smear-positive TB patients • Unimmunized child, especially for extrapulmonary TB • Incompletely treated known open case of TB • Patients on immunosuppressive therapy • Lack of diagnostic and treatment facilities	• Malnutrition • Smoking • Alcoholism	• Lack of political commitment • Poverty/low socioeconomic status • Overcrowding • War/famine • Illiteracy • Stigma associated with TB • Lack of community awareness • Males more than 45 years of age are at higher risk of developing TB

References

Diabetes and TB

1. Patel JC. Complication in 8793 cases of diabetics mellitus—a 14 years' study in Bombay Hospital, Bombay, India. *Indian Journal of Medical Sciences* 1989; **43:**177.
2. Ezung T, Devi NT, Singh NT, Singh TB. Pulmonary tuberculosis and diabeties mellitus—a study. *Journal of the Indian Medical Association* 2002;**100:**376.

HIV and TB

1. Raviglione MC, Harries AD, Msiska R, *et al*. Tuberculosis and HIV: Current status in Africa. *AIDS* 1997;**11:**S115–S123.
2. Elliott AM, Luo N, Tembo G, *et al*. Impact of HIV on tuberculosis in Zambia: A cross-sectional study. *British Medical Journal* 1990;**301:**412–15.
3. Harries AD. Tuberculosis in Africa—clinical presentation and management. *Pharmacology and Therapeutics* 1997;**73:**1–50.
4. Devdatta S, Dawson JJY, Fox W, *et al*. Attack rate of TB in a 5 year period among close family contacts of tuberculous patients under domiciliary treatment with H + PAS or H alone. *Bulletin of the World Health Organization* 1970;**42:**337–51.
5. Lutong L, Bei Z. Association of prevalence of tuberculin reaction with closeness of contact among household contacts of new smear-positive pulmonary tuberculosis patients. *International Journal of Tuberculosis and Lung Disease* 2000;**4:**275–7.

Malnutrition

1. Marrero A, Caminero JA, Rodriguez R. Towards elimination of tuberculosis in a low-income country: The experience of Cuba. 1962–97. *Thorax* 2000;**55:**39–45.

Poverty

1. Muniyandi M, Rajeswari R, Balasubramanian R. Tuberculosis Control Programme—is it pro-poor? *SAARC Journal of Tuberculosis, Lung Diseases and HIV* 2004;**1:**14–19.
2. Spence DP, Hotgchkiss J, Williams CS, *et al.* Tuberculosis and poverty. *British Medical Journal* 1993;**307:**789.
3. Barnes PF. Tuberculosis among the inner city poor. *International Journal of Tuberculosis and Lung Disease* 1998;**2:**41.
4. Parthania V, Almeida J, Nunn P, *et al.* The socioeconomic status of TB patients in India. Global TB Programme. Geneva: WHO; 1997.

Alcoholism

1. Santha T, Garg R, Frieden TR, *et al.* Risk factors associated with default, failure and death among tuberculosis patients treated in a DOTS programme in Tiruvallur District, South India, 2000. *International Journal of Tuberculosis and Lung Disease* 2002;**6:**780–8.
2. Balasubramanian R, Garg R, Santha T, *et al.* Gender disparities in tuberculosis: Report from a rural DOTS programme in south India. *International Journal of Tuberculosis and Lung Disease* 2004;**8:**323–32.

Smoking

1. Kolappan C, Gopi PG. Tobacco smoking and pulmonary tuberculosis. *Thorax* 2002;**57:**964–66.

Listing interventions for tuberculosis (by significance)

Medical interventions

1. Chemotherapy for tuberculosis
   a. Ambulatory, domiciliary treatment
   b. Supervise chemotherapy (Directly Observed Treatment [DOT]) as standard of care
   c. Intermittent chemotherapy
   d. Short-course daily regimen (6–8 months)
   e. Short-course intermittent regimen
   f. Evolved shorter duration (4 months) regimen with inclusion of ofloxacin
   g. DOT with patient-wise box concept in Revised National Tuberculosis Control Programme (RNTCP)
   h. Decentralized supply of drugs

2. Prophylaxis
   a. Chemoprophylaxis for young close contacts of sputum-positive patients
      Isoniazid 5 mg per kg body weight for 6 months
   b. Immunoprophylaxis—bacille Calmette-Guérin (BCG)

3. HIV and TB

   Antituberculosis treatment (ATT) as for HIV-negative TB patients and management with antiretroviral drugs

4. Diabetes mellitus and TB

   ATT as for non-diabetic TB patients and management of diabetes

5. Silicosis

   Screen for TB once in six months and ATT as for non-silicotic TB patients

Non-medical interventions

1. Alcoholism and smoking—focused information, education and communication (IEC) activity

Other interventions

1. Political commitment
2. Involvement of the private sector
3. Community awareness and involvement
4. Training and re-training of health personnel
5. Poverty alleviation programmes

Table 2. Listing interventions for tuberculosis (by significance)

		Non-medical interventions/prevention		
Outcome	Medical interventions	Exercise	Nutrition	Others
Manifestation type 1 (smear-positive pulmonary TB)	• Mainly chemotherapy, surgical intervention rarely required • Immunoprophylaxis—BCG • Chemoprophylaxis—isoniazid 5 mg/kg body weight daily for 6 months • Treatment of HIV-infected/AIDS cases • Strategies for prevention of HIV infection —as per NACO guidelines • Silicosis: Screen patients with silicosis once in 6 months and treat for TB when necessary	• Lifestyle modification so that the incidence of diabetes mellitus could be reduced • Interventions to deal with alcoholism and smoking	Interventions to prevent, treat/reduce malnutrition, both in children and adults	• Health education for raising community awareness about TB, its treatment (that treatment has to be completed), about the role of BCG vaccination, etc. • Interventions to reduce poverty and improve the economic condition of the people
Manifestation type 2 (smear-negative pulmonary TB)	Chemotherapy			• Training • Incentives
Manifestation type 3, etc. (extrapulmonary TB)	• Chemotherapy • Surgery			Monitoring and supervision

It would be good to relate this to the table wherein cases have been enumerated. Based on those causes, interventions could be derived, so that no intervention is missed.

References

Extrapulmonary TB

1. Rajeswari R, Balasubramanian R, Venkatesan P, *et al.* Short course chemotherapy in the treatment of Pott's paraplegia: Report on five-year follow-up. *International Journal of Tuberculosis and Lung Disease* 1997;**1:**152–8.
2. Jawahar MS, Sivasubramanian S, Vijayan VK, *et al.* Short-course chemotherapy for tuberculosis lymphadenitis in children. *British Medical Journal* 1990;**301:**359–62.
3. Balasubramanian R, Nagarajan M, Balambal R, *et al.* Randomised controlled clinical trial of short course chemotherapy in abdominal tuberculosis: A five year report. *International Journal of Tuberculosis and Lung Disease* 1997;**1:**44–51.
4. Rajeswari R, Sivasubramanian S, Balambal R, *et al.* A controlled clinical trial of short-course chemotherapy for tuberculosis of the brain. *Tubercle and Lung Disease* 1995;**76:**111–17.
5. Parthasarathy R, Sriram K, Santha T, *et al.* Short-course chemotherapy for tuberculosis of spine. A comparison between ambulant and radical surgery. *Journal of Bone and Joint Surgery* (British Volume) 1998;**81:**464.

HIV and TB

1. Harries AD. Tuberculosis in Africa: Clinical presentation and management. *Pharmacology and Therapeutics* 1997;**73:**1–50.
2. Alpert PL, Munsiff SS, Gourevitch NN, *et al.* A prospective study of tuberculosis and human immunodeficiency virus infection: Clinical manifestations and factors associated with survival. *Clinical Infectious Diseases* 1997;**24:**661–8.

Immunoprophylaxis

1. Brewer TF. Preventing tuberculosis with bacille Calmette–Guérin vaccine: A meta-analysis of the literature. *Clinical Infectious Diseases* 2000;**31** (Suppl. 3):S64–S67.

TB in children

1. Central TB Division. RNTCP: TB in children: Consensus guidelines of pediatricians, TB experts and TB control programme managers. Nirman Bhavan, New Delhi: Directorate General of Health and Family Welfare; 2004.

Males more than 45 years and TB

1. Tuberculosis Research Centre, Chennai. Association of initial tuberculin sensitivity, age and sex with the incidence of tuberculosis in south India: A 15-year follow-up. *International Journal of Tuberculosis and Lung Diseases* 2003;**7:**1083–91.
2. Balasubramanian R, Garg R, Santha T, *et al.* Gender disparities in tuberculosis: Report from a rural DOTS programme in south India. *International Journal of Tuberculosis and Lung Disease* 2004;**8:** 323–32.

Illiteracy

1. Rajeswari R, Balasubramanian R, Muniyandi M, *et al.* Socio-economic impact of tuberculosis on patients and family in India. *International Journal of Tuberculosis and Lung Disease* 1999;**3:**869–77.
2. Sudha G, Nirupa C, Rajasakthivel M, *et al.* Factors influencing the care-seeking behaviour of chest symptomatics: A community-based study involving rural and urban population in Tamil Nadu, South India. *Tropical Medicine and International Health* 2003;**8:**336–41.

Silicosis

1. Tam CM, Leung CC, Noertjojo K, *et al.* Tuberculosis in Hong Kong—patient characteristics and treatment outcome. *Hong Kong Medical Journal* 2003;**9:**83–90.

Table 3. Standard treatment protocol for tuberculosis

Conditions	Type	Tests (by type)	Drugs (dosage, type and duration)	Inpatient stay
Manifestation type 1	• Smear-positive pulmonary TB *M. tuberculosis*	Sputum smear for AFB and sputum culture for	• RNTCP-recommended treatment regimens Category I—isoniazid (600 mg), rifampicin (450 mg), pyrazinamide (1500 mg) and ethambutol (1200 mg), thrice a week on alternate days. Duration: 6 months Category II—Inj. streptomycin in addition to the above drugs. Duration: 8 months	Inpatient treatment generally not required. However, a small group of patients (10%) may need admission on account of severity of illness or complications of disease or treatment.
	• Re-treatment cases		• Paediatric dosage: Generally Category III regimen is prescribed for children. Isoniazid: 10 mg/kg, rifampicin: 10 mg/kg, Pyrazinamide: 30–35 mg/kg, ethambutol: 30 mg/kg, Streptomycin: 15 mg/kg	
Manifestation type 2	Smear-negative pulmonary TB	Sputum for smear preparation and culture, and chest X-ray	Category III—Regimen similar to Category I, except ethambutol. Duration: 6 months	
Others	Extrapulmonary TB	Tissue biopsy for histopatho-logical examination and culture for MTB, fine-needle aspiration smear for AFB and cytology	Category III (Duration: 6 months)	

AFB: acid-fast bacilli

References

Sputum smear for AFB

1. Host E, Mitchison DA, Radhakrishna S. Examination of smear for tubercle bacilli by fluorescence microscopy. *Indian Journal of Medical Research* 1959;**47:**495–9.
2. Baily GV, Savic D, Gothi GD, *et al.* Potential yield of pulmonary tuberculosis cases by direct microscopy of sputum in a district of South India. *Bulletin of the World Health Organization* 1967;**37:** 875–92.

Sputum culture for *M. tuberculosis*

1. Allen B, Baker FJ. *Mycobacteria*: *Isolation, identification and sensitivity testing*. London: Butterworth; 1968.
2. Levy H, Feldman C, Sacho H, *et al.* A re-evaluation of sputum microscopy and culture in the diagnosis of pulmonary tuberculosis. *Chest* 1989;**95:**1193–7.

X-ray

1. Larbaoui D, Grosset C, K Abderrahim, *et al.* The efficiency of methods of diagnosing pulmonary tuberculosis: An investigation in a chest clinic in Algiers. *Tubercle* 1970;**51:**403–11.
2. Grinspun M, Rojas L. Comparison between radiological and bacteriological findings in tuberculosis. *Revista Medica de Chile* 1973;**101:**797–805.

Treatment regimen

1. *RNTCP—Technical guidelines for tuberculosis*. New Delhi: Central TB Division; 2000.

Note: The focus of the National Commission on Macroeconomics and Health is on the health care delivery system at the primary and secondary levels of health care—from the district hospital downwards.

Responsibilities of health personnel at different levels

DTC–DTO

Preparation of quarterly report, supervision and monitoring, networking with other sectors, organization of training.

Primary health centre

Medical officer

- First visit—selection of TB suspects, history-taking, ordering sputum examination
- Second visit—categorization for treatment regimens, counselling, start treatment card, selection of DOT provider (DP), visit to the patient for default retrieval, weekly review, preparation of monthly report—programme management
- ANM/staff nurse: They have been identified as DPs and health educators.

Microscopy centre

- The role of the MO, staff nurse and ANM are the same as in the PHC.
- Laboratory technician
- Sputum microscopy—sputum for AFB, preparation of laboratory monthly report, disposal of infectious material.

The equipment required is a binocular microscope.

Subcentre

- VHN/ANM, MPW-M
- Duties as DP—provide drug under observation, maintain treatment card, carry out default retrieval and facilitate sputum examination at stipulated intervals.

Drugs—patient-wise box of RNTCP drugs, symptomatic drugs such as antihistamines, antacids, analgesics, etc.

Causal analysis and treatment protocols for vector-borne diseases

VECTOR CONTROL RESEARCH CENTRE, PONDICHERRY

1. Malaria

Table 1.1 Causes, symptoms and treatment of malaria

Condition	Symptoms	Causes	Treatment
Falciparum malaria ('malignant' tertian or subtertian)	• Febrile paroxysms • Intermittent chills rather than a clearly circumscribed cold phase, hot and sweating stages • Fever is remittent, daily or once in 3 days or twice every 3 days associated with chills • Non-specific symptoms such as fever, prostration, postural hypotension, a tinge of jaundice and tender hepatosplenomegaly	Malarial parasite: *Plasmodium falciparum*	• Treatment following microscopic confirmation • Chloroquine (CQ) 1500 mg orally in divided doses for 3 days in CQ-sensitive areas • Primaquine 45 mg single dose • In CQ-resistant areas—single dose of sulphadoxine (1500 mg) + pyrimethamine (7 mg) followed by primaquire (45 mg) • Appropriate management for other symptoms
Cerebral malaria	• History of several days of fever and non-specific symptoms as mentioned above • Complicated with gradual impairment of consciousness or generalized convulsions followed by persisting coma • Other manifestations (hyperpyrexia, hyperparasitaemia, hypoglycaemia, renal failure, hepatic dysfunction, pulmonary oedema, algid malaria, black water fever, etc.)	Malarial parasite: *Plasmodium falciparum*	• As in the case of falciparum malaria, treatment following microscopic confirmation • Supportive therapy (oxygen, and maintaining fluid and electrolyte balance) • Quinine IV drip (600 mg) repeated two or three times in 24 hours or • Artemether IV/IM (3.2 mg per kg on day 1; 1.6 mg per kg days 2–6) • CQ 1500 mg orally in divided doses for 3 days in CQ-sensitive areas • Primaquine 45 mg in single dose • In CQ-resistant areas: sulphadoxine–pyrimethamine compound—sulphadoxine (500 mg tablet) or sulphalene (500 mg) plus pyrimethamine (25 mg)—3 tablets as a single dose
Vivax malaria ('benign' tertian malaria)	• Intermittent fever with cold, hot and sweating stages; associated symptoms include headache, pain in the back, nausea and general malaise (the prodromal symptoms are mild or absent in relapses) • Febrile paroxysms occur in cycles of two days	Malarial parasite: *Plasmodium vivax*	• Treatment following microscopic confirmation • CQ 1500 mg orally in divided doses for 3 days • Primaquine 75 mg in 5 divided doses
Malariae malaria ('quartan' malaria)	• Clinical picture resembles that of vivax malaria but prodromal symptoms and rigors may be more severe • Anaemia is less pronounced • Febrile paroxysms occur in cycles of three days	Malarial parasite: *Plasmodium malariae*	• Treatment following microscopic confirmation • CQ 1500 mg orally in divided doses for 3 days • Primaquine 45 mg in a single dose
Ovale malaria (ovale tertian malaria)	• Clinical picture resembles that of vivax malaria but prodromal symptoms may be severe • Spontaneous recovery is more common with fewer relapses • Febrile paroxysms occur in cycles of two days	Malarial parasite: *Plasmodium ovale*	• Treatment following microscopic confirmation • CQ 1500 mg orally in divided doses for 3 days • Primaquine 75 mg in 5 divided doses

Table 1.2 Causes of conditions (by significance) due to malaria

Condition		Direct (normally clinical)	Indirect (diet, exercise, alcohol)	Distant (sociopolitical, economic, empowerment, gender, literacy, etc.)	
Falciparum malaria ('malignant' tertian or subtertian)	Main causes—*Plasmodium falciparum*	Multiplication of the malarial parasite and destruction of RBCs	Lack of nutrition, especially in children and pregnant women	• Poor economic conditions leading to creation of mosquitogenic condition favouring transmission • Occupation, sleeping habits leading to increased man/vector contact	• Low literacy associated with poor economic status leads to constraints in practising personal protection measures and seeking health care
Cerebral malaria	*Plasmodium* falciparum	• Multiplication of the malarial parasite and destruction of RBCs • Increased intravascular permeability/blockade of cebebral capillaries due to high parasitaemia leading to cerebral infarction, etc.			Lack of adequate and timely treatment leading to cerebral malaria
Vivax malaria ('benign' tertian malaria)	*Plasmodium vivax*	Multiplication of the malarial parasite and destruction of RBCs			Relapses in vivax malaria
Malariae malaria ('quartan' malaria)	*Plasmodium malariae*	Multiplication of the malarial parasite and destruction of RBCs			
Ovale malaria (ovale tertian malaria)	*Plasmodium ovale*	Multiplication of the malarial parasite and destruction of RBCs			
	Interaction with other causes	Anaemia			

References: Park K (ed). *Park's textbook of preventive medicine.* Jabalpur: M/S Banarasidas Bhanot; 2000:172–3, 215.
Bruce-Chwatt LJ. *Essential malariology.* London: William Heinmann Medical Books Ltd; 2002:85–126.

Table 1.3 Interventions (by significance) for various outcomes of malaria

			Non-medical interventions		
Condition	Outcomes	Medical interventions	Exercise	Nutrition	Others
Malarial fever (all the four plasmodium parasites)	Malaria and anaemia	• Antimalarials • Antipyretics, analgesics • Oral fluids and electrolytes	Nil	Balanced nutritious diet, vitamin supplements	• Bed rest • Sponging
	Cerebral malaria	• Antimalarials • Symptomatic • Oxygen therapy • Fluid and electrolyte balance			• Bed rest • Good nursing care

Table 1.4 Standard treatment protocols for malaria

Condition	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay
Malarial fever (paroxysms due to any one of the *Plasmodium* parasites)	• Trained nurses • Medical practitioner (1 week)	• Clinical signs and symptoms • Peripheral blood examination • ICT malaria test Pf serology • Molecular biological techniques (DNA/RNA probes)	• Chloroquine (CQ): In CQ-sensitive areas, 1500 mg orally in divided doses for 3 days • Primaquine 45 mg in single dose for Pf and Pm, and 75 mg in 5 divided doses for Pv and Po • SP compounds in other areas	May/may not be required
Cerebral malaria	• Medical practitioner • Referral hospitals (2 weeks)	• Clinical signs and symptoms • Peripheral blood examination • ICT malaria test Pf serology • Molecular biological techniques (DNA/RNA probes)	• Antimalarials (quinine, artemisinin, CQ depending on the indications) • Fluid replacement • Oxygen therapy when needed • Supportive therapy	Mandatory

References: Park K (ed). *Park's textbook of preventive medicine*. Jabalpur: M/S Banarasidas Bhanot; 2000:172–3, 215. Bruce-Chwatt LJ. *Essential malariology.* London: William Heinmann Medical Books Ltd; 2002:85–126.

Pf: *Plasmodium falciparum*; Pm: *Plasmodium malariae*; Pv: *Plasmodium vivax*; Po: *Plasmodium ovale*; SP: sulphadoxine–pyrimathamine; ICT: immunochromatographic card test

2. Dengue

Table 2.1 Causal analysis of dengue

Condition	Symptoms	Causes	Treatment
Asymptomatic virus carriers	Asymptomatic	Dengue virus (serotypes 1–4)	No treatment available
Dengue fever (DF)	• Suspected: Compatible with clinical description (2 or more of the following clinical features—headache, retro-orbital pain, myalgia, arthralgia, rash, haemorrhagic manifestations) • Probable: compatible with clinical description and either 1 or more of supportive serology (HI >1280), anti-dengue IgG/IgM, same location/same time as confirmed cases • Confirmed: Compatible with clinical description and laboratory tests confirmed	Dengue virus (serotypes 1–4)	• Symptomatic (antipyretics, analgesics) • Bed rest • Sponging • Oral fluids and electrolytes
Dengue haemorrhagic fever (DHF)	Probable and confirmed: one or more of positive tourniquet test, petechiae, ecchymosis or purpura, bleeding from mucosa, gastrointestinal tract, injection sites, haematemesis or melaena and thrombocytopenia (platelets <100,000/cmm), and evidence of plasma leakage (>20% rise in haematocrit, pleural effusion/ascites)	Dengue virus (serotypes 1–4)	• Symptomatic • Fluid replacement • Whole blood/platelet replacement
Dengue shock syndrome (DSS)	Same as DHF and evidence of circulatory failure manifested as rapid and weak pulse, pulse pressure <20 mmHg, etc.	Dengue virus (serotypes 1–4)	• Symptomatic • Sedation • Oxygen therapy • Fluid/whole blood/platelet replacement

Table 2.2 Causes of conditions (by significance) due to dengue

Condition		Direct (normally clinical)	Indirect (diet, exercise, alcohol)	Distant (sociopolitical, economic, empowerment, gender, literacy, etc.)
Dengue fever	Main causes	• Non-specific 'viral syndrome' with fever, headache, myalgia, chills, arthralgia, retro-orbital pain with or without rash • Virus multiplication in macrophages	Nil	• Practice of storing water in containers for domestic purposes • Low literacy associated with poor economic status leads to constraints in practising personal protection measures
	Interaction with other causes	Nil		
Dengue haemorrhagic fever	Main causes	• One or more of positive tourniquet test, petechiae, ecchymosis or purpura, bleeding from mucosa, gastro-intestinal tract, injection sites, haematemesis or melaena and thrombocytopenia (platelets <100,000/cmm), and evidence of plasma leakage (>20% rise in haematocrit, pleural effusion/ascites) • Increased vascular permeability resulting in plasma leakage (mainly into theperitoneal and pleural cavities), hypovolaemia and shock • Abnormal haemostasis due to vasculopathy, thrombocytopenia • Complement C3 and C5 levels depressed, C3a and C5a levels elevated	Nil	• Practice of storing water in containers for domestic purposes • Low literacy associated with poor economic status leads to constraints measures, partial immunity, in practising personal protection monsoon and post-monsoon (of late, dry summer season also) • Light to moderate rainfall, plains and plateau • Temperature: 20–25 ^{0}C/relative humidity >60%, both urban poor and rural areas favourable; urban: due to poor management of immediate environment, rural: poor water storage practices at household level in areas with erratic water supply contribute to breeding source for the incriminated vectors
	Interaction with other causes	Nil		
Dengue shock syndrome	Main causes	• Same as DHF and evidence of circulatory failure manifested as rapid and weak pulse, pulse pressure <20 mmHg, etc. • Increased vascular permeability resulting in plasma leakage (mainly into the peritoneal and pleural cavities), hypovolaemia and shock • Abnormal haemostasis due to vasculopathy, thrombocytopenia • Complement C3 and C5 levels depressed, C3a and C5a levels elevated	Nil	• Practice of storing water in containers for domestic purposes • Same as for DHF
	Interaction with other causes	Nil		

References: World Health Organization. *Prevention and control of dengue and dengue haemorrhagic fever: Comprehensive guidelines*. WHO regional publication, SEARO; 1999:3–53.
Park K (ed). *Park's textbook of preventive medicine*. Jabalpur: M/S Banarasidas Bhanot; 2000:172–3, 215.

Table 2.3 Interventions for various outcomes of dengue (by significance)

Outcome of dengue	Medical interventions	Non-medical interventions		
		Exercise	Nutrition	Others
Dengue fever	• Symptomatic (antipyretics, analgesics) • Oral fluid and electrolytes	Nil	Nil	• Bed rest • Sponging
Dengue haemorrhagic fever	• Symptomatic • Fluid replacement • Whole blood/platelet replacement			Good nursing care
Dengue shock syndrome	• Symptomatic • Sedation • Oxygen therapy • Fluid/whole blood/platelet replacement			• Bed rest • Good nursing care

Table 2.4 Standard treatment protocols for dengue

Condition	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay
Dengue fever (DF)	• Trained nurses • Medical practitioner	• Haemagglutination inhibition (HI) test • Complement fixation test • Neutralization test (NT) • ELISA for IgG and IgM • Isolation of the virus	• Antipyretics: 3–5 days • Analgesics: 3–5 days	May not be required
Dengue haemorrhagic fever (DHF)	• Medical practitioner • Hospital	• Clinical signs and symptoms • ELISA for IgG and IgM • Isolation of the virus	• Analgesics • Antipyretics • Fluid replacement • Whole blood/platelet replacement	Must
Dengue shock syndrome (DSS)	• Medical practitioner • Referral hospital	• Clinical signs and symptoms • ELISA for IgG and IgM • Isolation of the virus	• Analgesics • Antipyretics • Fluid replacement • Whole blood/platelet/plasma/replacement	Must

References: World Health Organization. *Prevention and control of dengue and dengue haemorrhagic fever: Comprehensive guidelines*. WHO regional publication, SEARO; 1999:3–53.
Park K (ed). *Park's textbook of preventive medicine*. Jabalpur: M/S Banarasidas Bhanot; 2000:172–3, 215.

3. Japanese encephalitis

Table 3.1 Causal analysis of Japanese encephalitis

Condition	Symptoms	Causes	Treatment
Prodromal	• Fever • Rigors • Headache • Nausea • Vomiting	JE virus (Flaviviridae family)	• Non-specific treatment, only symptomatic and supportive treatment is crucial • Should NOT use gamma-globulins and corticosteroids; physical therapy desirable
Acute encephalitic	• Convulsions • Neck rigidity • Muscular rigidity • Mask-like face • Abnormal movements • Dehydration	JE virus (Flaviviridae family)	(Supportive) • 1/2 normal saline infusion (children) • IV diazepam (0.3 mg/kg, every 4–6 hours) or phenobarbital (10%, 0.5 to 1 ml IM) • Antipyretic suppositories used • Cold/tepid sponging • Oxygen (1–2 litres/minute), if needed
Late stage	• Increased deep tendon reflexes • Thick and slow speech • Aphasia and paresis	JE virus (Flaviviridae family)	Rehabilitation during convalescence with physiotherapy

Table 3.2 Causes of conditions (by significance) due to Japanese encephalitis

Causes	Direct (normally clinical)	Indirect (diet, exercise, alcohol)	Distant (sociopolitical, economic, empowerment, gender, literacy, etc.)	
Main causes	• Viral multiplication in the local and regional lymph nodes • Dissemination to secondary sites and further multiplication leading to viraemia and invasion of the central nervous system	Nil	• Poor intersectoral coordination between health, agriculture, animal husbandry and local administration departments • Intensified wet agriculture activities facilitating breeding of vectors in rice fields, and pig (amplifier hosts) rearing favour transmission • Poor community participation in primary health care with respect to vector-borne disease control activities	• Illiteracy and ignorace about JE transmission leading to constraints in practising personal protection measures • *Culex* group of mosquitoes coming in contact with virus-infected birds
Interaction with other causes	No recorded reports			

Reference: Park K (ed). *Park's textbook of preventive medicine*. Jabalpur: M/S Banarasidas Bhanot; 2000:172–3, 215.

Table 3.3 Interventions (by significance) for Japanese encephalitis

Outcome	Medical interventions	Non-medical interventions		
		Exercise	Nutrition	Other
Prodromal	Supportive • IV fluids • Antipyretics	If needed	Yes	—
Encephalitic	Supportive • IV fluids • Antipyretics • Anticonvulsants • Tepid sponging	Yes	Yes	—

Reference: Rao PN. *Japanese encephalitis*. 2002.

Table 3.4 Standard treatment protocols for Japanese encephalitis

Condition	Personnel	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay	Non-medical inteventions/ prevention
Prodromal	Medical practitioner and nurse	• Serum and CSF • IgM—capture ELISA (MAC ELISA) • Immunofluorescent assay (IFA)	Supportive • IV fluids • Antipyretics • Anticonvulsants • Tepid sponging	Until complete recovery	Rest and good nutrition during convalescence, physiotherapy for neurological residual sequalae, if any, after complete recovery
Encephalitic	Medical practitioner	Reverse passive haemagglutination for confirmation			

(Cont.)

Table 3.4 (*cont.*) Standard treatment protocols for Japanese encephalitis

Condition	Personnel	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay	Non-medical inteventions/ prevention
		• Detection of the JE virus, antigen or genome in the tissue, blood or other body fluid by immunochemistry or immunofluorescence or PCR, or • JE virus-specific IgM in the CSF, or • Four-fold or greater rise in JE virus-specific antibody in paired sera (acute and convalescent phase) through IgM/IgG, ELISA, haemagglutination inhibition test, in a patient with no history of recent yellow fever vaccination and where cross-reactions to other flaviviruses have been excluded			

Reference: Rao PN. *Japanese encephalitis.* 2002.

1. General features

- Zoonotic viral disease
- Maintained in nature in animals and birds (pigs, egrets, pond herons)
- Transmitted primarily by mosquitoes of the genus *Culex*
- Human infection is only incidental, mostly not apparent, with 1 in 250–1000 infections resulting in disease
- Case fatality rate 20% and above; can be kept low by good management
- Until the early 1970s, reported only from south India; by 1978, from 21 States and UTs. Worst outbreak in 1988 in UP
- 10 states report incidence of JE regularly (AP, UP, Karnataka, TN, WB, Assam, Bihar, Goa, Manipur and Haryana)
- Population at risk >160 million
- Seasonal (May–October)
- Related to agricultural practices
- Children usually affected, adults affected in outbreaks in non-endemic areas
- Males affected more than females
- In endemic areas, 12%–44% of pigs are positive for the JE virus

2. Clinical features

There are three stages of JE:

- **Prodromal stage:** This stage is characterized by fever, rigors, headache, and nausea and vomiting. The stage lasts for 1–6 days.
- **Acute encephalitic stage:** This stage begins by the 3rd–5th day and is characterized by convulsions, altered sensorium, mask-like face, stiff neck, muscular rigidity, tremors (fingers, tongue, eyelids, eyes), abnormal movements of the limbs and speech impairment
- **Late stage:** During this stage, there is persistence of central nervous system (CNS) injury signs such as mental impairment, increased deep tendon reflexes, paresis, speech impairment, epilepsy, abnormal movements and behaviour abnormalities

3. Diagnosis of JE

- Collect the cerebrospinal fluid (CSF) (by lumbar puncture, in early acute phase, store at 4 °C in dry ice, transport to laboratory)
- Collect brain tissue from dead patients (immerse in 2 ml of 10% glycerol–saline (pH 7.4), transport in a sealed container in dry ice or liquid nitrogen for isolation of the virus)
- Collect blood (in acute phase and convalescence, serum to be separated and stored at 4 °C)

4. Case definition

Infection caused by JE virus may result in a febrile illness of variable severity associated with neurological symptoms ranging from headache to meningitis or encephalitis. Symptoms include headache, fever, meningeal signs, stupor, disorientation, coma, tremors, paresis (generalized),

Table 3.5 Year-wise and State-wise distribution of cases of Japanese encephalitis

State	1992		1993		1994		1995		1996		1997		1998		1999		2000		2001	
	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths
Andhra Pradesh	143	66	1175	467	260	131	986	270	332	108	982	247	527	192	965	200	343	72	0	0
Assam	259	93	96	30	230	145	11	11	64	29	88	26	26	6	11	2	158	69	241	113
Bihar	0	0	0	0	0	0	0	0	0	—	—	—	—	—	—	—	77	19	88	16
Delhi	352	103	106	49	54	31	3	1	—	—	1	—	—	—	—	—	—	—	1	0
Goa	43	8	37	10	10	1	11	3	4	0	40	8	0	0	0	0	15	3	2	0
Gujarat	0	0	0	0	0	0	0	0	—	—	1	—	—	—	—	—	—	—	0	0
Haryana	41	33	19	13	79	67	30	21	59	41	0	0	19	16	121	56	74	43	45	22
Karnataka	58	15	99	22	126	47	285	89	171	17	436	87	306	50	597	88	438	45	73	6
Kerala	0	0	0	0	0	0	0	0	105	31	121	19	103	24	214	4	164	2	0	0
Maharashtra	0	0	0	0	0	0	0	0	—	—	43	3	2	0	NA	NA		—	0	0
Manipur	29	0	11	1	11	0	268	62	20	2	0	0	28	1	42	1	1	0	0	0
Tamil Nadu	177	107	278	176	239	125	115	57	111	53	89	42	25	14	14	5	4	0	0	0
Uttar Pradesh	793	229	104	32	0	0	0	0	672	161	351	76	1051	195	1370	275	1170	253	625	132
West Bengal	537	234	366	123	234	93	1265	428	706	151	362	124	36	9	61	14	148	50	97	14
Total	2432	888	2291	923	1243	640	2974	942	2244	593	2514	632	2123	507	3395	645	2592	556	1172	303
CFR%	36.51		40.29		51.49		31.67		26.43		25.14		23.88		19.00		21.45		25.85	

NA: not applicable

Source: National Institute of Communicable Diseases, Delhi

hypertonia, loss of coordination. Clinically, the encephalitis cannot be distinguished from other CNS infections.

5. Detection and isolation of the virus

Detection of antigen from CSF

- Immunofluorescent assay (IFA) (can detect antigen as early as second day; useful when IgM antibodies are not detected in CSF)
- Reverse passive haemagglutination (RPHA) (within 10 days of onset)
- PCR (for virus genome)
- Inoculation into infant mice

Detection of antibody

From serum

- Haemagglutination inhibition (HI)
- Complement fixation(CF), kinetic CF
- Neutralization test
- Immunodiffusion

All based on a rise of IgG antibody titre (four-fold) in paired sera collected at 15–20 days' interval

Detection of antibody from serum and CSF

- IgM captured enzyme-linked immunosorbent assay (MAC ELISA) (IgM antibodies present after 7th day of illness)
- A single specimen is sufficient

6. Management of JE (only symptomatic)

- Fluid–electrolyte balance
- Temperature
- Convulsions
- Intracranial pressure

7. JE control strategy

- Surveillance
  —sero surveillance in animals/birds
  —vector surveillance (vector density, infection)
  —case surveillance
  1. Where no JE transmission is detected but vector is present (acute CNS syndromes, fever clustering)
  2. Where endemic or epidemic (weekly/monthly reporting on suspected/probable/confirmed cases
- Vaccination
- Vector control (indoor residual spraying, outdoor fogging)
- Diagnosis and management (including rehabilitation)
- Health education/distribution of information, education and communication (IEC) materials
- Training (health sector, other sectors)

8. JE vaccine

Three types of vaccines for JE are produced and used worldwide:

(i) Inactivated mouse brain vaccine (India–Nakayama strain) (0.5–1 ml SC, 2 doses, 1–4 weeks apart, booster after 1 year, subsequent boosters at 1–3-year intervals)
(ii) Inactivated hamster kidney cell vaccine
(iii)Live-attenuated hamster kidney cell vaccine

4. Kala-azar

Table 4.1 Causes, symptoms and treatment of kala-azar

Condition	Symptoms	Cause	Treatment
Kala-azar (visceral leishmaniasis)	• Fever • Hepatosplenomegaly • Anaemia • Wasting • Pigmentation • Cough • Diarrhoea	*Leishmania donovani*	• Antimony compounds 10 mg/kg IV/IM for 20 days • Antimony compounds 20 mg/kg IV/IM for 20 days • Liposomal amphotericin B • 2–3 mg/kg/dose/day for 10 days
Post kala-azar dermal leishmaniasis (PKDL)	• Hypopigmented erythematous macules • Nodular eruptions on the face	*Leishmania donovani*	Same as above
Cutaneous leishmaniasis	Nodule at the site of inoculation, central crust development, gradual healing	*Leishmania tropica*	Same as above

IV: intravenous; IM: intramuscular

Table 4.2 Causes of conditions (by significance) due to kala-azar

Condition		Direct (normally clinical)	Indirect (diet, exercise, alcohol)	Distant (sociopolitical, economic, empowerment, gender, literacy, etc.)
Kala-azar (VL)	Main causes	• Multiplication of *Leishmania* in macrophages of liver, spleen, bone marrow and lymphoid tissue • Granulopenia and thrombocytopenia	Nil	• Poor economic conditions leading to creation of vector breeding (sandfly) conditions favouring transmission • Low literacy associated with poor economic status leads to constraints in practising personal protection measures
	Interaction with other causes		• Anaemia • Immunosuppression • Association with other diseases such as TB and pneumococcal infection	• Mixed dwellings and proximity of human dwellings to cattle sheds
PKDL	Main causes	• Pathology same as above • Usually one to several years after apparent recovery from VL • Hypopigmented erythematous macules • Nodular eruptions on the face		• Poor economic conditions leading to creation of mosquitogenic conditions favouring transmission • Low literacy associated with poor economic status leads to constraints in practising personal protection measures
	Interaction with other causes		• Anaemia • Immunosuppression • Association with other diseases such as TB and pneumococcal infection	
Cutaneous leishmaniasis (CL)	Main causes	• Host immunity—immune cellular response • Tissue response (tissue damage due to antigen release) • Nodule at the site of inoculation, central crust development,gradual healing	Same as for PKDL	Same as for PKDL

References: Haslett C, Chilvers ER, Boon NA, Colledge NR (eds). *Davidson's principles and practice of medicine*. 19th ed. Churchill Livingstone; 2002:66–8.
World Health Organization. *Technical Report Series-701: Report of a WHO Expert Committee.* Geneva: WHO; 1984:14–23.
Manson's tropical diseases. 17th ed. USA: Williams and William; 1976:117.
Park K (ed). *Park's textbook of preventive medicine*. Jabalpur: M/S Banarasidas Bhanot; 2000:172–3, 215.

PKDL: post kala-azar dermal leishmaniasis; VL: visceral leishmaniasis

Table 4.3 Interventions for various outcomes of kala-azar (by significance)

		Non-medical interventions		
Outcomes	Medical interventions	Exercise	Nutrition	Others
VL	• Antimony compounds • Antipyretics • Amphotericin B	Nil	Correction of anaemia	Recurrent pneumococcal infection
PKDL	• Antimony compounds • Antipyretics • Amphotericin B		Correction of anaemia	
Cutaneous leishmanasis	• Antimony compounds • Antipyretics • Amphotericin B		Correction of anaemia	

References: Haslett C, Chilvers ER, Boon NA, Colledge NR (eds). *Davidson's principles and practice of medicine*. 19th ed. Churchill Livingstone; 2002:66–8.
World Health Organization. *Technical Report Series-701: Report of a WHO Expert Committee.* Geneva: WHO; 1984:14–23.
Manson's tropical diseases. 17th ed. USA: Williams and William; 1976:117.
Park K (ed). *Park's textbook of preventive medicine*. Jabalpur: M/S Banarasidas Bhanot; 2000:172–3, 215.

VL: visceral leishmaniasis; PKDL: post kala-azar dermal leishmaniasis

Table 4.4 Standard treatment protocols for kala-azar

Condition	Personnel	Tests (by type)	Drugs (dosage,type and time)	Inpatient stay	Others
Kala-azar	Medical practitioner	• LD bodies • Aldehyde tests • Serological tests • Leishmanin test • Haematological tests (leucopenia, anaemia, reversed albumin–globulin ratio,increased ESR, RBC–WBC ratio	• Antimony compounds 10 mg/kg IV/IM for 20 days • Antimony compounds 20 mg/kg IV/IM for 20 days • Liposomal amphotericin B 2–3 mg/kg/dose/day for 10 days	Required initially	Nil
PKDL	Medical practitioner	As above	As above	Not required	
Cutaneous leishmanasis	Medical practitioner	As above	As above	Not required	

References: Haslett C, Chilvers ER, Boon NA, Colledge NR (eds). *Davidson's principles and practice of medicine*. 19th ed. Churchill Livingstone; 2002:66–8.
World Health Organization. *Technical Report Series-701: Report of a WHO Expert Committee*. Geneva: WHO; 1984:14–23.
Manson's tropical diseases. 17th ed. USA: The Williams and William; 1976:117.
Park K (ed). *Park's textbook of preventive medicine*. Jabalpur: M/S Banarasidas Bhanot; 2000:172–3, 215.

LD: Leishman–Donovan; IV: intravenous; IM: intramuscular; ESR: erythrocyte sedimentation rate; RBC: red blood cell; WBC: white blood cell; PKDL: post kala-azar dermal leishmaniasis

5. Lymphatic filariasis

Table 5.1 Symptoms, causes and treatment of lymphatic filariasis

Condition	Symptoms	Causes	Treatment
Mf carriers	Asymptomatic	Circulating microfilaria in the peripheral blood	One course of diethylcarbazamine (DEC) at 6 mg/kg body weight per day for 12 days in three daily doses
ADL	Fever associated with adenolymphangitis	Secondary bacterial infection at the sites damaged by the filarial parasite in the lymphatic system	• Antibiotics (choice penicillin) • Antipyretics • Anti-inflammatory drugs
TPE	• Dry nocturnal cough • Exertional dyspnoea • Absolute eosinophil count more than 3000/cmm • Differential count more than 25%	Immunological (hyper) response	• Anthelminthic drug • One course of DEC at 6 mg/kg body weight per day for 12 days in three daily doses • Non-responders?
Lymphoedema I	• Reversible oedema in the limbs overnight • Skin normal	Damage to the lymphatic system (filarial parasite)	• Limb elevation • Manual massage • DEC?
Lymphoedema II	• Irreversible oedema in the limbs even with elevation • Skin normal	Repeated attacks of ADL due to filarial infection	• Limb hygiene, limb elevation, management of ADL • Physiotherapy
Lymphoedema III	• Irreversible oedema in the limbs • Skin thickened	Repeated attacks of ADL due to filarial infection	• Limb hygiene, elevation, management of ADL • Physiotherapy • Interferential therapy
Lymphoedema IV	• Irreversible oedema in the limbs • Skin-fold thickening, pigmentary changes, chronic ulceration and epidermal and subepi-dermal nodules • Myiasis	Repeated attacks of ADL due to filarial infection	• Topical application of antibiotics • Interferential therapy • Limb hygiene, elevation, management of ADL • Physiotherapy • Lymphonodal shunt
Hydrocele (filaricele) <15 cm	Swelling of the scrotum (tunica)	Accumulation of clear fluid in TVT due to filarial infection	Surgery
Hydrocele (filaricele) ≥15 cm	Swelling of the scrotum (tunica)	Accumulation of clear fluid in TVT due to filarial infection	Surgery
Chyluria	Milky urine	Obstruction to the lymphatic system	Antibiotics to prevent secondary infection

ADL: adenolymphangitis; TPE: tropical pulmonary eosinophilia; TVT: tunica vaginalis testis

Table 5.2 Causes of conditions (by significance) due to lymphatic filariasis

Condition	Causes	Direct (normally clinical)	Indirect (diet, exercise, alcohol)	Distant (sociopolitical, economic, empowerment, gender, literacy, etc.)
Lymphoedema I[1–6]	Main causes	• Damage to the lymphatic system caused by the filarial parasite • Consequence of acute episodes of ADL • Accumulation of lymph in the lower extremities due to the defective lymphatic vessels • Oedema normally disappears on elevation	• Physical exertion • Occupational—weavers, tailors who tend to sit for a long time with lower limbs in more exertion	• Poor economic conditions leading to creation of mosquitogenic conditions favouring transmission • Low literacy associated with poor economic status leads to constraints in practising personal protection measures
	Interaction with other causes	Nil		
Lymph-oedema II[1–4, 6, 7]	Main causes	• Damage to the lymphatic system caused by the filarial parasite • Obstructive and immunological effects • Pitting–oedema • Accumulation of lymph in the lower extremities due to defective lymphatic vessels • Oedema volume increases following each attack of ADL	• Physical exertion • Occupational—weavers, tailors who tend to sit for a long time with lower limbs in more exertion	• Poor economic conditions leading to creation of mosquitogenic conditions favouring transmission • Low literacy associated with poor economic status leads to constraints in practising personal protection measures
	Interaction with other causes	Nil		
Lymph-oedema III[1–4, 6–9]	Main causes	• Damage to the lymphatic system caused by the filarial parasite • Obstructive and immunological effects • Oedema—non-pitting • Loss of barrier facilitates penetration of bacteria • Accumulation of lymph in the lower extremities due to defective lymphatic vessels • Oedema volume increases following each attack of ADL	• Physical exertion • Occupational—weavers, tailors who tend to sit for a long time with lower limbs in more exertion	• Poor economic conditions leading to creation of mosquitogenic conditions favouring transmission • Low literacy associated with poor economic status leads to constraints in practising personal protection measures
	Interaction with other causes	Bacteria enter the tissues through entry lesions in the skin		
Lymph-oedema IV[1–4, 6–9]	Main causes	• Damage to the lymphatic system caused by the filarial parasite • Obstructive and immunological effects • Oedema—non-pitting • Loss of barrier function facilitates penetration of bacteria • Accumulation of lymph in the lower extremities due to defective lymphatic vessels • Skin-fold thickening • Hyperkeratosis • Hypo- or hypertrichosis • Pachyderma • Pigmentary changes • Chronic ulceration • Epidermal and subepidermal nodules	• Physical exertion • Occupational—weavers, tailors who tend to sit for a long time with lower limbs in more exertion	• Poor economic conditions leading to creation of mosquitogenic conditions favouring transmission • Low literacy associated with poor economic status leads to constraints in practising personal protection measures

(Cont.)

Table 5.2 (*cont.*) Causes of conditions (by significance) due to lymphatic filariasis

Condition	Causes	Direct (normally clinical)	Indirect (diet, exercise, alcohol)	Distant (sociopolitical, economic, empowerment, gender, literacy, etc.)
	Interaction with other causes	Bacteria enter tissues through entry lesions in the skin		
Hydrocele[1–4,6–9]	Main causes	• Damage to the lymphatic system caused by the filarial parasite • Obstructive and immunological effects • Accumulation of lymph in the scrotal sac due to the defective lymphatic vessels • Skin-fold thickening	• Physical exertion • Occupational—vendors using bicycles	• Poor economic conditions leading to creation of mosquitogenic conditions favouring transmission • Low literacy associated with poor economic status leads to constraints in practising personal protection measures
	Interaction with other causes	Bacteria enter tissues through entry lesions in the skin		

Table 5.3 Interventions (by significance) for lymphatic filariasis

		Non-medical interventions		
Outcomes	Medical interventions	Exercise	Nutrition	Other
ADL	• Antibiotics (choice penicillin) • Antipyretics • Anti-inflammatory drugs			
TPE	• Anthelminthic drugs • One course of DEC at 6 mg/kg body weight per day for 12 days in three daily doses • Non-responders?			
Lymphoedema I	• Elevation • Manual massage • DEC?			
Lymphoedema II	• Limb hygiene, elevation, management of ADL • Physiotherapy	• Limb elevation • Manual massage		
Lymphoedema III	• Limb hygiene, elevation, management of ADL • Physiotherapy • Interferential therapy	• Limb elevation • Manual massage		
Lymphoedema IV	• Topical application of antibiotics • Interferential therapy • Limb hygiene, elevation, management of ADL • Lymphonodal shunt			
Hydrocele (filaricele) <15 cm	Surgery			
Hydrocele (filaricele) ≥15 cm	Surgery			
Chyluria	Antibiotics to prevent secondary infection			

ADL: adenolymphangitis; DEC: diethylcarbamazine; TPE: tropical pulmonary eosinophilia

Table 5.4 Standard treatment protocols for lymphatic filariasis

Condition	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay
Asymptomatic mf carriers[1–4]	Medical practitioner	Night blood (peripheral) examination	• One course of diethylcarbamazine at 6 mg/kg body weight per day for 12 days in three daily doses	Not required
ADL[1–4]	Medical practitioner	Clinical signs and symptoms	• Antibiotics (choice penicillin) • Antipyretics	• Not required • Anti-inflammatory drugs
TPE [1,10,11]	Medical practitioner	Differential and absolute eosinophil count	• Antihelminthic drugs • One course of DEC at 6 mg/kg body weight per day for 12 days in three daily doses • Non-responders?	Required in case of recurrent attacks and breathing difficulty
Lymphoedema I[1–4]	Medical practitioner	Clinical signs and symptoms	• Elevation • Manual massage • DEC	Not required
Lymphoedema II[1–4]	Medical practitioner	Clinical signs and symptoms	• Limb hygiene, elevation, management of ADL • Physiotherapy	Not required
Lymphoedema III[1–4]	Medical practitioner	Clinical signs and symptoms	• Limb hygiene, elevation, management of ADL • Physiotherapy • Interferential therapy	Not required
Lymphoedema IV[1–4,12]	Medical practitioner	Clinical signs and symptoms	• Topical application of antibiotics • Interferential therapy • Limb hygiene, elevation, management of ADL • Physiotherapy • Lymphonodal shunt	Required when lymphonodal shunt is performed
Hydrocele (filaricele) <15 cm[1–4]	Trained surgeon	Clinical signs and symptoms	Surgery	Required
Hydrocele (filaricele) ≥15 cm[1–4]	Trained surgeon	Clinical signs and symptoms	Surgery	Required
Chyluria[1–4]	Medical practitioner	Urine test	Antibiotics to prevent secondary infection	Not required

ADL: adenolymphangitis; DEC: diethylcarbamazine; TPE: tropical pulmonary eosinophilia

References

1. World Health Organization. *Technical Report Series-821: Fifth Report of the WHO Expert Committee on Filariasis.* Geneva: WHO; 1992:14–23.
2. Pani SP, Yuvaraj J, Vijaylakshmi G, Subramanyam Reddy G. *Management of lymphatic filariasis—a manual for clinicians*. Vector Control Research Centre Miscellaneous Publications; 1997:1–22.
3. Dryer G, Noroes J, Figueredo-Silva J, Piessens WF. Pathogenesis of lymphatic disease in bancroftian filariasis: A clinical perspective. *Parasitol Today* 2000;**16:**544–8.
4. Shenoy RK, Sandhya K, Suma TK, Kumaraswami V. A preliminary study of filariasis related acute adenolymphangitis with special reference to precipitation factors and treatment modalities. *South East Asian Journal of Tropical Medicine and Public Health* 1995; **26:**301–5.
5. Radhakrishnan R, Srinivasan R, Krishnamoorthy K, Sabesan S, Pani SP. Myiasis in filarial lymphoedema due to *Chrysomyia bezziana*. *Natl Med J India* 1994;**7:**117–18.
6. Ramaiah KD, Radhamani MP, John KR, Evans DB, Guyatt H, Joseph A, *et al.* The impact of lymphatic filariasis on labour inputs in southern India: Results of a multi-site study. *Ann Trop Med Parasitol* 2000;**94:**353–64.
7. Olszewski WL, Jamal S, Manokaran G, Pani S, Kumaraswami V, Kubicka U, *et al.* Bacteriological studies of blood, tissue fluid, lymph and lymph nodes in patients with acute dermato-lymphangioadenitis (DLA) in course of 'filarial' lymphedema. *Acta Tropica* 1999;**73:**217–24.
8. Vaqas B, Ryan TJ. Lymphoedema: Pathophysiology and management in resource-poor settings—relevance for lymphatic filariasis control programmes. *Filaria J* 2003;**2:**4.
9. Burri H, Loutan L, Kumaraswami V, Vijayasekaran V. Skin changes in chronic lymphatic filariasis. *Trans R Soc Trop Med Hyg* 1996; **90:**671–4.
10. Kar SK, Mania J, Kar PK. Clinical filarial disease in two ethnic endemic communities of Orissa, India. *J Trop Med Hyg* 1993;**96:** 311–16.
11. Kar SK, Mania J. Tropical pulmonary eosinophilia in an Orissa village. *Natl Med J India* 1993;**6:**64–7.
12. Jamal S, Pani SP. Long term follow-up filarial oedema with (conservative) medical management. XVII International Congress of Lymphology under Filariasis, pp 144–6.

Controlling the HIV/AIDS epidemic in India

YING-RU J. LO, PADMAJA SHETTY, D.S.C. REDDY, SALIM HABAYEB

India is on the verge of having the greatest increase in the estimated number of people living with HIV/AIDS (PLWHA) in the world in the coming decades. With over 50 lakh PLWHA, India currently has the world's second largest number of cases. The Indian National AIDS Control Organization (NACO) projects that there will be 90 lakh HIV cases by 2010 (NACO 2005). By some projections, India is set to overtake South Africa as the country with the highest number of HIV/AIDS cases. This will create untold human suffering, cause severe stress on an already struggling public health system and have a catastrophic financial impact on families. One Indian study reports that families affected by HIV/AIDS can, on an average, spend 49% of household expenditure on treatment and this increases to 82% among low-income families (Duraisamy 2003).

In response, the Indian Government has undertaken a massive prevention and treatment programme targeting high-risk populations. The Government of India (GOI) has pledged to provide free antiretroviral drugs to 100,000 AIDS patients by the end of 2007. Global partners are also supporting the country's efforts to control the epidemic. Large-scale prevention programmes are being funded through the Bill and Melinda Gates Foundation and the Global Fund for AIDS, TB and Malaria (GFATM).

In the midst of these actions, however, there remain important systemic challenges to adequately scale-up the HIV/AIDS response to meet the potential increase in the epidemic in coming years. These challenges include, but are not limited to, an adequate health workforce, responsive infrastructure (including laboratory capabilities), comprehensive monitoring, and adequate and sustainable financing. This paper aims to provide an overview of the trends and characteristics of the HIV/AIDS epidemic in India, and the response to the epidemic by the GOI. Experiences of other countries in the region that have had success in controlling epidemics in their countries are included for comparison. Finally, the paper highlights some key health system constraints that face India in its attempt to alleviate the suffering of those living with HIV/AIDS and to meet the goal of the Tenth Five-Year Plan of 0% increase in HIV/AIDS by 2007.

World Health Organization
e-mail: shettyp@who.int

Method

This paper is based on a Medline search of original papers and reviews, information obtained from the internet, unpublished material, presentations and abstracts from recent meetings, reports of NACO, and personal communications with several programme managers and clinicians in India.

The global and regional HIV/AIDS situation

By the end of 2004, there were nearly 400 lakh PLWHAs worldwide; 22 lakh of them were children. More than 30 lakh deaths occurred due to HIV/AIDS in 2004, and each day nearly 14,000 new persons with HIV infections are added, with more than half of these occurring among young people under 25 years of age (UNAIDS 2004).

The South-East Asia Region (SEAR) is the second-most affected after sub-Saharan Africa, with more than 60 lakh PLWHA, 80% of whom live in India alone. There are multiple and diverse HIV epidemics in SEAR. Due to the large and dense population, and the presence of several factors that favour the spread of HIV, including poverty, gender inequality, mobility and social stigma, SEAR is likely to increasingly suffer the brunt of the epidemic. The majority of HIV infections in the region occur through unprotected sex between infected men and women. Commercial sex and injecting drug use are the main high-risk behaviours driving the HIV epidemic in Asia. In areas where the prevalence of the HIV infection has remained high among high-risk populations for some years, the infection has spread to lower-risk populations. There is a growing need for care and treatment of PLWHA in SEAR with an estimated 920,000 individuals in need. It is estimated that about 10% of eligible people are receiving antiretroviral treatment (ART) in the region (WHO 2005).

Although effective interventions to prevent HIV transmission and to provide care and treatment are currently being implemented, the HIV epidemic has not reversed in Asia with the notable exception of Thailand and Cambodia. The

key to success of the Thai HIV/AIDS control programme is political commitment at the highest level and allocation of sufficient resources for implementing a package of essential interventions aiming at national coverage. In Thailand, all sectors of society were involved in a multisectoral HIV/AIDS control programme, including strong advocacy and awareness campaigns. One key intervention was the targeted condom programme which promoted condom use among commercial sex workers (CSWs) in sex establishments, combined with empowerment of sex workers, screening for sexually transmitted infection (STI) and sanctions against brothel owners who did not comply (the 100% Condom Programme) (Rojanapithayakorn and Hanenberg 1996). Other interventions were the general strengthening of STI services, screening of blood units for blood-borne infections, HIV counselling and testing, prevention of mother-to-child transmission (PMTCT), a comprehensive continuum of care and ART since the early 1990s. None of the interventions was implemented in isolation but were part of a comprehensive prevention, care and treatment programme along with a strong HIV and STI surveillance system.

Recent epidemiological data from Cambodia indicate that the HIV epidemic has halted and begun to reverse. Cambodia followed the Thai example of establishing a multisectoral AIDS Commission under the chairmanship of the Prime Minister, allocating national resources, and implementing a package of essential interventions along with a strengthened advocacy and awareness campaign and HIV/STI surveillance. The '100% Condom Programme' is considered a key intervention contributing to the decreasing trend of HIV prevalence.

Although some progress has been made to prevent sexual transmission of HIV, HIV/AIDS among injecting drug users (IDUs) remains a neglected issue. Evidence shows that the HIV/AIDS epidemic among IDUs can be prevented, slowed, stopped and even reversed. For example, Dhaka, Bangladesh has maintained an HIV prevalence rate of below 5% among IDUs. The epidemic among IDUs in Nepal appears to have been delayed for several years. Studies in Asia have shown that the frequently adopted strategies of reduction of drug supply, enforcement of prohibitive laws and incarceration or forced detoxification programmes as a means of HIV prevention yield limited success (Centre for Harm Reduction and the Burnet Institute 2002).

The HIV/AIDS situation in India

Since the discovery of the first AIDS case in India in a female CSW in Tamil Nadu in 1986, HIV has now spread to all the States. Nationally, the HIV prevalence among adults (aged 15–49 years) is less than 1%, but with a population of more than 100 crore, India has the world's second-largest number of PLWHA—an estimated 51.34 lakh by the end of 2004 (NACO 2005b). In six States—Andhra Pradesh, Karnataka, Maharashtra, Manipur, Nagaland and Tamil Nadu—the HIV epidemic is classified as a generalized one, with more than 1% of women attending antenatal clinics (ANCs) being infected and an HIV prevalence among STI clinic patients of more than 5%.

The prevalence of HIV remains the highest among CSWs and their clients, men who have sex with men (MSM), IDUs, truck drivers and patients with STIs, whose behaviour puts them at high risk for contracting HIV. Mumbai has the country's largest brothel-based sex industry, with over 15,000 sex workers. Up to 70% of sex workers in Mumbai are HIV positive. Data on MSM and transgendered persons in Mumbai found that 17% of men and 68% of transgendered people were HIV positive. Twenty-two per cent of MSM were married, and 44% had visited female CSWs (Ekstrand *et al.* 2003). The IDU epidemic is perceived as a problem mainly in the north-eastern States with a reported HIV prevalence of more than 70% in sentinel surveillance sites during the past years (Eicher *et al.* 2000). However, selected surveys point out the increasing evidence of IDU in other parts of India, including border areas. Major metropolitan cities such as Mumbai, Kolkata, Chennai and Delhi have seen a diffusion of injecting drug use within the past decade. A recent study showed that among 4648 drug users interviewed in 14 cities across the country, 43% had injected drugs (National Household Survey 2002; Dorabjee and Samson 2000). By 2001, an estimated 15%–35% of truck drivers nationwide were HIV positive (UNAIDS 2002). HIV infection rates among STI patients were 19.6% in Andhra Pradesh and 13% in Manipur (NACO 2005c).

In India, infection may be due to both HIV-1 and the less pathogenic HIV-2. HIV-1 subtype C is predominant, while HIV-1 A/C recombinants have been described in Maharashtra. In the north-eastern States, other subtypes are present, including subtype B' (similar to the subtype found in Thai IDUs), E, C and B'/C recombinants.

AIDS case reporting

By the end of December 2004, 96,978 AIDS cases had been reported to NACO, with a significant increase in recent years, reflecting both the progression of the epidemic and the improvement in AIDS case reporting. The highest number of reported cases is among the 15–49 years age group. The overall male-to-female ratio showed a declining trend from 3.7 in 1998 to 2.3 in 2003 (NACO 2005).

HIV sentinel surveillance

Conducting HIV sentinel surveillance (HSS) in a country as enormous and diverse as India is a massive undertaking. In 1994, HSS was conducted in 55 sites and expanded to 180 sites in 1998. In 2003, 455 sites were conducting surveillance (NACO 2004). India's HSS system uses anonymous, unlinked blood sample screening for HIV antibodies to estimate the prevalence of HIV population groups in various States. Surveys are now conducted annually, and survey sites include

STI clinics and ANC, as well as sites that work with IDUs, CSWs and MSM. The most recent round of HSS was completed in 670 sites in 2004. These sites included 166 STI clinics, 271 ANCs, 124 rural ANC sites, 13 sites where IDUs were surveyed, 3 sites for MSM and 2 sites for CSWs (NACO 2004). The national working group on HIV estimations and projections commended NACO for adding sites that target IDUs, CSWs and MSM in addition to ANC and STI sites, to better monitor the trend of the HIV epidemic. The quality of HIV surveillance in India was described by an expert team of UNAIDS and WHO in 2001 as having the basic components of a high-quality surveillance system but lacking adequate representation, which has since been improved by adding rural sites and sites for core risk groups, and increasing the total number of sites.

Behavioural surveillance

NACO undertook the National Baseline Behavioural Surveillance Survey (BSS) from April through September 2001. Four groups were surveyed: general population, CSWs and their clients, MSM and IDUs. The general population included 3832 respondents 15–49 years of age (1916 males and 1916 females) with an equal number from urban and rural areas. A total of 5648 clients of CSWs and 5572 female CSWs were interviewed. In addition, control groups of CSWs were surveyed in Delhi, Mumbai, Kolkata and Andhra Pradesh, covering 1087 respondents. A total of 1387 MSM and 1355 IDUs were interviewed across all sampling units.

The National BSS found that the overall awareness of HIV/AIDS in India is 86%, though variance among States is significant (range 53.7%–99.2%) with major urban–rural (91%–74.6%) and gender differences (male-to-female: 86.2 to 73.1) (NACO 2001). Awareness among CSWs (94%), MSM (97%) and IDUs (97%) was much higher than in the general population. The high level of awareness contrasts with the low proportion of condom use during the last sexual contact in high-risk behaviour groups. Among CSWs, 50% reported consistent condom use with paying clients in the past 30 days. Among those brothel-based sex workers who had sex with a non-paying partner in the 3 months before the survey, only 21% reported using condoms consistently. Among MSM who had commercial sex in the month before the survey, only 13% reported consistent condom use with commercial male partners. This contrasts with the figure of 30% of MSM reporting consistent condom use with a non-commercial male partner in the month before the survey. Among the IDUs who reported sex with any non-regular partner in the 12 months before the survey, just 12% reported using condoms consistently with these partners (NACO 2001; NACO 2002).

The BSS found that among IDUs, 45.2% injected two to three times a day, whereas 16.1% injected more frequently. The majority of drug users in India are males. However, use of drug treatment data may underestimate the number of female drug users, who remain a hidden population. Women seeking assistance for drug use face heavy stigma and discrimination, and their ability to access treatment is hindered by their myriad responsibilities and workload (e.g. child care). Drug abuse by women in the north-east is believed to be growing (UNODC 2003).

Data from the BSS indicate some overlap between sex work and injecting drug use. Of the 6% of CSWs reporting ever trying any addictive drugs, almost one-third had injected drugs in the past 12 months.

High-risk behaviour groups such as sex workers, MSM and IDUs are populations that are difficult to access. Although sex work is not illegal, concomitant activities including soliciting and brothel-keeping are penalized. The Human Rights Watch (HRW) reports that Indian CSWs are treated with contempt and commonly subjected to violations of their fundamental rights by the police, both at the time of their arrest and while in detention. HRW also documents increasing violence against outreach workers and peer educators who work with CSWs and MSM. The national BSS found that 61% of female CSWs were illiterate. Homosexuality is a taboo topic in India, and MSM are severely marginalized. Injecting drug use is illegal and strategies focus on reduction of drug supply, enforcement of prohibitive laws and incarceration. Because of complex social and political dynamics, reaching CSWs, MSM and IDUs with HIV prevention services remains a major challenge.

HIV/AIDS estimations

India's HIV prevalence estimates are based on HSS taking into account certain assumptions such as STI prevalence in urban and rural areas, urban–rural differentials in HIV prevalence among STI patients, HIV prevalence among ANC patients, female-to-male differential in STI patients and HIV prevalence rates. Additional data are derived from targeted intervention sites and community-based studies on STI (Fig. 1). The assumptions were refined and validated in 2003. The latest HIV estimate in India for 2004 is 51.34 lakh.

The evolution of the HIV/AIDS programme in India

Since the first cases of AIDS were reported in India, the GOI and society have addressed the epidemic with increasing concern and resources.

National AIDS Committee

To formulate a strategy for the implementation of an HIV/AIDS prevention and control programme, the Ministry of Health and Family Welfare constituted the National AIDS Committee in 1986, under the chairmanship of the Union Ministry of Health and Family Welfare. The Committee brought together various ministries, non-governmental

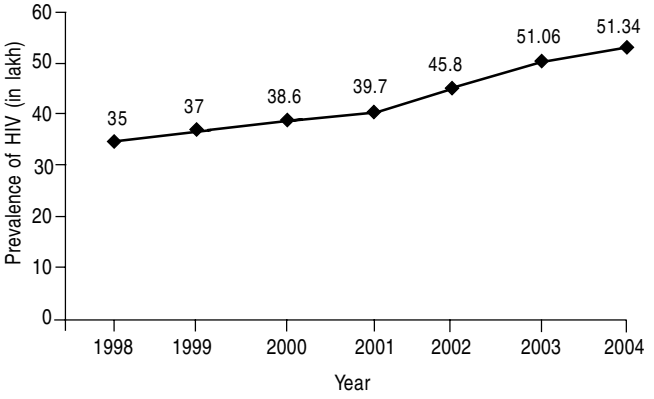

Fig. 1 HIV estimations in India 1998–2004

Source: National AIDS Control Organization 2005b

organizations (NGOs) and private institutions for effective coordination of programme implementation. The Committee acts as the highest-level body to oversee the performance of the Programme, to provide overall policy directions, and to forge multisectoral collaboration (NACO 2005).

Medium-term plan for HIV/AIDS control

In 1989, with the support of WHO, a medium-term plan for HIV/AIDS control was developed with a US$ 10 million budget provided from external sources. This plan was implemented in 4 States and Union Territories (UTs) that were most affected, namely Maharashtra, Tamil Nadu, West Bengal, Manipur and Delhi. Initial activities focused on the reinforcement of programme management capacities as well as targeted education and awareness campaigns (IEC) and surveillance.

Phase I of the National AIDS Control Programme

Following a series of discussions with the World Bank and WHO, GOI prepared a comprehensive five-year (1992–97) National Strategic Plan for the Prevention and Control of HIV/AIDS Phase I. This brought about the establishment of the NACO. During this stage, activities focused on preventing transmission of HIV through blood and blood products, control of hospital infections, increasing awareness of the dangers of unsafe sexual behaviours with multiple partners and sharing of needles for injecting drugs and strengthening of clinical services for both STI and HIV/AIDS (World Bank 2003; NACO 2005d).

Efforts to reduce bottlenecks in the implementation of the Programme were further strengthened with the formation of the State AIDS Societies with decentralized administrative and financial power for more focused programme implementation to address priority issues at the local level.

Phase II of the National AIDS Control Programme

Phase II of the National AIDS Control Programme (NACP) began in 1999. It is a completely centrally sponsored scheme implemented in 35 States and UTs by the State AIDS Control Societies and municipal corporations in the cities.

Table 1. Objectives of the national HIV/AIDS policy

1. Prevent the spread of HIV/AIDS and reduce its personal and social impact. The main activities include control of STIs; promotion of condom use; provision of HIV testing and counselling, care, and support for people with HIV/AIDS; surveillance; harm reduction for injecting drug users; provision of safe blood and blood products; and support for research and development.
2. Generate ownership of the control programme by governmental and non-governmental organizations at the national, State and local levels.
3. Create an enabling environment for prevention and treatment efforts.
4. Decentralize HIV/AIDS control activities.
5. Strengthen programme management at all levels; promote introduction of control activities in other government programmes.
6. Provide support to vulnerable groups.
7. Provide support including treatment to people with HIV/AIDS.
8. Work with multilateral and bilateral donors.
9. Promote better understanding of HIV/AIDS, especially among high-risk groups.

Source: Over *et al.* 2004

NACO assumed the responsibility for activities such as epidemiological surveillance for STIs and HIV/AIDS, training and capacity building, operational research, and monitoring and evaluation. NACO is also responsible for policy-level guidance, overseeing of the programme, allocation of public funds to the States, approval of proposed control activities and coordination with other donor partners. NACO works closely with the States and coordinates advocacy meetings. In 2002, the Government finalized and released the National AIDS Control Policy and the National Blood Policy. These policies were drafted following a wide range of consultations with governmental organizations (GOs) and NGOs, experts and partner agencies (Table 1) (Over *et al.* 2004).

Programme implementation largely depends on the capacities of State AIDS Control Societies, political commitment and administrative leadership, which vary across States. Administrative and financial management capabilities have been strengthened, but technical and management assistance are needed, mostly in strategic planning, priority setting and provision of key service delivery inputs to scaling-up interventions through public and private sector agencies.

The NACP is now planning Phase III, starting in 2006.

Targeted interventions to prevent HIV transmission

The main strategy in India to implement targeted interventions is to work through NGOs. By the end of 2004 (Khera *et al.* 2005), the State AIDS Control Societies had identified 930 NGOs to deliver targeted interventions among high-risk groups (Fig. 2). NACO estimates that 22 lakh people in high-risk groups/bridge populations were covered in 2004. These NGOs need technical support in understanding the complexities of HIV/AIDS issues and inputs for accelerating

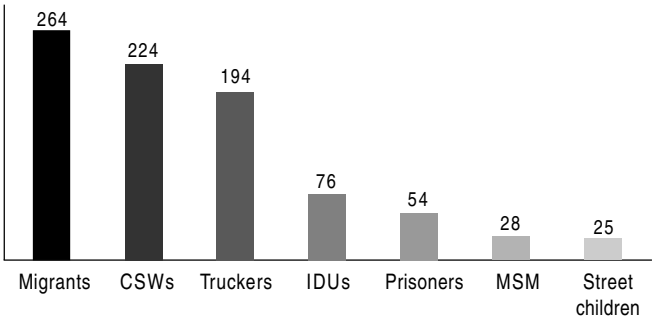

Fig. 2 Number of NGOs supporting interventions targeted at high-risk groups

NGO: non-governmental organization; CSWs: commercial sex workers; IDUs: injecting drug users; MSM: men having sex with men
Source: National AIDS Control Organization 2004

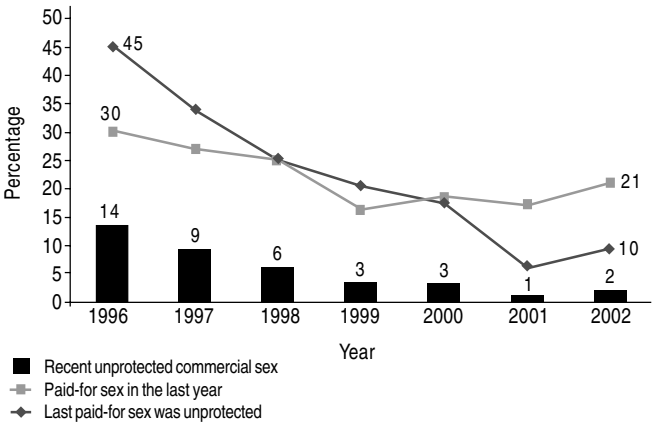

Fig. 3 Effect of high-profile prevention efforts in reducing unprotected commercial sex in Tamil Nadu, India

Source: MAP Report 2004

prevention-oriented outcomes. The interventions are aimed at CSWs, migrant workers, truckers, street children, MSM, IDUs and prisoners, mainly to decrease transmission by reducing high-risk behaviours.

Sex workers and their clients (specifically truck drivers)

The southern State of Tamil Nadu was one of the earliest to be affected by the HIV epidemic. The State Government joined with community groups and other partners to confront the epidemic, running high-profile public campaigns to discourage risky sexual behaviour and making condoms, STI screening and treatment services readily available for those in need. The result has been a significant drop in at-risk sexual behaviour. Figure 3 shows data from a BSS survey among truck drivers and their helpers in Tamil Nadu. In 1996, before the prevention campaigns began, 30% of these men reported sex with a female sex worker in the preceding 12 months, and just over half of them had used a condom at the last sexual contact. This suggests that nearly 14% of truck drivers reported recent unprotected sex with a sex worker. By 2002, this figure had fallen to 2%, partly because there was less sex with a CSW and because condom use rose from 55% to over 90% during the 6 years of prevention programming (MAP 2004).

In 1992, the All India Institute of Hygiene and Public Health launched a programme to reduce the transmission of HIV in Sonagachi, a red-light district in central Kolkata. The project began with two key interventions: a health clinic and outreach activities by peer educators. In 1992, consistent condom use with clients in Sonagachi was 1%. By 1998, this figure had reached 50%. During the same period, syphilis prevalence among CSWs covered by the project fell from 25% to 11%. In 1998, HIV prevalence among CSWs was 5%. A key element of success in the Sonagachi Project has been the participation of CSWs. The Sonagachi model for increasing condom use and maintaining low HIV prevalence rates among sex workers has been successfully implemented in other Indian settings, including in West Bengal (Basu *et al.* 2004).

Injecting drug use

The Indian Ministry of Social Justice and Empowerment has established 450 drug de-addiction centres in partnership with NACO. Only 100 of these centres are staffed with an outreach worker.

The NACO supports harm-reduction activities provided by NGOs. Common activities include needle and syringe exchange programmes (NSEP), substitution therapy, de-addiction, peer education and outreach, primary health care, counselling, drop-in centres and vocational rehabilitation programmes. NACO had approved NSEP operating in Manipur, New Delhi, Mumbai, Kolkata and Chennai. Substitution therapy with the use of sublingual buprenorphine is currently ongoing in five major Indian cities: New Delhi, Kolkata, Imphal, Mumbai and Chennai.

HIV/AIDS prevention and care services in the State of Manipur seem well established. In 1996, Manipur was the first State in India to formulate a State AIDS Policy (SAP), which explicitly included a 'harm reduction' approach to HIV/AIDS prevention among IDUs. Since 1998, the Manipur State AIDS Control Society has implemented a rapid intervention and care programme in partnership with 10 NGOs. The programme is a comprehensive strategy to prevent HIV/AIDS and provide care for those affected in Manipur. Its components are NSEP, condom promotion and provision, referral for HIV testing, STI treatment, home-based care and counselling. Even though the programme had limited geographical coverage within Manipur, it was one of the largest harm reduction efforts in Asia (Sharma *et al.* 2003). It has been reported that while heroin injectors may use the drug two to four times a day, those using buprenorphine and pharmaceutical mixes tend to inject less frequently as a result of the longer effect of these drugs. There is a discernible shift from injecting pure heroin to pharmaceuticals (Kumar *et al.* 2000; Kumar 2000).

The key challenge in scaling up targeted interventions is

to understand the level of effort needed, identify the size and location of high-risk groups/populations, determine what activities are required to expand the coverage of interventions targeting high-risk populations, and identify and contract a sufficient number of credible and committed NGOs to deliver those targeted interventions. In addition, monitoring of the performance of the programme is required. It remains absolutely critical to scale up well-designed and high-quality targeted interventions with adequate coverage.

HIV testing and counselling

NACO has been expanding Voluntary Counselling and Testing Centres (VCTC) since 1998. Significant progress has been made during 2003–04 with the technical support of WHO and UNAIDS. Seven hundred and nine VCTCs have been established nationwide (655 in NACO and 74 in other GOI settings). All districts in the six high-prevalence States have at least one VCTC. Since 2001, the total number of clients tested was more than 19.2 lakh; 80% received pre-test counselling and 13.5% tested HIV positive. During 2002–04, the proportion of those receiving pre-test counselling out of those being tested for HIV has increased from 61% to 96%. The proportion of HIV-seropositive persons remained around 13%–14% during that time (Fig. 4).

Although the availability of VCTC increased under the project, the number of trained counsellors is insufficient. NACO is planning to expand VCTC to the subdistrict level in all States to improve the accessibility of services. A monitoring and supervision system for the performance and quality of VCTC has now been established. Although NACO is scaling-up training and improving the quality of HIV testing and counselling in the public sector, the quality is very limited or unknown in the private sector with no regulating mechanism in place.

Prevention of mother-to-child transmission

With the support of UNICEF, NACO launched the national prevention of parent-to-child transmission (PPTCT) programme. The key components included antenatal care, HIV counselling and testing, safe delivery practices, administering nevirapine (NVP) to the mother and baby, and counselling for infant feeding options. The Programme covers 286 institutions including nationwide private and government medical colleges and all the districts in the six high-prevalence States of India. A summary of data collected during the period from January to September 2004 shows that out of the 1,962,255 women registered with the PPTCT Programme, 74% of women received counselling. The HIV testing acceptance rate was good (86%) but with great variations from State to State and from institution to institution. The overall prevalence observed is around 1.3% with State-wise variation. However, intervention uptake is moderate with only 41% of seropositive mother–baby pairs receiving nevirapine (Fig. 5).

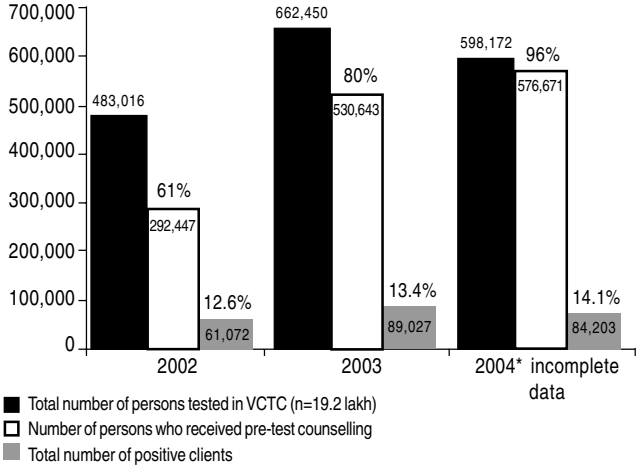

Fig. 4 HIV testing and counselling 2002–04

VCTC: Voluntary Counselling and Testing Centre
Source: National AIDS Control Organization 2004

Care and treatment

Since the launch of the second phase of the NACP in 1999, the GOI has demonstrated its commitment to provide low-cost care to PLWHA. This is reflected in the allocation of 12% of NACO's budget to care and support. The GOI started these activities with 25 community HIV/AIDS care centres across the country. A substantial amount of HIV/AIDS care and support is provided by NGOs and community-based organizations, including associations of PLWHA. These organizations deliver nutrition information, counselling for PLWHA and their families, school fee support, vocational training and, in some cases, provision of drugs for opportunistic infections (OIs). However, they struggle with highly inadequate financial and human resources, coupled with an increasing demand for their services. Many PLWHA experience difficulty in accessing such services because of the social stigma associated with HIV/AIDS (Solomon *et al.* 2002).

As mentioned earlier, 12% of NACO's budget is allocated to HIV/AIDS care, including prevention and treatment of OIs (about 16% of the total budget of the World Bank HIV/

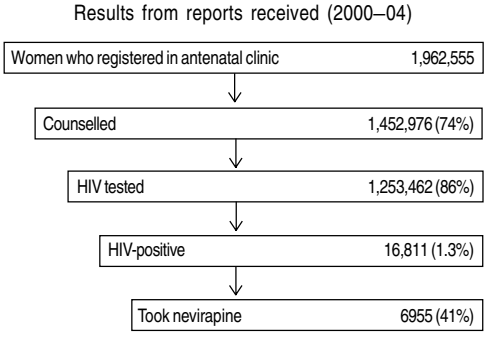

Fig 5. Prevention of parent-to-child transmission 2000–September 2004
Source: National AIDS Control Organization 2004

AIDS loan is designated for medicines to treat OIs). NACO's care strategy covers 30% of an estimated 500,000 PLWHA who seek treatment at government-run and some selected NGO hospitals (Over *et al.* 2004).

India's 2002 proposal to the GFATM states that only 1500 PLWHA are receiving (and adhering to) ART, and that another 8000–10,000 are intermittent users or poorly adherent (Kumarasamy *et al.* 1999). ART so far has primarily been prescribed for those who can pay or who are enrolled in research studies (Ekstrand *et al.* 2003).

During 2004, India made impressive progress in a short span of time since the announcement of a free ART programme on 1 December 2003. The target is that 100,000 patients should receive ART by the end of 2007. The GOI, with support from WHO, has held national and subnational technical consultations and review meetings, developed national treatment guidelines, prepared training materials and built up the capacity of medical teams. The free ART Programme was launched in April 2004 at eight tertiary hospitals in the six high-prevalence States of India plus the capital city, Delhi. Antiretroviral drugs were procured with the help of WHO. It is planned that by the end of 2005, the Programme will be expanded to 50 sites across the country and, by the end of 2005, to 50–100 tertiary hospitals. The remarkable progress made in such a short period of time is the result of administrative commitment, strategic planning, partnerships and, most importantly, the sheer hard work and compassion of the health workers at all levels. As of April 2005, a total of 7029 PLWHA have started treatment in the NACO-supported programme—34% of women and 4% children (NACO 2005). Analysis of data from a cohort of 143 patients from Tambaram, Chennai showed a three-fold increase in CD4 counts after 6 months of treatment, indicating marked clinical improvement after ART (Fig. 6).

Adherence and resistance

Treatment adherence is a critical issue. Poor adherence to ART leads both to poor clinical outcomes and to the transmission of drug-resistant viral strains, thus lowering the effectiveness of ART in the infected population. These considerations strongly suggest that ART programmes should invest sufficient resources in supporting high levels of adherence to counselling and monitoring. Concerns about adherence and the spread of resistant viral strains may be particularly pertinent in India because the generic, low-cost, triple-drug formulations available in India include non-nucleoside reverse transcriptase inhibitors (NNRTI) such as nevirapine. Evidence suggests that an easily acquired single-point mutation can confer resistance to all the agents in the NNRTI class when the virus becomes resistant to nevirapine alone.

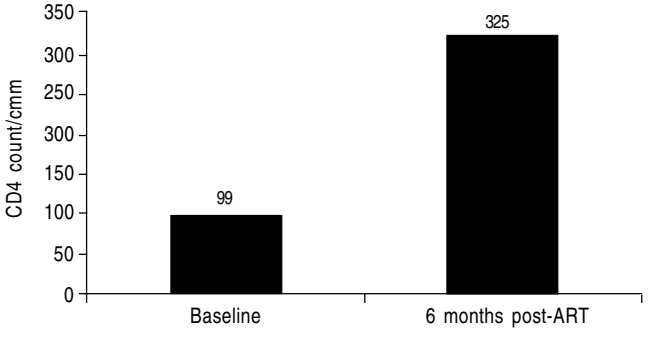

Fig 6. Pre- and post-ART (at month 6) CD4 counts in a cohort of 143 patients in Tambaram, Chennai in December 2004

ART: antiretroviral therapy

Although too few studies have been published on adherence to ART in resource-poor countries to draw firm conclusions, the results generated so far suggest that adherence rates are similar in resource-rich and resource-limited countries. Although high levels of adherence can be achieved, a wide range of adherence levels has been reported in both industrialized and developing countries. Drug cost can be a significant barrier to adherence. In a study on 100 patients on triple-drug ART treatment in India, 60% stopped treatment within a few months because of the high cost and because they preferred to take alternative treatment. Thus, operational research to identify effective adherence techniques specific to India is warranted (Ekstrand *et al.* 2003).

Access to medicines

The GOI has a mandate to provide access to treatment to all the employees working in various Central Government departments (Over *et al.* 2004). Some public sector provision is made through Central Government Health Scheme (CGHS), Employees' State Insurance Scheme (ESIS, the country's social security programme), the Railways and the Department of Defence. NACO has committed Rs 20 crore to antiretroviral medicines covering 25,000 PLWHA for the fiscal year 2005 in addition to GFATM funds.

To reduce prices, the Government is making efforts to exempt customs and excise duties on all antiretroviral drugs available in India. Indian pharmaceutical companies are currently manufacturing generic versions of ART and selling them at less than US$ 1 a day. The manufacture of generic ART drugs has been an essential element in the dramatic reduction of drug prices. However, India signed the agreement on Trade-Related Aspects of Intellectual Property Rights (TRIPS) as a member of the World Trade Organization in 1994. As a result, India had to change its patent law on 1 January 2005. In consequence, while Indian manufacturers may continue their existing production of generic versions of drugs that have been patented elsewhere in the world between January 1995 and December 2004, they may not be able to initiate the production of additional generic versions of drugs that fall into this category. This may affect some ART drugs, notably those used in second-line treatment regimens. Furthermore, future antiretroviral agents that may be developed will be even more affected; they will

receive a 20-year patent protection in India, and Indian companies will only be able to produce and sell generic versions of such new drugs after the relevant patents expire. These changes will affect not only the cost of ART programmes in India, but also in countries supplied inexpensive ART drugs by Indian companies.

Health system challenges to scaling up

India's public health system has struggled to provide a basic level of care for the poor and marginalized and, in the wake of a growing epidemic, the system will be under considerable more stress to provide testing, counselling, prevention, education, treatment and palliative care in a coordinated and sustained manner. The reasons for system constraints on scaling up HIV/AIDS interventions are complex, and reflect a number of broader public health system challenges as well as social challenges such as stigma and social exclusion.

Working Group 5 of the Commission on Macroeconomics and Health (Jha and Mills 2002) described the health system constraints that need to be removed to expand access to interventions at several levels, from the level of governance, public policies cutting across sectors, and health sector policy and strategic management to the levels of health services delivery, and community and household participation. This systematic review of constraints was applied to the scaling up of HIV/AIDS interventions in India and a modified and expanded version is presented in Table 2 (Sheshadri 2001).

The matrix is not exhaustive and the GOI and partners have made significant progress on many of these factors, as can be seen by the escalating control efforts over the past few years. Of course, the systemic challenges to the scaling up of HIV/AIDS mirror the broader public health system constraints that impact the prevention and care of any disease. For the purposes of this report, we will focus on issues at the level of health service delivery that have a significant impact on the control of the HIV/AIDS epidemic, including monitoring of the epidemic and impact of interventions, human resources, and the role of the private sector and NGOs.

Monitoring the epidemic

Tracking the epidemic and implementing effective programmes is made even more complex by the fact that the epidemic can be described as several localized subepidemics representing diverse risk factors and socioeconomic settings. A reliable and regular surveillance and data collection system on the prevalence, distribution and modes of transmission of HIV/AIDS is necessary for effective setting of priorities and planning a responsive control strategy (Bajpai and Goyal 2004). As described previously, NACO has expanded the sentinel surveillance programme in ANC and STI clinics. Still, it is generally thought that there is a need to strengthen and address the gaps in the surveillance system in the following areas:

- Conduct BSS at regular intervals;
- Expand sentinel surveillance to better represent high-risk groups including IDUs, CSW and MSM, and improve the mapping and size estimation of high-risk groups;
- Initiate better STI surveillance as an objective marker of behavioural change;
- Establish the pattern of disease in rural and migratory populations, high-risk occupational groups and other difficult-to-access groups;
- Understand the factors contributing to transmission of the disease from high-risk to low-risk groups;
- Carry out systematic data collection from private sector settings through establishment of public–private partnerships in surveillance (the private sector delivers over 80% of health care in India overall and delivers care to 80% of patients seeking treatment for STIs) (Ekstrand *et al.* 2003);
- Carry out external validation of the findings of the current surveillance system.

Data collection on the HIV/AIDS epidemic at the State and district levels is the key to effective planning and management in a decentralized programme, which needs to be operationalized by capacity building of personnel in State AIDS Control Societies. Tamil Nadu's early success in

Table 2. Health system constraints to scaling up HIV/AIDS interventions

Governance and overall policy framework	• Political will and commitment • Unwillingness of bureaucrats to involve themselves in a programme perceived as dealing with a taboo subject • Lack of a sense of urgency to tackle the impending epidemic
Health sector policy and strategic management	• Unpredictable funding from the Centre to the States • Inadequate technical and management capacity of programme managers • Inadequate advocacy efforts and sense of ownership at the State level
Health service delivery	• Inadequate capacity in terms of people who could manage the process as well as organizations such as NGOs to effectively implement well-designed programmes, for example, targeted interventions, care • Inadequate capacity for maintaining an accurate database and tracking the epidemic • Inability to regulate or monitor the provision of services by the private sector
Community/household demand/impact	• Non-availability of effective NGOs in high-risk areas due to lack of funding for such activities in the past • Lack of awareness among community members and implementers of the risk and unwillingness to admit that this is possible in their community

controlling the epidemic has been well recognized. The State's success has been attributed to an early autonomous and comprehensive strategy that built capacity on many levels including programme management, targeted interventions, community awareness and support, and surveillance. The Tamil Nadu State AIDS Control Society initiated a systematic data collection system early on, which included behaviour, awareness and sentinel surveys. These early surveillance efforts increased the understanding of the epidemic patterns, including spread to the general population, especially women and the increase in rural prevalence, helping to target the interventions by the State's AIDS control effort (Sheshadri 2001).

The surveillance for AIDS and STI, though started during the first phase of the Programme, needs strengthening. The number of cases with both STI and AIDS may not be accurate since the shame and stigma associated with these diseases deter patients from accessing government health services, and the majority seek relief from private practitioners who may not be qualified. Since there is no regulatory mechanism for obtaining information from NGOs and private clinics or testing centres, many cases are missed out.

Monitoring the programme

The effective implementation of ARV therapy is complicated by a lack of public infrastructure for monitoring adherence and resistance to, and outcomes of, therapy. Monitoring requires laboratory capabilities to confirm CD4 count levels, and the infrastructure and human resources to follow up adverse effects and patient tolerance to drug regimens. In light of the growing prevalence of drug-resistant HIV strains worldwide, the health system's capability to monitor and ensure drug availability and patient adherence to therapy is vital. Further, surveillance and evaluation of the impact and effectiveness of treatment of opportunistic infections as well as the interaction of other interventions in areas such as nutrition and sanitation also need to be targeted (Willbond *et al.* 2001).

Another area where monitoring is vital is the evaluation of the impact of an increasing emphasis on treatment and prevention, both at a programme level and with respect to personal behaviour. The strengthening of behavioural surveys will be important to monitor any decrease in preventive activities or increase in high-risk behaviours stemming from increased availability of treatment (Over *et al.* 2004).

Human resources and facilities

In an environment where access to primary care centres and health care workers is already low, an epidemic such as HIV/AIDS can put massive pressure on an already stretched Indian public health system. The related increases in deaths and infections will stress capacities of both primary care clinics and hospitals. Additionally, the public health system in India is experiencing critical staff shortage, especially in rural areas. Public health care staffing suffers from a lack of equitable geographic distribution, absenteeism, and monitoring of competence and quality of care. One estimate reports that out of almost 30,000 rural health posts for doctors, over 4000 remain unfilled and about 40% absenteeism has been estimated among doctors and other health care workers (Willbond *et al.* 2001). An unregulated and, for the most part, unaccountable health work force in the public and private sectors makes it difficult to monitor the impact and effectiveness of health service delivery.

Specifically, human resources for HIV/AIDS require specialized training in the disease, its risk factors and treatment options. Although India has a large pool of doctors and other health professionals, practical experience in the clinical management of AIDS patients or ART is limited (WHO 2003). Without the appropriate incentives, adequate training, and systematic tracking of health facilities and staff, successful expansion of the control programme will not be feasible. Prescriptions of wrong drug combinations or dosages and the inability to follow up patient treatment regimens decrease the effectiveness of treatment and increase the risk of drug resistance (Cohen 2004).

Role of the private sector and NGOs

The private sector in India plays a large and important role in the delivery of health care and is, overall, an important partner of the GOI in the care of patients with HIV/AIDS and associated infections. It has been estimated that 80% of doctors, 75% of dispensaries and 60% of hospitals in India are in the private sector (Ekstrand *et al.* 2003).

However, the prominence of the private sector in health delivery in India has also been an area of concern. Many Indian doctors and government officials believe that greater access to ART could lead, particularly in the largely unregulated private sector, to faulty prescription practices that might set the stage for the emergence of drug-resistant HIV strains (Mudur 2002). These concerns are reflected in the findings of a recent multicentric study of the causes of failure of ART in India. In the study, led by the personnel of the Grant Medical College and GT Hospital in Mumbai, only 10% of the patients were counselled before initiating ART. Adherence was observed in only 10% and all were on suboptimal regimens (Saple *et al.* 2002).

Another study led by this hospital examined the knowledge and practices of family physicians and consultants in three low- and high-prevalence States each. In the low-prevalence States, 70% of family physicians were unaware of the availability of an HIV-ELISA test, and 80% unaware of the various ARTs available except zidovudine (ZDV). CD4 counts and viral load monitoring facilities are unavailable and counselling concepts alien. In high-prevalence States, 85% of family physicians knew of ELISA and the Western blot tests. Elementary counselling concepts are known but seldom practised. Parameters to initiate therapy, drug

regimens, drug combinations and patient monitoring are poorly known. About 5% of family physicians attempt to use ART, with ZDV+3TC the most frequently used regimen, though monotherapy is also common. Internists, chest physicians and dermatologists/venereologists also practise HIV medicine, but only 60% of them know of HIV/AIDS drugs and regimens. Their knowledge of patient selection criteria and monitoring, including CD4 counts and viral load, is very limited. Over 90% are not familiar with salvage therapy (Vaidya and Deshpande 2002; Brugha 2002). Without more accountability from the private sector and better data from these sites, it is difficult to accurately say what percentage of HIV/AIDS diagnoses, treatment and care is imparted by the private sector and what is the impact. Therefore, capacity building should include strengthening the private sector.

As described previously, NACO has actively promoted community-based organizations and NGOs. NGOs are an integral partner in the scaling up of HIV/AIDS interventions, especially in their role as primary implementers of community-level interventions. The involvement of NGOs strengthens the implementation of the control programme at the grassroots level, improves its reach to marginalized populations and promotes greater community partnership and ownership. This involvement complements the decentralized approach through the State AIDS Control Societies. NGOs are involved in all aspects of HIV/AIDS control, including education, counselling, treatment and social support, especially among high-risk groups and schoolchildren through the School AIDS Education Programme (NACO 2005).

Capacity building should therefore include strengthening the role of NGOs at all levels of the control strategy—in policy development, implementation and evaluation of programmes—so that targeted interventions reach those most in need and greater political commitment and action can be advocated.

Financing challenges

If uncontrolled, the HIV/AIDS epidemic can have serious economic challenges for India, especially at the household level. The need for responsive, effective, and sustained preventive and treatment interventions necessitates commitment of national resources as well as sustained collaboration with international and national partners. Though the cost of treatment has been drastically reduced with the advent of generic drug production in India, the cost of therapy is still too expensive for millions of individuals and thus needs to be funded through public and other sources. As India's commitment to controlling HIV/AIDS and improving the quality of life of those suffering from the disease is growing, so are the challenges to finance the Programme.

Health sector resources

The GOI has expressed its commitment to increase its spending on social services including health, and to target the protection of the most vulnerable from the burden of expenditure on health. Currently, India spends 6.1% of its GDP on health (US$ 30 per capita). Of the total amount spent on health, general government expenditure accounts for 21.3% (US$ 6 per capita), while private spending contributes 78.7%. This is similar to what can be found in many developing countries where private health expenditure constitutes 73% of the total health expenditure (WHO 2004).

States account for 51% of general government spending and fund about 75% of total public health expenditures (World Bank 2001). However, the proportion of health to total expenditure in States through the 1990s actually declined from 6%–7% to just over 5% (Misra *et al.* 2003). Moreover, the level of Central Government spending for the States has not been found to reflect 'differences in health needs, performance of health systems or the amount of fiscal effort put in by the States' (Peters *et al.* 2003).

Of private expenditure, the majority is in the form of regressive out-of-pocket (OOP) spending (98% of private expenditure). This scenario is a familiar one in developing countries. Poor households are the most likely to require basic, essential services and the most likely to be pushed into poverty (or deeper into poverty) by OOP health spending. In 1995–96, nearly 40% of Indians who were hospitalized fell into debt to pay for hospital expenditures and nearly one-quarter fell below the poverty line as a result (Peters *et al.* 2002). A recent World Bank study on India concludes that OOP health expenses may push 2.2% of the population below the poverty line each year (World Bank 2001)

Over the past year, the GOI has made a commitment to increase investments in the health sector. In fact, this commitment has been promoted through the Government's Common Minimum Programme (CMP). The health component of the CMP calls for a doubling of public health spending over the next five years (with a focus on primary health care and communicable diseases), the development of a Universal Health Insurance scheme and increased emphasis on the poorest, least serviced districts. In its most recent budget, the Government has pledged an increase of Rs 10,280 crore for health.

Spending on HIV/AIDS

The NACP, including the National AIDS Control Project Phase II, and related projects by the US Agency for International Development (USAID) and the UK Department for International Development (DFID) require funding of Rs 1425 crore (phased in 1999–2005), of which a majority (Rs 1155 crore) is a combination of World Bank IDA credit (Rs 959 crore) and Government spending (Table 3). The funding that is allocated to each programme component is shown in Table 4. NACO has committed Rs 20 crore for antiretroviral medicines covering 25,000 PLWHA for the fiscal year 2005, in addition to GFATM funds.

Several donors have increased their funding for targeted

Table 3. Funding of National AIDS Control Project Phase II

Funding of the National AIDS Control Project Phase II	Rupees in crore
IDA credit (1999–2004)	1155
USAID assistance for AVERT Project in Maharashtra	166
DFID assistance for sexual health projects for the States of Andhra Pradesh, Gujarat, Kerala and Orissa	104
Total	1425

Source: National AIDS Control Organization 2005g

Table 4. Funding of the National AIDS Control Programme, by component

Component allocation of NACP-II	Rupees in crore
Targeted interventions for groups at high risk	265.6
Preventive interventions for the general community	389.1
Low-cost AIDS care	163.3
Institutional strengthening	286.5
Intersectoral collaboration	50.5

Source: National AIDS Control Organization 2005g

intervention programmes in India. The Bill and Melinda Gates Foundation has committed US$ 200 million to the initial 5 years of the Avahan Programme to reduce HIV-1 and STI transmission in selected high-risk populations (primarily female sex workers and their clients, and IUDs) in 65 high-prevalence districts in 4 States. The Programme will also support monitoring through behavioural and biological surveys. The Global Fund has approved US$ 100 million to India for counselling pregnant women and their families, and treatment to prevent MTCT. Such focused intervention strategies are supported by models that show upto 80% reduction in HIV incidence over the next three decades with consistent, high levels of condom use by CSWs, levels that are achievable with currently available interventions (Nagelkerke *et al*. 2002).

Though beyond the scope of this report, several challenges must be addressed, which have important implications for the sustainability of the scaling up and public financing of ART, even with subsidization of costs by external partners. These challenges include the implications of TRIPS-induced patent protection on the cost of drugs, facilities for testing and counselling, laboratory support for the monitoring of CD4 counts, training of and access to trained health care workers, and sustained strategies for procurement, transportation, and storage of medications. Already several countries such as Brazil have seen a massive increase in demand after the introduction of free or low-cost treatment (Willbond *et al*. 2001).

Overall, the per capita AIDS-related spending in India is lower than other countries that have had success in controlling their epidemic. India spends US$ 0.17 per person, as compared to US$ 0.55 in Thailand and US$ 1.85 in Uganda (*The Economist* 2004). Scaling up of the HIV/AIDS control intervention will require high expenditure and innovative schemes to protect poor families from an even greater financial burden. More information is needed on the HIV/AIDS budget to adequately comment on the efficiency in allocating resources or financing gaps. Also, further directed cost analysis is required to evaluate the financing of the scaling up of HIV/AIDS interventions in relation to the limited financial resources and capacity of overall health systems. However, it is evident that the Government needs a sustained commitment for funding HIV/AIDS control programmes, reflecting recent government pledges to increase overall health sector expenditures.

Discussion

There are an estimated 51 lakh PLWHA in India and the number, by all projections, is expected to rapidly increase. The increased burden of this disease on an individual basis will result in untold suffering, death and devastation of families. As witnessed in several sub-Saharan African countries, the HIV/AIDS epidemic in India has the potential of eliminating recent growth and development milestones. According to *Thailand Health Profile 1999–2000*, HIV/AIDS accounted for the most common cause of deaths among all age groups in 2000.

The GOI has necessarily implemented a comprehensive, targeted and ever-mounting HIV/AIDS Programme. The Government's commitment to controlling the disease is evidenced by the ambitious HIV/AIDS targets of its Tenth Five-Year Plan (India Planning Comminssion 2002). The goals are:

- 80% coverage of high-risk groups through targeted interventions
- 90% coverage of schools and colleges through education programmes
- 80% awareness among the general population in rural areas
- reducing transmission through blood to less than 1%
- establishing at least one VCTC in every district
- scaling up of PMTCT activities up to the district level
- achieving zero-level increase of HIV/AIDS by 2007.

These goals are to be viewed in the light of the launching of the National Rural Health Mission (NRHM), which has the ingredients to facilitate their successful fulfilment. Further, each of these challenges such as surveillance strengthening, especially in rural areas and the private sector, scaling up of public resources, infrastructure and trained human resources, increased involvement of NGOs and partnerships with the private sector can each be handled by the various components of the Programme. The strategies under the NRHM aim at decentralization of the Programme at the district level, especially in the Empowered Action Group (EAG) States, which are also the States vulnerable to HIV/AIDS—Uttar Pradesh, Bihar, Madhya Pradesh, Chhattisgarh, Jharkhand, Rajasthan, Himachal Pradesh, and Jammu and Kashmir. The list also includes the north-eastern States where HIV prevalence is either very high or on the rise.

The key components for operationalization envisaged under the Programme facilitate better implementation of prevention and control strategies for HIV/AIDS. There is provision in the Programme for a cadre of accredited social health activists (ASHA) in each village. Her job includes generation of awareness on HIV/AIDS and reproductive tract infection (RTI)/STI. Incidentally, the States chosen for focused implementation of the Programme are the very States where the BSS has noted that awareness among rural women is as low as 20%–27%. Under the Tenth Plan, NACO has fixed the target of achieving 80% awareness among the rural population. ASHA would act as agents for creating awareness at the grassroots level, and for bringing about a behavioural change for better reproductive health, including HIV/AIDS. They would also facilitate the detection of high-risk groups and behaviour in the local area, so that timely action can be taken. Besides, ASHA will offer counselling services, provide links with the primary health care system, private practitioners and others for meeting the needs of the beneficiaries. She will also be a source of condoms and other mother- and child-related services, the necessity for which would be acute, as more women and children are drawn into the HIV/AIDS epidemic in rural areas. She is likely to succeed since her earning is performance-linked; remuneration is to be given by the system for the work done.

Strengthening of the more than two thousand community health centres in the country to the level of Indian Public Health standards would eventually support the opening of VCTC, provision of PPTCT services and Directly Observed Treatment, Short-course (DOTS) under one roof and closer to the client in rural areas. The strengthening of district- and *taluk*-level hospitals can result in their acting as centres for dispensing and monitoring ART in the local area, thus solving issues regarding adherence to and monitoring of ART. Accredited private sector organizations with appropriate regulatory mechanisms, as envisaged in the NRHM, would pave the way to also utilize them for providing drugs and monitoring patients on ART in rural areas under the '3 by 5' initiative undertaken by NACO, whereby 1 lakh AIDS cases would be given ART by 2005 (NACO/MoHFW 2004).

Capacity building of programme managers to provide effective technical and managerial support at all levels from the national to the district level and below should provide the much-needed support to the Panchayat in the delivery of health care and for the initiation of District Health Mission and Village Health Committees. This would result in the formulation and implementation of area prevention strategies in localized pockets of high prevalence, and help to stall the progress of the epidemic.

Since the social and economic ramifications of HIV/AIDS are felt more than the health aspect, those affected would benefit immensely from health insurance schemes mooted for the rural poor. With the majority of the HIV-positive being from rural areas, and lower socioeconomic strata (NIHFW/NACO 2003), the introduction of such schemes would prevent families from being driven further into poverty.

Supportive policy reforms in the areas of Public Health Management, medical education, integrating Indian Systems of Medicine (ISM) into the mainstream and regulation of health providers can all be spheres for spearheading HIV/AIDS/STI programme components; e.g. in medical education, essential training of doctors in diagnosis, and in the management of ART among adults and children. It also provides an opportunity for initiating vocational courses on community-based counselling and providing care and support at home and in hospitals.

Although there is convergence of funds at all levels for the major health programmes under the NRHM, the funds for NACO would remain separate for all activities except for services integrated with the Reproductive and Child Health (RCH) Programme.

The integration of services for HIV/AIDS below the district level with the RCH and the common pooling of funds at the district level enable local authorities to spend funds according to the disease profile and other health needs of the community in the area.

Conclusion

In conclusion, it may be stated that though challenges exist, modalities and opportunities for handling them successfully are also available, and the formation of programme management units at the district level under the NRHM ensure that the pitfalls encountered earlier in the implementation of the HIV/AIDS Programme in India are prevented in the future.

The aim of this report has been to provide an overview of the HIV/AIDS epidemic in India, the direction of control efforts, relevant experiences from other countries, and to highlight some of the systemic constraints to scale-up that require immediate attention. It also discusses the potential of the newly introduced health programme, and the NRHM implemented from April 2005 (MoHFW 2005), which can help boost the implementation of the HIV/AIDS Programme in many spheres.

Over the past several years, India has shown a true commitment to improving and investing more in health and specifically for the control of HIV/AIDS. India's epidemic is multiple and diverse, and so the intervention strategies will have to be adapted to the diverse risk and behavioral characteristics. Consistent and regular information about the epidemic is needed, which well represents local data and high-risk groups. It is clear from experiences from other countries and the history of the Indian epidemic so far that to reach the MDG target of stopping the spread of HIV/AIDS by 2015 will require expansion of targeted interventions for high-risk populations, strengthening of the health system at large, improving partnerships with the private sector and civil society, and the backing of these activities with sufficient and sustained funding.

Acknowledgements

The authors acknowledge the contributions of Renu Garg, Karin Timmermanns, Jai P. Narain, Sergio Spinaci and Binod Mahanty.

References

Bajpai N, Goyal S. Primary health care in India: Coverage and quality issues. Center on Globalization and Sustainable Development. Working Paper No. 15, June 2004.

Basu I, Jana S, Rotheram-Borus MJ, Swendeman D, Lee SJ, Newman P, *et al.* HIV prevention among sex workers in India. *J Acquir Immune Defic Syndr* 2004;**36:**845–52.

Brugha R. Antiretroviral treatment in developing countries: The peril of neglecting private providers. *BMJ* 2002;**326:**1382–4.

Centre for Harm Reduction and the Burnet Institute. *Revisiting the hidden epidemic: A situation assessment of drug use in Asia in the context of HIV/AIDS.* Melbourne: Centre for Harm Reduction and the Burnet Institute; 2002.

Cohen J. HIV/AIDS: India's many epidemics. *Science* 2004;**304:**504–9.

Dorabjee J, Samson L. A multi-centre rapid assessment of injecting drug use in India. *Int J Drug Policy* 2000;**11:**99–112.

Duraisamy P. Economic impact of HIV/AIDS on patients and households in south India. 11th IAEN Face-to-Face Conference. Available from URL: http://www.iaen.org/papers (accessed in May 2005).

Eicher AD, Crofts N, Benjamin S, Deutschmann P, Rodger AJ. A certain fate: Spread of HIV among young injecting drug users in Manipur, north-east India. *AIDS Care* 2000;**12:**497–504.

Ekstrand M, Garbus L, Marseille E. *HIV/AIDS in India.* Country AIDS Policy Analysis Project. UCSF. San Francisco: AIDS Policy Research Center, University of California; 2003.

India Planning Commission. *Tenth Five-Year Plan, 2002–2007. Vol. 2*. Chapter 2.8; 2002:117. Available from URL: http://www.planningcommission.nic.in/plans/planrel/fiveyr/10th/default.htm (accessed in April 2005).

Jha P, Mills A. *Improving health outcomes for the poor*. Report of the Working Group 5 of the Commission on Macroeconomics and Health. New Delhi: WHO; 2002.

Khera, *et al*. Paper presented at National Review Meeting of HIV-TB Coordination 2, February 2005, Delhi, India.

Kumar MS, Mudaliar S, Thyagarajan SP, Kumar S, Selvanayagam A, Daniels D. Rapid assessment and response to injecting drug use in Madras, South India. *Int J Drug Policy* 2000;**11:**83–98.

Kumar S. Rapid situation assessment on injecting drugs use in Chennai, South India. Report to UNESCO, DAPPA and SHARAN. New Delhi, India, 2000.

Monitoring the AIDS Pandemic Network (MAP). *AIDS in Asia: Face the facts*. MAP report; 2004.

Misra R, Chatterjee R, Rao S. *India health report.* New Delhi: Oxford Press; 2003.

MoHFW. National Rural Health Mission Draft document, 2005.

Mudur G. India must change health priorities to tackle HIV. *BMJ* 2002;**325:**1132.

National AIDS Control Organization (NACO). An overview of the spread and prevalence of HIV/AIDS in India. 2005a. Available from URL: http://www.nacoonline.org/facts_overview.htm (accessed in May 2005).

NACO. Facts and figures. HIV estimates—2004. 2005b. Available from URL: http://www.nacoonline.org/facts_hivestimates04.htm (accessed in July 2005).

NACO. Facts and figures. Observed HIV prevalence levels State-wise: 1998–2004. 2005c. Available from URL: http://www.nacoonline.org/facts_statewise.htm (accessed in February 2005).

NACO. About NACO/National AIDS Control Programme Phase I (1992–99). 2005d. Available from URL: http://www.nacoonline.org/abt_phase1.htm.

NACO. 2005e. Available from URL: http://www.nacoonline.org (accessed on 25 February 2005).

NACO. 2005f. Available from URL: www.nacoonline.orgpartnership.htm (accessed in February 2005).

NACO. 2005g. Available from URL: www.nacoonline.org/prog_sche_finan.htm (accessed in March 2005).

NACO. 2005h. Monthly updates on AIDS (31 January 2005). Available from URL: http://www.nacoonline.org/facts_reportjan.htm (accessed on 22 February 2005).

NACO. National Baseline General Population Behavioural Surveillance Survey, 2001.

NACO. National Baseline High Risk and Bridge Population Behavioiural Surveillance. Parts I and II. 2001 and 2002.

NACO. State-wise HIV prevalence (1998–2003). Available from URL: http://www.nacoonline.org/ facts_statewise.htm (accessed on 22 February 2005).

NACO. Review of the Antiretroviral Treatment Programme, India, December 2004.

NACO/MoHFW. Programme implementation guidelines for a phased scale-up of access to antiretroviral therapy for people living with HIV/AIDS; 2004:10.

Nagelkerke NJ, Jha P, de Vlas SJ, Korenromp EL, Moses S, Plummer FA. Modelling HIV/AIDS epidemics in Botswana and India: Impact of interventions to prevent transmission. *Bull World Health Organ* 2002;**80:**89–96.

National Household Survey, 2002; RAS in 14 cities, 2002; Drug Abuse Monitoring System; Survey on the extended National Pattern of Drug Abuse and its consequences in India, 2002.

NIHFW/NACO. *HIV Sentinel Surveillance in India.* New Delhi: National AIDS Control Organization; 2003.

Over M, Heywood P, Gold J, Gupta I, Hira S, Marseille E. *HIV/AIDS treatment and prevention in India. Modeling the costs and consequences.* Washington, DC: The World Bank; 2004.

Peters DH, Rao KS, Fryatt R. Lumping and splitting: The health policy agenda in India. *Health Policy Plan* 2003;**18:**249–60.

Peters DH, Yazbeck AS, Sharma R, Ramana GNV, Pritchett L, Wagstaff A. *Better health systems for India's poor: Findings, analysis, and options.* Washington, DC: The World Bank; 2002.

Rojanapithayakorn W, Hanenberg R. The 100% condom program in Thailand. *AIDS* 1996;**10:**1–7.

Russell S. AIDS in India: South Asia's smoldering threat. Available from URL: www.sfgate.com (accessed in February 2005).

Saple DG, Vaidya SB, Kharkar RD. Causes of ARV failure in India. Abstract no. WePeB5860. XIV International Conference on AIDS, Barcelona, 7–12 July 2002.

Sharma M, Panda S, Sharma U, Singh HN, Sharma C, Singh RR. Five years of needle syringe exchange in Manipur, India: Programme and contextual issues. *Drug Policy* 2003;**14:**407–15.

Sheshadri SR. Constraints to scaling up health interventions: Country case study, India. CMH Working Paper Series, Working Paper No. WG5:15; 2001.

Solomon R, Solomon ARS, Vijaya S. Double impact: Integrating prevention, community sensitization and mobilization for sustainable community based care and support by service delivery for people living with HIV/AIDS and affected families in rural Andhra Pradesh, India. Abstract no. WePeF6740. XIV International Conference on AIDS, Barcelona, 7–12 July 2002.

Thailand Health Profile 1999–2000. Ministry of Public Health, Thailand.

The Economist. Could AIDS explode in India? Available from URL: *http://www.economist.com/opinion/displayStory.cfm?story_id=2603788* (accessed on 15 April 2004).

UNAIDS. *AIDS epidemic update*. Geneva: UNAIDS/04.45E; December 2004.

UNAIDS/WHO. *India: Partnership menu*. Geneva: UNAIDS; 2002. Available from URL: http://www.unaids.org/partnership/pdf/INDIAinserts.pdf.

UNDP. YouAndAIDS. Available at URL: http://www.youandaids.org/Asia%20Pacific%20at%20a%20Glance/India/index.asp (accessed on 7 March 2005).

UNODC. *Country profile India*. UNODC 2003. Available from URL: http://www.unodc.org/india/en/country_profile.html (accessed on 7 March 2005).

Vaidya SB, Deshpande AK. Antiretrovirals (ARVs) in India—a challenge with two edges. Abstract no. MoPeB3316. *XIV International Conference on AIDS*, Barcelona, 7–12 July 2002.

WHO Regional Office for South-East Asia. Available from URL: http://www.whosea.org (accessed on 24 February 2005).

WHO, India. Report of the first 3 by 5 mission. New Delhi, 8–12 December 2003.

WHO. *The World Health Report 2004: Changing history*. Geneva: WHO; 2004.

Willbond B, Thottingal P, Kimani J, ME Vaz L, Plummer FA. The evidence base of interventions in the care and management of AIDS in low and middle income countries. CMH Working Paper. Paper No. WG5:25. 2001.

World Bank. *India Country Assistance Strategy*. New Delhi: World Bank; 2001.

World Bank. *India National AIDS Control Project. Project performance assessment report* (credit 2350), 2 July 2003.

World Bank. *Raising the sights: Better health systems for India's poor. Health, nutrition, population sector unit*. India, South Asia Region, 2001.

HIV-1 trends, risk factors and growth in India

RAJESH KUMAR*, PRABHAT JHA†, PAUL ARORA†, NEERAJ DHINGRA†, INDIAN STUDIES OF HIV/AIDS WORKING GROUP

It is certain that the epidemic of human immunodeficiency virus type 1 (HIV-1) in India will increase. However, there remains considerable uncertainty about the prevalence and incidence of HIV-1 infection, and the determinants of infection. The future growth of HIV-1 also remains uncertain.

Currently, the official prevalence is estimated at just below 1% of the adult (15–49 years) population, based on data from female antenatal clinic (ANC) attendees.[1] The official National AIDS Control Organization (NACO) estimates suggest that 50 lakh adults are infected. The estimated prevalence is roughly double this figure in the southern States. Andhra Pradesh, Karnataka, Maharashtra and Tamil Nadu, and the north-eastern States of Nagaland and Manipur together comprise over 75% of all infections, even though they have less than 30% of the adult population.

Surveillance is central to HIV-1 control programmes. However, surveillance in general and high-risk populations has been limited to date. This report provides a systematic overview of several key sources of data, including all ANC data from 1998 to 2003, the national behavioural surveillance surveys, and published literature on risk factors for HIV-1. New mathematical modelling of the growth of HIV-1 for the next two decades is also provided.

These interim analyses will be followed by more complete analyses of all the data later in 2005. Specifically, primary data from ANC and sexually transmitted infection (STI) surveillance in 2004, and the primary data from behavioural surveillance have not yet been made available for analyses.

Objectives

- Study the current levels and recent trends (1998–2003) of HIV-1 in general populations based on ANCs.
- Describe what is known about the determinants of HIV-1 infection in general populations based on a meta-analysis of the literature and behavioural surveillance surveys.
- Provide projection data for HIV-1 in various risk groups based on mathematical models.
- Provide estimates of reduction in growth of HIV-1 from various prevention and treatment interventions.

Definition and sources of data

Definition of transmission dynamics

Transmission dynamics reveal how HIV-1 spreads in the population. We provide a brief review of transmission dynamics, as understanding this is central to the design of control programmes, choice of interventions and monitoring changes in transmission.

Sexual behaviour and HIV-1 infection are not evenly distributed in the population. Core groups of highly sexually active people drive the rate of transmission in HIV-1 epidemics. The basic reproductive rate, R_0, is the average number of infectious contacts by one infected individual. An infectious contact is a person who would transmit the infection if his/her partner is uninfected. For an epidemic to occur, each infected individual must on an average make infectious contacts with more than one individual (R_0 must exceed 1). The R_0 must be reduced to lower the prevalence of the infection, and brought below 1 to eradicate it from a population.[2]

R_0 is the product of three factors:

- transmissibility (β),
- rate of partner change (C), and
- duration of infection (D).

Each of these factors in turn depends on physiological and sociological events. The transmissibility, β, varies with the number of sexual acts per individual as well as the probability of infection. The rate of partner change, C, is dependent on the average number of unprotected partners and how much variation there is from the average.[2]

Thus, interventions can be directed at any aspect of these parameters.

1. Interventions *directed at* β enhance resistance to infection or decrease susceptibility. Barrier methods such as

*School of Public Health, Department of Community Medicine, Postgraduate Institute of Medical Education and Research, Chandigarh; e-mail: rajeshkum@sancharnet.in

†Centre for Global Health Research, St Michael's Hospital Toronto, Canada; e-mail: prabhat.jha@utoronto.ca

condoms, treatment of co-existent STIs, antiretrovirals that decrease transmission, circumcision and HIV-1 vaccines would reduce β.
2. Interventions *directed at C* aim to alter sexual behaviour, including decreasing the rate of partner change, decreasing concurrency (more than one partner at once), lengthening the time gap between serial partners, increasing the age of initiation of sexual activity, or decreasing high-risk behaviour.
3. Interventions *directed at D* aim to reduce the period of infectivity by the use of antimicrobials and antiretrovirals or via contact tracing or partner notification.

For HIV-1, the contribution of each individual to the value of R_0 is not proportional to his/her number of (unprotected) sex partners but to the approximate square of that number.[2] The average rate in the mean population is not high enough to sustain HIV-1 epidemics. However, C can be high enough in vulnerable groups (i.e. $C>1/\beta D$) to increase the epidemic above an R_0 of 1. Historically, there is little doubt that 'core transmitters' are central to the epidemiology of any STI including HIV-1.[3] This is not always well understood, and occasions much debate about the importance of such vulnerable groups, especially at high levels of the epidemic.[4,5]

Sources of data

Antenatal clinics and sexually transmitted infection sentinel sites

Data here refer to individual-level data from 319,097 visits of women to 266 public ANCs and 130,233 visits of men and women to public STI clinics from all 36 States and Union Territories in India from 1998 to 2003.

For 12 consecutive weeks, twice a year, unlinked, anonymous testing is done among 400 ANC and 250 STI clinic attendees from each ANC or STI clinic. HIV-1 status is measured in ANC and STI clinic attendees, following the World Health Organization (WHO) protocol for confirming HIV-1 status in a developing country: two positive HIV-1/2 enzyme immunoassays results confirm HIV-1/2 seroprevalence, while two negative tests confirm negative HIV-1/2 status.[1]

ANC-based HIV-1 data are used to monitor trends in HIV-1 prevalence over time among general populations. ANC attendees provide a large annual sampling frame, the characteristics of which stay relatively constant.[6–8] ANC attendees are assumed to be representative of the general population. However, ANC-based HIV-1 surveillance has been questionable in estimating prevalence in the general population as both over- and underestimation have been reported, when compared to population-based surveys.[9,10]

The major weakness in the ANC data is, however, the poor coverage. The number of sites has increased from 36 in 1998, 63 in 1999, 107 in 2000, 167 in 2001, 191 in 2002 to 266 in 2003. Only half the sites have been open for more than three years, making projections of trends unreliable before 2000/2001.

Changes in HIV-1 prevalence in the general population are approximated by trends in HIV-1 seroprevalence among ANC attendees aged 15–49 years. HIV prevalence among 15–24-year-old women attending ANCs has been selected by UNAIDS and WHO as a key indicator for the monitoring of HIV-1 prevention programmes.[11,12] While ANC data are valuable, several issues concerning selection biases for ANC data have been identified and must be taken into consideration when using them to estimate HIV-1 prevalence or incidence in the general population.[13–18]

ANCs provide care for expectant women. Ideal ANC care includes three or more visits—with at least one in the first trimester, two or more tetanus toxoid injections, and iron and folic acid supplements for at least three or more months. Women attending ANCs are supposed to receive a range of medical tests and counselling.

The population attending ANCs in India is diverse and thought to be, by and large, representative of the general population. The mean and median age of the attendees is 23 years. Public ANC coverage, defined as women having ever used a public ANC or been visited by a health worker prior to a birth, is approximately 65% nationally.[19] Women are less likely to visit an ANC if they are older, have high parity, are from scheduled tribes, illiterate, or poor.[19]

STI clinics provide care for persons with symptoms of STI or who are concerned about possible exposure. Persons attending STI clinics receive a physical examination for genital ulcerative/non-ulcerative disease and a Venereal Disease Research Laboratory (VDRL) slide test. STI clinic attendees are not representative of the general population. The mean and median age of STI clinic attendees during this time period is 28 years.

All results for ANC and STI populations are age-standardized to the 2001 Census.

Behavioural surveillance in general populations

NACO carried out two Behavioural Surveillance Surveys (BSS) in 2001 and 2002. One of the surveys assessed the behaviour of people from the general public. The general population BSS surveyed a total of 84,478 people, 42,125 of whom (49.9%) were residing in urban areas while 42,263 (50.01%) came from rural areas. Among the interviewed respondents, 42,631 were females (50.5%) while 41,847 were males (49.5%).[20] The median age of the respondents was 29 years for females and 30 years for males. The general population BSS used 22 sampling units from 35 States and Union Territories. Characteristics of those who did not agree to participate are not provided.

NACO also conducted BSS for high-risk people among 5572 commercial sex workers (CSWs) and 5648 of their clients. The median age of CSWs in the study was 27 years with a range of 11–49 years. The median age of clients of CSWs was 27 years with a range of 15–49 years. The high-

risk study was conducted in 21 sampling units in 32 States and Union Territories of the country. The study was not undertaken in areas of the country where the number of sex workers was considered to be insignificant. For various reasons, including those of study quality, the BSS for high-risk populations are not reported further here, but will be part of a forthcoming report examining all BSS among high-risk groups (Salil *et al.*, unpublished).

Review and meta-analysis of studies on risk factors for HIV-1 infection

We conducted a systematic review of existing epidemiological studies on risk factors for HIV-1 infection (Chen *et al.*, in preparation). The literature included in this study consisted of journal articles as well as reports and conference abstracts. We also contacted key control officers and non-governmental organizations (NGOs) to glean further information on risk factors. From 43 studies identified, we included 7 studies from 3 south Indian States with a total of 4212 adults as meeting the search criteria (having HIV-1 negative and HIV-1 positive populations, and also measured risk factors). Most studies drew their populations from STI clinics. We analysed the following risk factors: risky behaviour (paid sex, no condom use and history of STI), genital ulcer disease (GUD), lack of male circumcision and alcohol use. Standard statistical methods for analyses, chiefly pooled Mantel–Haenzel estimates, apply.

Dynamic compartment modelling for growth of HIV-1 and impact of interventions

We created a discrete, compartmental model incorporating high- and low-risk groups for HIV transmission (Weiss *et al.*, in preparation). High-risk groups included sex workers and men who often or sometimes visit them.

Low-risk groups comprised men who never visit sex workers and their wives. HIV spreads between these risk groups due to mixing; low-risk individuals can become high-risk by engaging in risky behaviour themselves (e.g. by becoming a sex worker), a high-risk individual can become low-risk as well (e.g. trucker chooses another profession). Different physiological and sociological characteristics of men and women are included in the model: condom use and efficacy, the presence of other STIs, the number of sexual contacts, and population growth with sex selection to favour male children.

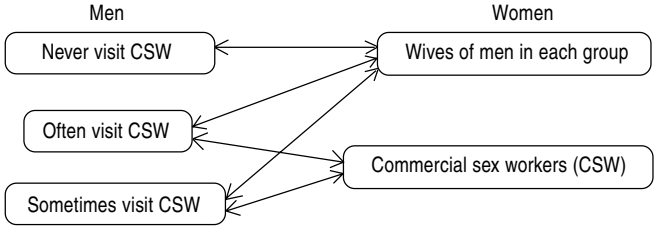

Fig. 1 Centre for Global Health Research (CGHR) mathematical model of HIV-1 spread

The data used for the dynamic compartment model draw on a careful review of all national data on HIV-1 transmission parameters, including various surveys (BSS), mapping studies, and other literature. They use conservative assumptions throughout, focused on modest HIV-1 growth, and have internal consistency checks on mathematical assumptions. The studies also included comparison to existing results, and a sensitivity analyses.

The equation to describe transmission is given in Fig. 2. Here, β is the probability that individual y, in risk group r, infects his/her partner x. The transmissibility, h, depends on the sex of the uninfected individual. The number of contacts per year for individual x is n. Both ulcerative (subscript 1, 3) and non-ulcerative (subscript 2, 4) STIs were considered in the model; STIs increase both the rate of transmission and acquisition of HIV. Above, w_x is the proportion of a given STI in the subgroup x, and m is the multiplication factor for a given STI. The transmission of HIV also depends on condom efficacy, E, and the frequency, f, of condom use between y and x. The proportion of partnering that occurs between individuals in group x and group y (with risk level r) is P_{yr}. The population of infected individuals in risk group yr is y_{ir}. When an uninfected individual becomes infected, through sexual contact with an infected individual, they move to the infected compartment with the same risk state and sex as before infection. Individuals can also move between risk levels.

Results of the routine ANC and STI surveillance from 1998 to 2003

Trends and variation of HIV-1 prevalence among ANC attendees

These were studied among 320,000 women attending ANCs from 1998–2003. Table 1 provides the overall results from 1998 to 2003 in the national ANC results. This shows steady but slow increases from 1998. The 2002 ANC HIV-1 prevalence is notably higher but this may reflect the rapid expansion of new sites in that year.

Figures 3a and 3b provide the ANC prevalence trends by high-prevalence States (Andhra Pradesh [AP], Karnataka, Maharashtra and Tamil Nadu [TN]) and the north-eastern

$$\beta_{yr\to x} = h_{yr\to x} \cdot n_x \cdot (w_x + w_{x1} \cdot m_1 + w_{x2} \cdot m_2) \cdot \left[(w_y + w_{yr1} \cdot m_3 + w_{yr2} \cdot m_4) \cdot (1 - E \cdot f_{yr\to x}) \cdot P_{yr} \cdot \frac{y_{ir}}{y_r} \right]$$

Fig. 2 Mathematical equation to describe the transmission of HIV-1

Table 1. Trends in HIV-1 prevalence among ANC attendees from 1998 to 2003

Year	Number positive/number tested	Prevalence %*	95% CI
1998	106/12,610	0.67	0.49–0.84
1999	205/24,241	0.68	0.48–0.89
2000	376/40,414	0.76	0.60–0.93
2001	492/64,568	0.75	0.60–0.90
2002	648/73,544	1.05	0.66–1.44
2003	897/103,452	0.80	0.69–0.91
All	2724/318,829		

ANC: antenatal clinic
*Age-standardized to the Indian 2001 Census

States (Manipur and Nagaland) and the rest of India, as well as within the high-prevalence States.

Marked variability in levels is seen from year to year, reflecting both the expansion of new ANC sites, as well as the small number of positives that need to occur to generate such variations. However, it is worth noting that levels in the high-prevalence States and the north-east are about 4–5-fold higher than those for the rest of India.[1] The important caveat is that the coverage in north India is limited to certain metropolitan cities. Figure 3b also suggests that, in contrast to much more marked variability (presumably reflecting an early, labile stage of the epidemic in Maharashtra, Karnataka and AP), there are lower levels of infection in TN, including some declines over the past few years.

Figures 4a, b and c provide the variation in HIV-1 prevalence in ANCs by education level, migration status and residence. There is a clear inverse association, with lower education levels having a 2–6-fold higher prevalence. The modest upward trend is seen most clearly in those with secondary or graduate education, narrowing the gap between education groups over time. There is little variation between migrant and non-migrant populations or by rural/urban residence.

Figure 5 shows the State-level extrapolations of prevalence in ANCs. This is the standard method used by NACO and others to create State-level estimates. As noted above, the geographical coverage of ANCs even in the high-prevalence States has been limited. Moreover, there is marked variation within a particular State. A more precise way of examining the ANC data is by 'focal-hot spots', as shown on the right side of Fig. 5. This implies that HIV-1 'hot spots' are localized to the following areas:

- The Mumbai–Karnataka corridor comprising about 6–7 districts
- Nagpur area of Maharashtra
- Nammakkal district of Tamil Nadu

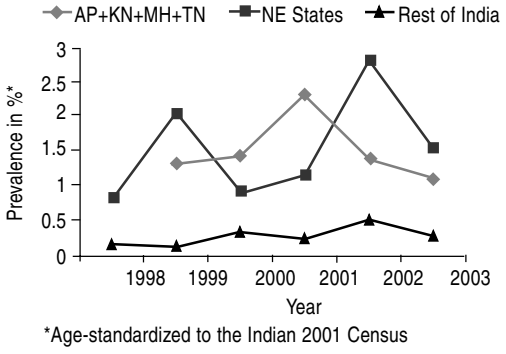

Fig. 3a Variation in HIV-1 prevalence in ANCs for major regions

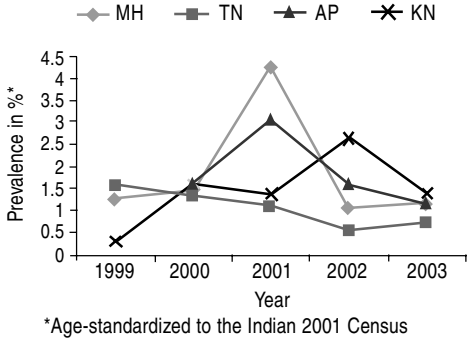

Fig. 3b Variation in HIV-1 prevalence in ANCs for selected States

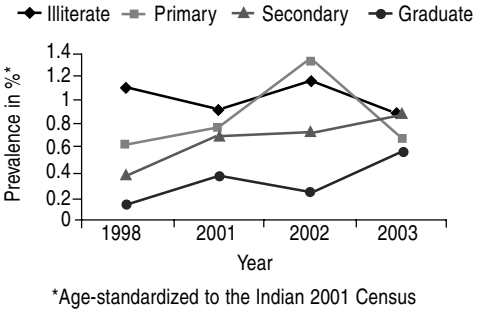

Fig. 4a Variation in HIV-1 prevalence in ANCs by education

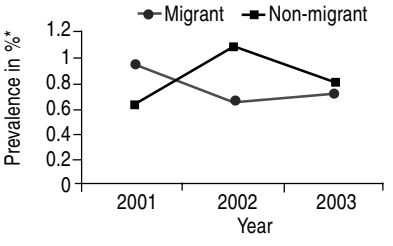

Fig. 4b Variation in HIV-1 prevalence in ANCs by migration status

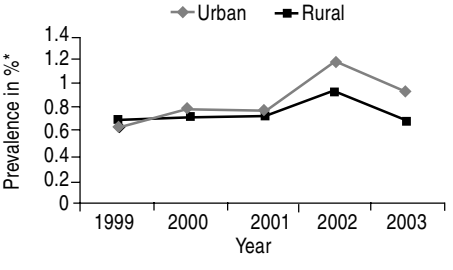

Fig. 4c Variation in HIV-1 prevalence in ANCs by residence

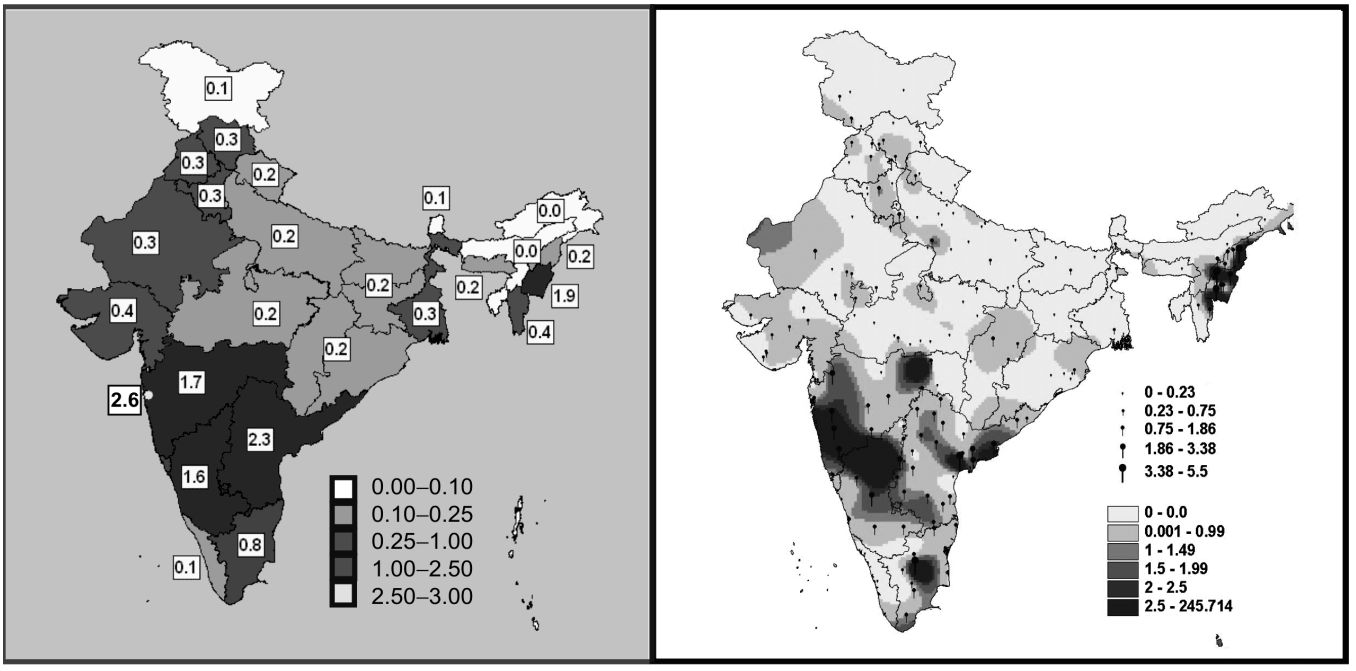

Fig. 5 State-level and focal area HIV-1 prevalence, 2000

Source: Williams *et al.* 2005[38]

- Eastern AP districts, specifically East and West Godavari
- Specific districts of the north-eastarn States of Manipur and Nagaland

The reasons for these particular 'hot spots' are not well understood, and may be a combination of migration patterns. For example, the 1991–2001 Census data trends suggest that a few districts in Karnataka have registered a growth rate lower than that seen for the State overall. The lower growth rate in some districts—Belgaum, Bellary, Bijapur and Raichur, for example—is presumably due to out-migration. (More detailed data are forthcoming from a re-analyses of the Census and Sample Registration System [SRS] data—Sethi *et al.*, forthcoming.) Similarly, some economically prosperous districts in Maharashtra show a higher population growth rate, perhaps suggesting increased in-migration. However, there might well be other factors accounting for such differences, including biological factors, variation in sexual networks in these areas, or better detection (or underdetection in some areas).

Finally, selection biases on who attends ANCs has been voiced as a possible concern, which means that the results of ANCs are unreliable.[18] However, the selection of additional sites has been based largely on the volume of ANC attendees, and as such should not introduce specific biases (as would geographical area-based selection). Moreover, our analyses of HIV-1 negative populations suggest there is little variation in distribution over 1998–2003 of key socioeconomic variables. Overall, about 10% were 15–19 years of age, 45% 20–24 years and 30% 25–29 years. About 30% were illiterate, 25% had primary education and 40% had secondary or higher education. About half were urban and half were rural, and 15% migrant and 85% non-migrant.

Trends and variation of HIV-1 prevalence among STI clinic attendees

These were estimated from among 130,000 adults attending STI clinics from 1998 to 2003. Table 2 provides the overall results from 1998 to 2003 of national STI clinics. The number of sites has increased from 31 in 1998, 52 in 1999, 94 in 2000, 127 in 2001, 159 in 2002 and 163 in 2003. Only 29 sites have been open for more than three years, making projections of trends unreliable before 2000/2001. The overall trends suggest no discernable increase in STI prevalence overall.

Figures 6a and 6b provide the STI HIV-1 prevalence trends by high-prevalence States, the north-eastern States and the rest of India, as well as within the high-prevalenc States. HIV-1 prevalence among STI attendees are almost 6–8-fold higher in the high-prevalence States, and about

Table 2. Trends in HIV-1 prevalence among STI clinic attendees from 1998 to 2003

Year	Number positive/number tested	Prevalence %*	95% CI
1998	329/5,439	5.60	4.94–6.26
1999	654/10,364	5.85	5.37–6.33
2000	1203/17,203	6.55	6.15–6.95
2001	1495/27,044	5.18	4.90–5.47
2002	1838/33,734	5.02	4.77–5.27
2003	2033/35,739	5.22	4.97–5.47
All	7552/129,523		

STI: sexually transmitted infection

*Age-standardized to the Indian 2001 Census

50% higher than in the north-eastern States. Among individual States, it is notable that levels of HIV-1 are much higher in AP than in other States.

As among the ANC populations, it was seen that lower education groups attending STI clinics have a higher HIV-1 prevalence with little change over time, and little differences by residence or migration status (data not shown).

Determinants of HIV-1 infection: Available literature

We analysed the following risk factors: risky behaviour (paid sex, no condom use and history of STI), GUD, lack of male circumcision, and alcohol use. Paid sex contacts were associated with an odds ratio (OR) of 4.7 (95% confidence interval [CI] 2.7–8.2). The OR for presence of GUD was 3.6 (CI 2.5–5.0), and that for lack of male circumcision was 2.1 (CI 1.3–3.5).

The potential for a reduction in the outcome of disease if a risk factor is removed is known as the attributable fraction. Attributable fractions in these populations for paid sex, history of and/or existing genital ulcers, lack of male circumcision, and alcohol use were 71%, 48%, 47%, 25%, respectively. Note that because there are several ways to avoid infection, the individual attributable fractions can add up to more than 100%. In addition, these attributable fractions are not population-based because of the selected nature of the population. The prevalence of each of these variables in the HIV-1 negative group was quite high, being 66%, 39%, 80%, 37% for paid sex contacts, history of and/or existing genital ulcers, lack of male circumcision, and alcohol use, respectively. This suggests that the control groups in these sites are also vulnerable populations, and that the true contribution of these risk factors may be much more substantial than shown in the meta-analysis here. Figure 7 summarizes these results.

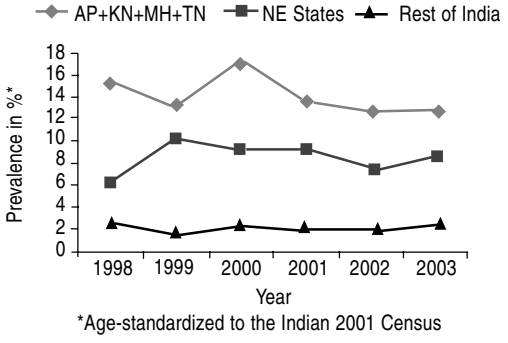

Fig. 6a Variation in STI clinic HIV-1 prevalence for major regions

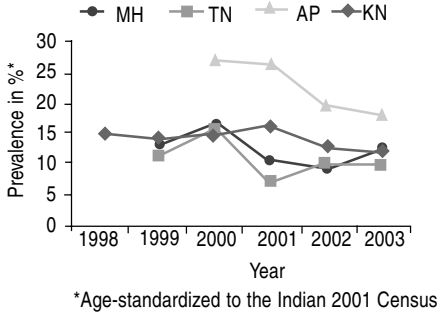

Fig. 6b Variation in STI clinic HIV-1 prevalence for selected States

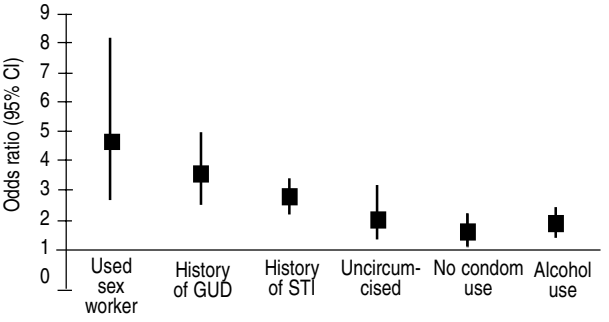

Fig. 7 Meta-analysis of risk factors for HIV-1 among high-risk groups

GUD: genital ulcer disease; STI: sexually transmitted infection
Source: Chen *et al.* (in preparation)

Indirect evidence that differences in male sexual behaviour, most commonly from use of sex work, can be found in the national BSS. Data from this survey is presented in Table 3. It indicates the percentage of 85,000 adults aged 15–49 years who report having a non-regular partner in the past 12 months by region. Overall, the high-prevalence States have markedly greater proportions reporting non-regular partners. This is particularly evident among males.

The major differences are even more evident when the numbers of reported sex partners are stratified by gender and region (Figs 8a and 8b). A much greater proportion of males in the high-prevalence States report having had 2 or more partners as compared to either the north-eastern States or the rest of India. Among females, differences between regions are much less evident. These findings are compatible with the idea that variation in patterns of sexual networks, particularly the use of sex work by males, are a major factor affecting State-level variation in HIV prevalence (especially in the south). Moreover, the wider dispersion of numbers of sexual partners among males (and, to a lesser extent, among females) in the high-prevalence States suggests that HIV-1 would be more likely to spread rapidly in these States (given that growth of HIV-1 is a function of the dispersion of sexual contacts).[2]

There are no direct estimates of what proportion of infections are due to sex work. Indirect estimates, based on assumptions derived from our previous work (of condom use, number of contacts, infectivity and overall HIV-1

Table 3. Prevalence of self-reported non-regular partner in the past 12 months, by gender and region, 2001

Gender	AP+KN+MH+TN	NE States	Rest of India
Male	12.9	2.6	7.9
Female	4.4	0.5	1.1

Source: Salil *et al.* (forthcoming)

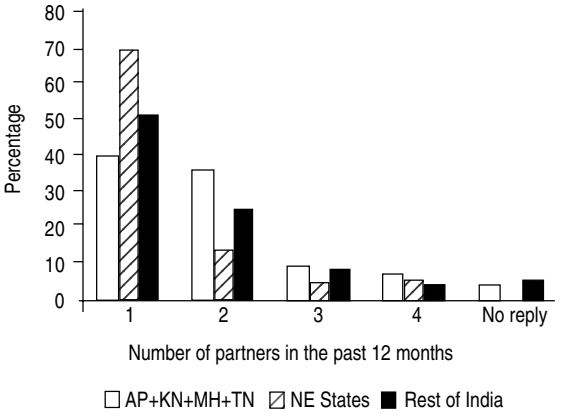

Fig. 8a Number of reported sexual partners in the past year, India, 2001 by region—Males

Source: Salil *et al.* (forthcoming)

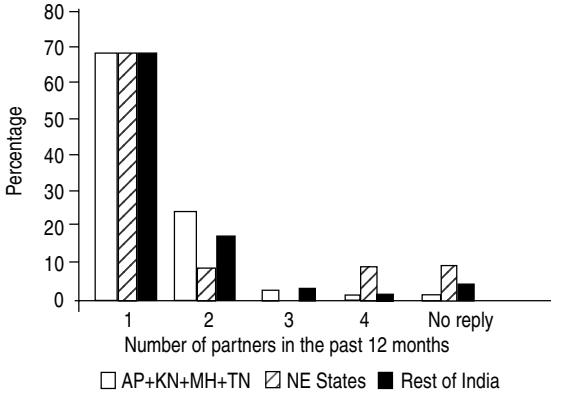

Fig. 8b Number of reported sexual partners in the past year, India, 2001 by region—Females

Source: Salil *et al.* (forthcoming)

infections) are presented in Table 4. This analysis also supports the hypothesis that most new infections in the high-prevalence States of the south are due to first- or second-generation spread through female sex workers and their male clients.

Future growth of HIV-1 infection and potential impact of interventions

Future growth of HIV-1 in India is, by its nature, difficult to predict. Various projection models have been done[21,22]

Table 4. Indirect estimates of the proportion of new infections due to sex work in the high-prevalence States

Prevalence of HIV-1 in female sex workers (%)	No. of female sex workers (in thousand)		Percentage of all infections due to female sex work	
	Low	High	Low	High
40	97	116	63	75
50	121	145	79	94
60	146	174	95	99

Source: Jha *et al.* (unpublished)

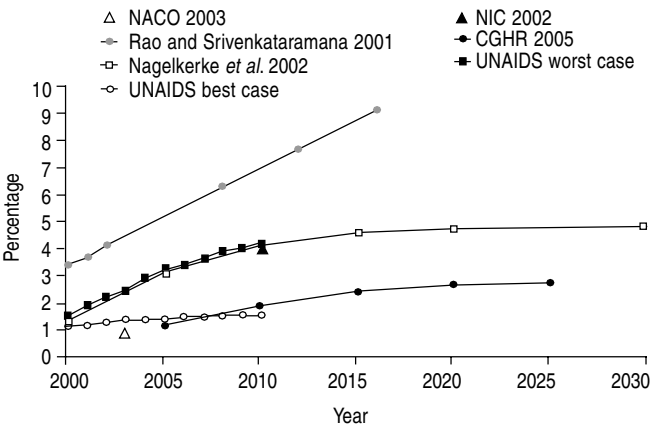

Fig. 9 Projections of HIV among adults in India. (NIC 2002,[21] Nagelkerke *et al.*[22] Rao and Srivenkataramana,[35] UNAIDS best-case and worst-case scenarios,[36] CGHR 2005[37])

and these are summarized in Fig. 9. The extremely rapid growth predicted some years ago does not appear to be occurring in India, based on the observed trends in ANC populations (*see below*).

A reasonable project model might involve the comparison of two scenarios: a worst-case and a best-case scenario. The worst-case scenario produced by UNAIDS in 1999 suggested growth to about 4% adult prevalence by 2010. More detailed work by Nagelkerke *et al.*[22] suggested this rate of increase would continue to about a 5% equilibrium prevalence by 2020.

The Centre for Global Health Research (CGHR) updated modelling uses the Nagelkerke scenarios and updates these using more up-to-date Indian data, and a slightly different projection model. These are less optimistic than the UNAIDS best-case scenario of 1999, and suggest that a little below 3% of the adult Indian population will be HIV-1 positive by 2025.

For the remainder of this report, we will refer to projections using the CGHR conservative model, and assess the impact of interventions with both the Nagelkerke 2002 and CGHR 2005 models.

Even with the modest growth scenario of about 3% equilibrium prevalence, about 500 lakh additional Indians will become HIV-1 infected over the next two decades (Fig. 10). This means that about 150–180 lakh Indians will be HIV-1 positive by 2015, making India the country with the largest absolute burden of HIV-1 infections in the world. Aside from China, no other population is likely to show such large absolute growth.

The CGHR models have not yet been adapted for specific regions of India and, with marked migration across States, such models are less robust than is the overall growth model. It can already be seen that the incidence of new infections will be 40% higher in 2015 than in 2005 (Fig. 11). Women have about a two-fold higher incidence than men in 2005, due to female sex work, as well as a higher biological susceptibility of high- and low-risk women to HIV-1

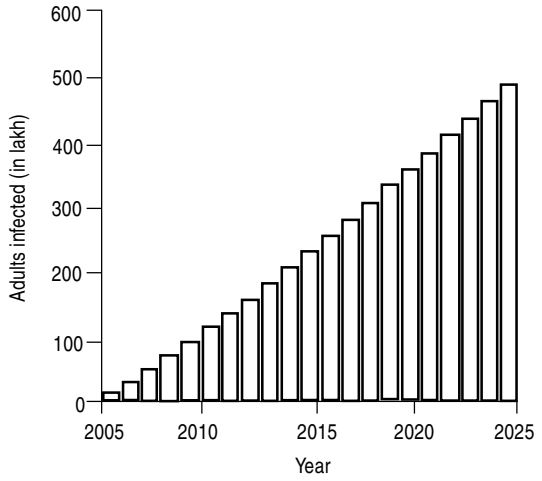

Fig. 10 Cumulative HIV-1 infections from 2005 to 2025 in India (in lakh)

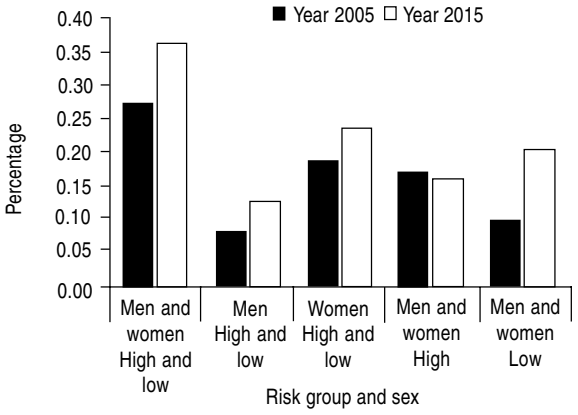

Fig. 11 HIV-1 incidence by gender and risk group in India, 2005 and 2015

infection.[23] The higher incidence among women is likely to be maintained to 2015, and more of the women infected will be low-risk women (chiefly wives of men who visit sex workers sometimes or often).

The CGHR and Nagelkerke models examine interventions to reduce HIV-1 growth (Table 5). These are the following:

Preventive interventions

1. Increases in consistent condom use by female sex workers from an estimated 25% to 40% to 75%.[3] Such reductions have been noted in numerous studies and most notably in places such as Sonagachi, Kolkata; Kibari and Pumwani, Kenya, Thailand and Cambodia.[24–28] The key intervention is peer-based outreach programmes (usually led by NGOs or CBOs) that reach female sex workers (FSWs), provide education, condoms, negotiating skills and access to basic health services. No specific new technologies are required. Sex workers are identified using participatory mapping efforts.
2. Reductions in HIV-1 transmission by about 30% due to community-based STI management using syndromic management and existing antibiotics. There continues to be controversy about the role of STI treatment in HIV-1 transmission, but a careful examination of all evidence to date suggests that STI treatment, especially for populations where HIV-1 is still dependent on sex work-based transmission, is effective.[29] We have used a modest 30% reduction in transmission parameters for this intervention.
3. Use of antiretrovirals to prevent mother-to-child HIV-1 transmission results in a transmission reduction of at least 50%. This includes provision of breast milk substitutes for transmission after delivery.
4. A 'Uganda' type intervention which results from mass change in behaviour of general populations leading to a 40% reduction in the uptake rate of commercial sex by 15-year-olds (both males paying for sex and females entering paid sex). Such an intervention is difficult, however, to reproduce outside of Uganda. In that setting, there was such a high level of infection (about one-fifth of adults), and deaths were so commonly reported that mass behaviour change followed.[30] Indirect analyses suggest that this change in behaviour was due to informal communication networks rather than the result of any specific information campaign. Nonetheless, to outline the potential impact of such an intervention, we have included this.
5. The hypothetical scenario of no commercial sex whatsoever. This is akin to the 'AB' or abstinence and be faithful components of the 'ABC' strategy proposed by the US administration as a possibly effective strategy. There is no clear way to tell if such an intervention is possible, but it is included nonetheless.
6. Hypothetical HIV-1 vaccines. For these, we assume 50% vaccine efficacy—that the vaccine reduces lifetime trans-

Table 5. New HIV-1 infections that can be avoided in India with various interventions over the next two or three decades

Interventions	Nagelkerke *et al.*[22] 2001/02 (%)	CGHR 2005[37] (%)
Preventive interventions		
75% consistent condom use by FSW	–83	–38
30% reduction in the transmission of STIs	–48	–39
50% reduction in mother-to-child transmission	–6	—
40% reduction in commercial sex work by 15-year-olds entering the adult population	—	–39
Hypothetical: no commercial sex work	—	–90
Hypothetical HIV-1 vaccine with 50% effectiveness, 95% coverage of		
General population with no adverse behaviour change	–57	—
FSW with no adverse behaviour change	–61	—
General population with full adverse behaviour change	+13	—
FSW with full adverse behaviour change	+27	—
Treatment: Antiretrovirals with 50% coverage in the general population and no adverse behaviour change	–19	

FSW: female sex worker; STI: sexually transmitted infection; CGHR: Centre for Global Health Research

mission by 50% among susceptible populations (this is not the same as 100% efficacy among half of all people vaccinated). The 50% efficacy estimate is arbitrary, but consistent with recent discussions of a feasible vaccine.[31] We assume vaccines can reach either 95% of the sexually active adult population, or 95% of sex workers, both within 7 years from start-up. With these, two other scenarios follow. The first is that there is no change in the proportion of commercial sex workers that uses condoms (that is, no 'adverse behaviour change'). The second is that those immunized stop using condoms during commercial sex, believing that they are protected from infection. There is already existing evidence of such 'treatment optimism' from the antiretroviral literature in western and developing populations,[32] and condom use among men who premedicate with antibiotics prior to sex work is much lower than those who do not take antibiotics.[33] Thus, adverse behaviour change is not an implausible scenario.

7. Antiretrovirals used among all eligible AIDS patients (i.e. late-stage ARV treatment) covering some 50% of the adult population. Here too, we assume that those who get treated receive additional advice and counselling and do not have adverse behaviour change.

The summary of the results for these interventions, using the relative reductions in new infections between 2000 and 2033 (for the Nagelkerke model) or 2005–2025 (for the CHGR model) are shown in Table 5, and selected interventions for the CGHR model are shown in Fig. 12.

The most effective strategy would be, of course, to have no commercial sex work at all. This would avoid 90% of future HIV-1 infections. However, increases in consistent condom use can achieve between 40% and 80% reduction in HIV-1 growth over the next few decades. Such strategies are widely practicable, and indeed form the basis for the Gates Foundation 'Avahan' programme.

The second most practicable strategy would be to accelerate increase in STI treatment, especially for GUD in India. Bacterial STIs are easily treatable. Trends in bacterial GUD or ulcerative herpes simplex virus (HSV)-2 in India are not well understood. Some African data suggest, for example, that a high prevalence of HSV-2 might account for a substantial proportion of HIV-1 infection.[4]

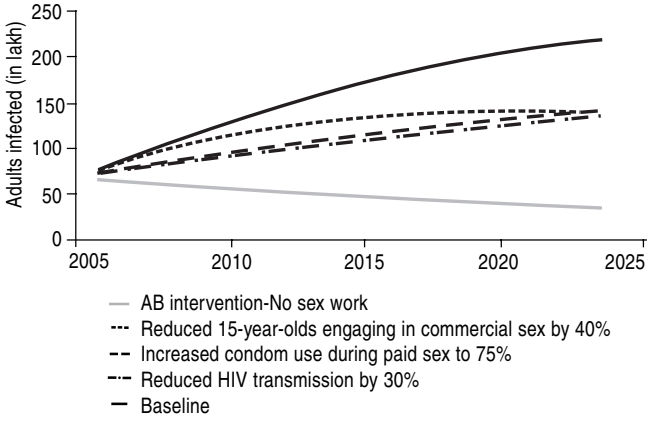

Fig. 12 Comparison of preventive interventions for HIV-1 in India

A 'Uganda' type intervention that changes young people's sexual behaviour is unlikely to be achieved through education alone. Careful reviews have concluded that intensive education programmes tend to raise awareness but not change behaviour among youth.[34] Moreover, such a strategy may be applicable only in advanced AIDS epidemics where many people are dying from AIDS, and this provides an important information signal to the uninfected. Such a strategy would be as effective as an STI strategy in our model.

Antiretrovirals are effective at reducing mother-to-child transmission, but the overall reduction in HIV-1 levels is only modest as the infected children do not pass the infection to others. Antiretrovirals for adult populations have a dramatic short-term impact on mortality, but as resistance appears, their overall effectiveness is limited (about 19% reduction in HIV-1 growth). Moreover, the assumption in the model that there is no adverse behaviour change due to the use of antiretrovirals is an optimistic one.

Similarly, even with a hypothetical HIV-1 vaccine, it can be seen that targeting such a vaccine to female sex work might be as effective as to the general population (in both about 60% of the HIV-1 growth is avoided). However, widespread use of a vaccine leading to behavioural disinhibition and a reduction in male condom use with FSWs could offset the potential benefits of a vaccine and the epidemic could worsen by 13% to 27%. Thus, even with an HIV-1 vaccine programme, strategies to reach CSWs and intervene using peer-based education programmes will be needed.

Figure 12 shows these results in graphic form, using the CGHR model for 2005–2025 and in terms of the annual prevalence of HIV-1 infections.

Further details of the model, including the results of sensitivity analyses will be published shortly.

Implications for HIV-1 and STI control and surveillance

The chief implications of these analyses are as follows:

1. Existing evidence suggests a modestly growing, but highly variable, HIV-1 epidemic in India. Currently, about 50–60 lakh people are infected, with the largest burden in a handful of States: AP, Karnataka and Maharashtra in particular, but also TN, Nagaland and Manipur.
2. Even with modest growth scenarios, perhaps some 500 lakh more Indians will become HIV-1 infected over the next two decades. This growth is very likely to affect specific hot-spot areas at much higher levels than the overall growth would suggest.

3. The chief determinant of the epidemic appears to be transmission from sex work networks, involving use by mobile males of FSWs and secondary infection of regular partners of such males.
4. The most effective and widely practicable strategies to reduce the growth of HIV-1 infection in India are to ensure high rates of consistent condom use among FSWs and their clients, and to increase access to syndromic management of STIs for the general population. These two strategies should be the cornerstone of any response to control AIDS in India.
5. Effective monitoring through routine, robust, reliable, low-cost and long-term epidemiological studies, and surveillance of risk behaviours for HIV-1 and STI are required in India. We endorse here the recommendations of a group of epidemiological experts (Kumar *et al.* forthcoming) which called for greatly expanded quality and coverage of routine ANC and STI surveillance, carefully designed general population studies, and specific integrated biological and behavioural surveys (*see* Appendix 1 for the full list of recommendations).

References

1. National AIDS Control Organization (NACO). *Annual Report 2002–2003, 2003–2004*. New Delhi: Ministry of Health and Family Welfare, Government of India; 2004:1–95.
2. May RM, Anderson RM. Transmission dynamics of HIV infection. *Nature* 1987;**326:**137–42.
3. Jha P, Nagelkerke JD, Ngugi EN, Prasada Rao JV, Willbond B, Moses S, *et al*. Public health. Reducing HIV transmission in developing countries. *Science* 2001;**292:**224–5.
4. Corbett EL, Steketee RW, ter Kuile FO, Latif AS, Kamali A, Hayes RJ. HIV-1/AIDS and the control of other infectious diseases in Africa. *Lancet* 2002;**359:**2177–87. *Erratum in Lancet* 2002;**360:**1178.
5. Munguti K, Grosskurth H, Newell J, Senkoro K, Mosha F, Todd J, *et al.* Patterns of sexual behaviour in a rural population in north-western Tanzania. *Social Science and Medicine* 1997;**44:**1553–61.
6. Boerma JT, Ghys PD, Walker N. Estimates of HIV-1 prevalence from national population-based surveys as a new gold standard. *Lancet* 2003;**362:**1929–31.
7. Zaba B, Boerma T, White R. Monitoring the AIDS epidemic using HIV prevalence data among young women attending antenatal clinics: Prospects and problems. *AIDS* 2000;**14:**1633–45.
8. Zaba BW, Carpenter LM, Boerma JT, Gregson S, Nakiyingi J, Urassa M. Adjusting ante-natal clinic data for improved estimates of HIV prevalence among women in sub-Saharan Africa. *AIDS* 2000;**14:**2741–50.
9. Fylkesnes K, Ndhlovu Z, Kasumba K, Mubanga MR, Sichone M. Studying dynamics of the HIV epidemic: Population-based data compared with sentinel surveillance in Zambia. *AIDS* 1998; **12:**1227–34.
10. Thomas K, Thyagarajan SP, Jeyaseelan L, Varghese JC, Krishnamurthy P, Bai L, *et al*. Community prevalence of sexually transmitted diseases and human immunodeficiency virus infection in Tamil Nadu, India: A probability proportional to size cluster survey. *National Medical Journal of India* 2002;**15:**135–40.
11. UNAIDS. National AIDS Programmes: A guide for monitoring and evaluation. Geneva: UNAIDS; 1999.
12. World Health Organization (WHO). *Evaluation of a National AIDS Programme. A methods package. 1. Prevention of HIV infection*. Geneva, Switzerland: WHO; 1994.
13. Campbell T, Bernhardt S. Factors that contribute to women declining antenatal HIV testing. *Health Care for Women International* 2003;**24:**544–51.
14. Changalucha J, Grosskurth H, Mwita W, Todd J, Ross D, Mayaud P, *et al.* Comparison of HIV prevalences in community-based and antenatal clinic surveys in rural Mwanza, Tanzania. *AIDS* 2002;**16:**661–5.
15. Glynn JR, Buve A, Carael M, Zaba B. Adjustment of antenatal clinic HIV surveillance data for HIV-associated differences in fertility. *AIDS* 1999;**13:**1598–9.
16. Gregson S, Terceira N, Kakowa M, Mason PR, Anderson RM, Chandiwana SK, *et al.* Study of bias in antenatal clinic HIV-1 surveillance data in a high contraceptive prevalence population in sub-Saharan Africa. *AIDS* 2002;**16:**643–52.
17. Saphonn V, Hor LB, Ly SP, Chhuon S, Saidel T, Detels R. How well do antenatal clinic (ANC) attendees represent the general population? A comparison of HIV prevalence from ANC sentinel surveillance sites with a population-based survey of women aged 15–49 in Cambodia. *International Journal of Epidemiology* 2002;**31:**449–55.
18. Glynn JR, Buve A, Carael M, Musonda RM, Kahindo M, Macauley I, *et al.* Factors influencing the difference in HIV prevalence between antenatal clinic and general population in sub-Saharan Africa. *AIDS* 2001;**15:**1717–25.
19. Roy TK, Arnold F, Kulkarni S, Kishor S, Gupta K, Mishra V, *et al.* (eds). *National Family Health Survey-2*. Mumbai, India: International Institute for Population Sciences; 2002.
20. NACO. *National Baseline General Population Behavioural Surveillance Survey 2001*. New Delhi: National AIDS Control Organization, Ministry of Health and Family Welfare, Government of India; 2001.
21. Gordon DF (ed). *The next wave of HIV/AIDS: Nigeria, Ethiopia, Russia, India, and China.* Intelligence Community Assessment, September. National Intelligence Council; 2002.
22. Nagelkerke NJ, Jha P, de Vlas SJ, Korenromp EL, Moses S, Blanchard JF, *et al.* Modelling HIV/AIDS epidemics in Botswana and India: Impact of interventions to prevent transmission. *Bulletin of the World Health Organization* 2002;**80:**89–96.
23. Gray RH, Wawer MJ, Brookmeyer R, Sewankambo NK, Serwadda D, Wabwire-Mangen F, *et al.* Probability of HIV-1 transmission per coital act in monogamous, heterosexual, HIV-1-discordant couples in Rakai, Uganda. *Lancet* 2001;**357:**1149–53.
24. Jha P, Mills A. Improving health of the global poor. *The Report of Working Group 5 of the Commission on Macroeconomics and Health, 2002*. Geneva and London. London, UK: London School of Hygiene and Tropical Medicine, Commission on Macro-economics and Health; 2002.
25. Kaul R, Kimani J, Nagelkerke NJ, Fonck K, Keli F, MacDonald KS, *et al.* Reduced HIV risk-taking and low HIV incidence after enrollment and risk-reduction counseling in a sexually transmitted disease prevention trial in Nairobi, Kenya. *Journal of Acquired Immune Deficiency Syndrome*: *J AIDS* 2002;**30:**69–72.
26. Nelson KE, Celentano DD, Eiumtrakol S, Hoover DR, Beyrer C, Suprasert S, *et al.* Changes in sexual behavior and a decline in HIV infection among young men in Thailand. *New England Journal of Medicine* 1996;**335:**297–303.
27. Arora P, Cyriac A, Jha P. India's HIV-1 epidemic. *Canadian Medical Association Journal* 2004;**171:**1337–8.
28. WHO, Ministry of Health, Cambodia. *Controlling STI and HIV in Cambodia: The success of condom promotion*. Manila: WHO, WIPRO; 2001.

29. Grosskurth H, Gray R, Hayes R, Mabey D, Wawer M. Control of sexually transmitted diseases for HIV-1 prevention: Understanding the implications of the Mwanza and Rakai trials. *Lancet* 2000;**355:**1981–7.
30. Stoneburner RL, Low-Beer D. Population-level HIV declines and behavioral risk avoidance in Uganda. *Science* 2004;**304:**714–18.
31. World Bank. Consultative meeting on accelerating the development of an HIV/AIDS vaccine for developing countries: Issues and options for the World Bank. New Delhi: World Bank; 1999.
32. Crepaz N, Hart TA, Marks G. Highly active antiretroviral therapy and sexual risk behavior: A meta-analytic review. *Journal of the American Medical Association* 2004;**292:**224–36.
33. Lowndes CM, Alary M, Gnintoungbe CA, Bedard E, Mukenge L, Geraldo N, *et al.* Management of sexually transmitted diseases and HIV prevention in men at high risk: Targeting clients and non-paying sexual partners of female sex workers in Benin. *AIDS* 2000;**14:**2523–34.
34. Jha P, Vaz LM, Plummer FA, Nagelkerke NJ, Willbond B, Ngugi E, *et al. The evidence base for interventions to prevent HIV infection in low and middle-income countries.* Working Group Five Paper no. 2. Geneva: Commission on Macroeconomics and Health; 2002.
35. Rao CN, Srivenkataramana T. Projection of HIV infections in India: An alternative to back-calculation; *Current Science* 2001; **81**:25–34.
36. Jha P. Project Appraisal Document: Second National HIV/AIDS Control Project. World Bank, May 1999.
37. CGHR. Mathematical modeling for HIV/AIDS in India. 2005. Available from URL: www.cghr.org.
38. Williams BG, Granich R, Chauhan LS, Dharmshaktu NS, Dye C. The impact of HIV/AIDS on the control of tuberculosis in India. *Proc Natl Acad Sci USA* 23 June 2005. Available from URL: http://www.pnas.org/cgi/reprint/0501615102v1.

Appendix 1

Improving the monitoring of HIV/AIDS in India in the high-risk and general population
Ad Hoc Review by Epidemiological Experts*

Summary

An expert group of epidemiological experts provides ten specific recommendations on how to improve the monitoring of HIV-1 infection and its determinants among high-risk and low-risk populations in India. Monitoring the evolution of HIV-1 in India is central to establishing if control programmes are working (or not). The major recommendations of this report include:

- Re-analyses of the considerable number of existing behavioural and other data at district or subdistrict levels;
- Ensuring that integrated biological and behavioural surveillance is done in a way to ensure that target populations are reliably sampled, and that biases in reporting condom use are recorded;
- A pilot programme of 40 districts for enhanced ANC testing for HIV-1 and STIs among the general population, with quality control, re-sampling and creation of central repositories;
- Ensuring than future population-based behavioural or biological surveys are done with adequate methodological attention so that they provide meaningful data representative of the population and with adequate sample size; and
- Ensuring that the surveillance efforts are integrated into overall capacity building and sustainable monitoring strategies to monitor what is clearly a very heterogeneous epidemic.

Background

Several epidemiologists from India and around the world met in Ahmedabad on 6 January 2005 and in Bangalore from 11 to15 January 2005 to review the epidemiology of HIV-1 in India, and the best options to improve the monitoring of incidence, prevalence and mortality from HIV-1 infection. The Bangalore meeting was held concurrently with the Monitoring and Evaluation (M&E) meeting of the Bill and Melinda Gates Foundation (BMGF) Avahan programme.

The Avahan programme is a US$ 200 million five-year control programme focusing on 71 high-prevalence districts

*Dr Rajesh Kumar (Chair), Professor and Head, School of Public Health, Post Graduate Institute of Medical Education and Research, Chandigarh (experience with large-scale population studies on child and adult mortality, reproductive health, and HIV-1 epidemiology); Dr Prabhat Jha (Rapporteur), Canada Research Chair of Health and Development, Centre for Global Health Research, University of Toronto (principal investigator of the RGI–CGHR prospective study of 6 million Indians, and studies of HIV-1 transmission among 45,000 adults in Tamil Nadu, and former Team Leader of the World Bank-financed Second National HIV/AIDS Project); Dr Prakash Bhatia, Professor, Professor of Community Medicine, Osmania Medical College, Hyderabad (Regional Coordinator of NACO surveillance); Dr Lalit Dandona, Director, Centre for Public Health Research, Administrative Staff College of India, Hyderabad (PI of IBBS among 6500 sex workers and 6500 MSM); Dr Neeraj Dhingra, Resident Director, RGI–CGHR Prospective Study, New Delhi; Dr Vendhan Gajalakshmi, Director, ERC, Chennai (PI of prospective study of 500,000 adults in Chennai, retrospective studies of 80,000 deaths, and HIV-1/TB studies, and Co-PI of Tamil Nadu VCTC studies); Dr Prakash Gupta, Director, Healis-Seksaria Institute of Public Health, Mumbai (PI of Mumbai prospective study of 150,000 adults); Dr V. Lakshimi, Professor, Nizam's Institute of Medical Sciences, Hyderabad (Microbiologist in charge of 13,000-population-based biological testing in AP); Dr Prem Mony, Epidemiologist, Institute of Population Health and Clinical Research, St John's National Academy of Health Sciences, Bangalore (experience in population-based research on male sexual behaviour and TB epidemiology); Dr Nico Nagelkerke, Professor, United Emirates University, UAE (mathematical modelling of HIV-1 and biostatistician to the Kenya HIV/AIDS intervention projects); and Dr S. Sreevidya, Epidemiologist, HSIPH (Co-PI of studies on tobacco use among antenatal clinic populations and Mumbai prospective study)

(a combined population of 1450 lakh). The primary target groups will be approximately 300,000 FSWs and their clients. Specific changes in HIV-1 prevalence will be monitored through integrated behavioural and biological surveys (IBBS) among 100,000 people (over years 1, 3 and 5), detailed studies in general populations and special surveys in high-risk populations. Mathematical models will be used to evaluate 'counter-factual' scenarios, and cost-effectiveness of interventions. Much of the IBBS work will be implemented by Indian Council of Medical Research (ICMR) institutions in various States of India, led by the National AIDS Research Institute (NARI).

Concurrently, the National AIDS Control Programme (NACO) has made a decision to focus its communication and NGO-led peer-based education components of the Second National AIDS Control Project (US$ 200 million/ 5 years, expiring 2005) on improving coverage for FSWs and clients. India is also a core country for WHO's '3 by 5' programme for antiretroviral access, with the goal of placing 300,000 people on ARVs by the end of 2005. NACO has also begun to discuss a Third IDA credit with the World Bank for HIV/AIDS control. Importantly, NACO has also set an ambitious goal 'Zero by Seven', assumed to mean there will be no new increases in the HIV-1 infection prevalence rate above the levels of 2006.

Thus, for a variety of stakeholders, there is a fresh need to examine the methods to expand and strengthen the epidemiology of HIV-1 in India to monitor the evolution of what is already clearly a heterogeneous epidemic, to inform the success of control programmes and new interventions (such as the introduction of antiretrovirals), and to identify research questions that are central to new interventions (such as HIV-1 vaccines).

The epidemiological group focused on the following items in its discussion and made specific recommendations on each:

- The existing evidence on HIV-1 growth in India, including variation and the implication for the variation for control programmes;
- Methodological strengths and areas requiring further attention for monitoring HIV-1 among general and high-risk populations;
- Links of the current monitoring efforts to longer-term capacity building for epidemiological studies in India.

Existing evidence on HIV-1 epidemiology in India

The known epidemiology of HIV-1 growth in India will be highlighted in detail in a forthcoming report by the National Commission on Macroeconomics and Health. Salient highlights include the following:

- HIV-1 prevalence is estimated to be about 1% of the adult population (15–49 years), but this is highly variable. The States of Andhra Pradesh, Karnataka, Maharashtra and Tamil Nadu are estimated to contain most of the prevalent infections (but gaps in routine surveillance in north India are substantial).
- Geographical and demographic 'hot spots' have been identified. 'Hot spots' of HIV-1 from sexual transmission are most notable in the Maharashtra/Mumbai–northern Karnataka corridor, in/around the Nagpur area in Maharashtra, coastal districts of Andhra Pradesh, in Namakakkal in Tamil Nadu. 'Hot spots' from injecting drug use are notable in Manipur and Nagaland.
- As of now, the demographics of these hot spots (age, sex and occupational risk-groups at a minimum) have not been described. Moreover, given the migratory nature of the populations at high risk, and gaps in routine surveillance in several States, caution is needed to state that only selected areas are 'hot spots'.
- The overall growth of HIV-1 from 1998 to 2003 has been modest but steady. Future growth is uncertain, and will be informed by the 2004 ANC data.
- Model-based projections of growth of HIV-1 suggest that at a minimum, 500 lakh *new* infections can be expected over the next two decades in India overall. However, the major increase is likely to occur in selected areas of certain States.
- Control of these hot spots remains critical to curbing overall HIV-1 growth in India.
- State-level prevalence averages rely on political/administrative units and are likely meaningless.

Monitoring HIV-1 prevalence and incidence, and risk factors among high-risk populations

High-risk, vulnerable populations such as sex workers (SW) and their clients are central to the growth of HIV-1 in India. Indirect estimates suggest that most new infections in the heterosexual population arise from the male use of FSWs. However, these data are debated, and more direct epidemiological confirmation is needed.

Over the past few years, major surveillance efforts for these populations have focused on STI clinics (about 250 consecutive males and females tested anonymously and in an unlinked fashion twice a year in about 163 sentinel sites at public facilities), mapping of these populations in several districts/States, and on behavioural surveys in several districts/States. Voluntary counselling and testing centres (VCTCs) are available in almost each district in the southern States, and a growing number of northern States. Both these clinics have substantial selection biases from the populations that attend them. However, they can still be useful in tracking information on selected risk factors in self-selected, high-risk populations.

The Avahan programme's planned expansion to include biological samples (blood for HIV-1, HSV-2 and syphilis; urine for *C. trachomatis* and *N. gonorrhoeae*) from a 100,000 strong high-risk population over 5 years (2005,

2007 and 2009) should provide a substantial amount of new and important information on these otherwise hard-to-reach populations. Similarly, Avahan plans to implement BSS (questions only) annually in each of the 71 Avahan districts, which should provide substantial new evidence on such populations.

The group identified the key gaps in this approach:

- Caution in trying to design questions to measure intimate and complex sexual behaviour. Ultimately, many of these behaviours are not observable with even detailed survey instruments. Results from a few participants are not readily applicable to other populations.
- The sample frame designed may not be the one implemented due to the very mobile nature of sex work. The Frontiers project IBBS among 6500 FSWs and 6500 MSM in over a dozen districts of Andhra Pradesh found that actual sampling frames could not always be actually implemented due to changes in mobile populations between mapping/sampling and survey.
- There is too great a complexity in the IBBS, such as including genital examination on a subset of women and too long a questionnaire. The IBBS should retain as its core focus a simple, widely practicable questionnaire that can be reproduced in subsequent years. Specific needs for the mathematical model should not lead to too many questions (some of which are not answerable).
- Only 27 of the 71 Avahan districts are covered under the IBBS, and the selection of these districts is based on programme coverage and other criteria. This risks introducing subtle biases where intervention programmes focus more sharply on the IBBS districts to show good performance/quality.
- The wide variation in sex work practices even within a district site implies that IBBS results may be hard to generalize for that district. For example, the Frontiers project experience showed nearly three-fold differences in condom use among FSWs in the same districts.
- There is likely overreporting of condom use by FSWs in existing surveys. This implies that the IBBS design may not have optimal power to detect changes in condom use. Moreover, as the Avahan programme expands, and community participation increases, more plausible, but lower levels of condom use might be reported. This may lead to the false conclusion that the interventions *lowered* condom use.
- To avoid erroneous conclusions, some simple validation/re-survey methods focusing on condom use need to be introduced throughout the IBBS to understand under- or overreporting. These 'correction factors' cannot be extrapolated safely to other geographical areas or to other specific subpopulations (such as street-based or brothel-based FSWs). Thus, considerable caution will be required in interpreting the results.
- Reported STI levels may be lower than anticipated, so that the IBBS may not have adequate power for detecting changes in STI outcomes of interest for each district. Detailed power calculations using lower STI levels would help evaluate at what level the changes in STI outcomes could be detected with the planned sample size, e.g. for groups of districts or at the State level.
- There is limited coverage of the IBSS or BSS in the northern areas of India, particularly in cities with more than 10 lakh population and, presumably, having large migration levels.

Recommendations

1. An expert group assemble, re-analyse and synthesize all of the existing BSS and IBBS data, mapping studies, and other information using as disaggregated levels as possible (district at a minimum). The expert group should identify which districts/cities that are not covered by existing BSS/mapping or other related surveys require such surveys.

   The re-analyses should also review the age, sex, socioeconomic background of the respondents and compile any available data on non-response so as to better understand who participates in these surveys, and why. The re-analyses should also aim to document the variation in reported condom use by specific sites or types of sex workers.

   NACO and the owners of these primary data should work to make the primary and raw data from these completed surveys (most of which were publicly funded) available to the study team. (Indeed, the full anonymous and unlinked results should be freely available on the website of NACO—akin to the easy access to the raw data files of the NFHS-2.)
2. The selection of the Avahan districts for inclusion in the first IBBS should be done by an independent statistical panel, who can also advise on related options for future surveys (such as rolling sample frame with partial overlap). This is the procedure used for the Registrar General's Sample Registration System and other major surveys.
3. The early pilot results of the Avahan IBBS should be reviewed critically for compliance rates, feasibility and other aspects so as to help simplify the IBBS implementation in the first and second rounds. Avahan should keep open the possibility of simplifying procedures so that more districts with a larger sample size are included in subsequent rounds (especially if the power calculations show that condom use and STI prevalence are not as predicted).
4. HIV-1 incidence testing through 'detuned' methods may well be very useful in monitoring changes between the 2005, 2007 and 2009 surveys. Here again, caution is needed to ensure that the enrolled populations (which will certainly differ over the years) are comparable.
5. Despite considerable selection biases among STI and VCTC attendees, this population has a high HIV-1

positivity rate and is therefore useful to study high-risk populations. Specific, case–control studies among these populations should be designed, focusing on risk factors for HIV-1 infection, including sex work contact, age at first sexual exposure, male circumcision status, STI of various types, consistency of condom use and use of HIV-1 preventive or treatment services. These studies can also help validate if surrogate questions such as time away from home are a useful proxy for unprotected sex contact with a non-regular partner.

Monitoring HIV-1 prevalence, incidence and risk factors among general populations

The ultimate success of control programmes and the mitigation of the social and economic costs of HIV/AIDS can only be determined by prevalence, incidence and mortality among general populations. The chief source of epidemiological information are female ANC attendees, and proposed general population surveys. Each is reviewed in turn.

ANC surveillance

ANC sites have expanded from 38 in 1998 to 266 as of 2003. These rely on semi-annual anonymous, unlinked testing of about 400 consecutive women attending ANC sites at public institutions. Only half of the sites have been open for more than three years. Overall, only about 65% of women used public facilities for antenatal care, but this proportion is much higher in the southern States and Maharashtra, where HIV-1 levels appear higher.

The ANC data have been able to demonstrate, despite weakness shown below, that the HIV-1 epidemic is concentrated in certain areas in States where it is measured (*see* the section on 'Background' above). The ANC data have also been able to provide reasonable, but by no means fully certain, evidence that in some urban settings like Delhi, Chandigarh and Gujarat, HIV-1 levels in the general population have not increased dramatically (staying well below 0.5% for several years).

The biases among women attending ANCs have not been well studied in India. However, preliminary re-analyses by NACO suggest that there was little variation in age, education, residence and migration patterns between 1998 and 2003 among HIV-1 negative women.

Additional analyses are required to confirm if these groups are representative of local levels of the enrolment area's ever-married women (forthcoming analyses). Similarly, the amount of information that is possible to collect (including limited questions on sexual behaviour, risk-taking) is limited.

The chief weaknesses of the ANC data are two: first, they do not capture information on males. Second, they are more useful for detecting trends, and have not been well validated for measurement of HIV-1 incidence or prevalence in the general community. Results from the Tamil Nadu Community Prevalence Survey suggest that ANC prevalence is two-fold lower than the overall State level, but the Bagalkot Community Survey suggests that the two are comparable (*see below* for caveats of general population surveys). Similarly, there are reports of inconsistent quality in testing (including adherence to ELISA testing procedures), sampling frame (including recruiting non-representative populations), and overall supervision.

Because the ANC data represent the best existing and reasonably low-cost method for tracking changes in the general population, these represent the most cost-effective and practicable base from which to expand. Already, ANC has expanded to all districts in the high-prevalence States for 2004.

Recommendations

1. Considerable efforts be made to improve the quality and completeness of ANC data testing from all parts of India. These would include:

- Test in at least one public facility and ideally one private/NGO facility in all districts of India.
- Add focused questions on sexual behaviour (including knowledge of paid sex, husband's time away from home plus wife's time away from home during pregnancy when the husband is alone in the house, religion and male circumcision status, HIV-1 testing, use of antiretrovirals, condom use, access to HIV-1 interventions).
- Add questions on tobacco and alcohol use by husband.
- Add routine measurement for other viral and bacterial STIs, specifically HSV-2, syphilis, and possible *C. trachomatis* antibodies (as a marker of sexual risk).
- Add incidence testing ('detuned') in some areas.
- Create a central long-term biorepository of a random percentage of tested samples for future biological testing (the last three to be done in a phased manner after adequate pilots).

2. A network of academic institutions, working with NARI, NACO and others conduct a pilot quality control study covering some 40 districts (including identified 'hot spot districts' as given in the section on 'Existing evidence on HIV-1 epidemiology in India').

   The pilot would aim to provide training and standardization to ANC staff in proper epidemiological questions, and in specimen collection. The network would introduce innovation such as use of a rolling sample (not simply twice a year), training of ANC staff in interview methods and good quality HIV-1 and STI testing, use of a 10% re-sample, central archiving of all positive samples, a random percentage of negatives for specialized testing (including detuned assays) and quality control. The central samples could be used for focused biological research (for example, on variation in viral and host factors that may explain the remarkable heterogeneity of HIV-1 in India).

3. Based on the pilot, ICMR/NACO consider expansion and quality control of ANC sites as the central pillar of the proposed third World Bank IDA credit.

General population surveys, including the planned NFHS-3

Major completed activities for general population surveys include a National Behavioural Survey in General Population (NBS) covering 85,000 adults in major States of India completed in 2001, a forthcoming National Community Prevalence Study (the results of which are not yet available), and a planned National Family Health Survey-3 (NHFS-3), which would include HIV-1 testing in about 125,000 adults in high-prevalence States.

The group concluded that there were considerable methodological challenges in general population surveys. The central goal of such surveys is to generate prevalence rates and risk profiles representative of the general population, but it is precisely in this area that weaknesses are most notable. Consider the following:

- It is well known that those who refuse to join such surveys are different from those who participate. This may be due to three factors: non-response; non-availability of economically and physically active young males during surveys; and the limited ability to capture migrant populations, hence possible underrepresentation of these in general population surveys. Thus, higher-risk, more mobile females or males may often not be captured in the house listing procedures used. The Male Sexual Behaviour General Population Survey (GPS) in Vellore showed a response rate of 87% and that non-respondents were more likely to be older (35–40 years of age), married, and having white-collar jobs or affluent private businesses. Surrogates of risk-taking (smoking or alcohol use) were not different between responders and non-responders). No other large-scale BSS or IBBS that we reviewed has reported on the characteristics of the non-respondents (including age, sex, marital status, occupation, education, time away from home) and surrogates of risk-taking (such as smoking or alcohol use). The NBS does not provide any table that compares the interviewed population to the Census characteristics.
- The experience from the General Population Survey conducted in the Bagalkot district of Karnataka is telling. The original study design aimed for 7000 adults, based on a census-based house listing. Only about 4600 agreed to participate and, of these, only about 3500 provided blood samples. Thus, with the HIV-1 status available in only about half of the target population, the study cannot be representative.
- Experience from various studies suggests that while behavioural data, or blood or urine samples may be obtained (including from the NHFS-2), obtaining both creates numerous problems with community participation. This implies that any survey which attempts to do both needs to be designed with considerable care, and only after use of extensive pilots. Similarly, while past NFHS surveys have had high participation rates among those selected, the planned introduction of HIV-1 testing in NHFS-3 may well lower overall response rates, and thus make difficult or impossible the comparison of fertility and other indicators to earlier NHFS rounds.

Adequate sample sizes are needed for general population surveys, given that the estimated prevalence of HIV-1 among adults is still quite low (2% as an upper level in most settings). The specific objectives of general population surveys need to be taken into account in sample size estimates. For example, generating a State-level average for HIV-1 prevalence is of limited use, given that it is already evident that HIV-1 epidemics are remarkably heterogeneous within a State (or even a district). Consider the following:

- The NHFS-3 proposal calls for testing some 125,000 adults in high-prevalence States or, on an average, about 6400 men and 6400 women in each of the high-prevalence States. However, this would generate only about 60 positive cases among women and men each, or only 2–3 positives among women in each district for an average State. This number of positives is too small to validate local ANC results.
- Similarly, the design effect is likely underestimated as the marked heterogeneity in HIV-1 levels within a State mean that sampling errors could lead to underestimation, and inability to reproduce the findings in future surveys.
- The absolute number of HIV-1 positive persons is too small to do any meaningful bivariate analyses except at the national level.

The design document for the NHFS-3 is not clear in the efficiency of testing strategies. A very large number of samples (125,000) will need to be tested to generate only a few positive results (some 1000–2500). Methods to increase the use of pooled samples and their relevance to blood spots are not clear. Similarly, the NHFS-3 proposes, quite wrongly, to destroy the samples after testing.

A better sample frame for large HIV-1 prevalence surveys is likely to be the Sample Registration System (SRS). The SRS covers about 10–11 units per district on an average, each with about 1000–1500 people (of which about 40% would be 15–49 years of age). The SRS is representative of the population. Because house lists are known, they can provide meaningful information on the non-response characteristics of those surveyed. Moreover, the new SRS sample frame will be followed to 2014, thus enabling prospective follow-up.

Table A1.1 NHFS-3 sample size estimates for each of the high-prevalence States

Group	Assumed HIV prevalence rate (%)	Assumed design effect*	Proposed minimum sample size	Confidence interval		Absolute number of positives	
				Lower (%)	Upper (%)	Lower bound	Upper bound
Women and men separately	1.50	1.25	6,400	1.1	1.9	70	122
	1.25	1.25	6,400	0.9	1.6	58	102
	0.75	1.25	6,400	0.5	1.0	32	64
Both women and men together	1.50	1.25	12,800	1.2	1.8	154	230
	1.25	1.25	12,800	1.0	1.5	128	192
	0.75	1.25	12,800	0.6	0.9	77	115

*Note that the design effect used is optimistic—it may well be two-fold or higher

Recommendations

1. A NACO/Avahan expert group should review the results of all existing community prevalence studies, including appropriate information on non-respondents, sample size estimates, validation of results of sexual behaviour, and other aspects.
2. The utility of general population surveys of HIV-1 and STIs in conjunction with other biological tests be first evaluated in well-designed pilot studies in about 3–4 States. These pilots would pay attention to the following options:

- Surveys focused only on blood-based household health surveys (comprising blood pressure and other physical measurements, along with a blood spot on filter paper), with limited sexual behavioural questions (but with likely high compliance rates).
- Validation of indirect questions on sexual behaviour (for example, use of surrogate markers such as time away from home for use of sex work).
- Use of biological markers to define sexual behaviour risk (for example, *C. trachomatis* or HSV-2 antibodies correlate with lifetime number of sexual partners in western settings).
- Alternative sampling strategies, such as sampling only men, or sampling men and women in alternative households.
- Careful attention to methods for collection, processing and long-term biorepository of collected samples, including choice of samples (lowering the costs of testing with pooling methods), cheaper indigenous assays and other aspects of laboratory quality control.
- Careful attention to methods for the use of large survey datasets within the SRS to create anonymous, unlinked subsamples for population-based, age, sex, and region or hot spot-specific HIV-1 and STI prevalence.

Monitoring AIDS mortality

Especially due to expanding access to antiretrovirals, studying mortality from AIDS will be required. The forthcoming Registrar General of India (RGI)–Centre for Global Health Research (CGHR) prospective study of 60 lakh Indians, capturing some 150,000 deaths from 2001 to 2003 and an additional 75,000 deaths from 2004 to 2005 should provide robust and direct estimates of AIDS mortality, and the socioeconomic and limited behavioural correlates of such mortality.

Capacity-building for monitoring HIV-1 and sustainability of efforts

The expert group strongly endorsed the view that stand-alone monitoring and evaluation efforts, while required for project monitoring and donor accountability, need to be integrated into an overall nationally led strategy. Such a strategy would build capacity for routine, robust, low-cost, and long-term monitoring of a remarkably heterogeneous HIV-1 epidemic. The expert group believes that the above set of efforts, with close coordination between NACO, ICMR, Avahan and others can be a model for developing countries to build sustainable monitoring systems.

Causal analysis and treatment protocols for sexually transmitted infections, HIV/AIDS and opportunistic infections

NATIONAL AIDS RESEARCH INSTITUTE, PUNE

1. Sexually transmitted infections

Table 1.1 Causes of sexually transmitted diseases (by significance)

	Direct (normally clinical)	Indirect (diet, exercise, alcohol)	Distant (sociopolitical, economic, empowerment, gender, literacy, etc.)
	• Sexual—unprotected sex with casual partners or HIV-infected person • Ignorance about STDs	• Practice of risky sexual behaviour • Alcohol or other substance use • Social stigma associated with risky sexual behaviours, sexually transmitted diseases and 'at-risk' subpopulations	• Migration—single male member migration • Poverty • Natural calamities such as famine and earthquake • Low empowerment as seen among 'at-risk' subpopulations • Social taboo on discussions on sexuality • Low coverage of quality life skills' education to adolescents in and out of school
Interaction with other causes	*(by descending order of proportionate morbidity)*		
STDs	• Enhance the risk of acquisition/transmission of HIV infection by two- to ten-fold • Generally a higher risk of transmission is associated with ulcerative diseases 1. Genital ulcer disease —Herpes genitalis —Chancroid —Syphilis —Lymphogranuloma venereum —Granuloma inguinale 2. Genital discharge syndrome —Trichomoniasis —Chlamydial urethritis —Gonorrhoea 3. Genital warts 4. Reproductive tract infections	• Poor treatment-seeking behaviour including delayed treatment-seeking, incomplete treatment for STDs, low acceptability of STD clinics, etc. • Poor health-seeking behaviour among 'at-risk' subpopulations • Low access to quality risk reduction counselling • Low acceptability/awareness about syndromic management guidelines among health care providers	• Receptive sexual partners—females as well as males (in men having sex with men) • Low coverage of quality targeted interventions among 'at-risk' subpopulations • Poor control over unqualified traditionally accepted practitioners for treatment of STDs • Poor coordination between the private and public sectors
Transfusion-associated HIV infection			
1. Interacting diseases	• Haemophilia • Thalassaemia • Postpartum haemorrhage	• Practice of providing plasma • Lack of wide availability of HIV-tested blood products • Rarely, non-adherence to rational use of blood	
2. Injecting drug use		• Peer pressure • Low adherence to biosafety precautions	• Unemployment • Poverty • Social instability
3. Occupational exposure		• Ignorance about postexposure prophylaxis • Access to postexposure prophylaxis drugs	• Low availability of protective equipment at the workplace
MTCT	• Pregnancy when a woman recently acquires HIV • Breastfeeding	• Vaginal delivery • Ignorance about MTCT • Lack of access to HIV testing and counselling	

STD: sexually transmitted disease; MTCT: mother-to-child transmission

Table 1.2 Interventions (by significance) for the management of sexually transmitted diseases

				Non-medical interventions/prevention	
Outcome		Medical interventions		Prioritized (targeted) interventions	Subpopulations
STDs	Syndromic management of STDs (at all levels of health care)[1]	Aetiological management (not favoured as it leads to missed opportunities)	Establishment of STD surveillance to assess the pattern and proportionate morbidity of STDs and drug-resistance profile to suitably modify choice of drugs in the syndromic approach	• Generic package of prioritized intervention that includes (i) Management of STDs using the syndromic approach (ii) Peer-based education on STDs/HIV/AIDS (iii) Promotion and distribution of condoms (iv) Empowerment of socially challenged groups (v) Creation of an enabling environment • Life-skills' education that includes peer-based approaches to create enabling environment and empowerment	• Sex workers • Men who have sex with men • Truckers • Migrant subpopulations • Adolescents
Transfusion-associated HIV infection	Rational use of blood and blood products	NA	NA	Promote rational use of blood/blood products	• Promote voluntary blood donation • Intensify self-deferral strategy • Treat potential causes of anaemia aggressively
Injecting drug use-associated HIV	NA	NA	NA	Intensify and saturate with targeted interventions aiming at harm reduction, such as needle exchange, harm-reduction strategies, enabling environment, using peer-based approaches	
Occupational exposure	Provide anti-retrovirals on needle-stick exposure	• Establishment of national needle-stick registry • Provide combination ART for 4 weeks	NA	• Periodic biosafety training • Initiate needle-stick audit • Access to post-exposure prophylaxis • Counselling	
Mother-to-child transmission of HIV	Antiretroviral/s for PMTCT	Counselling on infant feeding practices	NA	• Enhance access to HIV testing and counselling • Peer-based interventions for exclusive breastfeeding	

STD: sexually transmitted disease; AIDS: acquired immunodeficiency syndrome; PMTCT: prevention of mother-to-child transmission; ART: antiretroviral therapy

[1]National AIDS Control Organization (NACO). *Simplified STI/RTI treatment guidelines*. New Delhi: Government of India; 1998.

Table 1.3 Standard treatment protocols for the management of conditions occurring due to sexually transmitted diseases

Condition	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay
Genital ulcer disease (syndromic approach)	• About 10 minutes per consultation of a physician • About 20–30 minutes per consultation of a counsellor • About 20 minutes of a laboratory technician	• RPR/VDRL (at all levels of health care) • Dark-ground microscopy (up to the district level) • Tzanck preparation (at the tertiary level)	• Inj. benzathine penicillin 2.4 MU IM × 2 + erythromycin 500 mg qid × 14 days • If the lesions look like herpes genitalis, give acyclovir 200 mg 5 times/day × 7 days OR famciclovir 250 mg bd × 7 days	Not required
Genital discharge syndrome	• About 10 minutes per consultation of a physician	• Not needed at the PHC level	Use norfloxacin 800 mg stat OR	Not needed

(Cont.)

Table 1.3 (*cont.*) Standard treatment protocols for the management of conditions occurring due to sexually transmitted diseases

Condition	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay
	• About 20–30 minutes per consultation of a counsellor • About 20 minutes of a laboratory technician	• Gram stain • Wet mount (up to the district level)	Inj. ceftriaxone 250 mg IM stat + Cap. doxycycline 100 mg bd × 7 days	
Vaginal discharge	• About 15 minutes per consultation of a physician • About 20–30 minutes per consultation of a counsellor • About 20 minutes of a laboratory technician	• Per speculum examination • Gram stain • Wet mount	• E/o cervicitis norfloxacin 800 mg stat + doxycycline 100 mg bd × 7 days + metronidazole 200 mg tds × 7 days • E/o vaginal infection alone; treated as candidial; give fluconazole 150 mg stat	Not needed
Inguinal bubo	• About 10 minutes per consultation of a physician • About 20–30 minutes per consultation of a counsellor	Clinical diagnosis	Use doxycycline 100 mg bd × 14 days OR erythromycin 500 mg qid for 1 day OR tetracycline 500 mg qid × 14 days	
Lower abdominal pain among women (pelvic inflammatory disease)	• About 10 minutes per consultation of a physician • About 20–30 minutes per consultation of a counsellor • About 20 minutes of a laboratory technician	• Clinical diagnosis • Gram stain • Wet mount	Inj. ceftriaxone 250 mg IM + doxycycline 100 mg bd × 7 days + metronidazole 200 mg tds or 400 mg bd × 14 days	
Swelling of the scrotum	• About 10 minutes per consultation of a physician • About 20–30 minutes per consultation of a counsellor	Clinical diagnosis	Inj. ceftriaxone 250 mg stat + doxycycline 100 mg bd × 7 days	Not needed
Ophthalmia neonatorum	• About 10 minutes per consultation of a paediatrician • About 20 minutes of a laboratory technician		Inj. ceftriaxone 50 mg/kg, maximum dose: 125 mg stat	Not necessary
Post-exposure prophylaxis	• About 10 minutes per consultation of a physician • About 20–30 minutes per consultation of a counsellor	• HIV ELISA of patient as well as the provider at the baseline and at 3 months (up to the district level) • HIV-1 DNA PCR at a tertiary health care centre	Within 15 minutes to 4 hours (ideally) or 24 hours minimally, provide: zidovudine 300 mg bd + lamivudine 150 mg bd + indinavir 800 mg tds for 4 weeks[1]	Not necessary
Prevention of mother-to-child transmission	• About 10 minutes per consultation of a physician • About 20–30 minutes per consultation of a counsellor	• HIV ELISA of pregnant woman (up to the district level) • HIV-1 DNA PCR of the baby at 2 and 4 months in a non-breastfed baby or one month after stopping breast milk (available only in select tertiary care centres)	• Currently, NACO provides nevirapine 200 mg one tablet during labour and single dose of nevirapine 2 mg/kg of body weight of baby within 72 hours of birth.[2] However, in view of the reports of emergence of nevirapine-resistant mutations in mothers that preclude the use of a nevirapine-based regimen, WHO recommends the use of zidovudine 300 mg bd + lamivudine 150 mg bd for at least a week after birth to the mother • Elective caesarean section	Hospitalization for an average of 5 days if the mother undergoes elective caesarean section additionally

RPR: rapid plasma reagin; VDRL: Venereal Disease Research Laboratory; Inj.: injection; ELISA: enzyme-linked immunosorbent assay; PCR: polymerase chain reaction

[1]NACO. *Specialist training and reference module*. New Delhi; Government of India; 2002.

[2]Guay LA, Musoko P, Fleming T, *et al*. Intrapartum and neonatal single dose nevirapine compared with zidovudine for prevention of mother-to-child transmission of HIV-1 in Kampala, Uganda: HIVNET 012 randomised trial. *Lancet* 1999;**354:**705–8.

2. HIV/AIDS

Table 2.1 Interventions (by significance) for the treatment of advanced HIV disease (CD4 count <200 cells/cmm)

	Non-medical interventions/prevention		
Medical interventions	Exercise	Nutrition	Others
• Chemoprophylaxis against opportunistic infections (at all levels of health care) • Treatment of opportunistic infections if they occur (at all levels of health care) • Antiretroviral therapy (only up to the district level)	NA	Green leafy vegetables, good food hygiene, potable water	Psychosocial support

Table 2.2 Standard treatment protocol for the management of advanced HIV disease

Condition	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay
Advanced HIV disease (acquired immunodeficiency syndrome)	• About 15 minutes per consultation of a physician • About 30 minutes per consultation of a counsellor • About 15 minutes per consultation of a nurse and laboratory technician	• Specific tests 1. CD4/CD8 counts every 6 months (at tertiary and district levels only) 2. Plasma viral load (not recommended currently due to high cost) • Supportive tests 1. Routine haemogram (at all levels) at every visit 2. Liver function tests —ALT —AST —Alkaline phosphate (up to the district level; initially 14 days after initiating ART and later symptom-directed) 3. Chest X-ray to rule out TB before initiating ART at all levels of care and later symptom-directed 4. Test the sputum for the presence of AFB to rule out TB before initiating ART at all levels of care and later symptom-directed 5. USG of the abdomen wherever required 6. ELISA or PCR for HCV at a tertiary care centre among injecting drug users or transfusion-transmitted HIV 7. ESR at all levels—symptom-driven	Chemoprophylaxis • All patients whose CD4 count is <200 cells/cmm or those having a history of any AIDS-defining illness in the past receive co-trimoxazole double strength tablet once a day OR (if sensitive to sulpha drugs) dapsone 100 mg od until the CD4 count increases beyond 350 cells/cmm after initiating ART (given at all levels of health care) • ART (given up to the district level) • Those who do not have concurrent TB and whose haemoglobin level is above 8 g% are given zidovudine 300 mg bd + lamivudine 150 mg bd + nevirapine 200 mg od for 14 days. If the patient does not develop any severe skin rash or hepatotoxicity, provide a fixed-drug combination of: zidovudine 300 mg bd + lamivudine 150 mg bd + nevirapine 200 mg bd until the patient develops signs of immunological/clinical failure (generally a patient takes about 2.5 years to develop failure) • In case the haemoglobin level is <8 g%, substitute zidovudine with stavudine 30/40 mg bd depending on the body weight of the patient (30 mg if the weight is <60 kg) • If the patient has concurrent TB, to avoid drug–drug interaction between nevirapine and rifampicin, give efavirenz 800 mg od in place	NA

(Cont.)

Table 2.2 (*cont.*) Standard treatment protocol for the management of advanced HIV disease

Condition	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay
			of nevirapine in the regimen described above *Note:* The efavirenz-based regimen is more potent and the response is durable. However, it costs three times more than the nevirapine-based regimen	
Treatment failure (salvage regimen)	• About 15 minutes per consultation of a physician • About 15 minutes per consultation of a counsellor • About 15 minutes per consultation of a nurse and laboratory technician	• Specific tests 1. CD4/CD8 counts every 6 months (at tertiary and district levels only) 2. Plasma viral load (not recommended currently due to high cost) • Supportive tests 1. Routine haemogram (at all levels) at every visit 2. Liver function tests —ALT —AST —Alkaline phosphate (up to the district level; symptom-directed) 3. Chest X-ray (symptom-driven, at all levels of care) 4. Test the sputum for the presence of AFB to rule out TB (symptom-driven, at all levels of care) 5. USG of the abdomen wherever required 6. ESR at all levels (symptom-driven) 7. Other tests are driven by the symptoms with which the patient presents	• Antiretroviral drugs selected for salvage therapy are initiation regimen-specific • Though there are 20 different antiretroviral drugs, the most widely accepted regimen for salvage is as follows: abacavir 300 mg bd + didanosine EC 250/400 mg od (weight <60 kg—250 mg) + indinavir 800 mg bd + ritonavir 100 mg bd (IDV/rtv) • In place of IDV/rtv, nelfinavir 1250 mg bd or lopinavir 400 mg bd + ritonavir 100 mg bd (LPV/rtv) can also be used • About 30% of patients may fail after 3 years. After failure, antiretroviral medicines may be chosen for treatment based on the prior treatment pattern	NA Admission would not be required to initiate therapy and thereafter. However, depending on the opportunistic infection that the patient may develop as a sign of treatment failure, one may require hospitalization

ALT: alanine transaminase; AST: aspartate transaminase; ART: antiretroviral therapy; AFB: acid-fast bacilli; USG: ultrasonography; HCV: hepatitis C virus; ESR: erythrocyte sedimentation rate; TB: tuberculosis; ELISA: enzyme-linked immunosorbent assay; PCR: polymerase chain reaction

Table 2.3 Some complications of antiretroviral therapy, and personnel, tests and drugs required for their treatment

Complication	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay	Advice
Lipodystrophy	About 15 minutes in a directed examination	• Waist–hip ratio (at all levels of care) • USG of the abdomen (up to the district level) • Dual-energy X-ray absorptiometry (DEXA) (available at select tertiary-level centres)	• Change the NRTI or PI • Continue metformin 500 mg bd • Restorative surgery	Not required	Low fat diet
Lactic acidosis	About 10–15 minutes in a directed examination	• Anion gap (available at select tertiary care centres) • Lactic acid level (available at select tertiary care centres)	• Stop NRTIs and NNRTIs if severe renal failure • Hydration • Use riboflavin 50 mg/day till improvement	Potentially a fatal disorder. Requires hospitalization for weeks depending on the severity of lactic acidosis	

(Cont.)

Table 2.3 (*cont.*) Some complications of antiretroviral therapy

Complication	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay	Advice
Hyperlipidaemia	About 10–15 minutes in a directed examination	Lipid profile (at tertiary care centres)	• Change the PIs to NRTIs/ NNRTIs • Use atorvastatin 10 mg/day OR pravastatin 20 mg/day OR fenofibrate 54–160 mg qid OR gemfibrozil 600 mg bd		
Insulin resistance		• No standardized methods • Frequent fasting blood glucose levels while receiving PIs	Use metformin depending on the blood sugar level	Not reported	Dietary advise

USG: ultrasonography; NRTI: nucleoside reverse transcriptase inhibitor; NNRTI: non-nucleoside reverse transcriptase inhibitor

Bibliography

1. Bartlett JG, Gallant JE. *Medical management of HIV infection*. Baltimore, USA: Johns Hopkins University; 2003.
2. *DHSS guidelines for the use of antiretroviral agents in HIV-1 infected adults and adolescents*. USA: Department of Health & Human Services; 10 November 2003.

3. Opportunistic infections

Table 3.1 Interventions (by significance) for the management of opportunistic infections

	Medical interventions		Non-medical interventions/prevention		
Outcome	Primary chemoprophylaxis	Secondary chemoprophylaxis	Exercise	Nutrition	Other
Tuberculosis	Not advocated excepting to the close contacts of TB cases	Not advocated	NA	NA	NA
Candidiasis	Not required	Use fluconazole 150–200 mg od; once a week in patients with oesophageal candidiasis	NA	Non-spicy diet	NA
Herpes zoster	Not available	Not available	NA	NA	NA
Cryptosporidial diarrhoea	Not available	Not available	NA	NA	Potable water
Cryptococcal meningitis	Not available	Maintenance regimen with fluconazole 200–400 mg/day	NA	NA	Avoid contact with pigeon excreta (debatable)
Pneumocystis carinii pneumonia (PCP)	Use trimethoprim–sulphamethoxazole DS 1 od OR dapsone 100 mg od	For life, unless CD4 counts increase beyond 200 cells/cmm for at least 3 months while receiving antiretroviral therapy (ART)	NA	NA	Do not share a hospital room with a person having PCP
Toxoplasmic encephalitis	Use trimethoprim–sulphamethoxazole DS 1 od OR dapsone 50 mg od + pyrimethamine 50 mg/week + leucovorin 25 mg/week	For life, unless CD4 counts increase beyond 200 cells/cmm for at least 6 months while receiving ART	Do not eat raw or undercooked meat, particularly lamb, beef and pork	NA	Avoid handling litter and cat's excreta

(Cont.)

Table 3.1 (*cont.*) Interventions (by significance) for the management of opportunistic infections

	Medical interventions		Non-medical interventions/prevention		
Outcome	Primary chemoprophylaxis	Secondary chemoprophylaxis	Exercise	Nutrition	Other
Isospora belli diarrhoea	Use trimethoprim–sulphamethoxazole DS 1 od	For life, unless CD4 counts increase beyond 200 cells/cmm for at least 3 months while receiving ART	NA	NA	Potable water
Cytomegalovirus disease (most often retinitis or oesophagitis)	Use ganciclovir 1 g tds	For life, unless CD4 counts increase beyond 150 cells/cmm for at least 3 months while receiving ART	NA	NA	NA
Herpes simplex infection	Not recommended	• Use acyclovir 400 mg bd OR famciclovir 250 mg bd up to 2 years (safety data available only up to 2 years) • Treatment with antiretroviral drugs is effective in reducing recurrences	NA	NA	NA
Progressive multifocal leucoencephalopathy (PML)	NA	NA	NA	NA	NA
Hepatitis B	HBV vaccine to those who are not infected with hepatitis B virus	NA	NA	NA	Practise safe sex
Hepatitis C	NA	NA	NA	NA	Injection safety, especially needle exchange programme among IDUs
Cancer of the cervix	NA	NA	NA	NA	Cauterize genital warts in females and conduct yearly Pap smear examination of HIV-positive women
Pneumococcal pneumonia	Pneumococcal conjugate vaccine	NA	NA	NA	NA

NA: not applicable; IDU: injection drug user; Pap: Papanicolaou

Table 3.2 Personnel, tests, drugs and duration of inpatient stay for opportunistic infections

Condition	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay
Tuberculosis	• About 10 minutes per consultation of a physician • About 15 minutes per consultation of a counsellor (currently not available at TB clinics)	• Directed by symptoms/clinical suspicion • Chest X-ray (up to the district level) • Sputum for AFB (at all levels) • USG of the abdomen (up to the district level) • FNAC of lymph nodes (up to the district level) • CSF examination (up to the district level) • Ascitic fluid examination (up to the district level)	• Chemoprophylaxis is not advisable excepting to close contacts of a TB case —Category 1: $2(EHRZ)_3 + 4(HR)_3$ —Category 2: $2(SEHRZ)_3 + 1(EHRZ)_3 + 5(HRE)_3$ —Category 3: $2(HRZ)_3 + 4(HR)_3$ • The maintenance phase may be prolonged in TB meningitis and extrapulmonary TB (depending on the response to treatment) • Treatment to be provided at all levels of health care	There are no publications reporting the number of days of hospitalization among HIV–TB patients in India

(Cont.)

Table 3.2 (*cont.*) Personnel, tests, drugs and duration of inpatient stay for opportunistic infections

Condition	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay
Oropharyngeal candidiasis	• About 10 minutes per consultation of a physician • On an average, a patient may develop candidiasis at least 5 times if she/he does not receive antiretroviral therapy	• KOH/fresh mount preparation (up to the district level) • Culture—Sabouraud agar (if drug-resistant species is expected) (only at the tertiary level) • Rarely, endoscopy (only at the tertiary level)	• Localized mucocutaneous candidiasis —fluconazole 150 mg od × 10–14 days • Oesophageal candidiasis —fluconazole 150–200 mg × 10–14 days OR —itraconazole 100 mg od × 10–14 days OR —ketoconazole 200 mg bd × 10–14 days OR —amphoptericin B (rarely)	Hospitalization may not be required for candidiasis *per se*
Herpes zoster	• About 10 minutes for each consultation of a physician • About 10 minutes for each consultation of a counsellor	• Clinical diagnosis mostly • Tzanck test (at the tertiary level)	• Use famciclovir 500 mg tds × 7–10 days OR acyclovir 200 mg 5 times a day × 7–10 days OR valacyclovir 1 g tds × 7–10 days • Use prednisolone or amitriptyline in case of severe neuralgia	Mostly not required
Cryptosporidial diarrhoea	• About 10 minutes for each consultation of a physician • About 10 minutes for each consultation of a counsellor • About 15 minutes of a laboratory technician	Stool: Modified Ziehl–Nielsen stain (can be done up to the district level)	• In advanced HIV disease, ART is the best treatment. However, in its absence, the patient can be given: —azithromycin 1200 mg × 2 days followed by 1200 mg/day for 27 days OR —nitazoxanide 500 mg bd for 2–3 weeks • Oral rehydration (at all levels of care)	• No Indian publication on the duration of hospitalization is available • On an average, a patient spends about 2–3 days in hospital during each episode • Episodes can be frequent in patients who are not receiving ART
Cryptococcal meningitis	• About 10 minutes per consultation of a physician • About 15 minutes of a laboratory technician	• India ink preparation (up to the district level) • Routine CSF examination (up to the district level) • CSF: Cryptococcal antigen (up to the tertiary level) • Serum cryptococcal antigen (up to the tertiary level)	• Use amphotericin B 0.7 mg/kg/day IV with or without 5-flucytosine 100 mg/kg/day for 14 days followed by fluconazole 400 mg/day for 8 weeks • This is followed by a maintenance regimen with fluconazole up to 200–400 mg/day	Indian studies not available. However, on an average, 2 weeks of hospitalization are required
Pneumocystis carinii pneumonia	• About 10 minutes per consultation of a physician • About 15 minutes of a laboratory technician	• Chest X-ray (up to the district level) • Induced sputum for silver methanamine stain or Giemsa (tertiary centres) • Therapeutic trial with trimethoprim (TMP)/ sulphamethoxazole (SMX) at any level of health care	• Use trimethoprim–sulphamethoxazole (TMP 15 mg/kg/day, SMX 75 mg/kg/day) tds × 21 days OR primaquine 30 mg qid × 21 days + clindamycin 300–450 mg qid × 21 days	• Lack of Indian data • On an average, 7 days of hospitalization are needed
Toxoplasmic encephalitis	About 10 minutes per consultation of a physician	• MRI (tertiary centres) • Therapeutic trial at all levels of health care	• Use pyrimethamine 200 mg loading dose, then 75 mg/day + leucovorin 10–20 mg/day + sulphadiazine 1–1.5 g qid × 3–6 weeks	• Lack of Indian data on hospitalization

(Cont.)

Table 3.2 (*cont.*) Personnel, tests, drugs and duration of inpatient stay for opportunistic infections

Condition	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay
			• Maintenance regimen: Continue the above-mentioned drugs, if not receiving ART	• On an average, 7 days of hospitalization
Isospora belli diarrhoea	About 10 minutes per consultation of a physician	Stool microscopy—AFB stain (up to the district level)	Use trimethoprim–sulphamethoxazole DS tablet tds × 2–4 weeks	Generally hospitalization would not be required. However, the patient may be admitted for rehydration for a day or two.
Cytomegalovirus disease	15 minutes per consultation of a physician			• Indian data not available • On an average hospitalisation for 3–6 weeks is required for each of these conditions
CMV retinitis		Fundoscopy (up to the district level)	• Use foscarnet 90 mg/kg bd IV × 14–21 days OR ganciclovir 5 mg/kg IV bd × 14–21 days • Maintenance with valganciclovir 900 mg/day However, the use of ART is known to be efficacious and cost-effective compared to others	Not required
CMV oesophagitis or colitis		• Culture (not feasible) • Diagnosis by exclusion	• Use ganciclovir 5 mg/kg IV bd × 2–3 weeks OR foscarnet 60 mg/kg tds IV × 2–3 weeks • Maintainance dose, if the patient is not put on ART	Up to 3 weeks
CMV encephalitis/ radiculomyelopathy		• CSF examination (up to district level) • PCR (not available in the public sector)	• Use ganciclovir 5 mg/kg IV bd + foscarnet 90 mg/kg bd IV for 3–6 weeks with maintenance However, ART effectively prevents further disease progression	Up to 6 weeks
Herpes simplex infection	About 10 minutes per consultation of a physician	Tzanck test (tertiary care)	• Use acyclovir 400 mg tds × 7–10 days • Use famciclovir 250–500 mg bd × 5–10 days	Not required unless the patient has encephalitis
Progressive multifocal leuco-encephalopathy	About 15 minutes per consultation of a physician	• MRI (tertiary level) • CSF examination (up to district level) • PCR for JC virus (not available)	• Preferred treatment is with antiretroviral therapy • Interferon-alpha is costly and less effective	• Indian data not available • On an average, 1 week of hospitalization
Hepatitis B	• About 5 minutes at the first consultation of a physician • About 10 minutes at the first consultation with a counsellor • Average time spent for testing and sera preparation is about 15 minutes of a laboratory	• HBsAg test (available up to the district level) • Anti-HBeAg (may be available at the tertiary level) • Anti-HBc (may be available at the tertiary level) • HBV DNA PCR (may be	• Use lamivudine 100 mg od for one year • Interferon alpha-2b—5 million units SC 3 times per week × 4 months is costly • Administration of ART impacts HBV infection and is cost-effective	No data

(Cont.)

Table 3.2 (*cont.*) Personnel, tests, drugs and duration of inpatient stay for opportunistic infections

Condition	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay
	technician (tested in batches. However, varies by sophistication of the test)	available at the tertiary level) • Liver function tests		
Hepatitis C	• About 5 minutes at the first consultation of a physician • About 10 minutes of first consultation with a counsellor • Average time spent for testing and sera preparation is about 15 minutes of laboratory technician (tested in batches. Varies by sophistication of the test)	• HCV EIA (tertiary care) • HCV RNA testing (select tertiary care centres) • Liver function tests	Interferon-alpha 3 million units 3 times a week + ribavirin 800–1200 mg/day for 48 weeks OR Pegylated interferon 1.5 µg/kg SC per week for 48 weeks	Data not available
Non-Hodgkin lymphoma	• About 20 minutes in directed examination of a physician • Laboratory technician and pathologist	• Biopsy (up to the district level) • CT scan (at the tertiary level) • X-ray (up to the district level)	Standard cyclophosphamide, doxorubicin, adriamycin, vincristine, prednisolone (CHOP) OR methotrexate, bleomycin, doxorubicin, cyclophosphamide, vincristine, dexamethasone, G-CSF (M-BACOD) OR etoposide, prednisolone, vincristine, cyclophosphamide, doxorubicin (EPOCH)	Variable on clinical presentation and stage
Cancer of the cervix		• Pre-invasive surgical options including hysterectomy • If invasive, depending on the stage, radiation and chemotherapy are advocated		
		Other common conditions in HIV-infected individuals		
Wasting disease	• 10 minutes in directed examination of a physician • 20 minutes per consultation of a nutritionist	Generally clinical	Use cyproheptidine 2–4 mg bd up to 2 weeks OR megestrol acetate 800 mg/day × 12 weeks OR dronabinol 2.5–10 mg bd	Not required
Seborrhoeic dermatitis	About 5 minutes in directed consultation of a physician	Clinical	Use hydrocortisone (2.5%) locally + ketoconazole (2%) cream locally + tar-based shampoo	Not required
Thrombocytopenia	• 10 minutes in directed consultation of a physician • 10 minutes of a laboratory technician	Platelet count (up to the district level)	Use prednisolone 40–60 mg per day in divided doses to be tapered off within 2 weeks OR IVIG 400 mg/kg/day on days 1, 2, 14 followed by every 2–4 weeks OR Splenectomy OR ART, blood or platelet transfusion	Not reported but may need a day or two for transfusion
Salmonella infection	About 15 minutes in directed examination of a physician	Culture (up to the district level)	Use ciprofloxacin 500–750 mg bd × 14 days OR ceftrioxime 2 g/day IV OR cefotaxime 4–8 g/day IV × 14 days	Not available, but on an average 4–5 days
Bacterial pneumonias	About 15 minutes in directed examination of a physician	• X-ray (up to the district level)	Use ceftrioxime 2 g/day IV OR	Up to 2 weeks

(Cont.)

Table 3.2 (*cont.*) Personnel, tests, drugs and duration of inpatient stay for opportunistic infections

Condition	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay
		• Blood culture (up to the district level) • Sputum—Gram stain (up to the district level)	cefotaxime 4–8 g/day IV × 2 weeks	
Aphthous ulcers	About 5 minutes in a given consultation of a physician	Clinical	Use prednisolone 5–10 mg bd × 5–7 days OR thalidomide 100–200 mg/day for about 4 weeks OR ART	Not required
Taenia cruris	About 5 minutes in directed consultation of a physician	Generally clinical diagnosis	Use fluconazole 150–200 mg once a week × 4 weeks + local miconazole application. Duration depends on the extent and sites involved	None

AFB: acid-fast bacilli; USG: ultrasonography; FNAC: fine-needle aspiration cytology; CSF: cerebrospinal fluid; MRI: magnetic resonance imaging; PCR: polymerase chain reaction; SC: subcutaneous; IVIG: intravenous immunoglobulin; G-CSF: granulocyte colony stimulating factor; CT: computerized tomography; EIA: enzyme immunoassay; HBV: hepatitis B virus; HCV: hepatitis C virus; ART: antiretroviral therapy

Bibliography

1. *USPHS/IDSA guidelines for the prevention of opportunistic infections in persons infected with human immunodeficiency virus.* USA: USPHS; 2001.
2. Bartlett JG, Gallant JE. *Medical management of HIV infection.* Baltimore, USA: Johns Hopkins University; 2003.

Review of women and children's health in India: Focus on safe motherhood

RAJANI R. VED*, AVTAR SINGH DUA†

A comprehensive consideration of women's health in India is to view it through a holistic perspective that spans reproductive health needs beyond maternity and fertility, such as reproductive morbidities ranging from sexually transmitted diseases (STDs) to uterine prolapse, other diseases, such as tuberculosis and cancers, gender-based violence and the health needs of older women. A well-coordinated and efficient health system is required to address all these issues. In taking a systems approach, one could argue that strengthening the system through a quality and rights perspective, and linking women, families and households would contribute significantly to addressing women's health needs.

Interventions to reduce the maternal mortality ratio (MMR), infant mortality rate (IMR) and child deaths, and to lower the fertility rate, use tried and tested approaches that have demonstrated success globally and in parts of India. These interventions are focused and require effectively functioning health systems.

This paper is based on the premise that a short-to-medium term focus on reductions in the maternal and infant mortality through strengthening health systems and ensuring competent and trained providers at the grassroots and primary health centre levels will yield benefits that can be realized for other health needs of women. For example, ensuring delivery by skilled attendants* contributes to reductions in maternal morbidity as well as mortality, since it is estimated that for every maternal death there are 50–100 cases with morbidity.[1]

India has made substantial gains in improving the overall health of men, women and children in the fifty-eight years since Independence. Despite these gains, maternal and child deaths constitute a significant burden of disease among women and children. According to World Health Organization (WHO) estimates, India contributes about 24 lakh to the 108 lakh global child deaths and accounts for 25% of the 529,000 global maternal deaths.[2] Although India has a long history of programmatic efforts to improve the health of mothers and children, it appears that these efforts lack focus and consistency, and interventions have not always been evidence-based. The continuing high MMR and IMR can be attributed to shifting policies and programmes as well as to the level of the complexity of the technical strategies required to address safe motherhood and child survival.

Community-based, primary health care preventive and promotive approaches such as nutrition education, environmental sanitation and the use of technologies that need relatively low skill levels such as immunization and oral rehydration therapy (ORT), and which can be delivered practically at the doorstep, have contributed considerably to improving infant and child health. Maternal health interventions, on the other hand, require more complex clinical and surgical interventions, and depend on a well-functioning health system that provides adequate clinical back-up for outreach interventions.

Background

The history of India's family welfare programmes (Box 1) illustrates the focus and changes of programme and policies, and provides evidence that gains in some indicators were achieved at the cost of others.[3] Although successive Five-Year Plans have articulated a strong maternal and child health (MCH) component, National- and State-level policies and programmes were mostly characterized by the domination of population concerns.

*Public Health Consultant
New Delhi
e-mail: vedarya@vsnl.com

†Member, National Commission on Macroeconomics and Health, Government of India, New Delhi
e-mail: avtarsinghdua@yahoo.co.in

*The term 'skilled birth attendant' refers exclusively to people with midwifery skills (for example, doctors, midwives, nurses) who have been trained to proficiency in the skills necessary to manage normal deliveries and diagnose or refer medical complications. Depending on the setting, other health care providers, such as auxiliary nurse–midwives, community midwives, village midwives and health visitors may also have acquired appropriate skills if they have been specially trained. (*Source: Reduction of maternal mortality: A joint WHO/UNFPA/UNICEF/World Bank Statement.* Geneva: World Health Organization; 1999)

Box 1. History of India's family welfare policy and programmes, 1951–2003

1950s	Training institutions for population analysts/family planning workers established, rural and urban clinics set up.
1960s	Facilities for sterilization increased through clinics, sterilization camps, outreach and hiring auxiliary nurse–midwives (ANMs). The approach was largely family planning oriented.
1970s–1980s	Multipurpose workers responsible for providing a set of family planning, maternal and child health and other primary health care services. The Medical Termination of Pregnancy Act was introduced in 1971.
1985	The three-tiered health infrastructure became the norm across the country.
1986	Universal Immunization Programme introduced.
1992–1996	Child Survival and Safe Motherhood Programme launched.
1993–1995	MS Swaminathan Committee recommendations were submitted, the International Conference on Population and Development, and the Beijing Conference on Women were held which influenced policy.
1997	Reproductive and Child Health (RCH)-I Programme introduced.
1997–2004	RCH-I implemented, National Polio Eradication Campaign in full swing, prevalence of HIV/AIDS increased, particularly in the South, West and the North-East, RCH-II design in process.

Source: Measham and Heaver 1996

The Expanded Programme on Immunization (EPI) of 1979 and the Universal Immunization Programme (UIP) of 1985 focused on child health. It was only in the Child Survival and Safe Motherhood (CSSM, 1992–1995) programme that interventions for maternal health and maternal mortality were actually considered.

Even while this agenda remained unfinished, in the 1990s, India's family welfare programme made a paradigm shift from being demographically driven to one that incorporated the recommendations of the Cairo and Beijing Conferences, namely, that an integrated approach to link population to development, women's empowerment, gender equality and reproductive rights would result in improved health of women and their families. Thus, instead of continuing with efforts to reduce the MMR as a clear and focused agenda, the Reproductive and Child Health-I (RCH-I, 1997–2004) project was launched which expanded the scope of the Programme.[4]

This expanded scope incorporated a range of critical and necessary dimensions of women's health care. However since the system was not equipped to handle existing programmes, this sudden expansion of programme portfolio, with dramatic policy shifts, led to a dilution in efforts to reduce maternal and child mortality. This resulted in a nearly stagnant MMR and a slowing decline in the IMR. The plateau in reduction of the IMR can be attributed to poor neonatal survival, closely linked to care during labour and in the postpartum period. Commensurate with the focus of the programme, the total fertility rates (TFR) have shown declines at the national level (Table 1). The data demonstrate that consistent and focused interventions are necessary for reductions to occur in the IMR and MMR.

This paper reviews the current status of maternal and infant health, including causes of death; summarizes evidence-based interventions to improve maternal and child health, and reduce the MMR and IMR using examples from global experiences and research; analyses technical strategies, policies, human resources and health infrastructure required for CSSM and the RCH-I Programmes; reviews the extent to which RCH-II has incorporated evidence-based strategies in specific contexts to reduce the MMR and finally proposes options to most effectively address reductions in these key indicators to achieve the goals of the National Population Policy (NPP 2000)[7] and the Millennium Development Goals (MDGs) outlined in Table 2. Using the 1992–1993 National Family Health Survey (NFHS)-1 data, which show an under-five mortality rate (U5MR) of 111.9 and an MMR of 424, India would have to achieve a U5MR of 37 and an MMR of 106 (from the present 86.9 and 540, respectively) by 2015 to achieve the MDGs. The focus of this paper, both in analysis and recommendations, is skewed towards health system strengthening. However, this does not in any way minimize the enormous potential and sure success of social mobilization for improved health care in pushing service delivery systems. Indeed, the effectiveness of health sector reform is likely to be limited unless there is a commensurate push to engage civil society institutions to ensure community participation.

Table 1. Changes in key indicators (1971–2002)[5,6]

Year	Total fertility rate (%)	Infant mortality rate (per 1000)	Maternal mortality ratio
1971–1981	5.0 in 1971–75 and 4.5 in 1976–80 (RGI)	134 in 1971–75 and 124 in 1976–80 (RGI)	
1981–1991	4.5 in 1981–85 and 4.3 in 1986–90 (RGI)	104 in 1981–85 and 91 in 1986–90 (RGI)	580 (1982–1986) Bhat *et al.* 1995
1991–1995	3.39 (NFHS1992–1993)	79 (NFHS1992–1993)	424 (NFHS1992–1993)
1996–1999	2.85 (NFHS1998–1999)	64 (NFHS1998–1999)	540 (NFHS1998–1999)
1999–2002		64 (SRS 2002)	

Sources: National Family Health Survey (NFHS), Rounds 1 and 2; Bhat *et al.* 1995; RCH-II National Programme Implementation Proposal (PIP); Registrar General of India (RGI) 1999a; Sample Registration System (SRS) 2002

Table 2. Goals of the National Population Policy 2000, Tenth Plan, RCH-II and Millennium Development Goals

Indicator	Tenth Plan goals (2002–2007)	RCH-II goals (2004–2009)	National Population Policy 2000	Millennium Development Goals
Population growth	16.2			
Infant mortality rate	45/1000	35/1000	30/1000	—
Under-five mortality rate	—	—	—	Reduce from two-thirds of the 1990 levels
Maternal mortality ratio	200/100,000	150/200,000	100/100,000	Reduce by three-fourths by 2015
Total fertility rate	2.3	2.2	Meet 100% needs	—
Couple protection rate	65%	65%	Meet 100% unmet needs	—

PART I

Status of safe motherhood, infant and child health, and family planning interventions

Safe motherhood interventions

Maternal mortality is the outcome of a complex web of causal factors that include social, economic, educational, political and cultural causes as well as issues such as gender inequity, state of physical infrastructure, geographic terrain and the health system. Evidence from parts of India and elsewhere demonstrates that it is possible to substantially reduce maternal mortality by addressing health system factors alone to ensure that all women have access to safe delivery services. Over the past two decades, new understanding regarding interventions for maternal mortality has emerged (Box 2).[8]

Global experiences in reducing maternal mortality

Some countries in the developed world[9] have a better success story in reducing maternal mortality than others and these hold some lessons for India. Despite a low gross national product (GNP), Sri Lanka, Malaysia and Thailand have also mirrored these successes to some extent (Table 3).[10]

Sweden achieved an MMR of 228/100,000 by the turn of the twentieth century before modern surgical techniques, antibiotics or blood transfusion became available. This was achieved largely through increasing the number of births attended by certified, trained midwives, primarily at home. A medical doctor who could be called upon in case of complications supervised them. In countries such as the United States, these reductions were delayed until the advent of modern technologies and midwives were actively discouraged until the Second World War. In the UK, there was competition between midwives and doctors for access to deliveries. Maternal mortality stayed persistently high despite improvements in sanitation, nutrition and provision of antenatal care, until the 1940s when antibiotics, newer obstetric procedures and blood banking became possible (Fig. 1).

Success in reducing maternal mortality has also been demonstrated in Tamil Nadu, under the RCH-I Programme, where the health department creatively utilized a package of flexible funding to post nurses round the clock at primary health centres (PHCs), engage anaesthetists from the private sector and supported community-based organizations to manage ambulance services. These efforts led to increases in the number of institutional deliveries, increase in the number of caesarean sections for obstetric emergencies and enabled rural women with obstetric complications to reach a secondary care facility in time.[11]

The main lesson to be drawn from these successes is the 'professionalization' of delivery care in bringing about substantial reduction in the maternal mortality in a relatively short time-frame. However, it was not the technological intervention *per se*, but the framework of equity within which the technology was located, widespread provision of information, strong political will, empowered, trained midwives, the backing of public health professionals,

Box 2. Current understanding of interventions to reduce the maternal mortality ratio

- It is not possible to predict which woman will develop complications in pregnancy.
- Antenatal care alone cannot prevent maternal mortality, and every woman must be cared for by a skilled attendant during labour.
- Transport to link all levels of maternal health care, for emergencies to basic emergency obstetric care or comprehensive emergency facilities must be readily available.
- Basic emergency obstetric services must be available in primary/community health centres.
- Comprehensive obstetric services must be available in first referral units/secondary care facilities.
- Facilities must have the requisite infrastructure and human resources to handle the major direct causes of maternal deaths.
- Record-keeping and audits must be done to allow periodic assessment of performance so that appropriate action can be taken to improve the efficiency and effectiveness of interventions.
- Births, maternal and perinatal deaths must be registered so that the situation is reviewed regularly and priorities identified.

Table 3. Time taken to halve the maternal mortality ratio (MMR), Malaysia and Sri Lanka, 1949–1992[10]

Interventions	MMR—Malaysia	MMR—Sri Lanka
In 3–7 years	1950: 534	1947: 1056
• Establishment of systems to train and supervise midwives, and regulate midwifery practices	1957: 282	1950: 486
• Introduce accountability for results, systems for monitoring births and deaths		
• Models for effective communication with women and communities		
• Better obstetric techniques for those who already had access		
• Introduction of modern medical advances into existing services—general health improvement including malaria control, introduction of antibiotics		
In the next 13 years	1957: 282	1950: 486
• Improved access for the rural population. The critical elements of obstetric care were made available to the bulk of the rural population through the development of a widespread rural network of trained skilled midwives as its backbone, along with hands-on support from supervisory staff competent in basic obstetrics and a system for prompt access to facilities that could treat obstetric complications	1970: 148	1963: 245
From the 1970s onwards	1976: 78	1973: 121
• Use of strategies to increase utilization of existing services through better management, a focus on quality and systemic responsiveness to public needs and expectations	1985: 37 1991: 18	1981: 58 1992: 27

Source: Pathmanathan *et al.* 2003[10]

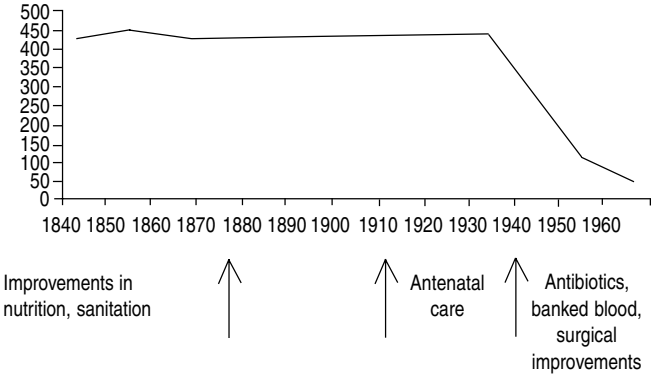

Fig. 1 Maternal mortality: UK 1840–1960

Source: Maine 1999

pressure by civil society and accountability of the health system, which brought about the reduction.

Status and trends in maternal mortality

Reliable data on maternal mortality (beset by its relative rarity, problems in definition, underreporting, misclassification) are not available for India. Existing estimates show widespread variations in the MMR among regions and States, with the south and west showing a lower MMR than the north and east. However, even here, there is little decline during the years studied (Table 4).

Causes of maternal mortality

A direct obstetric death is one due to complications of pregnancy, delivery, or the postpartum period, including complications of abortion. An indirect obstetric death is one due to existing medical conditions that are made worse by the pregnancy or delivery. On an average, one-quarter of maternal deaths are classified as indirect deaths. Indian data for 1998 show this to be slightly higher at 27.4% (Table 5). Around 72.4% of maternal deaths are due to direct causes—haemorrhage (in the antepartum or postpartum period), eclampsia, pre-eclampsia, infection, obstructed labour and complications of labour.[12]

Haemorrhage is the commonest cause of maternal death; it is likely to occur with no warning and can result in death within two hours from the onset of bleeding. Interventions

Table 4. Maternal mortality ratio, India and selected States

Region/State	MMR (1997)	MMR (1998)
India	408	407
North and central		
Rajasthan	677	670
Haryana	105	103
Punjab	196	199
Madhya Pradesh	498	498
Uttar Pradesh	707	707
East/North-east		
Orissa	361	367
West Bengal	264	266
Bihar	451	452
Assam	401	409
West		
Gujarat	29	28
Maharashtra	135	135
South		
Andhra Pradesh	154	159
Karnataka	195	195
Kerala	195	198
Tamil Nadu	76	79

MMR: maternal mortality ratio

Sources: Registrar General of India. *SRS Bulletin* 1999;33

Table 5. Causes of maternal mortality

Causes of maternal death	Deaths (%)
Direct causes	
Haemorrhage	29.65
Puerperal complications (including sepsis)	16.10
Obstructed labour	9.50
Abortion	8.90
Toxaemia of pregnancy	8.30
Indirect causes	
Anaemia	19.00
Pregnancy with tuberculosis, malaria and hepatitis B	6.20
Other causes	2.20

Source: SRS Bulletin 2000

require rapid transport to a comprehensive emergency obstetric care (CEmOC) facility. Deaths due to puerperal sepsis could be reduced through hygienic practices during delivery and administration of antibiotics. Obstructed labour and hypertensive disease of pregnancy require early identification and prompt referral. These causes are amenable to direct interventions through the delivery of a well-defined package of services which, in turn, are dependent on a well-developed health system linking skilled birth attendants at the community level through referral and transport to basic and CEmOC centres.

Performance of Safe Motherhood Programmes

Reduction in the MMR has been an articulated goal of the CSSM and RCH-I. Safe Motherhood interventions were antenatal care (ANC), safe delivery services (institutional and domiciliary), postpartum care and safe abortion services. Appendix 1 gives State-wise data on antenatal care coverage, place of delivery, attendant at delivery and postpartum care.

Antenatal care

Currently, ANC is provided by the auxiliary nurse–midwife (ANM) at the static clinic in the subcentre or through field outreach. Only 33% of pregnant women in India were contacted by a health worker in the first trimester, only 20% received the recommended antenatal package (consisting of three antenatal visits, two doses of tetanus toxoid [TT] and iron–folic acid [IFA] tablets/syrup for three months). The coverage of TT was 66.8% and IFA 58%. There are wide variations across States. Even in States with good indicators such as Tamil Nadu, only 50% of women receive all the services. Outreach services and their quality, which are cross-cutting across interventions, need more attention.

Delivery care

Evidence from the NFHS-2 shows that despite the high levels of maternal mortality, access of women to emergency obstetric care (EmOC) remains low. Appendix 1 also provides State-wise data on home deliveries, institutional deliveries, and deliveries conducted by skilled attendants. Only 33% of deliveries in India take place in institutions. Only 30% of all deliveries are conducted by a doctor and 11% by a nurse, ANM, midwife or lady health visitor (LHV). This figure is actually lower since the ANM does not meet the qualification of a skilled attendant who should be able to perform the functions of essential obstetric care. About 60% of deliveries are still attended by unskilled birth attendants, including traditional birth attendants (TBAs).

Global evidence points to the fact that ANC has no effect on reducing the MMR. However, in India, data show that among women who had had four or more antenatal check-ups, 70% delivered in institutions compared to 7% for those who had had no antenatal check-up.[13] This makes a case for improving antenatal services as a means of encouraging women to deliver in institutions or at least to seek the services of a skilled provider. Table 6 shows the correlation between any type of ANC received and delivery by a skilled attendant or in a medical facility in a few selected States. Clearly, the greater the reach of ANC, the more the likelihood of delivering in safe hands or in a facility.

Table 6. Correlation between antenatal care, presence of skilled birth attendant, and institutional delivery in selected States

States	Mothers who received antenatal care (%)	Institutional births	Deliveries attended by a skilled attendant (%)
Andhra Pradesh	92.7	49.8	65.2
Gujarat	86.4	46.3	53.5
Rajasthan	47.5	21.5	35.8
Bihar	36.3	14.6	23.4
All India	44.0	33.6	42.3

Source: NFHS-2 1998–1999

If institutional deliveries need to be promoted, women must have access to an institution capable of offering EmOC at a reasonable distance from their homes. Considerable infrastructural and human resources will need to be pumped in to ensure functioning referral sites that can offer EmOC at first referral units (FRUs) and community health centres (CHCs) (Table 7).

Postpartum care

The recommended protocol for postpartum care in the current RCH Programme is three postpartum visits in the first eight weeks after birth. They are expected to take place within the first 72 hours, the first week of birth and at about eight weeks. If antenatal and safe delivery care is at best limited, then the status of postpartum care for women immediately following birth and even a week after the birth is woefully inadequate. The NFHS-2 data show

Table 7. Status of availability of equipment, infrastructure and human resources for emergency obstetric functions at district health centres, first referral units and community health centres

Staff, equipment, infrastructure	District hospitals (*n* = 210) (%)	First referral units (*n* = 760) (%)	Community health centres (*n* = 886) (%)
Operation theatre	98	93	86
Regular blood supply	60	12	8
Delivery facility	91	89	73
Separate aseptic labour room	44	36	28
Functional vehicle	91	73	61
Obstetrician/gynaecologist	78	48	28
Paediatrician	78	37	19
Anaesthesiologist	70	22	10
Medical staff trained in emergency obstetric care	19	17	11

Source: Adapted from the Facility Survey, GOI 2003

that despite the existence of postpartum complications (bleeding in 11% and high fever in 13%), only 16.5% of women received any form of postpartum care during the first eight weeks after delivery. Of these, less than 15% were seen during the first 48 hours and about 30% within the first week. Lack of postpartum care by a trained provider also implies lack of newborn care.

Safe abortion services and post-abortion care

Unsafe abortions constitute about 9% of maternal mortality. Although a favourable legal environment exists, policies that govern human and physical resources contribute to a mismatch between the demand and supply for abortion services. This results in many women seeking services at centres that are not recognized as providing safe abortion services. The number of abortion centres has risen, but they continue to be in the private sector and in urban areas. Medical termination of pregnancy (MTP) in India can only be provided by a medical doctor certified for the purpose. Registration and certification of facilities as legal abortion-providing sites is fraught with cumbersome and bureaucratic delays.

The predominant method of abortion used is dilatation and curettage. Efforts have been made in RCH-I to train medical providers in the use of manual vacuum aspiration (MVA) but these have not been widespread. There has been resistance to enabling mid-level providers to use MVA. This ignores that fact that there is a significant body of experience globally to suggest that mid-level providers can successfully use MVA.

Although it has been documented that the majority of abortions in this country are due to unwanted pregnancies among married women, the facilities for post-abortion care, particularly post-abortion contraception, are limited.

Programme and policy review

Table 8 reviews the programme strategies of two national-level flagship projects—CSSM and RCH-I, which did attempt to address MMR through the promotion of institutional deliveries and access to essential obstetric care. However, there were no significant declines in the MMR and IMR.[11,15]

Lack of success in the CSSM and RCH-I Programmes can be attributed to the following factors, ranging from community involvement to management and accountability:

Community involvement

- Little community mobilization around MMR
- Despite funding support for transport, little community advocacy or monitoring of *panchayats* to ensure that the funds were utilized by those who need them the most.

Policy and planning

- Continuing focus on interventions that made no contribution to reduction in the MMR, such as training of TBAs
- Lack of specialists in FRUs has been a long-standing issue. Until recently, no policies have been articulated to look at options such as nurse–anaesthetists or training MBBS doctors in obstetrics and anaesthesia. Fear of public litigation following the Consumer Protection Act and medical negligence are real issues. However, there is no policy to look into mechanisms for providing indemnity to providers taking risks to manage EmOC.
- Availability of blood remains an issue. Stringent requirements to establish blood-banking units are a deterrent and there is limited exercise of creativity in identifying options such as storage of blood.
- Bureaucratic hurdles to obtaining recognition as a safe abortion site and lack of adequate training of medical officers and paramedicals in newer non-surgical abortion techniques.

Management

- Centralized planning of interventions with consequent lack of ownership at the State level
- Decentralization occurred in RCH-I to a certain extent, but efforts were severely constrained due to lack of State capacity for planning and management.
- Poor management skills among medical professionals in administrative positions led to lack of coordination among training, supplies and monitoring.
- Tardy and disjointed interventions to provide EmOC and equip FRUs resulted in partly equipped FRUs, with either human resources or infrastructure or both lacking.

Table 8. Objectives and performance of the CSSM and RCH-I Programmes

Programme strategy	CSSM (1992–1996)	RCH-I (1997–2004)
MMR goal	Reduce MMR to below 200/100,000	Reduce MMR to 300/100,000 by 2002
Objectives	Improve safe motherhood through promoting timely identification and treatment of maternal complications, institutional deliveries, strengthening FRUs, training TBAs and other providers. Blanket approach across the country	Provision of essential obstetric care closer to the place of residence (such as PHCs) and EmoC at CHCs, FRUs, and adapt midwifery care at PHCs Districts categorized as A, B and C Financial envelope to better-performing States
Skilled birth attendant (SBA) at the community level	TBA training focused on five cleans, no identification of complications or building linkages with referral, only short-term training of ANMs. No training of existing ANMs, LHVs or nurses in midwifery skills, no attention to upgrading subcentre as a possible delivery site with an SBA	In C category (or areas with poor health indicators) districts: —midwifery skills of ANMs to be upgraded —private sector ANMs to be trained in midwifery —community-maintained, clean delivery rooms (done as pilots in a few States, and that only in the past one year)
Basic EmOC	Plans were focused only on comprehensive services at higher levels of care. Not considered at any level below FRU and no providers other than obstetrician/gynaecologists and anaesthetists considered for provision of emergency care	In B category districts (where the trend is towards institutional deliveries): —enhance PHC to provide BEmOC —train staff in midwifery skills —hire additional staff on contract basis —strengthen referral linkages with FRU No upgrading of skills of MBBS doctors, except in a few pilot cases. Very little actually happened in the absence of policy environment change.
Comprehensive EmOC	2500 FRUs planned; location of 1700 identified, only 600 started functioning, uncoordinated inputs	Make FRUs more effective to universalize institutional deliveries. Lack of human resources remains a significant issue. Only recently is there a move to train MBBS doctors in anaesthesia and caesarean section, so as to ensure trained providers at BEmOC and CEmOC levels.
Transport	No funds for transport	In all States, funds for transport provided to *panchayat* and national maternity benefit scheme announced, very little expenditure incurred

CSSM: Child Survival and Safe Motherhood; RCH: Reproductive and Child Health; MMR: maternal mortality ratio; TBA: trained birth attendant; FRU: first referral unit; PHC: primary health centre; EmOC: emergency obstetric care; CHC: community health centre; ANM: auxiliary nurse–midwife; LHV: lady health visitor
Sources: World Bank 1997; Reproductive and Child Health (RCH)-II 2004; Registrar General of India 1998

Expanding access and improving quality

- Failure to reduce the number of deliveries taking place in unskilled hands by expanding the number of skilled providers at the community level
- Training of providers not focused on competency building of skills to address pregnancy and delivery complications
- No effort to train the ANM in essential obstetric functions, which could serve to prevent deaths due to some complications and stabilize women before referral to FRUs.

Monitoring and accountability

- Mechanisms for reporting and auditing maternal deaths do not exist. Unless large-scale surveys (expensive and cumbersome) are conducted, the true picture at the district level and below is largely unknown. Even in FRUs it is difficult to obtain data on the number of emergency cases and interventions performed.
- Programme monitoring and supervision at all levels is not clearly articulated, resulting in lack of accountability of staff.

Linkages with providers in other sectors

- The private sector, which provides a significant share of Safe Motherhood interventions, is largely out of the realm of regulation and accountability. Few linkages have been established with the private sector, beyond contracting staff on an individual basis.
- Non-governmental organizations (NGOs) that work closely with communities to raise awareness about maternal mortality issues are hampered by the lack of active referral linkages with primary or secondary centres in the public sector.

Pattern of expenditure on interventions for women's health

Interventions were introduced under the RCH Programme to address the third delay for maternal mortality. Equipment kits and drugs were provided to FRUs for managing maternal complications, funds were released for need-based construction of operation theatres (OTs) and labour rooms at CHCs/district hospitals. An abstract summary of funds released to the States under the RCH Programme from 2001–02 to 2003–04 shows that only about 55% of the funds released for Civil Works—Major (for construction of OTs and labour rooms) and Minor (for repairs)—were utilized under the Programme (Appendix 7). Funds were released for hiring the services of anaesthetists but overall only 42.7% could be utilized because anaesthetists are not available even in the private sector at subdistrict and CHC levels in many districts.

Funds were also provided for hiring the services of obstetricians/gynaecologists in the private sector on contract, but only 56.24% of the funds could be utilized. About 30% of maternal deaths occur due to haemorrhage and require blood transfusion. However, because of the stringent rules and regulations under the Drugs and Cosmetics Act following the increased occurrence of HIV infection/AIDS, the availability of blood at the subdistrict/CHC levels became difficult. Under the RCH Programme, pilot projects were planned for setting up a regular and reliable supply of blood to PHCs/CHCs by linking them with the nearest District Blood Bank. However, action in this regard could be initiated only towards the end of 2003, when the Central Government issued the 'Guidelines for Setting up Blood Storage Centres at First Referral Units/CHCs'.

The scheme of 24-hour delivery services for promoting institutional deliveries was introduced, under which an honorarium was given to the Medical Officer, ANM and a Class IV employee for conducting deliveries at PHCs/CHCs at night. Under this scheme, an arrangement was made for the doctor and at least one nurse to be available on call beyond normal working hours. However, only 47.01% of funds released for this activity could be spent. For strengthening institutional services for maternal and reproductive health care, hiring the services of ANMs, Public Health Nurse/Staff Nurse and Laboratory Technicians (LTs) on contract was also provided for. All States spent over 90% of the funds released under these heads, thereby indicating the need for increasing the capacity of their institutions.

To address the second delay causing maternal mortality, a scheme for referral transport of pregnant women was introduced for hiring a vehicle to transport the pregnant mother to the appropriate health facility. However, the utilization of funds under the scheme was only 18.77% of the Rs 5.99 crore released for this activity. Even today, many mothers die on their way to a health facility. This was primarily because funds were initially released to *panchayats* but they were not properly sensitized and second, funds under this scheme were available only in some districts and that too not in the entire district, thereby making information, education and communication (IEC) and social mobilization efforts very difficult. In most cases people did not know that such funds were available in their area.

According to the NFHS-2, only 33.6% of deliveries were institutional (with figures ranging from 14.6% in Bihar to 93.0% in Kerala) and only 42.3% of deliveries are attended by a health professional (21.4% in Assam to 94.0% in Kerala). A scheme for training of *dais* was introduced in September 2000 in about 150 districts where safe delivery rates were less than 30%. According to this scheme, *dais* were to be trained for 10 days in two phases (of 6 and 4 days, respectively, with an interval of 4–12 weeks) at PHCs which conducted about 50 deliveries per month on an average (about 600 deliveries per year). It was assumed that each district would have about 20 such PHCs with the requisite number of deliveries so that *dais* (in batches of 10) could have hands-on experience in conducting safe deliveries. At least 12,000 deliveries (20 PHCs × 50 deliveries per month × 12 months) were assumed to be taking place in these 20 PHCs alone; this was assumed to be in addition to the deliveries conducted at CHCs and district hospitals. However, taking 10–15 lakh as the population of an average district and a crude birth rate of 30/1000, about 30,000–45,000 deliveries could be expected in the district annually (and at a safe delivery rate of less than 30%, 9000–13,500 deliveries could be expected to be safe—that too, institutional and home deliveries by trained personnel combined). Whether institutions could be identified in such districts where safe delivery rates were less than 30% is doubtful, but many States including UP, Uttaranchal, West Bengal and Orissa could utilize a significant proportion of funds available under this scheme. Once the *dais* had been trained, they should have been provided disposable *dai* delivery kits for conducting deliveries; however, these kits were not provided in most areas.

Limited IEC activities were conducted for increasing the awareness of mothers about danger signs during pregnancy and early recognition of maternal complications so that a timely decision could be taken of transporting the mother to an appropriate health facility. There is also a need to introduce the concept of 'birth preparedness' so that all planning for delivery is done in advance—where the woman will deliver, who will conduct the delivery, where to go in case a complication arises, who will accompany her, how will she be transported, what will be the source of funds and who will donate blood if required.

Action was also initiated under the RCH Programme for the treatment of reproductive tract infections (RTIs)/sexually transmitted infections (STIs). Kits for the diagnosis of RTIs/STIs and drugs for their treatment were supplied to the States. Funds released to the States for hiring the services of LTs on contract were fully utilized, thereby

increasing their capacity. However, this could be a short-term measure and State Governments need to fill up existing vacancies of LTs at CHCs and PHCs and sanction posts of LTs at those CHCs and PHCs where they have not already been sanctioned.

How has maternal mortality been addressed in the RCH-II Programme?[16]

The RCH-II Programme places considerable emphasis on reducing the maternal mortality with articulation of evidence-based strategies, improvements in managerial capacity, and policy changes to ensure a larger pool of providers for BEmOC and CEmOC. Several initiatives to guide policy modifications that enable a larger pool of providers to handle obstetric emergencies and to facilitate access to blood are under way. Blood storage guidelines have been drawn up and pave the way for easier access to blood at secondary-level facilities. The six-month training for medical officers in anaesthesiology has been tested and is likely to be scaled up. There are ongoing discussions on allowing ANMs to administer oxytocin and injectable antibiotics, opening several possibilities in upgrading an ANM to function as a skilled birth attendant (SBA), provided competency-based training and appropriate supervision mechanisms are in place. Overall, there is serious commitment to reducing the MMR. The document also shows that some of the lacunae of RCH-I have been addressed while planning for RCH-II. Some key issues of concern that might hamper efforts to reduce the MMR are discussed below.

Decentralized planning

In planning for RCH-II, States have been asked to develop their own strategies, based on a framework of interventions provided by the Centre. Experience shows that in several States, guidelines and frameworks provided by the Centre are unquestioningly used, regardless of the context and locale. This is particularly so in the poor-performing States where management capacity among health planners needs substantial strengthening.

Expanding access to safe deliveries

RCH-II will promote safe delivery through SBAs at the domiciliary level or in institutions. SBAs from the private sector will be trained as entrepreneurs, but there is little understanding of how they will link to referral centres and what are the supervisory and accountability mechanisms. RCH-II places a high priority on strengthening FRUs and enabling 50% of PHCs to function as 24-hour service delivery centres with the flexibility to contract out services. There is little discussion on the delegation of responsibilities beyond medical officers. Nurses are still left out of the realm of provision of obstetric services. Discussion on mapping and prioritization based on utilization is inadequate. Interventions for safe abortion are still restricted to public sector facilities and medical doctors, despite the evidence that mid-level providers can use MVA and thus bring abortion services closer to women.

Role of ANMs in RCH-II vis-à-vis expectations in MMR reduction

The frontline worker for all interventions is the ANM. The ANM will be strengthened to deliver essential obstetric care, but will continue to perform the same set of duties she currently performs. There is no reflection of concern for the high workload of the ANM. Focus on one element invariably leads to neglect of other issues. One of the critical issues for poor service access is the fact that ANMs do not stay at designated subcentres for a variety of reasons, ranging from security, poorly equipped centres, or lack of accountability. None of those reasons have been seriously considered. Little is discussed beyond behaviour change communication (BCC) on the imperative, and the advantage of engaging communities through NGOs, community groups and other mechanisms to improve community awareness, and use community resources in referral and monitoring. The document falls substantially short on this aspect.

Issues of management and accountability

PHCs are to be upgraded and staffed by a team of medical officers (MOs), LHVs and ANMs. There is no discussion on accountability, or a strategy on how to deal with abseentism, both of which are issues which contribute to the low utilization of PHCs. It is proposed to engage management graduates to strengthen management at the district level. However, general management professionals will have little impact on the day-to-day management of clinical services. Medical professionals need an ongoing infusion of public health management principles that apply to day-to-day functioning of the health services. Provision of incentives for good performance is suggested in the RCH-II document. However, motivation for improved organizational and professional commitment, particularly where low morale leads to absenteeism and poor service quality has not been considered.

Involvement of the private sector

RCH-II places considerable emphasis on increased private sector involvement in a range of RCH services, including safe delivery. There is little discussion on accreditation and monitoring of private providers, particularly with regard to equity issues.

Review of past programmes, performance and design of future programmes for reducing the MMR show that unless there is sustained commitment, a favourable policy environment to address outreach and improve technical competence, bolstered by adequate financial resources for infrastructure and equipment, the goal will remain elusive.

Infant and child health interventions

Unlike in maternal mortality, India's performance in improving infant and child health and survival has been significant. However, the slow pace of decline in the IMR, widespread infant and child malnutrition, and slow progress in complete immunization are key areas of concern.

Status and trends in IMR and U5MR

Infant mortality can be classified as perinatal (from 28 weeks' gestation to seven days of birth), neonatal (birth till the first four weeks of life), and post-neonatal (after the first four weeks of life). Perinatal and neonatal mortality are largely determined by gestational age and care at delivery. As infant mortality declines, post-neonatal mortality shows more rapid declines. Perinatal and neonatal mortality constitute a larger proportion of the IMR. The rate of reduction in infant mortality has slowed down considerably. The average decrease in IMR was around 3% each year in the two decades preceding 1992. In the subsequent ten years, the decline has been of the order of 1.5% each year.[15] Data from the 2002 Sample Registration Survey (SRS) show an IMR of 64. Of this, about 69% occur in the neonatal period. The U5MR has also dropped from 144 per 1000 in the early 1980s to 89 per 1000 in 1999.

Table 9 shows that about 50% of all under-five deaths occur in the first month of life. Progress in reducing the IMR and U5MR has been uneven across States, with the poor-performing States in the north, central and east showing far higher rates than those of the better-performing States in the south and west (Appendix 2).

Causes of neonatal, infant and under-five deaths

Infant and child deaths are the result of several risk factors. Proximate determinants of infant and child survival include a mix of preventive and some curative interventions: maternal immunization with TT, safe delivery, birth spacing of more that 24 months, breastfeeding and appropriate weaning, complementary feeding, access to safe water and sanitation, immunization, administration of vitamin A, ORT, and antibiotics for neonatal sepsis, and respiratory and other infections.

Table 9. Under-five child deaths in India

Age completed	Under-5 child deaths (cumulative)
Day 1	20%
Day 3	25%
Day 7	37%
Day 28	50%
1 year	75%
5 years	100%

Source: Reproductive and Child Health (RCH)-II Programme Implementation Proposal—extrapolated from the Indian Council of Medical Research (ICMR) study data (2003)

Neonatal mortality

Young maternal age, short birth intervals and lack of safe delivery are factors that contribute to high neonatal mortality. When the maternal age is less than 20 years, the neonatal mortality rate (NMR) for this age group is 66; where the birth interval is less than 24 months the NMR is 77% compared to 37% when the birth interval is between two and four years. The NMR is lower among babies born to women who receive ANC, deliver in the hands of a skilled attendant, and receive postnatal care (22%), compared to 54% among those born to mothers who receive no care, and 37% for those who receive one or two types of care.

A review of the Registrar General of India's (RGI's) Cause of Death Survey for rural India shows that conditions originating in the perinatal period were among the top 10 killers of infants and included diseases of the respiratory system (62.5%), other infectious diseases (15.5%) and parasitic diseases (4.8%).[17] Only about 20% of deaths were due to congenital malformations, central nervous system, cardiovascular, haematological and metabolic disorders or trauma.

Among children in the age group of 1–4 years, about half of the deaths are accounted for by respiratory diseases, anaemia, diarrhoea, dysentery, malaria, typhoid and influenza. This implies that, given prompt recognition at home, accurate diagnosis (clinical and laboratory-based), and early and complete therapy, the majority of these conditions are completely amenable to treatment.[17]

Malnutrition is responsible for 56% of under-five deaths. The risk of death rises among children who are mildly, moderately and severely malnourished. On an average, a child who is severely underweight is 8.4 times more likely to die from infectious diseases than a well-nourished child.[18] Approximately 47% of children under three years of age in India were undernourished and 45% had stunted growth. Exclusive breastfeeding is an important child survival intervention. According to the NFHS-2, the proportion of breastfed infants was 37%, and at 6 months about 19%.

Review of infant and child health programmes

The package of interventions in CSSM and RCH included essential newborn care, immunization, administration of vitamin A, and appropriate management of diarrhoeal diseases and acute respiratory infections (ARIs). Nutritional supplementation for children in the age group of 0–6 years is provided through the Integrated Child Development Services (ICDS) scheme. The major thrust of programmes so far has been on reduction of post-neonatal mortality. Neonatal mortality, which is closely linked to safe motherhood interventions, has been largely untouched.

Essential newborn care

Well-trained (TBAs), who can provide the essential package of newborn care, can contribute to dramatic reductions in the NMR and IMR. Home-based care of sick newborns, education of mothers and training of birth attendants have been largely unsuccessful. Training of TBAs in newborn care has been implemented sporadically.

Immunization

Immunization has been a consistent thrust of programmes since the early 1980s. The rates of children immunized against all the six major vaccine-preventable diseases have fallen (NFHS-1 and NFHS-2). While the large majority of children receive one or more of the antigens, only 42% are completely covered with all the six antigens (Appendix 3). Data from the Evaluation and Intelligence Division (2002) of the Ministry of Health and Family Welfare (MOHFW) in 176 districts showed an average decrease of 15.4% in full immunization rates over the past five years.[15] Only 66 districts showed an average increase of 9.4% in immunization coverage over the five-year period. In 2001, the average BCG–measles drop-out rate was 14.4%, indicating low acceptability or demand for immunization services. The pulse polio campaign contributed its share to low immunization coverage. Community sensitization generated the belief that intake of polio vaccine was sufficient to protect against all vaccine-preventable diseases, and thus resulted in low turnout for other vaccines.

Management of acute respiratory infections (ARI) and diarrhoeal diseases

Diarrhoeal diseases and ARIs constitute a significant cause of morbidity and mortality in infants and children. Although several factors contribute to this, ready availability of ORT and antibiotics for ARI can mitigate the impact to a certain extent. The NFHS data reveal that mothers' knowledge of the signs of diarrhoea that prompt care-seeking is low (63%), few mothers (29%) know that there should be an increased intake of fluids, and one-third of mothers restricted fluid intake. The use of oral rehydration salt (ORS) was low among children who had diarrhoea (26%) even though 62% of the mothers were aware of the need to use ORS. The data also suggest that 36% of children with ARI were not taken to a facility or provider.

Programme and policy review of past programmes

Reduction in the IMR has been a goal of programmes even before the CSSM Programme was initiated. This paper reviews child survival interventions in the CSSM and RCH-I Programmes.

Table 10 shows that no specific or focused efforts were implemented in RCH-I despite the findings from the CSSM project. Possible reasons include:

Community involvement

- Community needs assessment, a valuable planning tool, focused almost exclusively on contraceptive needs.
- Community-based interventions, such as home management of diarrhoea, early recognition of ARI, home-based care of the newborn, were neither comprehensive nor implemented effectively.

Planning

- The shift in focus of RCH-I to a range of interventions with the target-free approach as its main thrust area resulted in poor planning and attention to child health interventions.
- There were no clear programmatic outcomes for child survival interventions in RCH-I, except for the overall reduction in the IMR and reaching immunization targets.
- Increasing the responsibilities of the ANM reduced the amount of time for community and outreach interventions necessary for child survival.
- Campaign approach to polio immunization caused disruptions in routine immunization programmes.

Improving the quality of services

- Lack of skills training for birth attendants to manage newborn care
- Newborn interventions were limited to facilities.

Issues of governance, accountability and management capacity have already been discussed in the section on Programme and Policy Review.

Pattern of expenditure on interventions for child health

To increase the outreach of maternal and child care services, the Government of India released funds to States for conducting RCH camps in remote PHCs. These camps were so successful that States requested the Centre to allow them to conduct more such camps. Accordingly, expenditure under this scheme was over 130% of the allocated budget.

For services to children, vaccines, drug kits and equipment kits were supplied by the Central Government. Interventions for child health have been overwhelmed by the Intensified Pulse Polio Immunization (IPPI) Programme. About 87.8% of the allocation for child health under the RCH Programme was for the IPPI and if other activities for immunization are included, about 90.2% of all allocation was for the IPPI Programme and immunization-related activities. Enormous savings would be made once poliomyelitis is eradicated from the country. As a result of the IPPI campaigns, cases of poliomyelitis have also come down significantly but poliomyelitis has not been eradicated even after 10 years of implementation of the Programme; other countries where such campaigns were quite successful could manage to eradicate poliomyelitis within a span of 3–5 years. Of late, cases of poliomyelitis have been found in those States

Table 10. Child health interventions—objectives and achievements

Programme strategy	CSSM Programme (1992–1996)	RCH-I Programme (1997–2004)
IMR and U5MR goal	Reduce the IMR to below 60/100,000 and U5MR to 70 IMR in 1996: 72/1000; U5MR: 115	Reduce the IMR to below 60/100,000 by 2002 IMR in 2002: 64/1000
Objectives	• UIP Plus—immunize against all the six VPDs and maternal TT • Management of diarrhoea and ARIs • Supplementation with iron and vitamin A • Essential newborn care	• Essential newborn care • Adapt and introduce IMCI • Immunization (support for eradication of polio) • Supplement vitamin A
Immunization	• Immunization programmes received significant emphasis, improving quality of the cold chain, improving coverage in identified pockets of low coverage. • Maternal TT coverage increased from 53.8% to 67%. Complete coverage for children rose to 52.4% from 35.4% (NFHS-1) (MICS)	• Continued emphasis on immunization programme, introduction of polio campaigns • Reversal in complete immunization coverage
ORT/ARI	• Increase in the supply of ORS • Social marketing for ORS • The number of children with diarrhoea who received ORS packets ranged from 30.6% (NFHS) to 34.5% (MICS) • Co-trimoxazole supplied to ANMs • Medical and paramedical workers trained in the management of ARIs • Impact data NA	• IMCI adapted for the Indian context but late in the life of the project • Interventions similar to those in CSSM • Marginal change in the indicators
Administration of vitamin A	• Five prophylactic doses of vitamin A for children in the age group of 9 months to five years, and prophylactic iron • Vitamin A coverage dropped from 64% to 62%.	• Interventions similar to those in CSSM • Marginal change in the indicators
Essential newborn care	• Training medical and paramedical personnel to achieve reduction in the NMR • NMR dropped from 51.1% (NFHS) to 47% (SRS)	The NFHS data show that only 16.5% of women were visited in the first two days of birth and only 42% of deliveries were conducted by skilled personnel (who would know how to manage newborn complications)

CSSM: Child Survival and Safe Motherhood; RCH: Reproductive and Child Health; IMR: infant mortality rate; U5MR: under-five mortality rate; UIP: Universal Immunization Programme; VPDs: vaccine-preventable diseases; TT: tetanus toxoid; ARI: acute respiratory infection; IMCI: Integrated Management of Childhood Illnesses; NFHS: National Family Health Survey; MICS: Multi Indicator Cluster Survey; ORT: oral rehydration therapy; ORS: oral redydration salt; ANM: auxiliary nurse–midwife; NMR: neonatal mortality rate; SRS: Sample Registration System

that had no case since the past 2–3 years, which is disturbing. Because of undue emphasis on IPPI campaigns, most other interventions for maternal and child care have taken a back seat—full immunization coverage rates have fallen consistently in all States and people in communities have become complacent that it is the responsibility of the Government to provide immunization; for one IPPI campaign all workers are occupied for 7–10 days. If four such rounds are conducted in a year, considerable time is spent on IPPI activities alone.

The National Poliomyelitis Surveillance Project (NPSP) was started in 1997 and Surveillance Medical Officers (SMOs) were recruited and placed at State and district levels. WHO is supporting them on a contract basis. The SMOs have definitely added to the capacity of the health system, but the question of sustainability will always be there because they are on contract and can be withdrawn at any time. In the long run, the capacity of programme managers at all levels—the Centre, the States and the districts—will have to be increased through recruitment/placement of full-time, regular government officers.

How has infant and child mortality been addressed in RCH-II?

The RCH-II design document recognizes that strengthening the health systems is critical for effective child health interventions. The focus is on reduction of the NMR, through implementing an Integrated Management of Neonatal and Child Illnesses (IMNCI) Programme, and strengthening the Universal Immunization Programme (UIP). The IMNCI will focus on systems strengthening, facility support as well as household management of the sick newborn and child. Substantial management and logistics support is planned. The Programme has the following concerns:

- Interventions for child health lack focus.
- There is no discussion on measures to address malnutrition either through health interventions or through convergence activities.
- Neonatal mortality is proposed to be addressed through the IMNCI rather than through focused interventions as part of maternal mortality reduction.

- Global reviews of the IMCI have shown that constraints of the health systems severely limit the effectiveness of the approach. One of the fundamental factors underlying poor progress in health indicators in India is the weakness of the health system.
- Pre- and in-service training shows several lacunae. The adapted Indian version of the IMNCI has added on the element of neonatal care and reduced the period of training from a maximum of 11 days to a maximum of 8 days and thus the efficacy of the training programme is limited, particularly for paramedical workers.
- It is proposed to add the IMNCI to the ANM's list of duties, without reviewing her already overloaded job chart.
- It is proposed to involve *anganwadi* workers (AWWs) in the management of ARIs and diarrhoeal diseases. It is unclear what type of village-level monitoring mechanisms will be put in place and how much training and supervision will be provided.

Child health interventions need focusing and laying down of clear priorities, and short- and medium-term goals to address the high and stagnant IMR and U5MR.

Family planning and reduction in the total fertility rate (TFR)

Family planning has been part of India's family welfare programmes and has received the most attention, albeit with mixed results. Family planning was originally conceived as constituting sharply targeted programmes and formed a substantial portion of the day-to-day workload of peripheral health workers. With the introduction of the target-free approach, the pressure has lessened somewhat but it still continues to be an important component of the programme. Population stabilization is a key goal of all policies.

Family planning programmes can reduce the MMR in two ways: by reducing the number of pregnancies and by reducing 'high-risk' pregnancies—at young ages and with short birth intervals, which have a higher likelihood of becoming obstetric emergencies, with consequences for maternal and child survival. Family planning reduces maternal deaths by reducing the number of unwanted pregnancies. It has been estimated that about 35% of all maternal mortality in Asia could be reduced by family planning services.[12]

Status and trends in TFR

The TFR has declined in all States since the early 1970s. It has declined by almost half a child in the six-and-a-half years between NFHS-1 and NFHS-2. However, the national progress must be seen in the context of the performance of individual States. Replacement-level fertility, or close to this level, has been reached in Kerala, Tamil Nadu, Karnataka, Goa, Andhra Pradesh, Himachal Pradesh, Delhi and Punjab (Appendix 4).

Data from the NFHS-2 (Appendix 5) show that the total wanted fertility rate was lower by 25% than the TFR. This means that if the women who considered their pregnancy unwanted had a means of prevention, the TFR could conceivably have dropped to about 2.1, very close to the replacement-level fertility, barring Uttar Pradesh, Bihar, Rajasthan, and a few States in the north-east.

Interventions to reduce TFR

Key interventions to reduce fertility are: increasing the use of contraceptives, raising the age at marriage, provision of a reproductive health care package to address other health needs, legalizing abortion, improving child survival and enabling education of girls. In addition, several States have now articulated coercive measures to enforce the two-child norm, seen as necessary for reaching replacement-level fertility.

Performance review of family planning programmes

The challenge is in meeting unmet needs, through expanding contraceptive choices, providing high-quality holistic reproductive services including counselling and follow up, and promoting male responsibility in using contraceptives, especially condoms, given the HIV/AIDS situation in the country. Providing contraceptive services to a population of a few hundred million adults is a daunting task and the Family Planning Programme in India has been able to achieve considerable success in bringing about changes in many demographic indicators.

Use of contraceptives

Overall, the use of contraceptives (modern methods) has risen to 42.8% (NFHS-2). Female sterilization is by far the most popular method of contraception practised in India, accounting for 75% of contraceptive use. The Family Planning Programme is skewed towards women, with male methods of contraception (condoms and vasectomy) accounting for 5% of total contraceptive use. The use of contraceptives increases with age, peaking at 67% for women in the age group of 35–39 years.

Data from the NFHS-2 on the unmet need for contraceptive services are about 8.3% for spacing and 7.5% for limiting methods. The State-level variations for unmet need range from 7% in Punjab to 25% in Uttar Pradesh and Bihar, and about 36% in Meghalaya (Appendix 5). Unmet need is the highest among young women below 20 years of age (27%).

Almost one-third of births occur within 24 months of the previous birth. Evidence shows that shorter birth intervals are associated with a higher IMR.

Choice of contraceptive method

The choice of the method of contraception is dependent on the quality of services, including information on side-effects, follow up, informed choice and a broad range of

Table 11. Critical interventions for mothers and children

Critical interventions for all pregnant women	Critical interventions for infants and children
1. Establishing contact in the first trimester with every pregnant woman for provision of comprehensive antenatal care (including early registration, a minimum of three antenatal check-ups, of which one is conducted at a primary health centre or similar institution and the woman is seen by a medical officer). For details on ANC at each level *see* Appendix 6. 2. Access to safe delivery through a skilled birth attendant at home and appropriate transport and communication link with a suitable back-up facility	1. Ensure that all women have access to safe deliveries to ensure newborn care. 2. Detect and manage early signs of newborn illness through regular postpartum visits. 3. Mothers and caretakers must be trained in home-based care of the newborn. 4. Improve immunization coverage through mobilization of the community and unfailing, regular immunization visits. 5. Use of appropriate treatment algorithms for diarrhoea and acute respiratory infections. 6. Re-invigoration of school health programmes to periodically examine children for helminthic and other infections, and malnutrition, especially anaemia among girls. 7. Concerted efforts must be made to reduce malnutrition through convergence with the Integrated Child Development Services (ICDS) Scheme, and community support to reduce the burden of malnutrition and attendant ill health.

methods. Other than sterilization, options in the Programme for women are limited and include oral contraceptive pills and intrauterine devices. NFHS-2 data show that only 21.7% of women who accepted a method were told about the side-effects of the method and 70% received follow-up services.

How does RCH-II address population stabilization?

RCH-II proposes a range of interventions to address reduction in the TFR. They include: expanding the method mix, involving private providers in the provision of services including sterilization, increasing the overall pool of providers for family planning services, intersectoral convergence with the AWW to serve as a village-level depot holder, increasing compensation payments to acceptors, advocacy through the mass media, social marketing, and involving Panchayati Raj institutions (PRIs) to serve as depot holders. Issues of concern include the following:

- Implementing the gamut of interventions will require considerable administrative and managerial capacity.
- The underlying assumption appears to be that expanding the supply will result in increased demand. This may not be entirely true. Substantial efforts at the community level through interpersonal approaches by the ANM or LHV might be more effective.
- Involvement of the private sector carries the risk of lack of accountability.
- The increase in compensation for wages lost may result in corruption and/or coercion.
- The newer methods to be introduced (standard days method [SDM], lactational amenorrhoea method [LAM]) will require that potential users be highly motivated, fully aware of the side-effects and have the wherewithal to switch methods. Thus, the ANM needs to have the time and skills to motivate and address the needs of users of these methods. Currently, the functions of counselling and education do not find a place in the ANM's heavy workload.
- There is no description of how planning for contraceptive delivery will be done in a community. The earlier Community Needs Assessment (CNA) approach does not seem to find a place in the document.
- Involvement of the mass media and satisfied couples to propagate the message of family planning has been tried in the past and has limited reach for positive behaviour change. Larger community mobilization efforts such as through community groups of women will be necessary and more effective.

It appears that population stabilization in RCH-II is a stand-alone effort. It is conceptually difficult to link it to interventions for maternal mortality and child health. Yet, system providers will need to integrate this at each level to ensure smooth functioning and achieve the reductions in TFR that will contribute to reduced MMR and IMR.

PART II

Issues related to the auxiliary nurse–midwife (ANM) and primary health centre (PHC)

Auxiliary nurse–midwife

The ANM is a worker who is most directly in touch with the community. The ANM is expected to implement a range of programmes and her duties range from conducting the community needs assessment to conducting delivery at the subcentre or at home. Community- or field-level outreach is the responsibility of the ANM. She also provides preventive, promotive and selected curative services for safe motherhood, child health, immunization and family planning. Her duties include education, motivation, counselling and service provision.

Training and effectiveness of ANMs

ANMs receive an initial 18-month training, supplemented by in-service training, often focusing on additional duties. Over the years, the training programme of ANMs has moved from being knowledge-based to a hands-on, skill-building approach.

In an observational study of ANMs conducted across the country, where services were graded—excellent (>75% of subtasks performed well), satisfactory (>50%–75% of tasks well done) and poor (50% of tasks well done)—none of the ANMs performed excellently in any of the major services (ANC, delivery, prenatal care [PNC], immunization and contraception).[19] While about 80% scored a satisfactory grade for delivery, immunization and family planning, 44% scored poorly on ANC. This finding is validated by other studies where it has been pointed out that the ANM's focus is primarily on family planning and immunization, leading to attrition of skills in other areas. A study in Andhra Pradesh pointed out that there is a gap of about four years between the ANM's training and being appointed to her job.[20]

Reporting requirements and supervisory support

Several studies (before and after the target-free approach) found that the stress on achieving sterilization targets left them with little time to attend to their other duties.[21] Outreach services are uneven and often only to houses close to the road, or in the vicinity of the subcentre or *anganwadi* centre. The ANM has to maintain thirteen registers and submit seven reports to the PHC. Supervision is focused on the reports and little other support is provided. The LHV is expected to support the ANM. However, her own lack of professional competence and the high priority accorded to records and achievement of family planning targets have rendered her more of an administrative supervisor than a clinically competent paramedical provider who can support the ANM in her day-to-day clinical duties.

Constraints in operation

Overburdening of the ANM has been a consistent finding in several studies. Lack of physical infrastructure, equipment and basic amenities at the subcentre are major reasons for the low motivation and absenteeism among ANMs.

The majority of subcentres do not have the facility for storing vaccines. Thus, the ANM has to procure the vaccine from the PHC on the days when immunization is offered at the outreach sessions or at the static clinic. Unless the ANM has a vehicle or can prevail upon somebody to give her a ride, she spends the better part of the day in going to the PHC, obtaining a supply of vaccines and then travelling to the habitation/village. Even assuming that the distance from the subcentre to the PHC is about 5 km, lack of adequate transport and the weight of the supplies (estimated to be about 15 kg for the essential equipment required to conduct ANC and immunization) make the task difficult. It is clear that there is no time to conduct village-level education sessions or house-to-house visits for newborn and postnatal care.

Lack of support and an increasing workload

The ANM shoulders more than a fair share of her burden since the workload has not decreased. In fact, there is ever-increasing responsibility, as new and emerging diseases appear on the horizon. Thus, Directly Observed Treatment, Short-course (DOTS) for tuberculosis, follow up, and now identification of women in need of prevention of parent-to-child transmission of HIV/AIDS are also her responsibility.

Primary health centre issues

The PHC is expected to serve as a point where the ANM receives her support, supervision, equipment, guidance and is the referral link for all cases, including provision of BEmOC and stabilization/referral of emergency conditions. It appears that the PHC is the weakest link in the chain. A majority of PHCs do not have the full complement of staff. In addition, shortages and misuse of transport funds preclude the ability of the MO to travel to the field and back up the ANM with the requisite service delivery and supervisory support. The glaring gap in the number of women physicians at every level of the chain is a critical factor in the gender inequity in access to services. Several studies indicate that utilization of PHC services for maternal and child health services is poor (less than 25%), and they either have a negative image or are unknown in many communities. One PHC study in Karnataka showed that most MOs do not stay at the PHC but commute, thus spending little time at the PHC and hardly any time travelling to the villages. Other issues include lack of equipment and supplies, poor drug availability, and low budgets for maintenance and repair.

PART III

Recommendations

Interventions to reduce the MMR, IMR and child mortality are well known and evidence-based. Where a spectrum of services starting from the community level through referral and linked to appropriate policy changes have been implemented in a framework of equity and access, maternal, infant and child mortality have come down significantly (Appendix 6, Table 11). The challenge is in implementing these interventions, making policy and programme changes to fit local needs, and ensuring functional health systems.

Increase in financial resources is necessary to expand the number of facilities that can provide EmOC and facility-based neonatal and child care; providers need to be reoriented and retrained to enable a high degree of technical competence and acquire multiple skills; sound and reliable measurement standards and monitoring systems need to be instituted to enhance performance and accountability. Availability of supplies, drugs and equipment must be ensured to enhance the quality of care and, finally, a high level of community involvement through direct contact with people and convergence with other sectors is necessary to ensure utilization.[22]

To ensure these interventions, certain actions are proposed at the level of the community, subcentre, PHC and CHC. These actions need to be promoted within a strong organizational and management framework for public health care at all levels, complemented by social mobilization efforts through effective engagement with civil society. The strengthening of this framework is critical for service delivery sites to offer equitable and high-quality services. Accountability is the second crucial factor in the improvement of service delivery and is related to the overall standard of governance. Key areas that need to be addressed include the following:

- Political and policy commitment to making mothers and children the focus of programmes within a well-functioning public health system;
- Clear-cut distinctions and role clarity between clinical service providers, public health managers, paraprofessional staff, and an appropriate mix of each in all facilities;
- Review and revamping of institutions (medical and nursing colleges, ANM training schools, schools and departments of public health), curricula and teaching/training methods for medical and paramedical personnel to ensure that they cater to the public health needs of the country;
- Policy changes (with appropriate incentives, deterrents and decentralized accountability mechanisms) to ensure that all personnel in the public sector system actually are in place at the facilities they are posted to;
- Improving management capacity at the State, district and block levels to ensure that all inputs are coordinated, and that day-to-day functioning of health facilities is not disrupted by lack of staff or supplies;
- Appropriate back-up is present at each successive higher facility for prompt referral and action. Wherever possible, introduce the use of newer technologies such as the internet and mobile phones, to facilitate rapid communication and response systems, thus circumventing some limitations such as long distances and inadequate or expensive transport;
- Mainstreaming of gender sensitivity and equity into all aspects of training and services;
- Availability of and adherence to standards and protocols at each level of the system;
- Technical support and supervision of all services to maintain a high quality of service;
- Unhampered supply of drugs and equipment;
- Decentralization of procurement systems for items of a certain value;
- Frequent refresher training through onsite or distance learning to ensure that all providers are up-to-date with knowledge and skills in service provision;
- Effective monitoring and evaluation systems to ensure that valid, reliable and current data are available at every level and are being used to further programme implementation and correction;
- Increasing community involvement in ensuring high quality could be through processes such as involvement in registration of births and deaths, death audits, particularly maternal and newborn deaths, and periodic review meetings between the community and staff (the PRI could be an appropriate mechanism).

Community-level interventions

At the community level, two separate sets of interventions are necessary. The first set of interventions can only be implemented through education, sensitization and mobilization, and is focused towards advocacy for maternal and child health. The objective is to make communities aware that all deliveries need a skilled attendant at birth; that management of sick newborns and children involves a set of skills that mothers and other caretakers need; and ready access to a health care facility through transport and financial resources is essential. This can be done through community groups such as self-help groups (SHGs), with the active involvement of *panchayat* representatives.

The second set of interventions is related to providers at the community level. The following options can be considered:

- Posting a female community health worker at each village and habitation, who serves as the link between the ANM

and the community, and is empowered by a set of skills (basic clinical and communication), commodities and drugs that earn her credibility as a valued resource person in the eyes of the community;
- Developing and strengthening a team of village workers including the AWW, TBA, and the village registered medical practitioner (RMP) (after appropriate training and certification);
- Posting a village-level female health worker to head the village-level team and strengthening the team through training and exposure visits.

Domiciliary delivery

Where domiciliary deliveries are still high, women must be encouraged to access an SBA. The following are recommended:

- Reorientation of the TBA, with community sensitization to enable her to play an active role in identification of complications and supporting the skilled provider;
- All communities should have arrangements for transport and funds for referral, particularly for the poor and marginalized, which should be known to all;
- Skilled birth attendants should be available at the community level to manage safe delivery and sick newborns. Wherever possible, the ANM should be trained as the SBA, and supported by adequate referral;
- Sufficient ANMs must be trained in essential obstetric care and located at subcentres, within the easy reach of communities.

Institutional delivery

Promotion of institutional deliveries and ready access to institutional care for home deliveries with complications are two key strategies. While the choice of workers and service delivery site locations are dependent on the terrain, infrastructure, mobility and connectivity, distance–time and workload should be the considerations for the location of facilities rather than the population size. No community or habitation should be more than two hours away from an FRU that is equipped for emergency services.

Organization of clinical services

Clinical services can be organized in one of the following ways:

Subcentre strengthening

- The number of subcentres need to be expanded, so that one subcentre with the appropriate staff and infrastructure is available within a distance of a few kilometres of a community/habitation.
- Community education, counselling and motivation require a different skill mix and should be delinked from clinical service delivery functions for preventive and promotive health services. ANMs should use their skills for service delivery and outreach should be provided by a community-level worker.
- Subcentre services to be strengthened by posting a team of two ANMs, both trained in midwifery skills, with one being present at the centre and one providing outreach services on a rotating basis. One keeps the subcentre open and the other does outreach for clinical services such as ANC, immunization, or home visits for deliveries. The subcentre functions as a site for delivery, supply of intrauterine devices (IUDs) and possibly as an MVA site. The subcentre should always have one ANM with support from a village-level team or worker.
- For EmOC, referrals are made to a designated PHC/CHC/FRU (or similar set-up in the private sector) no more than 2–3 hours away. Wherever possible, subcentres should have telecommunication facilities so that plans for referral are foolproof.
- Supervisory and technical support is provided by a roving team located at the PHC, whose function is largely supervisory. It also provides assistance, surveillance and coordination, and supplies and logistics rather than service delivery.
- A proportion of the maintenance and supplies budget should be handed over to ANMs so that there is flexibility in the purchase of drugs and equipment to tide over periods of shortage or disruptions in supply.
- The *panchayat* should be the first level of accountability for village-level providers and frontline health workers, and the repository of health surveillance and death and quality audits.

Primary health centre

Currently, the high level of absenteeism and the low utilization of PHCs preclude their use as referral sites for EmOC. However, in many States, the PHC is the closest referral site. Thus, they need to be strengthened. Delegation of skills and functions should be done, given that medical professionals are reluctant to stay in rural areas, particularly the more remote sites. The following options are proposed:

- At PHCs, a team of at least two MOs trained in BEmOC should be available round the clock.
- As an alternative, the possibility of training nurses in BEmOC could be considered, in areas where there is a shortage of medical personnel or as back-up when MOs are not available. One option could be to train nurses in public health/advanced clinical skills to function as Nurse Practitioners, and perhaps manage PHCs, under guided supervision from the CHC. More MOs can then be moved up to the CHC level and form outreach teams to provide support to such Nurse Practitioners.

Community health centres and first referral units

The CHC is envisaged as the hub of the network of service institutions. They perform three major functions:

- Provide secondary-level clinical inpatient and laboratory services for RCH (including sterilization and EmOC) and a range of other health conditions;
- Provide technical support and supervision of staff at subcentres and PHCs;
- Provide managerial and logistic support for drugs, supplies and infrastructure maintenance.

Each district should have 2–3 FRUs depending on the terrain and travel time required to reach them. These FRUs have blood bank facilities, facilities for general and spinal anaesthesia, and specialists/trained MOs who can perform emergency surgical procedures, including caesarean sections. Another option would be to staff CHCs with about 4–6 MOs with short-course training in obstetrics, anaesthesiology and MTP services. At least 30% of medical providers should be women. In addition, CHCs should have counselling centres for HIV/AIDS, gender-based violence and family planning. In areas with a high prevalence of HIV/AIDS, interventions for prevention of parent-to-child transmission also need to be included.

Common to all interventions is the provision of female community health workers who take on the responsibility of community needs assessment, mobilization, counselling and education, reaching out to the most vulnerable, providing treatment for minor ailments, escorting or facilitating referral for maternal and newborn emergencies, and maintaining village/habitation-level records. This worker is also responsible for coordinating with the AWW, TBA and village-level informal providers to ensure minimal harmful practices and better regulation. Technical support is provided by the ANM, but the worker is accountable to the *panchayat* and/or community groups such as SHGs or other common interest groups.

Conclusion

To enable these recommendations, a clearly articulated strategy and a medium- to long-term objective for safe motherhood, consistency in pursuit of the objective regardless of distractions, decentralized planning, budgeting and management, technical support to administrators and medical providers, coordination among inputs to various elements, a long-term preservice training strategy and human resource development are required.

References

1. World Bank. *Improving women's health in India.* Development in Practice Series. Washington, DC: World Bank; 1996.
2. WHO/UNICEF/UNFPA. *Maternal mortality in 2000: Estimates developed by WHO, UNICEF, and UNFPA*. Geneva: World Health Organization; 2004.
3. Measham AR, Heaver AR. *India's Family Welfare Programme: Moving to a reproductive and child health approach*. Washington, DC: World Bank; 1996.
4. World Bank. Project appraisal documents (India): Reproductive and Child Health Project. Human Resources Division, South Asia Country Department II, South Asia Region, April 1997.
5. International Institute of Population Sciences (IIPS) and ORC Macro. *National Family Health Survey 2 (NFHS-2), 1998–1999*. Mumbai: IIPS, ORC Macro; 2000.
6. Bhat PN, Navaneetham M, Irudaya Rajan S. Maternal mortality in India: Estimates from a regression model. *Studies in Family Planning* 1995;**5:**217–32.
7. Government of India. National Population Policy 2000. New Delhi: Ministry of Health and Family Welfare.
8. Jill G, *et al.* What works: A policy and program guide to the evidence on family planning, safe motherhood and STI/HIV/AIDS interventions. Module 1: Safe Motherhood, Policy Project, 2003.
9. DeBrouwere V, Van Lerberghe W. *Safe motherhood strategies: A review of the evidence.* Antwerp: ITG Press; 2001.
10. Pathmanathan I, Liljestrand J, Martins JM, Rajapaksa LC, Lissner C, de Silva A, *et al. Investing in maternal health—learning from Malaysia and Sri Lanka.* Development in Practice Series. The World Bank, East Asia and the Pacific; 2003.
11. Assessment of RCH schemes and Financial Envelope (unpublished). Reproductive and Child Health Programme I. New Delhi: World Bank; 2003.
12. Maine D. *Safe motherhood programs: Options and issues.* New York: Center for Population and Family Health, Columbia University; 1991.
13. Sugathan KS, Mishra V, Retherford RE. *Promoting institutional deliveries in rural India: The role of antenatal care services.* National Family Health Survey Reports, Number 20. Mumbai: IIPS and Hawaii, USA: East West Population Center; 2001.
14. CSSM Programme Evaluation, Mother Care Matters, Mother Care Project. Arlington, VA: John Snow; 1996.
15. RCH II National Programme Implementation Proposal (PIP)—Draft, October 2004.
16. *Survey of Causes of Death (Rural), India. Annual Report, 1998*. Office of the Registrar General, Vital Statistics Division, 1998.
17. Pelletier DL, Frongill EA, Schroeder DG, Habicht JP. The effects of malnutrition on child mortality in developing countries. *Bulletin of the World Health Organization* 1995;**73**:443–8.
18. Visaria L. From contraceptive targets to informed choice. In: Radhika R, Jeejeebhoy S (eds). *Women's reproductive health in India*. Jaipur and New Delhi: Rawat Publications; 2000:331–82.
19. *Impact of integrated skill training (IST) and specialized skill training (SST) under RCH programme in the country*. *Evaluation Report*. New Delhi: National Institute of Health and Family Welfare; 2004.
20. Rangarao AP. Role and efficacy of ANM and male worker in primary health care in Andhra Pradesh. A Qualitative Study. Commissioned by the Department for International Development, (DFID), India, 2003.
21. Koenig M, Khan ME (eds). *Improving the quality of care in India's Family Welfare Programme: The challenge ahead*. New York: Population Council; 1999.
22. Misra R, Chatterjee R, Rao S. *India health report*. New Delhi: Oxford University Press; 2003.

Appendix 1
Maternal care indicators by State

Maternal care indicators for births during the three years preceding the survey by State, India, 1998–99

State/Region	Percentage who received all recommended types of antenatal care[1]	Percentage of births delivered in a medical institution	Percentage of deliveries assisted by a health professional[2]	Percentage of non-institutional deliveries with a postpartum check-up within two months of birth[3]	Percentage of non-institutional deliveries with a postpartum check-up within two days of birth[3]
India	20.0	33.6	42.3	16.5	2.3
North					
Delhi	32.8	59.1	65.9	19.5	2.1
Haryana	20.8	22.4	42.0	15.7	2.5
Himachal Pradesh	30.2	28.9	40.2	21.2	2.9
Jammu and Kashmir	30.7	35.6	42.4	27.4	1.1
Punjab	31.7	37.5	62.6	20.3	5.7
Rajasthan	8.3	21.5	35.8	6.4	0.5
Central					
Madhya Pradesh	10.9	20.1	29.7	10.0	0.5
Uttar Pradesh	4.4	15.5	22.4	7.2	1.5
East					
Bihar	6.4	14.6	23.4	10.0	1.4
Orissa	21.4	22.6	33.4	19.2	2.2
West Bengal	19.7	40.1	44.2	31.6	7.1
Assam	15.8	17.6	21.4	25.5	0.5
West					
Goa	60.6	90.8	90.8	41.0	6.9
Gujarat	25.0	46.3	53.5	10.4	1.6
Maharashtra	31.0	52.6	59.4	29.8	6.9
South					
Andhra Pradesh	35.6	49.8	65.2	44.9	1.6
Karnataka	41.5	51.1	59.1	35.3	3.6
Kerala	64.9	93.0	94.0	27.4	7.5
Tamil Nadu	50.8	79.3	83.8	53.0	10.1

Note: Table includes only the two most recent births during the three years preceding the survey.

[1] Three or more antenatal check-ups (with the first check-up within the first trimester of pregnancy), two or more tetanus toxoid injections, and iron and folic acid tablets or syrup for three or more months

[2] Doctor, auxiliary nurse–midwife, nurse, midwife, lady health visitor, or other health professional

[3] Based on births in the 2–35 months preceding the survey

Source: NFHS 1998–99

Appendix 2
Infant and child mortality by State

Neonatal, postneonatal, infant and under-five mortality rates for the five-year period preceding the survey by State, India, 1998–99

State/Region	Neonatal mortality	Postneonatal mortality 1	Infant mortality ($_1q_0$)	Under-five mortality ($_5q_0$)
India	43.4	24.2	67.6	94.9
North				
Delhi	29.5	17.4	46.8	55.4
Haryana	34.9	21.9	56.8	76.8
Himachal Pradesh	22.1	12.3	34.4	42.2
Jammu and Kashmir	40.3	24.7	65.0	80.1
Punjab	34.3	22.8	57.1	72.1
Rajasthan	49.9	30.9	80.4	114.9
Central				
Madhya Pradesh	54.9	31.2	86.1	137.6
Uttar Pradesh	53.6	33.1	86.7	122.5
East				
Bihar	46.5	26.4	72.9	105.1
Orissa	48.6	32.3	81.0	104.4
West Bengal	31.9	16.8	48.7	67.6
Assam	44.6	24.9	69.9	89.5
West				
Goa	31.2	5.5	63.7	46.8
Gujarat	39.6	23.0	62.6	85.1
Maharashtra	32.0	11.7	43.7	58.1
South				
Andhra Pradesh	43.8	22.1	65.8	80.5
Karnataka	37.1	14.4	51.5	69.8
Kerala	13.8	2.5	16.3	18.8
Tamil Nadu	34.8	13.3	48.2	63.3

Source: NFHS 1998–99

Appendix 3
Childhood vaccination by State

Percentage of children 12–13 months of age who received specific vaccinations at any time before the interview (according to the vaccination card or the mother) and percentage with a vaccination card that was shown to the interviewer by State, India, 1998–99

	Percentage vaccinated											
			DPT			Polio						Percentage showing
State/Region	BCG	Polio 0	1	2	3	1	2	3	Measles	All*	None	vaccination card
India	71.6	13.1	71.4	65.0	55.1	83.6	78.2	62.8	50.7	42.0	14.4	33.7
North												
Delhi	92.0	36.9	90.8	88.3	79.9	93.8	91.7	81.0	77.5	69.8	5.1	43.7
Haryana	86.8	6.1	89.5	84.5	71.1	90.1	87.4	74.3	72.2	62.7	9.9	24.4
Himachal Pradesh	94.6	4.2	96.7	96.1	88.8	97.2	97.2	89.8	89.1	83.4	2.8	54.6
Jammu and Kashmir	85.6	4.8	85.7	83.6	72.3	88.3	85.4	74.3	68.9	56.7	10.4	51.1
Punjab	88.7	11.2	88.4	87.3	82.0	90.5	88.5	83.6	76.5	72.1	8.7	43.0
Rajasthan	53.9	3.2	47.8	40.2	26.1	75.5	67.3	44.6	27.1	17.3	22.5	14.7
Central												
Madhya Pradesh	64.9	10.1	62.8	52.3	37.0	85.4	79.0	56.7	35.5	22.4	13.9	25.1
Uttar Pradesh	57.5	4.7	57.3	46.5	33.9	66.5	60.3	42.3	34.6	21.2	29.5	20.4
East												
Bihar	37.7	3.6	39.7	33.4	24.2	18.3	71.7	41.0	16.6	11.0	16.8	17.4
Orissa	84.7	14.6	80.1	74.8	61.9	88.7	84.8	68.4	54.0	43.7	9.4	46.2
West Bengal	76.5	2.1	77.9	70.1	58.3	83.9	76.5	61.7	52.4	43.8	13.6	58.0
Assam	53.5	3.1	57.4	48.5	37.5	61.8	53.6	37.9	24.6	17.0	33.2	32.5
West												
Goa	99.2	31.6	97.6	95.2	93.4	99.2	98.4	95.8	84.3	82.6	0.0	69.7
Gujarat	84.7	5.3	83.1	75.4	64.1	90.2	82.5	68.6	63.6	53.0	6.6	31.8
Maharashtra	93.7	8.3	94.9	91.7	89.4	97.2	94.7	90.8	84.3	78.4	2.0	48.9
South												
Andhra Pradesh	90.2	5.3	89.8	86.9	79.5	93.8	90.9	81.6	64.7	58.7	4.5	41.3
Karnataka	84.8	26.4	87.0	84.8	75.2	91.9	89.0	78.3	67.3	60.0	7.7	41.2
Kerala	96.2	60.6	96.0	94.4	88.0	96.9	95.2	88.4	84.6	79.7	2.2	63.2
Tamil Nadu	98.6	85.5	98.6	97.5	96.7	99.7	99.5	98.0	90.2	88.8	0.3	45.8

DPT: diphtheria, pertussis and tetanus

Note: Table includes only surviving children from among the two most recent births in the three years preceding the survey

* BCG, measles, and three doses each of DPT and polio vaccines (excluding Polio 0)

Source: NFHS 1998–99

Appendix 4
Wanted fertility rates by State

Total wanted fertility rate and total fertility rate for the three years preceding the survey by State, India, 1998–99

State/Region	Total wanted fertility rate	Total fertility rates
India	2.13	2.85
North		
Delhi	1.72	2.40
Haryana	2.10	2.88
Himachal Pradesh	1.50	2.14
Jammu and Kashmir	1.74	2.71
Punjab	1.55	2.21
Rajasthan	2.57	3.78
Central		
Madhya Pradesh	2.40	3.31
Uttar Pradesh	2.83	3.99
East		
Bihar	2.58	3.49
Orissa	1.90	2.46
West Bengal	1.78	2.29
Assam	1.75	2.31
West		
Goa	1.47	1.77
Gujarat	2.08	2.72
Maharashtra	1.87	2.52
South		
Andhra Pradesh	1.88	2.25
Karnataka	1.56	2.13
Kerala	1.81	1.96
Tamil Nadu	1.71	2.19

Note: Rates are based on births during 1–36 months preceding the survey to women in the age group of 15–49 years.
Source: NFHS 1998–99

Appendix 5
Need for family planning services by State

Percentage of currently married women with unmet need, met need, and total demand for family planning (FP) services, and percentage of total demand satisfied, according to State, India, 1998–99

State/Region	Unmet need for FP[1]			Met need (currently using)[2]			Total demand for FP			Percentage of demand satisfied
	For spacing	For limiting	Total	For spacing	For limiting	Total	For spacing	For limiting	Total	
India	8.3	7.5	15.8	3.5	44.7	48.2	11.8	52.2	64.0	75.3
North										
Delhi	5.9	7.5	13.4	7.8	56.0	63.8	13.7	63.5	77.1	82.7
Haryana	2.9	4.7	7.6	4.1	58.3	62.4	7.0	62.9	69.9	89.2
Himachal Pradesh	3.6	4.9	8.6	3.5	64.2	67.7	7.1	69.2	76.3	88.8
Jammu and Kashmir	7.4	12.6	20.0	4.5	44.6	49.1	11.9	57.1	69.0	71.1
Punjab	2.8	4.5	7.3	5.2	61.5	66.7	8.0	66.0	74.0	90.1
Rajasthan	8.7	8.9	17.6	2.4	37.9	40.3	11.1	46.8	57.9	69.6
Central										
Madhya Pradesh	8.9	7.3	16.2	2.2	42.1	44.3	11.1	49.4	60.5	73.2
Uttar Pradesh	11.8	13.4	25.1	3.0	25.1	28.1	14.7	38.5	53.2	52.8
East										
Bihar	12.6	11.9	24.5	1.4	23.1	24.5	14.0	35.0	49.1	50.0
Orissa	8.7	6.8	15.5	2.4	44.4	46.8	11.1	51.2	62.3	75.1
West Bengal	6.3	5.5	11.8	9.8	56.9	66.6	16.0	62.4	78.4	85.0
Assam	7.0	10.0	17.0	7.9	35.3	43.3	14.9	45.3	60.2	71.8
West										
Goa	7.3	9.8	17.1	7.1	40.4	47.5	14.4	50.2	64.6	73.5
Gujarat	4.8	3.7	8.5	4.9	54.2	59.0	9.7	57.8	67.5	87.4
Maharashtra	8.1	4.9	13.0	3.1	57.8	60.9	11.2	62.7	74.0	82.4
South										
Andhra Pradesh	5.2	2.5	7.7	0.5	58.9	59.6	5.9	61.4	67.3	88.5
Karnataka	8.3	3.2	11.5	2.1	56.2	58.3	10.4	59.4	69.8	83.5
Kerala	6.9	4.9	11.7	6.2	57.5	63.7	13.1	62.4	75.5	84.4
Tamil Nadu	6.6	6.4	13.0	2.2	49.9	52.1	8.8	56.3	65.1	80.1

[1] Unmet need for spacing includes pregnant women whose pregnancy was mistimed, amenorrhoeic women whose last birth was mistimed, and women who are neither pregnant nor amenorrhoeic and who are not using any method of family planning and who say they want to wait two or more years for the next birth. Also included in unmet need for spacing are women who are unsure whether they want another child or who want another child but are unsure when to have it. Unmet need for limiting refers to pregnant women whose pregnancy was unwanted, amenorrhoeic women whose last child was unwanted, and women who are neither pregnant nor amenorrhoeic who are not using any method of family planning and who want no more children.

[2] Met need for spacing refers to women who are using some method of family planning and say they want to have another child or are undecided whether to have another. Met need for limiting refers to women who are using some method and who want no more children. Note that spacing and limiting refer to the reason for using contraception rather than to the particular method used.

Source: NFHS 1998–99

Appendix 6
Levels of service delivery for reduction in maternal and infant mortality

RCH intervention	Community	Subcentre/village clinic	Primary health centre/ community health centre	First referral unit
	Community mobilization, education, motivation, counselling and surveillance	Service delivery—needing personnel trained in clinical, diagnostic and management skills, specialized equipment, and training in record-keeping		
Antenatal contact	Early registration, nutrition education, supplements through ICDS, mobilization, referral and transport, knowledge of and access to schemes to facilitate transport and referral, complication recognition, psychological and emotional support, IFA provision and compliance, ANC card, birth planning	Tetanus toxoid, haemoglobin status, obstetric examination, maternal weight/height, urine protein, blood pressure, general physical examination	ANC care including laboratory-based STI screening, malaria, TB testing, VCT access, urine for infections	All functions at the previous level
Normal delivery	Accompany patient, ensure sensitive treatment, identify follow-up issues	Could be at home or subcentre or village clinic—ANM to conduct normal delivery using partograph, active management of 3rd stage, parenteral oxytocin, misoprostol, and antibiotics		
Complicated delivery	Accompany patient to the referral site, in case she is BPL ensure appropriate remuneration and other benefits	Stabilize patient and refer after communicating with CHC/FRU	MOs capable of performing BEmOC functions	Specialists or MOs trained in caesarean section and aneasthesiology, blood bank—PPTCT
Postpartum care	Complication recognition, breastfeeding support, family planning motivation and counselling, nutrition education, supplements	Visits as scheduled, antibiotics for sepsis, referral for complications	Management of PPH, manual removal of placenta	
Newborn care	Education on feeding, temperature control, and infection prevention, complication recognition, arrangements for referral	Management of newborn asphyxia, pneumonia, hypothermia, visits as scheduled, early recognition of complications and management/ referral	Care of sick newborn, IV, oxygen	Emergency newborn care
Family planning services	Education, individual and couple counselling, enough information to discuss myths and misconceptions, enable method switch, ensure follow up, facilitate access to ANM or PHC, maintain eligible couple register, distribute pills, condoms, follow up,	Subcentre—IUD, emergency contraception, injectables, LAM, SDM	Male and female sterilization	

(Cont.)

	facilitate access for those in need of sterilization		
Safe abortion services	Enable access to sites providing safe abortion services, identify signs of post-abortion complications, counsel on post-abortion contraception	Manual vacuum aspiration, menstrual regulation, management of post-abortion complications, referral, post-abortion contraception	D&C, medical abortion, management of post-abortion complications, referral, post-abortion contraception
Child health	Nutrition education—early and exclusive breastfeeding, timely introduction of complementary feeds, access to supplementation, mobilization for immunization services, support ANM in outreach immunization, transport vaccines, mobilize children, listing of surveillance for children with VPD, at-risk children, education on diarrhoeal disease prevention, ORT, distribute ORS, recognition of ARI/pneumonia, facilitate access to ANM	Subcentre immunization, outreach immunization, vitamin A supplementation, antibiotics for ARI, IV for mild to moderate dehydration and referral for severe diarrhoea	Same Management of severe respiratory and gastrointestinal infections

ICDS: Integrated Child Development Services; IFA: iron–folic acid; ANC: antenatal care; STI: sexually transmitted infection; TB: tuberculosis; VCT: voluntary counselling and testing; BPL: below poverty line; CHC: community health centre; FRU: first referral unit; MO: medical officer; PPTCT: prevention of parent-to-child transmission; BEmOC: basic emergency obstetric care; PPH: postpartum haemorrage; IUD: intrauterine device; LAM: lactational amenorrhoea method; SDM: standard days method; VPD: vaccine-preventable diseases; ORT: oral rehydration therapy; ORS: oral rehydration salt; ARI: acute respiratory infection; D&C: dilatation and curettage

Appendix 7

Table A7.1 State-wise funds released and utilized under the RCH-I Programme from inception till 2003–04 (Rs in lakh)

State/Union Territory	Funds released	Expenditure	(%)
Andhra Pradesh	14,413.52	10,848.54	(75.27)
Arunachal Pradesh	1,400.77	1,227.92	(87.66)
Assam	6,110.99	4,612.31	(75.48)
Bihar	20,140.43	10,076.56	(50.03)
Goa	227.04	185.37	(81.65)
Gujarat	15,656.32	8,537.16	(54.53)
Haryana	8,978.17	6,093.82	(67.87)
Himachal Pradesh	3,426.48	1,803.19	(52.63)
Jammu and Kashmir	2,694.27	1,691.15	(62.77)
Karnataka	9,463.15	7,774.02	(82.15)
Kerala	5,862.62	5,034.66	(85.88)
Madhya Pradesh	19,081.06	13,618.88	(71.37)
Maharashtra	14,097.04	9,120.91	(64.70)
Manipur	2,842.95	2,912.86	(102.46)
Meghalaya	784.04	591.51	(75.44)
Mizoram	3,509.33	3,001.07	(85.52)
Nagaland	1,127.21	1,042.45	(92.48)

Table A7.1 (*cont.*) State-wise funds released and utilized under the RCH-I Programme from inception till 2003–04 (Rs in lakh)

State/Union Territory	Funds released	Expenditure	(%)
Orissa	8,349.26	4,539.24	(54.37)
Punjab	3,690.81	3,026.05	(81.99)
Rajasthan	16,944.13	9,879.51	(58.31)
Sikkim	607.06	436.15	(71.85)
Tamil Nadu	9,383.83	6,379.18	(67.98)
Tripura	1,508.1	1,072.18	(71.09)
Uttar Pradesh	52,649.67	35,757.48	(67.92)
West Bengal	13,514.13	9,906.54	(73.31)
Andaman and Nicobar	342.11	170.55	(49.85)
Chandigarh	360.1	156.2	(43.38)
Dadra and Nagar Haveli	120.31	87.28	(72.55)
Daman and Diu	254.37	77.63	(30.52)
Lakshadweep	240.59	69.39	(28.84)
Pondicherry	295.5	254.67	(86.18)
Delhi	2,746.83	1,865.43	(67.91)
Total	240,822.19	161,849.86	(67.21)

Source: Department of Family Welfare, Government of India

Table A7.2 Analysis of utilization of funds under the RCH-I Programme from inception till 2003–04 (Rs in lakh)

Activities	Released		Expenditure		% utilization
Administration					
Computer and Furniture	191.92		187.18		97.53
SCOVA staff	994.94		758.84		76.27
Immunization cards	173.36		121.63		70.16
EC registers	173.79		131.93		75.91
Transportation charges of drugs	5.88		0		0.0
MCH registers	207.33		53.67		25.89
Other oper. charges	496		248.25		50.05
Contingency	177.07		176.01		99.40
Review meeting/mob. support	603.05		71.19		11.80
Subtotal	3,023.34	(1.26%)	1,748.7	(1.08%)	57.84
Maternal health					
RCH drugs	715.385		208.725		29.18
Adsorbent cotton	532.6		397.12		74.56
Dai training	1,025.42		641.03		62.51
RCH training	3,962.145		3,006.039		75.87
Contractual ANMs	3,124.236		2,769.648		88.65
Contractual PHNs	883.038		944.229		106.93
Contractual laboratory technicians	732.4		732.34		99.99
Pethidine injection	23.8		6.11		25.67
SM consultants	354.98		199.65		56.24
Anaesthetists	101.31		42.82		42.27
24-hour delivery	1,323.11		621.93		47.01
Referral transport	598.65		112.39		18.77
RTI/STI consumables	63		32.14		51.02
RCH camps	290.883		360.342		123.88
Vanaspati van	1,750.33		75.99		4.34
Subtotal	15,481.287	(6.43%)	10,150.503	(6.27%)	65.57
Child health					
RCH drugs	715.385		208.725		29.18
Outreach services	825.38		445.59		53.99
Cold chain maintenance/injection safety	2,018.56		1,074		53.21
Imm str project	1,033.938		1,419.98		137.34
PPI	112,160.7		80,095.88		71.41
Contractual ANMs	4,165.648		3,692.864		88.65
Contractual PHNs	1,177.384		1,258.972		106.93
RCH camps	387.844		480.456		123.88
RCH training	5,282.86		4,008.052		75.87
Subtotal	127,767.699	(53.05%)	92,684.519	(57.27%)	72.54
Family planning					
CNAA/TFA	508.52		236.97		46.60
ZSS	961.76		329.86		34.30
Contractual ANMs	3,124.236		2,769.648		88.65
Contractual PHNs	883.038		944.229		106.93
RCH camps	290.883		360.342		123.88
RCH training	3,962.145		3,006.039		75.87
Subtotal	9,730.582	(4.04%)	7,647.088	(4.72%)	78.59
Area projects					
EC sector reforms	32,677.55		15,583.94		47.69
Financial envelopes	1843.12		1,655.78		89.84
Sub-project	23,738.75		18,226.58		76.78
Subtotal	58,259.42	(24.19%)	35,466.3	(21.91%)	60.88
Infrastructure					
Minor civil works	7,853.45		4,365.28		55.58
Major civil works	18,262.39		9,751		53.39
Moped adv to ANMs	444.57		36.35		8.18
Subtotal	26,560.41	(11.03%)	14,152.63	(8.74%)	53.28
Grand total	240,822.738		161,849.74		67.21

Source: Department of Family Welfare, Government of India

Table A7.3 Abstract summary of funds released to States under the RCH Programme, 2001–02 to 2003–04 (Rs in lakh)

State/Union Territory	Year											
	2001–02				2002–03				2003–04			
	In cash	In kind	Grants-in-aid to Soc/NGOs	Total	In cash	In kind	Grants-in-aid to Soc/NGOs	Total	In cash	In kind	Grants-in-aid to Soc/NGOs	Total
Andhra Pradesh	2,338.69	2,555.30	112.01	5,006.00	1,509.69	2,323.65	75.17	3,908.51	3,144.73	96.34	135.52	3,376.59
Arunachal Pradesh	156.45	154.98	0	311.43	306.94	69.30	29.87	406.11	44.29	0.04	1.00	45.33
Assam	1,334.67	1,308.94	35.70	2,679.31	793.03	1,693.10	56.13	2,542.26	1,282.36	143.71	36.64	1,462.71
Bihar	1,842.97	4,867.76	85.50	6,796.23	4,137.94	6,414.17	135.04	10,687.15	3,731.31	2,221.77	180.73	6,133.81
Chhattisgarh	964.42	0	25.00	989.42	476.61	1,413.35	22.07	1,912.03	1,085.25	44.61	22.75	1,152.61
Goa	39.74	45.66	0	85.40	15.47	38.37	0	53.84	16.67	0.25	0	16.92
Gujarat	8,060.05	1,936.03	168.60	10,164.68	1,235.12	1,997.67	65.98	3,298.77	1,742.49	605.99	23.64	2,372.12
Haryana	1,336.25	880.23	46.61	2,263.09	1,007.50	930.14	58.92	1,996.56	1,916.85	856.04	47.30	2,820.19
Himachal Pradesh	417.21	289.97	36.50	743.68	410.41	410.80	22.71	843.92	615.29	2.18	44.89	662.36
Jammu and Kashmir	569.56	442.00	22.00	1,033.56	425.53	570.01	13.53	1,009.07	218.57	21.14	22.72	262.43
Jharkhand	37.00	0	4.00	41.00	903.73	1,763.39	27.06	2,694.18	1,003.11	425.82	0	1,428.93
Karnataka	1,835.64	2,055.00	16.77	3,907.41	2,478.22	2,171.42	94.94	4,744.58	633.90	42.17	30.00	706.07
Kerala	875.88	907.88	94.92	1,878.68	711.76	850.37	128.93	1,691.06	605.84	11.44	104.34	721.62
Madhya Pradesh	1,590.24	2,656.67	94.47	4,341.38	1,969.26	2,438.18	131.31	4,538.75	2,097.69	844.94	208.12	3,150.75
Maharashtra	3,352.62	3,331.47	182.07	6,866.16	1,523.07	4,249.74	116.16	5,888.97	3,172.43	20.68	145.95	3,339.06
Manipur	860.86	194.66	0	1,055.52	230.36	124.68	59.10	414.14	113.40	0	2.00	115.40
Meghalaya	195.12	193.93	0	389.05	155.45	70.80	0	226.25	78.27	0.51	0	78.78
Mizoram	780.29	67.31	0	847.60	706.38	62.91	0	769.29	175.98	0	0	175.98
Nagaland	196.51	133.01	0	329.52	217.88	87.89	15.01	320.78	164.25	26.96	1.00	192.21
Orissa	2,169.46	1,655.66	105.21	3,930.33	690.55	1,812.87	209.99	2,713.41	953.19	183.69	199.29	1,336.17
Punjab	729.69	928.38	0	1,658.07	275.45	1,174.90	17.97	1,468.32	313.41	37.20	13.00	363.61
Rajasthan	4,054.24	2,532.43	31.73	6,618.40	2,138.67	2,234.20	69.29	4,442.16	4,119.19	1,072.42	5.00	5,196.61
Sikkim	89.49	58.36	10.00	157.85	92.90	64.02	23.71	180.63	35.10	0.04	24.71	59.85
Tamil Nadu	796.52	2,002.78	63.55	2,862.85	1,288.45	1,774.94	19.05	3,082.44	783.27	103.00	55.38	941.65
Tripura	468.16	192.67	15.00	675.83	177.61	145.95	17.80	341.36	78.61	1.46	18.80	98.87
Uttar Pradesh	7,572.74	9,567.68	149.86	17,290.28	9,843.55	10,362.28	88.39	20,294.22	11,716.92	3,573.61	179.14	15,469.67
Uttaranchal	462.04	0	0	462.04	424.61	728.41	33.93	1,186.95	496.02	86.77	5.75	588.54
West Bengal	2,208.45	3,118.51	34.51	5,361.47	1,424.30	3,042.49	145.96	4,612.75	3,245.93	947.80	159.84	4,353.57
Andaman and Nicobar	151.58	18.02	0	169.60	13.53	20.68	0	34.21	25.88	0	0	25.88
Chandigarh	27.57	26.81	0	54.38	17.04	66.83	6.46	90.33	18.80	0.04	37.09	55.93
Dadra and Nagar Haveli	19.47	13.15	0	32.62	6.18	9.12	0	15.30	3.26	36.00	0	39.26
Daman and Diu	10.25	8.18	0	18.43	25.41	9.32	0	34.73	6.99	0.74	0	7.73
Lakshadweep	11.42	10.56	0	21.98	16.04	220.59	0	236.63	7.33	0	0	7.33
Pondicherry	29.17	29.13	0	58.30	25.85	25.08	0	50.93	35.73	18.02	0	53.75
Delhi	365.49	367.17	188.90	921.56	344.50	480.59	63.44	888.53	566.62	609.90	102.57	1,279.09
Total	45,949.91	42,550.29	1522.91	90,023.11	36,018.99	49,852.21	1747.92	87,619.12	44,248.93	12,035.28	1807.17	58,091.38

Source: Department of Family Welfare, Government of India

Causal analysis and treatment protocols for maternal complications

SARALA GOPALAN, SHALINI GAINDER

1. Bleeding in pregnancy

Table 1.1 Causes of bleeding in pregnancy[1–3]

Condition	Direct causes	Indirect causes	Distant causes
Bleeding in early pregnancy (at less than 20 weeks of gestation)	• Threatened abortion • Inevitable abortion • Incomplete abortion • Ectopic pregnancy • Molar pregnancy • Post-medical termination of pregnancy complication • Injury to the genital tract		
Bleeding in pregnancy (after 20 weeks of gestation)	• Placenta praevia • Abruptio placentae • Local cause • Undetermined antepartum haemorrhage	• Multiparity • Chronic hypertension and gestational hypertension increase the chances of abruptio placentae • Elderly woman	

Table 1.2 Personnel, drugs, equipment and other supplies required for the management of bleeding in pregnancy at various levels of care[4]

Level of health care	Personnel required	Drugs/equipment/other supplies
Village/subcentre	• Health worker • Skilled birth attendants • Midwife/nurse • Adequately trained *dais*	Refer without examination
PHC	• Doctor • Nurse • Health worker • Laboratory technician • Pharmacist • Driver	• Disposable needles • IV cannula • IV set • Inj. normal saline • Inj. Ringer lactate • Inj. tetanus toxoid • Inj. anti-D • Plasma expanders: hetastarch, haemaccel, dextran 70 • Cap. ampicillin • Gloves • Speculums • D&C sets • Boilers • Vehicle for transportation
CHC	• Specialist • Doctor	• Disposable needles • IV cannula

(Cont.)

Postgraduate Institute of Medical Education and Research
Chandigarh 160012
e-mail: pgimer@chd.nic.in

Table 1.2 (*cont.*) Personnel, drugs, equipment and other supplies required for the management of bleeding in pregnancy at various levels of care[4]

Level of health care	Personnel required	Drugs/equipment/other supplies
	• Nurse • Health assistant • Laboratory technician • Pharmacist • Driver	• IV sets • Inj. normal saline • Inj. Ringer lactate • Inj. tetanus toxoid • Inj. anti-D • Plasma expanders: hetastarch, haemaccel, dextran 70 • Inj. ampicillin • Inj.gentamicin • Inj. metronidazole • Gloves • Speculum • D&C sets • Boilers • Vehicle for transportation
District hospital	• Specialist • Doctor • Anaesthetist • Pathologist • Nurse • Health assistant • Laboratory technician • Pharmacist	• Disposable needles • IV cannula • IV sets • Blood transfusion sets • Inj. normal saline • Inj. Ringer lactate • Inj. tetanus toxoid • Inj. anti-D • Plasma expanders: hetastarch, haemaccel, dextran 70 • Inj. ampicillin • Inj. gentamicin • Inj. metronidazole • Gloves • D&C sets • Boilers • Working operation theatres • Vehicle for transportation

PHC: primary health centre; CHC: community health centre; IV: intravenous; Inj.: injection; Cap.: capsule; D&C: dilatation & curettage

Table 1.3 Management of conditions occurring due to bleeding in pregnancy at various levels of care

Condition	Village/subcentre	PHC	CHC	District hospital
A pregnant woman reports to a TBA/MHW/ midwife, nurse with bleeding	Refer immediately to a PHC or the nearest health facility having a doctor	• Doctor or midwife/nurse to attend to the case • History Duration of the pregnancy, amount of bleeding, passage of products of conception, abdominal pain, severity of the pain, h/o any syncopal attack, h/o interference, contraception • Examination —Pallor, temperature, pulse, blood pressure —P/A: for any tenderness, free fluid, size of the uterus —P/S: amount of bleeding, products of conception seen or not —P/V: size of the uterus, any	• Specialist to attend to the case • History Duration of the pregnancy, amount of bleeding, passage of products of conception, abdominal pain, severity of the pain, h/o any syncopal attack, h/o interference, contraception • Examination —Pallor, temperature, pulse, blood pressure —P/A: for any tenderness, free fluid, size of the uterus —P/S: amount of bleeding, products of conception seen or not —P/V: size of the uterus, any	• Specialist to attend to the case • History Duration of the pregnancy, amount of bleeding, passage of products of conception, abdominal pain, severity of the pain, h/o any syncopal attack, h/o interference, contraception • Examination —Pallor, temperature, pulse, blood pressure —P/A: for any tenderness, free fluid, size of the uterus —P/S: amount of bleeding, products of conception seen or not —P/V: size of the uterus, any

(Cont.)

Table 1.3 (*cont.*) Management of conditions occurring due to bleeding in pregnancy at various levels of care

Condition	Village/subcentre	PHC	CHC	District hospital
		adnexal tenderness • Investigations Hb estimation	adnexal tenderness • Investigations Hb estimation, blood grouping and Rh typing, BT, CT, RFT	adnexal tenderness • Investigations Hb estimation, blood grouping and Rh typing, BT, CT, RFT
Shock	ANM to start an IV line and refer immediately to a PHC or the nearest health facility having a doctor	• Establish an IV line and start rapid IV fluids (normal saline or Ringer lactate) • Arrange for transportation and midwife to accompany the patient • Transfer the patient to the nearest district/tertiary referral centre	• Start rapid IV fluids till the pulse improves • Give blood if cross-matching and storage facilities are available and monitor the vital signs. If blood is not available, quickly assess the vital signs and refer to the nearest district/tertiary referral centre • Continue IV fluids • Arrange for a nurse or midwife to accompany the patient to ensure IV fluids during transportation	• Secure two IV lines; start rapid IV fluids till blood is cross-matched • Give blood. Quickly assess the cause of shock and treat • If the diagnosis is uncertain, refer to a medical college or nearest tertiary referral centre • Arrange for a nurse to accompany the patient to ensure IV fluids during transportation
Threatened abortion	Refer immediately to a PHC or the nearest health facility having a doctor	If the bleeding is minimal and the uterine size corresponds to the period of gestation, refer to the district hospital for USG to confirm the viability of the pregnancy	If the bleeding is minimal and the uterine size corresponds to the period of gestation, refer to the district hospital for USG to confirm the viability of the pregnancy	• If the bleeding is minimal and the uterine size corresponds to the period of gestation, confirm viability by USG. Advise rest and give Inj. anti-D 100 μg if the mother is Rh negative • Missed abortion: If the foetus is not viable, repeat the coagulogram weekly for 3 weeks, till the woman aborts spontaneously or evacuate using misoprostol or suction evacuation
Inevitable abortion	Refer immediately to a PHC or the nearest health facility having a doctor	If the doctor has the skill to perform MTP, do so, otherwise refer to the nearest CHC or district hospital	• Pregnancy less than 12 weeks: Wait till spontaneous expulsion begins. Start augmentation with oxytocin and perform manual aspiration followed by check curettage • Pregnancy more than 12 weeks: Start oxytocin augmentation. Once the foetus is expelled, examine the foetus and placenta and check whether it is complete or not. If there are retained products then perform gentle curettage. Keep the patient under observation —Give Inj. TT prophylaxis —If the mother is Rh negative, give anti-D —Antibiotic cover: Cap. ampicillin or Tab. co-trimoxazole as per the availability • Advise to report if bleeding continues or if she develops fever	• Pregnancy less than 12 weeks: Give oxytocin augmentation followed by dilatation and evacuation or suction evacuation • Pregnancy more than 12 weeks: Give oxytocin augmentation till the foetus is expelled. Examine whether the placenta is complete or not. If there are retained products, perform curettage. —Give Inj. TT prophylaxis —Give anti-D to Rh-negative mother —Antibiotic cover: Give Cap. ampicillin or Tab. co-trimoxazole according to the availability

(Cont.)

Table 1.3 (*cont.*) Management of conditions occurring due to bleeding in pregnancy at various levels of care

Condition	Village/subcentre	PHC	CHC	District hospital
Incomplete abortion	Refer to a PHC	• If the bleeding is heavy and there is passage of the products of conception then manage as below —Secure an IV line, refer to the nearest CHC/ district hospital —If the doctor is trained to perform D&C then perform evacuation at the PHC	• If the bleeding is heavy and there is passage of the products of conception then manage as below —Specialist to perform dilatation and evacuation if the pregnancy is less than 12 weeks —If the pregnancy is more than 12 weeks and the foetus has not been expelled, give oxytocin augmentation and wait for spontaneous expulsion. If some products are still retained, perform gentle curettage. Give * Inj. TT * Inj. anti-D to Rh-negative mother * Tab. co-trimoxazole DS 1BD for 5 days * Alternatively, give Cap. ampicillin 500 mg 6-hourly x 5 days —Advise to report back if bleeding persists or the woman develops fever	• If the bleeding is heavy and there is passage of the products of conception then manage as below —If the pregnancy is less than 12 weeks, give oxytocin augmentation followed by dilatation and evacuation or suction evacuation —If the pregnancy is more than 12 weeks, give oxytocin augmentation till the foetus is expelled. Examine whether the placenta is complete or not. —If there are retained products, perform check curettage —Give * Inj. TT prophylaxis * Inj. anti-D to Rh-negative mother * Antibiotic cover: Cap. ampicillin or Tab. co-trimoxazole as per the availability
Complete abortion	Refer to a PHC	• Confirm h/o passage of products of conception • Conduct a P/V examination under aseptic conditions to assess the size of the uterus • Ask to report in case of excessive or countinous bleeding or fever	• Confirm h/o passage of products of conception • Conduct a P/V examination under aseptic conditions to assess the size of the uterus • Ask to report in case of excessive bleeding or fever • Check the blood group and Rh typing; if Rh negative, give Inj. anti-D 100 μg	• Confirm h/o passage of products of conception • Conduct a P/V examination to confirm the size of the uterus • Perform USG to rule out the presence of any retained products • Advise to report in case bleeding continues or fever develops • Give Inj. anti-D to Rh-negative woman
Molar pregnancy	Start an IV line and refer to a PHC or the nearest health facility having a doctor	• H/o passage of grape-like products • Check the vital signs; the per abdominal uterine size may be larger than the duration of pregnancy • Management Start an IV line and refer to a district hospital	• H/o passage of grape-like products • Check the vital sings; the per abdominal uterine size may be larger than the duration of pregnancy • Management Start an IV line and refer to a district hospital	• H/o passage of grape-like products • Check the vital signs; the per abdominal uterine size may be larger than the duration of pregnancy • Review history • Check vital signs; conduct a P/A examination to check the size of the uterus • Investigations —Hb estimation, blood grouping and Rh typing, RFT, BT, CT, chest X-ray —USG to confirm the diagnosis • Management —Specialist to be present

(Cont.)

Table 1.3 (*cont.*) Management of conditions occurring due to bleeding in pregnancy at various levels of care

Condition	Village/subcentre	PHC	CHC	District hospital
				—IV line to be established —Anaesthetist should be available —Blood to be cross-matched —Suction evacuation to be done —Give 10 U oxytocin in 500 mg normal saline at the time of the procedure —Products must be sent for histopathology to a medical college or a tertiary centre • Send the patient to a tertiary centre for follow up • If the patient has any complication, refer to a higher centre
Post MTP/post-abortal complications	Refer to a PHC	• Secure an IV line • Refer to a tertiary hospital	• Secure an IV line • Refer to a tertiary hospital	• Review history • Examine the patient • If gut injury is suspected or the patient is in shock, manage as shock and refer without delay to a tertiary centre • Refer with a referral slip
Ectopic pregnancy	• H/o abdominal pain, spotting, fainting • Refer to a PHC	• H/o pain, fainting attack, spotting • Examination —Pallor, pulse, blood pressure —P/A: Look for tenderness, free fluid —P/V: Tenderness on cervical movement, the uterus is normal in size, presence of adnexal mass, any tenderness • Management —Refer to a higher centre —If shock is present, treat as shock and refer to a district hospital	• H/o pain, fainting attack, spotting • Examination —Pallor, pulse, blood pressure —P/A: Look for tenderness, free fluid —P/V: Tenderness on cervical movement, the uterus is normal in size, presence of adnexal mass, any tenderness • Management —Refer to a higher centre —If shock is present, treat as shock and refer to a district hospital	• H/o pain, fainting attack, spotting • Examination —Pallor, pulse, blood pressure —P/A: Look for tenderness, free fluid —P/V: Tenderness on cervical movement, the uterus is normal in size, presence of adnexal mass, any tenderness • Management —Establish an IV line —Treat shock if present —Confirm diagnosis by USG —Culdocentesis can be done to confirm haemoperitoneum —Carry out blood transfusion if shock is present or Hb <7 g% —Perform laparotomy; if the family is complete, perform bilateral salpingectomy else perform conservative surgery —Observe the vital signs and urine output • If there is an unruptured ectopic pregnancy and the patient's vital signs are stable, medical management can be considered if the family is not complete; for this refer to a higher centre
Woman with bleeding P/V and pregnancy more than 20 weeks	• MHW/midwife/TBA to attend • Do not attempt any	• Attending doctor or trained midwife should review history quickly	• Quick history by the doctor • Check the vital signs	• History-taking by a specialist • Duration of the pregnancy • Previous BP records

(Cont.)

Table 1.3 (*cont.*) Management of conditions occurring due to bleeding in pregnancy at various levels of care

Condition	Village/subcentre	PHC	CHC	District hospital
	examination • Transfer to a PHC immediately • Arrange for transportation	• Check the vital signs • Do not do a P/V examination • Start an IV line with Inj. normal saline • Arrange for transportation • Refer to a district/medical college hospital whichever is nearer, without delay • Nurse/midwife to accompany if the bleeding is heavy or if the patient is in shock	—Pallor, pulse, BP, fundal height, feel of the uterus, any tenderness, foetal heart rate —Start an IV line • Arrange for transportation • Refer to a district/medical college hospital whichever is nearer, without delay • Transfer the patient with a nurse or someone who can look after an IV line and resuscitate the patient	• Duration and amount of bleeding • Any previous episode of bleeding • Painless, unprovoked bleeding as in placenta praevia or pain followed by bleeding and continuous or intermittent pain • Perception of foetal movements • Any previous surgery • Examination —Look for pallor; check the pulse and BP —Check the fundal height, feel of the uterus, any tenderness, foetal heart rate • Investigations —Hb estimation —Blood grouping and Rh typing —BT, CT, clot retraction time —USG
Patient in shock		• Start an IV line and give rapid IV fluids—plasma expanders are to be given • Refer to a district hospital • Send a trained midwife or health worker with the patient	• Start an IV line and give rapid IV fluids—plasma expanders are to be given • If blood is available, do cross-matching and start giving blood • Catheterize the bladder • Refer to a district hospital • Send a trained midwife or health worker with the patient	• Start 2 IV lines • Start IV crystalloids • Cross-match blood and start transfusion • Catheterize the bladder • Do USG for diagnosis of abruptio placentae or placenta praevia
Placenta praevia (in shock)				• Prepare for a caesarean section immediately • Keep adequate blood ready • Take written consent • Arrange adequate oxytocin, Inj. carboprost, Tab. misoprostol for rectal use, Inj. methergin • Perform a caesarean section as a life-saving surgery for the mother • Post-operative —Watch for PPH —Monitor the vital signs every hour —Monitor the urine output for 24 hours —Replace blood adequately by repeat Hb evaluation
Bleeding is stopped and the pregnancy is less than 32 weeks (placenta praevia)				• Admit the patient —Give steroids for foetal lung maturity (Inj. betamethasone two doses of 12 mg 24 hours apart) and observe for 48 hours • If there is no further bleeding, advise complete rest • If the patient is stable for 48

(Cont.)

Table 1.3 (*cont.*) Management of conditions occurring due to bleeding in pregnancy at various levels of care

Condition	Village/subcentre	PHC	CHC	District hospital
				hours then —If type 1/type 2 anterior placenta praevia is present and the patient has easy access to a hospital * Discharge and manage at home * Get written consent to report immediately in case of bleeding and ask the patient to take strict bed rest —In case of type 2 posterior/ type 3 or 4 placenta praevia * Continue observation of the patient in hospital
Pregnancy is between 32 and 36 weeks				• Light bleeding, maternal and foetal condition normal, not in labour —Conservative management • Minimal exertion after three days if stable —Deliver at 36 weeks or more if the bleeding is heavy and/or the maternal–foetal condition is not stable or the woman is in labour or in case of intra-uterine death, deliver immediately • If USG reveals type 2 posterior/ 3 or 4 placenta praevia —If patient has bleeding —Give blood if needed and perform an LSCS • If USG reveals type 1 or 2 anterior placenta praevia —If patient has bleeding —Give blood if needed; keep the operation theatre ready for a caesarean section if indicated; conduct a P/V examination in the theatre —Induce labour (if indicated)/ LSCS —Active management of the third stage of labour —Monitor for PPH
Pregnancy beyond 36 weeks				• Delivery indicated whenever diagnosis of placenta praevia is made • LSCS to be performed in type 2 posterior and central placenta praevia, and types 3 and 4 placenta praevia
Drugs available/ equipment		• IV sets • Inj. normal saline	• IV sets • Inj. normal saline	• IV sets • Inj. normal saline

(Cont.)

Table 1.3 (*cont.*) Management of conditions occurring due to bleeding in pregnancy at various levels of care

Condition	Village/subcentre	PHC	CHC	District hospital
		• IV cannula • Plasma expanders: Inj. haemaccel, hetastarch or dextran 70	• IV cannula • Plasma expanders: Inj. haemaccel, hetastarch or dextran 70	• IV cannula • Blood transfusion set • Plasma expanders: Inj. haemaccel, hetastarch or dextran 70 • Inj. oxytocin, carboprost, methergin, antibiotics, Tab. misoprostol • Working operation theatre
• Abruptio placentae, shock/signs of shock If the bleeding is light to moderate, pregnancy is less than 34 weeks, condition of the mother is stable and there is no foetal distress	Manage as shock and refer to the nearest health centre having facilities for a caesarean section	Manage as shock and refer to the nearest health centre having facilities for a caesarean section	Perform a caesarean section operation/ARM, if required or refer to a district hospital	• Manage shock and deliver the patient early • Check the vital signs, establish an IV line, arrange fresh blood and assess the clotting status • If there is any clotting problem, transfuse fresh blood • Give Inj. betamethasone two doses of 12 mg 12 hours apart for foetal lung maturity
At >34 weeks of pregnancy				• Perform ARM • Augment labour • If the cervix is not ripe or there is foetal distress, perform LSCS • If there is heavy bleeding and the foetus is alive, perform an LSCS • If the foetus is dead, induce labour and give blood transfusion • Monitor for complications (DIC, renal failure) —Repeat CT/CRT after 4-hours —Give fresh blood transfusion —Monitor the progress 4-hourly —Conduct assisted delivery by vacuum extraction to hasten delivery —If the progress is unsatisfactory after 4 hours, refer to a higher centre —If there is decreased urine output or coagulopathy, refer with a nurse or midwife to a medical college hospital —Monitor for complications (DIC, renal failure) —After delivery, monitor for urinary output and PPH

PHC: primary health centre; CHC: community health centre; TBA: traditional birth attendant; MHW: multipurpose health worker; h/o: history of; P/A: per abdomen; P/S: per speculum; P/V; per vaginam; BT: bleeding time; CT: clotting time; RFT: renal function tests; ANM: auxiliary nurse–midwife; IV: intravenous; USG: ultrasonography; Inj.: injection; MTP: medical termination of pregnancy; TT: tetanus toxoid; Tab.: tablet; Cap.: capsule; Hb: haemoglobin; BP: blood pressure; PPH: postpartum haemorrhage; DIC: disseminated intravascular coagulation; CRT: clot retraction time; LSCS: lower segment caesarean section; ARM: artificial rupture of membranes

References

1. *Williams Obstetrics, Vol. 21*. International Edition. McGraw-Hill Companies, Inc.; 2003:619.
2. Arulkumaran S, Singh K. Antepartum haemorrhage. In: *The management of labor*. Chennai: Orient Longman Ltd.; 1996:157.
3. Vaginal bleeding in later pregnancy and labor S-17. *Managing complictions in pregnancy and childbirth*. IMPAC December 2002 WHO/SEARO, India.
4. Life-saving management of bleeding in pregnancy. Standards of Midwifery/Practice for safe motherhood. Standard document, WHO. Regional Publication, SEARO, No. 38, New Delhi; 1999:87.

2. Postpartum haemorrhage (PPH)

Table 2.1 Causes of postpartum haemorrhage

Direct causes	Indirect causes	Distant causes
Traumatic • Injuries to the genital tract *Atonic* • Retained products of conception • Prolonged labour • Multiparity (multiple pregnancies) • Polyhydramnios • Abruptio placentae • Underlying coagulopathy • Big baby • Induction of labour	• Underlying anaemia • Delivery at home • Non-availability of oxytocics, methergin • Underestimation of blood loss by the birth attendant • Delay in transportation • Absence of blood bank facilities	• Ignorance • Delay in seeking treatment • Distance from the hospital

Table 2.2 Management of postpartum haemorrhage (PPH) at various levels of health care

Village level/subcentre	PHC	CHC	District hospital
Preventive measures • All deliveries should be institutional and home deliveries should be discouraged by explaining the advantages of institutional deliveries • All deliveries should be conducted by a trained midwife/ skilled birth attendant • The ANM/HW (F) should do counselling in the antenatal period and as delivery nears • Free supply of oxytocics, methergin injections and misoprostol tablets to the personnel who perform delivery • High-risk cases should be referred to a CHC or district hospital for delivery • If a high-risk woman goes into labour then the attending HW(F)/ midwife should accompany the patient with drugs to the referral hospital • If the bleeding continues following delivery: —Transport the woman to a PHC	• Only a doctor or trained midwife/ nurse should conduct the delivery • Active management of the third stage of labour should be practised by the doctor or trained midwife/ nurse conducting the delivery • If the bleeding continues following delivery —Check the pulse, BP, pallor, retraction of the uterus —Shift the patient to the delivery table —Empty the bladder —Establish an IV line and give oxytocin infusion 20 U in 500 ml normal saline —Give Inj. methergin 0.2 mg IM or through the IV line —If Tab. misoprostol is available 800 μg can be given per rectum —Quickly explore the cervix for any tears. If a tear is present, suture it • If the bleeding continues: —Call for help for transportation —Insert an indwelling catheter	• If the bleeding continues following delivery: —Look for pallor, check the pulse, BP, temperature —Conduct a P/A examination to check whether the uterus is retracted —Shift the patient to the delivery table —Empty the bladder using a catheter —Start IV Inj. normal saline with oxytocin 20 U continuous drip —Give Inj. methergin 0.2 mg IM or through the IV line —Explore the vagina and cervix for tears. If a tear is found, suture it —If the uterus is atonic, give Inj. carboprost or Tab. misoprostol 800 μg per rectum —Continue oxytocin drip • If the bleeding continues, i.e. PPH —Check the clotting status —If blood is available, start transfusion —Put an indwelling urinary	• If the bleeding continues following delivery, i.e. patient is referred as a case of PPH —Specialist to be present —Check pallor, pulse, BP, fundal height of the uterus —Catheterize the patient —Start 2 IV lines —Give 20 U oxytocin IV —Give blood for cross-matching —Check the clotting status • Patient requires exploration under general anaesthesia —Do a P/V examination and remove any retained bits of placenta, clots —Do a P/S examination and suture any tears that are seen —Look for any vaginal tears • In case of uncontrolled haemorrhage —Give blood transfusion —Continue oxytocin drip —Give Inj. carboprost IM —Give Tab. misoprostol 800 μg per rectum

(Cont.)

Table 2.2 (*cont.*) Management of postpartum haemorrhage (PPH) at various levels of health care

Village level/subcentre	PHC	CHC	District hospital
without delay —During this period the trained personnel (midwife/nurse/birth attendant) can (i) conduct vaginal examination and explore for retained products of conception (ii) continue uterine massage (iii) perform bimanual compression • Drugs and supplies —Gloves —Disposable syringes with needles —Inj. methergin —Tab. misoprostol —Inj. oxytocin	—Continue uterine massage —Continue IV oxytocin drip; give plasma expanders if the bleeding is heavy —Transfer the patient —Perform bimanual compression —Accompany the patient to the district hospital (midwife/nurse) • If the bleeding stops: —Do Hb estimation —If the patient requires blood transfusion transfer to the district hospital —During transportation, a doctor/nurse/midwife should accompany the patient. They should carry IV fluids, oxytocin and methergin injections • Drugs and supplies —Inj. oxytocin —Inj. methergin —Tab. methergin —Tab. misoprostol 200 μg —Inj. normal saline —Foley catheter —Red rubber catheter —IV cannula —IV set —Disposable syringes and needles —Suture material: chromic catgut no. 1-0 —Needle holder —3 sponge holders —2 Sim speculums —Light or headlamp —Delivery table with stirrups	catheter —Transfer the patient to a higher centre • Laboratory investigations —Haemoglobin (Hb) estimation —Blood grouping and cross-matching • If a patient with PPH is pale and the bleeding is not controlled, she —Requires exploration under anaesthesia —Requires blood transfusion • If these facilities are not available, transfer the patient on plasma expanders to a district or a medical college hospital, whichever is nearer • During transportation —Doctor/nurse/midwife to accompany the patient. They should carry IV fluids, oxytocin, methergin injections and plasma expanders —Continue oxytocin drip —Give Inj. carboprost, Tab. misoprostol 800 μg per rectum • Requirements at the CHC —Blood storage and cross-matching facility, and a pathologist to do cross-matching • Drugs and supplies —Inj. carboprost which requires refrigeration —Tab. misoprostol 200 μg —Inj. methergin ampoules, —Inj. oxytocin —Inj. normal saline —Plasma expanders —IV cannula —IV set —Foley catheter —Urobag —Suture material: chromic catgut no. 1-0 —2 Sim speculums —3 sponge holders —Good head lamp or light source for exploration —Delivery table with stirrups	—Perform bimanual compression —Continue uterine massage —If the uterus is well-contracted but there is local oozing from the cervix and vagina, do tight packing and arrange for transportation • If the cervical tear appears to extend to the broad ligament, the patient needs laparotomy and may require hysterectomy • Depending on the attending obstetrician's skill and availability of blood, the patient can either be referred or laparotomy can be performed with full consent as a life-saving procedure • When transferring such as patient, a nurse should accompany her —Continue oxytocin drip, blood transfusion —Continue uterine massage —Refer with a referral slip with details of examinations done and medication given • Requirements at the district hospital —Anaesthetist —Pathologist —Blood bank —Functioning operation theatre • Drugs and supplies —Inj. carboprost which requires refrigeration —Tab. misoprostol 200 μg (advantage: it is cheap and can be stored at room temperature) —Inj. methergin —Inj. oxytocin —Inj. normal saline —Plasma expanders —IV cannula —IV sets —Blood transfusion sets —Foley urinary catheter —Urobag —Disposable syringes and needles

PHC: primary health centre; CHC: community health centre; ANM: auxiliary nurse–midwife; HW(F): health worker (female); BP: blood pressure; Inj.: injection; Tab.: tablet; IV: intravenous; IM: intramuscular

References

1. W*illiams obstetrics, Vol. 21*. International Edition. McGraw-Hill Companies, Inc.; 2003:619.
2. Arulkumaran S, Haththotuwa R, Chua S. The management of postpartum haemorrhage. *The management of labor*. 1996:183.
3. Vaginal bleeding after childbirth. S-35. *Managing complications in pregnancy and childbirth*. IMPAC publication WHO/SEARO; Regional Publication, SEARO, No. 38; 2002.
4. Life-saving management of primary postpartum haemorrhage. Standards of midwifery: Practice for safe motherhood. Standard Document. (Std. 7) WHO; 1999:108.

3. Obstructed labour

Table 3.1 Causes of prolonged/obstructed labour[1,2]

Direct causes	Indirect causes	Distant causes
• Cephalopelvic disproportion • Malposition and malpresentation • Foetal anomalies • Cervical dystocia • Prolonged rupture of membranes • Uterine dysfuntion	• Short statured mother • Bad obstetric history • Multiparity	• Unbooked case • Home delivery • Distance from hospital • Ignorance

Table 3.2 Interventions for the management of prolonged/obstructed labour[2–4]

Medical interventions	Non-medical interventions
• Identify signs of obstructed labour • Correct the cause, e.g. oxytocin drip or artificial rupture of the membranes • Operative/caesarean delivery • Train to use the partograph • Use the partograph	• Refer high-risk cases before the onset of labour • Refer in time • Arrange transportation

Table 3.3 Personnel, drugs and supplies required for the management of prolonged/obstructed labour[4]

Personnel	Drugs and supplies
Village/subcentre • Trained midwives • Trained health workers • Skilled birth attendants • Well-trained *dais*	• Partographs • Gloves • Clean instruments for delivery • Cap. ampicillin
PHC • Doctor • Trained midwives/nurses • Pharmacist • Driver	• Partographs • Gloves • Clean instruments for delivery • IV set, IV cannula, disposable syringes and needles, IV fluids • Inj. ampicillin 500 mg, Inj. metronidazole, Inj. gentamicin
CHC • Specialist • Nurse • Driver	• Partographs • Gloves • Clean instruments for delivery • IV set, IV cannula, disposable syringes and needles, IV fluids • Inj. ampicillin 500 mg, Inj. metronidazole, Inj. gentamicin
District hospital • Specialist • Anaesthetist • Nurse • Pathologist • Pharmacist	• Working operation theatres • Blood bank • Partographs • Gloves • Clean instruments for delivery • IV set, IV cannula, disposable syringes and needles, IV fluids • Inj. ampicillin 500 mg, Inj. metronidazole, Inj. gentamicin

PHC: primary health centre; CHC: community health centre; Cap.: capsule; Inj.: injection; IV: intravenous

Table 3.4 Management of prolonged/obstructed labour at various levels of health care

Management protocol	Village/subcentre	PHC	CHC	District hospital
Prevention	• Increasing the awareness among pregnant women about the advantages of institutional delivery through posters, the media, health workers • Delivery by —trained midwife/nurse/ skilled birth attendant • Train *dais* to use partograph —Basic principle of its use —Referral of high-risk cases	• Record-keeping and auditing of all cases of obstructed labour by the health assistant • Reinforcement of institutional delivery, i.e. well supervised delivery and postpartum period by doctor or nurse where a doctor is available for assistance	• Record-keeping and auditing of all cases of obstructed labour • Reinforcement of institutional delivery	• Record-keeping and auditing of all cases of obstructed labour • Reinforcement of institutional delivery
Women in labour	• Refer all high-risk women • Maintain partograph during labour • Encourage oral hydration • If the latent phase is prolonged, i.e. more than 8 hours —Refer to the PHC —Do not give enema or any injections for augmentation —Per abdomen examination of the foetal head in fifths should be practised —Minimal per vaginal examination should be done using asepsis and wearing gloves —If the per vaginal findings are the same after 4 hours, refer to the CHC —If the woman does not improve after 12 hours of labour she should be referred to the district hospital —If there is foetal distress, refer to the district hospital	• Refer all high-risk women • Maintain partograph during labour • Encourage oral hydration • If the latent phase is prolonged, i.e. more than 8 hours —If there has been no change in cervical findings, the patient must be in false labour —If the cervical findings have changed, then refer to the CHC —Per abdomen examination of the foetal head in fifths should be practised —Minimal per vaginal examination should be done using asepsis and wearing gloves —If the per vaginal findings are the same after 4 hours, refer to the CHC —If the woman does not improve after 12 hours of labour she should be referred to the district hospital —If there is foetal distress, refer to the district hospital	• Maintain partograph • Encourage oral hydration • If the latent phase is prolonged —If there has been no change in the cervical findings, the woman must be in false labour —If the cervical findings have changed, then rupture the membranes and start Inj. ampicillin 500 mg 6-hourly —If the membranes have already ruptured, start oxytocin and reassess contractions after 2 hours —If there is no further change in the cervical findings, refer to the district hospital • If the active phase is prolonged —If there is no change in cervical findings in 4 hours or progress is delayed then rupture the membranes and reassess after 4 hours —Start oxytocin if the membranes have already ruptured —Monitor labour and increase pitocin till contractions improve and reassess after 4 hours —If there is no progress after the above intervention, refer to the district hospital —If there are signs of obstruction, refer immediately • Refer to the district hospital if there is foetal distress and the woman requires a caesarean section • If the cervix is fully dilated	• Maintain partograph • Encourage oral hydration • If the latent phase is prolonged —If there has been no change in the cervical findings, the woman must be in false labour —If the cervical findings have changed, then rupture the membranes and start Inj. ampicillin 500 mg 6-hourly —If the membranes have already ruptured, start oxytocin and reassess contractions after 2 hours —If there is no further change in the cervical findings, critically assess the cause —If there is cephalopelvic disproportion then perform LSCS • If the active phase is prolonged —If there is no change in cervical findings in 4 hours or progress is delayed then rupture the membranes and reassess after 4 hours —Start oxytocin if the membranes have already ruptured —Monitor labour and increase pitocin till contractions improve and reassess after 4 hours —If there is no progress after the above intervention, critically assess the cause —If there is cephalopelvic disproportion perform LSCS • If there are signs of obstruction immediately

(Cont.)

Table 3.4 (*cont.*) Management of prolonged/obstructed labour at various levels of health care

Management protocol	Village/subcentre	PHC	CHC	District hospital
			and there is no descent of the head assess whether there is cephalopelvic disproportion; if yes, refer • If instrumental delivery is required then perform the same. • Give ampicillin 500 mg 6-hourly to any woman with ruptured membranes • Give ampicillin 500 mg + Inj. genatmicin 1.2 mg/kg body weight 8-hourly to any woman with ruptured membranes for more than 12 hours • Refer immediately to the district hospital if there is malpresentation • Arrange for transportation	perform LSCS • If there is foetal distress and the woman requires caesarean section perform LSCS immediately • If the cervix is fully dilated and there is no descent of the head, assess whether there is cephalopelvic disproportion; if yes, immediately perform LSCS • If instrumental delivery is required then perform the same • Give ampicillin 500 mg 6-hourly to any woman with ruptured membranes • Give ampicillin 500 mg + Inj. genatmicin 1.2 mg/kg body weight 8-hourly to any woman with ruptured membranes for more than 12 hours • If the foetus is dead and instrumental delivery is an option then refer to a tertiary hospital • If labour is prolonged and obstructed then refer to a tertiary centre

PHC: primary health centre; CHC: community health centre; Inj.: injection; LSCS: lower segment caesarean section

References

1. The puerperium (Chapter 17). In: *Williams Obstetrics, Vol. 21*. International Edition. McGraw-Hill Companies, Inc.; 2003:423.
2. Rao KB. Prolonged and obstructed Labor. In: *The management of labor*. Chennai: Orient Longman Ltd.; 1996:301.
3. Unsatisfactory progress of labor S-57. Managing Complications in Pregnancy and Childbirth. IMPAC. December, 2002 publication WHO/SEARO.
4. World Health Organization. *Std.-7 Life-saving management prolonged and obstructed Labor of. Pg 93*. Standards of Midwifery Practice for Safe Motherhood; New Delhi, 1999.

4. Puerperal sepsis

Table 4.1 Causes of puerperal sepsis[1]

Direct causes	• Prolonged labour • Prolonged leakage • Chorioamnionitis • Multiple unclean per vaginal examination • Manual removal of the placenta • Retained placental tissue
Indirect causes	• Delivery by an untrained person • Unclean/unsterile instruments • Lack of adequate antibiotic therapy after delivery
Distant causes	• Low socioeconomic status • Distance from the hospital • Delay in seeking treatment • Delay in referral
Associated factor	Underlying anaemia

Table 4.2 Interventions for various grades of puerperal sepsis

Manifestation	Medical interventions	Non-medical interventions
Grade I puerperal sepsis	Antibiotics	• Protein-rich diet • Analysis of the cause/s and auditing
Grade II puerperal sepsis	• Admission • IV antibiotics	• Protein-rich diet • Analysis of the cause/s and auditing
Grade III puerperal sepsis	• Admission • IV antibiotics • Management of complications such as septic shock, ARF, MODS • Timely referral • Surgical intervention, when essential	• Protein-rich diet • Supportive care • Transportation

IV: intravenous; ARF: acute renal failure; MODS: multiorgan dysfunction syndrome

Table 4.3 Personnel, drugs and tests required for the management of puerperal sepsis[1,2]

Grade of puerperal sepsis	Personnel	Tests	Drugs	Inpatient stay
Grade I	• Nurse • Doctor • Laboratory technician • Pharmacist	• Hb estimation • TLC and DLC	• Cap. ampicillin 500 mg or amoxicillin 6-hourly + • Tab. metronidazole 400 mg 8-hourly + • Inj. gentamicin 60 mg 8-hourly (or 1.5 mg/kg 8-hourly)	—
Grade II	• Nurse • Specialist • Laboratory technician	• Hb estimation • TLC and DLC	• Inj. cefotaxime 1 g 8-hourly • Inj. ampicillin 500 mg 6-hourly • Inj. metronidazole 400–500 mg • Inj. gentamicin 60 mg 8-hourly (or 1.5 mg/kg 8-hourly)	3–7 days
Grade III	• Specialist • Nurse • Radiologist • Pathologist • Anaesthetist	• Hb estimation • TLC and DLC • Blood culture • Urine culture • Pus culture • X-ray of the chest • X-ray of the abdomen • USG of the abdomen	Broad-spectrum antibiotics, IV cefotaxime + aminoglycoside + metronidazole till the culture reports are available	Usually prolonged

Hb: haemoglobin; TLC: total leucocyte count; DLC: differential leucocyte count; Cap.: capsule; Tab.: tablet; Inj.: injection; IV: intravenous; USG: ultrasonography

Table 4.4 Protocol for the management of puerperal sepsis at different levels of health care[1,2,4,5]

Management protocol	Village/subcentre	PHC	CHC	District hospital
Preventive measures	• ANM/nurse/midwife to follow up every delivery after 1 week • Woman delivered by a *dai* in an unclean environment must receive Cap. ampicillin 500 mg 6-hourly x 5 days from a health worker • If manual removal of the placenta has been done, give ampicillin + metronidazole 400 mg 8-hourly and Inj. gentamicin 1.2 mg/kg body weight 8-hourly (refer to a PHC) • If the membranes rupture before the second stage or multiple P/V examinations are conducted before delivery, give antibiotics	• Every woman who has delivered should receive antibiotic cover: Cap. ampicillin 500 mg 6-hourly x 5 days • If the delivery is conducted by a *dai*, give the woman Cap. ampicillin 500 mg 6-hourly • If manual removal of the placenta has been done, give Inj. ampicillin 500 mg 6-hourly + Inj. metronidazole 400 mg 6-hourly + Inj. gentamicin 1.2 mg/kg 8-hourly • If the membranes rupture before the second stage or multiple P/V examinations are conducted before delivery, give antibiotics	• Every woman who has delivered should receive antibiotic cover: Cap. ampicillin 500 mg 6-hourly x 5 days • If the delivery is conducted by a *dai*, give the woman Cap. ampicillin 500 mg 6-hourly • If manual removal of the placenta has been done, give Inj. ampicillin 500 mg 6-hourly + Inj. metronidazole 400 mg 6-hourly + Inj. gentamicin 1.2 mg/kg 8-hourly • If the membranes rupture before the second stage or multiple P/V examinations are conducted before delivery, give antibiotics	• Every woman who has delivered should receive antibiotic cover: Cap. ampicillin 500 mg 6-hourly x 5 days • If the delivery is conducted by a *dai*, give the woman Cap. ampicillin 500 mg 6-hourly • If manual removal of the placenta has been done, give Inj. ampicillin 500 mg 6-hourly + Inj. metronidazole 400 mg 6-hourly + Inj. gentamicin 1.2 mg/kg 8-hourly • If the membranes rupture before the second stage or multiple P/V examinations are conducted before delivery, give antibiotics
History	• Any woman with fever, foul-smelling discharge, increased bleeding, diarrhoea/vomiting/ constipation, abdominal distension • Refer to a PHC	• Any woman with fever, foul-smelling discharge, increased bleeding, diarrhoea/vomiting/ constipation, abdominal distension • Doctor to attend	• Any woman with fever, foul-smelling discharge, increased bleeding, diarrhoea/vomiting/ constipation, abdominal distension • Specialist to attend	• Any woman with fever, foul-smelling discharge, increased bleeding, diarrhoea/ vomiting/constipation, abdominal distension • Specialist to attend
Examination		• Look for pallor, icterus • Check the pulse and BP • Note the temperature • Examine the respiratory and cardiovascular systems	• Look for pallor, icterus • Check the pulse and BP • Note the temperature • Examine the respiratory and cardiovascular systems	• Look for pallor, icterus • Check the pulse and BP • Note the temperature • Examine the respiratory and cardiovascular systems
Puerperal sepsis Grade I		• P/A examination: Feel of the abdomen, distension, any tenderness, guarding, rigidity, bowel sounds, size of the uterus • P/V examination: Size of the uterus, tenderness, presence of any foreign body, any retained products • Examination of the breasts, episiotomy wound and lower limbs for swelling • Cap. ampicillin 500 mg 6-hourly + Tab. metronidazole 400 mg 8-hourly + Inj. gentamicin 1.2 mg/kg body weight 8-hourly • If there is no improvement in 24–48 hours or worsening, refer	• P/A examination: Feel of the abdomen, distension, any tenderness, guarding, rigidity, bowel sounds, size of the uterus • P/V examination: Size of the uterus, tenderness, presence of any foreign body, any retained products • Examination of the breasts, episiotomy wound and lower limbs for swelling • Cap. ampicillin 500 mg 6-hourly + Tab. metronidazole 400 mg 8-hourly + Inj. gentamicin 1.2 mg/kg body weight 8-hourly • If there is no improvement in 24–48 hours or worsening, admit	• P/A examination: Feel of the abdomen, distension, any tenderness, guarding, rigidity, bowel sounds, size of the uterus • P/V examination: Size of the uterus, tenderness, presence of any foreign body, any retained products • Examination of the breasts, episiotomy wound and lower limbs for swelling • Cap. ampicillin 500 mg 6-hourly + Tab. metronidazole 400 mg 8-hourly + Inj. gentamicin 1.2 mg/kg body weight 8-hourly • If there is no improvement in 24–48 hours or worsening, admit
Puerperal sepsis Grade II			• Monitor the vital signs/ temperature • Give IV antibiotics: cefotaxime (1 g 8-hourly) + gentamicin (60 mg 8-hourly) + metronidazole	• Monitor the vital signs/ temperature • Give IV antibiotics: cefotaxime (1 g 8-hourly) + gentamicin (60 mg 8-hourly) + metronidazole

(Cont.)

Table 4.4 *(cont.)* Protocol for the management of puerperal sepsis at different levels of health care[1,2,4,5]

Management protocol	Village/subcentre	PHC	CHC	District hospital
			(400–500 mg 8-hourly) • Maintain an input/output chart of the patient	(400–500 mg 8-hourly) • Maintain an input/output chart of the patient
Investigations			• Hb, TLC and DLC, X-ray of the chest, X-ray of the abdomen	• Hb, TLC and DLC, X-ray of the chest, X-ray of the abdomen, USG
Puerperal sepsis Grade III		• Secure an IV line • Start antibiotics • Refer to district hospital	• Secure an IV line • Start antibiotics • Refer to district hospital	• Secure an IV line • Start broad-spectrum IV antibiotics —Inj. cefotaxime 1 g 8-hourly —Inj. gentamicin 1.2 mg/kg —Inj. metronidazole 400 mg 8-hourly • Maintain a record of the vital signs and temperature • If there is no improvement in 24–48 hours, refer to a tertiary centre • If the patient has intraperitoneal pus collection or pelvic abscess, refer to a tertiary centre
Investigations			• Hb, TLC and DLC, X-ray of the chest, X-ray of the abdomen	• Hb, TLC and DLC, X-ray of the chest, X-ray of the abdomen, USG
Septic shock	Refer to a higher centre	• Secure an IV line (doctor and nurse) • Start IV antibiotics (broad-spectrum) • Refer to a tertiary centre	• Secure an IV line (doctor and nurse) • Start IV antibiotics (broad-spectrum) • Catheterize the bladder and refer to a tertiary centre	• Secure an IV line • Start IV antibiotics (broad-spectrum) • Catheterize the bladder • Refer to a tertiary centre
Availability of drugs	• Soap, essential equipment for delivery • Gloves • Cap. amoxicillin 500 mg • Cap. ampicillin 500 mg • Tab. metronidazole 400 mg	• Gloves • Inj. ampicillin 500 mg • Inj. gentamicin 80 mg • Inj. metronidazole 500 mg • IV set • Disposable syringes • Cap. ampicillin 500 mg • Tab. metronidazole 400 mg	• Gloves • Foley catheter • Urobag • Inj. ampicillin 500 mg • Inj. gentamicin 80 mg • Inj. metronidazole 500 mg • IV set • Disposable syringes • Cap. ampicillin 500 mg • Tab. metronidazole 400 mg	• 3 pairs of gloves • Foley catheter • Urobag • Inj. ampicillin 500 mg • Inj. gentamicin 80 mg • Inj. metronidazole 500 mg • IV set • Disposable syringes • Cap. ampicillin 500 mg • Tab. metronidazole 400 mg

PHC: primary health centre; CHC: community health centre; ANM: auxiliary nurse–midwife; Cap.: capsule; Inj.: injection; Tab.: tablet; P/A: per abdomen; P/V: per vaginam; TLC: total leucocyte count; DLC: differential leucocyte count; USG: ultrasonography

References

1. Puerperal infection (Chapter 26). *William Obstetrics, Vol. 21*. International edition. McGraw-Hill Companies, Inc.; 2003:671.
2. American College of Obstetricians and Gynecologists. *Antimicrobial therapy for obstetric patients.* ACOG Educational Bulletin No. 245, March 1998.
3. Safe motherhood needs assessment. Available from URL: who.int/reproductive–health/MNBH/smna_annexa.en.html.
4. World Health Organization. Life-saving management of puerperal sepsis. In: *Standard of midwifery practice for safe motherhood, Vol. I.* New Delhi: WHO, SEARO, Standard Document Regional Publication No. 38;1999:116.
5. World Health Organization. Peritonitis. In: *Integrated management of pregnancy and childbirth. A guide for midwives and doctors.* New Delhi: WHO, SEARO; 2002:S-108.

5. Septic abortion

Table 5.1 Causes of septic abortion[1]

Direct causes	Indirect causes	Distant causes
• Foreign bodies inserted in the genital tract • Injury and sepsis of the genital organs • Retained foetal tissue • Perforation of the uterus	• Abortion by untrained persons • Illegal abortion • Unclean/unsterile instruments • Use of abortifacients	• Ignorance • Unmarried girl • Prenatal sex determination • Fear of loss of confidentiality

Table 5.2 Interventions for various grades of septic abortion

Manifestation	Medical interventions	Non-medical interventions
History of intervention or abortion by unskilled person	• Give oral antibiotics • Rule out injury • Confirm complete abortion	• Give follow up advice
Grade I sepsis	• Give antibiotics	• Refer if history of intervention by unskilled person
Grade II sepsis	• Admit • Give IV antibiotics	• Refer if history of intervention by unskilled person
Grade III sepsis	• Admit • Give IV antibiotics	• Provide supportive care • Arrange for transportation

Table 5.3 Personnel, investigations and drugs required to treat septic abortion[1,2]

Grade of septic abortion	Personnel	Tests	Drugs	Inpatient stay
Grade I	• Nurse • Doctor • Laboratory technician	• Haemoglobin estimation • Total and differential leucocyte counts	• Cap. ampicillin 500 mg OR amoxicillin 6-hourly + • Tab. metronidazole 400 mg 8-hourly + • Inj. gentamicin 60 mg 8-hourly (or 1.5 mg/kg 8-hourly)	
Grade II	• Nurse • Specialist • Laboratory technician	• Haemoglobin estimation • Total and differential leucocyte counts	• Inj. cefotaxime 1 g 8-hourly • Inj. ampicillin 500 mg 6-hourly • Inj. metronidazole 400–500 mg • Inj. gentamicin 60 mg 8-hourly (or 1.5 mg/kg 8-hourly)	3–7 days
Grade III	• Specialist • Nurse • Radiologist • Pathologist • Anaesthetist	• Haemoglobin estimation • Total and differential leucocyte counts • Blood biochemistry • Urine examination • Pus for culture and antibiotic sensitivity • X-ray of the chest • X-ray of the abdomen • Ultrasonography of the abdomen	• Broad-spectrum antibiotics cefotaxime + aminoglycoside + metronidazole till culture reports are available	Usually prolonged

Cap: capsule; Tab.: tablet; Inj.: injection

Table 5.4 Management of septic abortion at various levels of health care[1–3]

Management protocol	Village/subcentre	PHC	CHC	District hospital
Preventive measures	• All abortions to be performed by registered, qualified personnel • MTP facilities to be made more readily available • Promotion of these facilities • Confidentiality to be maintained in case the woman is unmarried • Promotion of contraceptive facilities • Awareness of complications associated with abortion • Auditing all cases of septic abortion • Continued vigilance against mid-trimester abortion	• All abortions to be performed by registered, qualified personnel • MTP facilities to be made more readily available • Promotion of these facilities • Confidentiality to be maintained in case the woman is unmarried • Promotion of contraceptive facilities • Awareness of complications associated with abortion • Auditing all cases of septic abortion • Continued vigilance against mid-trimester abortion • Prolonged rupture of the membranes or multiple per vaginal examinations before delivery • Antibiotics	• All abortions to be performed by registered, qualified personnel • MTP facilities to be made more readily available • Promotion of these facilities • Confidentiality to be maintained in case the woman is unmarried • Promotion of contraceptive facilities • Awareness of complications associated with abortion • Auditing all cases of septic abortion • Continued vigilance against mid-trimester abortion • Prolonged rupture of the membranes or multiple per vaginal examinations before delivery • Antibiotics	• All abortions to be performed by registered, qualified personnel • MTP facilities to be made more readily available • Promotion of these facilities • Confidentiality to be maintained in case the woman is unmarried • Promotion of contraceptive facilities • Awareness of complications associated with abortion • Auditing all cases of septic abortion • Continued vigilance against mid-trimester abortion
Septic abortion	• Any woman with a h/o abortion performed by untrained personnel must be examined by a doctor • Refer immediately	• Any woman with a h/o abortion performed by untrained personnel must be examined by a doctor	• Any woman with a h/o abortion performed by untrained personnel must be examined by a specialist	• Any woman with a h/o abortion performed by untrained personnel must be examined by a specialist
Examination		• Look for pallor, icterus • Record the pulse, BP, temperature • Examine the respiratory and cardiovascular systems	• Look for pallor, icterus • Record the pulse, BP, temperature • Examine the respiratory and cardiovascular systems	• Look for pallor, icterus • Record the pulse, BP, temperature • Examine the respiratory and cardiovascular systems
Grade I sepsis	Refer immediately to the nearest health facility having a doctor	• P/A examination: feel of the abdomen, distension, presence of tenderness, guarding, rigidity, bowel sounds • Size of the uterus • P/V examination: size of the uterus, tenderness, any foreign body, any retained products of conception • Examination of the breasts, lower limbs for swelling • Start oral antibiotics: ampicillin 500 mg 6-hourly + metronidazole 400 mg 8-hourly + Inj. gentamicin 80 mg IM BD • If no improvement is seen in 24–48 hours or the patient's condition worsens, refer • If abortion done by untrained personnel refer to the district hospital	• P/A examination: feel of the abdomen, distension, presence of tenderness, guarding, rigidity, bowel sounds • Size of the uterus • P/V examination: size of the uterus, tenderness, any foreign body, any retained products of conception • Examination of the breasts, lower limbs for swelling • Start oral antibiotics: ampicillin 500 mg 6-hourly + metronidazole 400 mg 8-hourly + Inj. gentamicin 80 mg IM BD • If no improvement is seen in 24–48 hours or the patient's condition worsens, refer • If abortion done by untrained personnel refer to the district hospital	• P/A examination: feel of the abdomen, distension, presence of tenderness, guarding, rigidity, bowel sounds • Size of the uterus • P/V examination: size of the uterus, tenderness, any foreign body, any retained products of conception • Examination of the breasts, lower limbs for swelling • Start oral antibiotics: ampicillin 500 mg 6-hourly + metronidazole 400 mg 8-hourly + Inj. gentamicin 80 mg IM BD • If no improvement is seen in 24–48 hours or the patient's condition worsens, refer • If abortion done by untrained personnel refer to the district hospital

(Cont.)

Table 5.4 (*cont.*) Management of septic abortion at various levels of health care[1–3]

Management protocol	Village/subcentre	PHC	CHC	District hospital
Grade II sepsis	Refer immediately to the nearest health facility having a doctor	Refer to CHC	• Admit • Monitor vital signs/temperature • Give IV antibiotics cefotaxime (1g 8-hourly) + gentamicin (60 mg 8-hourly) + metronidazole (400–500 mg 8-hourly) • Maintain an input/output chart of the patient	• Admit • Monitor vital signs/temperature • Give IV antibiotics cefotaxime (1 g 8-hourly) + gentamicin (60 mg 8-hourly) + metronidazole (400–500 mg 8-hourly) • Maintain an input/output chart of the patient
Investigations			• Estimate Hb, TLC and DLC, and do an X-ray of the chest and abdomen	• Estimate Hb, TLC and DLC, and do an X-ray of the chest and abdomen, and ultrasound of the abdomen and pelvis
Grade III sepsis	Refer to district hospital or tertiary centre	Refer to district hospital or tertiary centre	• Secure an IV line • Start broad-spectrum antibiotics —Inj. cefotaxime 1 g 8-hourly —Inj. metronidazole 400 mg 6-hourly —Inj. gentamicin 1.2 mg/kg • Refer to district hospital	• Secure an IV line • Start broad-spectrum antibiotics —Inj. cefotaxime 1 g 8-hourly —Inj. metronidazole 400 mg 6-hourly —Inj. gentamicin 1.2 mg/kg • Maintain vital signs and an input, output record • If no improvement in 24–48 hours, refer • If the patient has inraperitoneal pus collection or a pelvic abscess, refer
Investigations			• Estimate Hb, TLC and DLC, and do an X-ray of the chest and abdomen	• Estimate Hb, TLC and DLC, and do an X-ray of the chest and abdomen
Septic shock	• Refer to a higher centre	• Secure an IV line (doctor and nurse) • Start broad-spectrum IV antibiotics —Inj. cefotaxime 1 g 8-hourly —Inj. metronidazole 400 mg 6-hourly —Inj. gentamicin 1.2 mg/kg • Refer to a tertiary centre	• Secure an IV line • Start broad-spectrum IV antibiotics —Inj. cefotaxime 1 g 8-hourly —Inj. metronidazole 400 mg 6-hourly —Inj. gentamicin 1.2 mg/kg • Catheterize the bladder • Refer to a tertiary centre	• Secure an IV line • Start broad-spectrum IV antibiotics —Inj. cefotaxime 1 g 8-hourly —Inj. metronidazole 400 mg 6-hourly —Inj. gentamicin 1.2 mg/kg • Catheterize the bladder • Refer to a tertiary centre
Availability of drugs	• Cap. amoxicillin 500 mg OR • Cap. ampicillin 500 mg • Tab. metronidazole 400 mg • Gloves	• Inj. ampicillin 500 mg • Inj. gentamicin 80 mg • Inj. metronidazole 500 mg • IV set • Disposable syringes • Cap. ampicillin 500 mg • Tab. metronidazole 400 mg	• Inj. ampicillin 500 mg • Inj. gentamicin 80 mg • Inj. metronidazole 500 mg • IV set • Disposable syringes • Cap. ampicillin 500 mg • Tab. metronidazole 400 mg • Foley catheter • Urobag	• Inj. ampicillin 500 mg • Inj. gentamicin 80 mg • Inj. metronidazole 500 mg • IV set • Disposable syringes • Cap. ampicillin 500 mg • Tab. metronidazole 400 mg • Foley catheter • Urobag

Inj: injection; Tab.: tablet; Cap.: capsule; MTP: medical termination of pregnancy; BP: blood pressure; h/o: history of; P/A: per abdomen; P/V: per vaginam; IV: intravenous; IM: intramuscular; Hb: haemoglobin; TLC: total leucocyte count; DLC: differential leucocyte count; PHC: primary health centre; CHC: community health centre

References

1. Abortion (Chapter 33). *Williams Obstetrics, Vol. 21*. International edition. McGraw-Hill Companies, Inc.; 2003:877.
2. American College of Obstetricians and Gynecologists. *Antimicrobial therapy for obstetric patients*. ACOG Educational Bulletin No. 245, March 1998.
3. Safe motherhood needs assessment. Available from URL: who.int/reproductive–health/MNBH/Smna annexa.en.html.

6. Hypertension and eclampsia in pregnancy

Table 6.1 Causes of hypertension and eclampsia in pregnancy[1,2]

Main causes

Direct
- Genetic predisposition
- Immunological causes
- Unknown aetiology

Indirect
- Calcium intake

Distant
- Lack of antenatal care
- Failure to record the blood pressure during antenatal visits can cause worsening of hypertension to pre-eclampsia

Interaction with other causes
- Essential hypertension
- Associated diabetes
- Elderly woman
- Underlying renal disorder
- Systemic lupus erythematosus

Table 6.2 Manifestations and management of hypertension in pregnancy[3,4]

Manifestations	Management
Chronic hypertension	Refer to a tertiary-level health centre
Gestational hypertension	Manage at a CHC or district hospital
Pre-eclampsia	Refer to a tertiary-level health centre
Eclampsia	Immediate care at subcentre/PHC/CHC/ district hospital followed by prompt referral to a tertiary centre

CHC: community health centre; PHC: primary health centre

Table 6.3 Resource requirement for the management of hypertension in pregnancy

Condition	Personnel	Tests	Drugs/other interventions	Inpatient stay
Chronic hypertension	• Midwife • Nurse • Doctor • Specialist	• Renal function tests • Platelet count • Urine examination for proteinuria	• Tab. alphamethyldopa 250 mg 8-hourly to 4 g/day • Tab. nifedipine 5 mg, 15 to 120 mg per day • Tab. nifedipine retard 20–120 mg per day	Refer
Gestational hypertension	• Midwife • Nurse • Doctor • Specialist • Laboratory technician	• Renal function tests • Platelet count • Urine examination for proteinuria	• Magnesium sulphate ampoules containing 1 g in 2 ml as 50% solution (Dosage as scheduled on page 134) • Others: Ensure that the sphygmomanometers with all personnel doing antenatal check-up are in working condition. Check the availability of the following: —Weighing machine —Equipment for urine examination by boiling method or Uristix —Oxygen cylinders —Disposable syringes —Mouth gag/airway —For sedation give Inj. phenergan and Inj. pethidine	2–3 weeks
Pre-eclampsia	• Midwife • Nurse • Doctor • Specialist • Laboratory technician • Driver	• Renal function tests • Platelet count • Urine examination for proteinuria		Refer
Eclampsia	• Midwife • Nurse • Doctor • Specialist • Laboratory technician • Driver	• Renal function test • Platelet count • Urine examination for proteinuria		

Tab.: tablet; Inj.: injection

Table 6.4 Management of hypertension in pregnancy at various levels of health care[3–6]

Management	Subcentre	PHC	CHC	District hospital
BP ≥140/90 mmHg and gestation period <20 weeks	Refer to a medical college	Refer to a medical college	Refer with a referral slip to a medical college or a tertiary centre	Refer with a referral slip to a medical college or a tertiary centre
BP≥140/90 mmHg and gestation period >20 weeks	Refer	Refer	Refer	• Advise —Daily recording of BP at home —Rest at home —Follow up every 3 weeks till 28 weeks then every 2 weeks to monitor the foetal growth clinically —Perform an ultrasonography when required to monitor foetal growth —Advise the woman to stay near the hospital —Advise the woman to report to the hospital if the BP is ≥150/100 mmHg • Explain to the patient the symptoms and signs of impending eclampsia and ask to report to the hospital if such symptoms/signs develop —Admit patients who are non-compliant or for whom follow up is difficult —Induce labour at term, i.e. at 38 weeks
If the diastolic BP is >100 mmHg	Refer immediately without delay	• Start antihypertensive drug: Tab. alphamethyldopa 250 mg 8-hourly and refer the patient • Check the urine for proteinuria	• Start antihypertensive drug: Tab. alphamethyldopa 250 mg 8-hourly and refer the patient • Check the urine for proteinuria	• Admit the patient • Start Tab. alphamethyldopa 250 mg 8-hourly • Increase the dosage as per requirement • Give nifedipine in addition, if required • Monitor the BP, urine for proteins, foetal growth by ultrasonography and do RFT • Induce labour at term if the BP is controlled on drugs and the foetal growth is adequate
If the diastolic BP is ≥110 mmHg	Refer immediately without delay	• Check the urine for proteinuria • Start an anti-hypertensive drug: Tab. nifedipine 5 mg stat and then 8-hourly • Confirm that the patient has no signs of impending eclampsia • Refer immediately with a referral slip to district hospital or tertiary centre	• Check the urine for proteinuria • Start an antihypertensive drug: Tab. nifedipine 5 mg stat and then 8-hourly • Confirm that the patient has no signs of impending eclampsia • Refer immediately with a referral slip to district hospital or tertiary centre	• Admit the patient • Check the urine for proteinuria • Start antihypertensive drugs: —Tab. nifedipine retard 10 mg BD or Tab. nifedipine 5 mg 8-hourly —Add Tab. alphamethyldopa if the BP is not controlled —Increase the dosage if required

(Cont.)

Table 6.4 (*cont.*) Management of hypertension in pregnancy at various levels of health care[3–6]

Management	Subcentre	PHC	CHC	District hospital
				—Monitor the foetal growth —Monitor the BP 12-hourly, test the urine for proteinuria, do RFT and check the platelet count • Refer if proteinuria is present and the BP is not controlled with drugs • If there is worsening of hypertension, terminate the pregnancy • If the foetal growth is compromised, terminate the pregnancy
Patient with increased BP with symptoms and signs of impending eclampsia	Refer quickly	• Sedation • Antihypertensive drug: Tab. nifedipine 5 mg stat • Refer	• Specialist to attend in the following cases: —If the BP is increased —Proteinuria is present —Patient shows symptoms/ signs of impending eclampsia • Give a loading dose of magnesium sulphate and refer to a tertiary centre or give sedation and antihypertensives and refer • Before giving magnesium sulphate, confirm that —the knee jerks are present —the urine output is more than 30 ml/hour —the RR is >16/minute then refer to a tertiary centre	• Specialist to attend in the following cases: —If the BP is increased —Proteinuria is present —Patient shows symptoms/ signs of impending eclampsia • Give a loading dose of magnesium sulphate and refer to a tertiary centre or give sedation and antihypertensives and refer • Before giving magnesium sulphate, confirm that —the knee jerks are present —the urine output is more than 30 ml/hour —the RR is >16/minute then refer to a tertiary centre
Woman with eclampsia	• MHW/nurse/midwife to attend • Turn the woman to the left • Keep her on the floor to prevent her from falling • Arrange for transportation and transfer to a PHC • Explain to the family/ relatives that the condition is related to increased BP in pregnancy	• Doctor to attend • History: Confirm the presence of prodromal symptoms, any record of increased BP, h/o passing adequate urine • Rule out any previous h/o epilepsy, fever • Examination —State of consciousness —Oedema —Pulse —BP —Cardiovascular system —RR —Knee jerks • P/A examination: Examination can be delayed till the patient settles • Management: Treat any woman as eclampsia if	• Specialist to attend • History: Confirm the presence of prodromal symptoms, any record of increased BP, h/o passing adequate urine • Rule out any previous h/o epilepsy, fever • Examination —State of consciousness —Oedema —Pulse —BP —Cardiovascular system —RR —Knee jerks • P/A examination: Examination can be delayed till the patient settles • Management: Treat any woman as eclampsia if	• Specialist to attend • History: Confirm the presence of prodromal symptoms, any record of increased BP, h/o passing adequate urine • Rule out any previous h/o epilepsy, fever • Examination —State of consciousness —Oedema —Pulse —BP —Cardiovascular system —RR —Knee jerks • P/A examination: Examination can be delayed till the patient settles • Management: Treat any woman as eclampsia if

(Cont.)

Table 6.4 (*cont.*) Management of hypertension in pregnancy at various levels of health care[3–6]

Management	Subcentre	PHC	CHC	District hospital
		she presents with convulsions and increased BP, and has no past h/o convulsions • Give oxygen • Turn the patient to the left side • Secure the airway if possible • Carry out oral suction to clear the airway • Secure an IV line • Give a loading dose of magnesium sulphate as detailed below • Give an antihypertensive drug: Tab. nifedipine 5 mg (not simultaneously with magnesium sulphate) • Arrange for transportation and refer to a higher centre • Transfer with a nurse • Catheterize the bladder 2 hours after giving magnesium sulphate	she presents with convulsions and increased BP, and has no past h/o convulsions • Give oxygen • Turn the patient to the left side • Secure the airway if possible • Carry out oral suction to clear the airway • Secure an IV line • Give a loading dose of magnesium sulphate as detailed below • Give an antihypertensive drug: Tab. nifedipine 5 mg (not simultaneously with magnesium sulphate) • Arrange for transportation and refer to a higher centre • Transfer with a nurse • Catheterize the bladder 2 hours after giving magnesium sulphate	she presents with convulsions and increased BP, and has no past h/o convulsions • Give oxygen • Turn the patient to the left side • Secure the airway if possible • Carry out oral suction to clear the airway • Secure an IV line • Give a loading dose of magnesium sulphate as detailed below • Give an antihypertensive drug: Tab. nifedipine 5 mg (not simultaneously with magnesium sulphate) • Arrange for transportation and refer to a higher centre without delay • Transfer with a nurse • Catheterize the bladder 2 hours after giving magnesium sulphate

PHC: primary health centre; CHC: community health centre; BP: blood pressure; RFT: renal function tests; BP: blood pressure; MHW: multipurpose health worker; h/o: history of; RR: respiratory rate; P/A: per abdomen; Tab.: tablet; IV: intravenous

Loading dose of magnesium sulphate

- If the respiratory rate is >16/minute, the urine output is ≥30 ml/hour and knee jerks are present, give 4 g of 20% magnesium sulphate intravenous, slowly over 5–7 minutes, i.e. 4 g of 50%, i.e. 8 ml of magnesium sulphate solution diluted in 12 ml of normal saline.

  +

  5 g of 50% magnesium sulphate, i.e. 10 ml in each buttock deep intramuscular (IM) with 1 ml of 2% lignocaine (a total of 10 g IM)
- Mention the dose and time of giving the loading dose on the referral slip when the patient is referred.

Follow-up dose

- Check that
  —the urine output is ≥30 ml/hour
  —the knee jerks are present
  —the respiratory rate is >16/minute.
- Then give 5 g of 50% solution of magnesium sulphate, single dose, deep IM injection.
- Repeat the dose every 4 hours.
- Transfer the patient to a tertiary centre without delay.

References

1. Dekker GA, Sibai BM. Etiology and pathogensis of pre-eclampsia: Current concepts. *Am J Obstet Gynecol* 1998;**179:**1359.
2. Hypertensive disorders of pregnancy. In: *Williams Obstetrics.* International edition. McGraw-Hill Companies, Inc.; 2003:567.
3. World Health Organization. Life-saving management of eclampsia. In: *Standard of midwifery practice for safe motherhood, Vol. I.* New Delhi: WHO, SEARO, Standard Document, Regional Publication No. 38;1999:90.
4. World Health Organization. *Integrated management of pregnancy and childbirth. A guide for midwives and doctors.* New Delhi: WHO, SEARO; 2002.
5. Arulkumaran S, Ratnaum SS. Management of eclampsia. In: *The management of labour*. Chennai: Orient Longman; 1996:330.

7. Anaemia in pregnancy

Table 7.1 Causes of anaemia in pregnancy

	Direct[1,2]	Indirect[1,2]	Distant[1,2]
Main causes	Iron deficiency anaemia	• Poor intake of iron • Intolerance to iron • Poor iron reserves among women • Multiparity • Lack of birth spacing	• Poor socioeconomic status • Illiteracy • Unawareness and non-compliance • Teenage marriage and pregnancy • Poor supply of iron tablets
Associated causes	• Malabsorption • Worm infestation • Malaria • Other anaemias	—	—

Table 7.2 Interventions for mild, moderate and severe anaemia

Manifestation	Medical interventions	Non-medical interventions
Mild anaemia	• Oral iron therapy: 100 mg elemental iron tablet + folic acid 0.5 mg, twice daily • Deworming: Tab. mebendazole 100 mg, BD x 3 days • Prophylaxis and treatment for malaria	Dietary supplementation of iron and protein
Moderate anaemia	• Oral iron therapy: 100 mg elemental iron tablet + folic acid 0.5 mg, twice daily • Deworming: Tab. mebendazole 100 mg, BD x 3 days • Prophylaxis and treatment for malaria	Dietary supplementation of iron and protein
Severe anaemia	• Oral iron therapy or intramuscular iron injections or blood transfusion	Dietary supplementation of iron and protein
Severe anaemia with circulatory failure	Referral to a tertiary health care centre	

Table 7.3 Personnel, tests and drugs required for management of anaemia in pregnancy

Anaemia in pregnancy	Personnel	Tests	Drugs	Inpatient stay
Routine antenatal check-up for women[3]	• Nurse/midwife/ skilled birth attendant at the village/subcentre • Doctor • Specialist • Laboratory technician • Pharmacist	Hb estimation	Iron tablet containing 100 mg elemental iron + 0.5 mg folic acid × 100 days after the first trimester	—
Mild anaemia[4,5]	• Nurse/midwife • Doctor • Specialist • Laboratory technician • Pharmacist	• Hb estimation • Peripheral blood film • Urine: Routine and microscopy	• Tab. iron containing 100 mg elemental iron + 0.5 mg folic acid, BD, till the anaemia is corrected • Tab. mebendazole 100 mg BD × 3 days • Tab. chloroquine for treatment and prophylaxis of malaria	—
Moderate anaemia[4,5]	• Nurse • Doctor • Specialist • Laboratory technician • Pharmacist	• Hb estimation • Peripheral blood film • Stool examination • Urine: Routine and microscopy	• Iron tablet containing 100 mg elemental iron + 0.5 mg folic acid, BD, till the anaemia is corrected • Tab. mebendazole 100 mg BD × 3 days • Tab. cloroquine for treatment and prophylaxis of malaria • Iron dextran injections for IM use, 6–8 injections per person	1–2 weeks

(Cont.)

Table 7.3 (*cont.*) Personnel, tests and drugs required for management of anaemia in pregnancy

Anaemia in pregnancy	Personnel	Tests	Drugs	Inpatient stay
Severe anaemia[4,5,6]	• Nurse • Doctor • Specialist • Pathologist • Laboratory technician • Pharmacist • Driver	• Hb estimation • Peripheral blood film • TLC and DLC • Stool examination • Urine culture • Iron studies	• Blood transfusion • Iron injections IM • Tab. iron • Tab. mebendazole • Tab. chloroquine • Oxygen cylinders • IV sets, BT sets • Disposable syringes and needles • Inj. frusemide • Inj. methergin • Inj. oxytoxin • Tab. misoprostol • 250 mg methergin • Ventouse • Outlet forceps	1–2 weeks

Hb: haemoglobin; Tab.: tablet; Inj.: injection; IV: intravenous; TLC: total leucocyte count; DLC: differential leucocyte count

Table 7.4 Protocol for management of anaemia in pregnancy

Protocol for management	Village level/subcentre	PHC	CHC	District hospital
Screening				
History	Diet, previous menstrual cycles, previous childbirth, fever, worms in stool, breathlessness	Diet, previous menstrual cycles, previous childbirth, fever, worms in stool, breathlessness	Diet, previous menstrual cycles, previous childbirth, fever, worms in stool, breathlessness	Diet, previous menstrual cycles, previous childbirth, fever, worms in stool, breathlessness
Examination	• Look for pallor • Examine the conjunctiva, nails • Examine the tongue • Look for oedema of the feet • Note the weight • Check the pulse and blood pressure • Examine the abdomen	• Look for pallor • Examine the conjunctiva, nails • Examine the tongue • Look for oedema of the feet • Note the weight • Check the pulse and blood pressure • Examine the abdomen	• Look for pallor • Examine the conjunctiva, nails • Examine the tongue • Look for oedema of the feet • Note the weight • Check the pulse and blood pressure • Examine the abdomen	• Look for pallor • Examine the conjunctiva, nails • Examine the tongue • Look for oedema of the feet • Note the weight • Check the pulse and blood pressure • Examine the abdomen
Investigations	Hb estimation	Hb estimation	• Hb estimation • Peripheral blood film, TLC and DLC • Urine: Routine and microscopy • Stool examination	• Hb estimation • Peripheral blood film, TLC and DLC • Urine: Routine and microscopy • Stool examination • Urine culture
Mild anaemia Hb level: 8–10 g%	• Double dose of iron tablets • Follow up at 4 weeks by giving an appointment • Confirm compliance at 2 weeks by home visit by health worker • Deworming • Malaria prophylaxis in endemic areas • Check the Hb level again after 4 weeks • Inform about the case to the AWW for dietary benefits	• Double dose of iron tablets • Follow up at 4 weeks by giving an appointment • Confirm compliance at 2 weeks by home visit by health worker • Deworming • Malaria prophylaxis in endemic areas • Check the Hb level again after 4 weeks • Inform about the case to the AWW for dietary benefits	• Double dose of iron tablets • Follow up at 4 weeks by giving an appointment • Confirm compliance at 2 weeks by home visit by health worker • Deworming • Malaria prophylaxis in endemic areas • Check the Hb level again after 4 weeks • Inform about the case to the AWW for dietary benefits	• Double dose of iron tablets • Follow up at 4 weeks by giving an appointment • Confirm compliance at 2 weeks by home visit by health worker • Deworming • Malaria prophylaxis in endemic areas • Check the Hb level again after 4 weeks • Inform about the case to the AWW for dietary benefits

(Cont.)

Table 7.4 (*cont.*) Protocol for management of anaemia in pregnancy

Protocol for management	Village level/subcentre	PHC	CHC	District hospital
Moderate anaemia Hb level: 6.5–8 g% (pregnancy less than 8 months)	Refer to a PHC	• Double dose of iron tablets • Deworming • Record the case and inform the health assistant • Confirm compliance at 2 weeks • If compliance is good, continue treatment and check the Hb level again at 4 weeks • If compliance is poor, give iron injections IM • If there is no improvement, refer with a referral slip • If the Hb level is less than 8 g% record this and the registration number in the report register in a different coloured ink (green) • Inform the health assistant who should form a link with an MHW of the village for follow up and auditing	• Double dose of iron tablets • Deworming • Record the case and inform the health assistant • Confirm compliance at 2 weeks • If compliance is good, continue treatment and check the Hb level again at 4 weeks • If compliance is poor, give iron injections IM • If there is no improvement, refer with a referral slip • If the Hb level is less than 8 g% record this and the registration number in the report register in a different coloured ink (green) • Inform the health assistant who should form a link with an MHW of the village for follow up and auditing	• Double dose of iron tablets • Deworming • Record the case and inform the health assistant • Confirm compliance at 2 weeks • If compliance is good, continue treatment and check the Hb level again at 4 weeks • If compliance is poor, give iron injections IM • If there is no improvement, refer with a referral slip to medical college • If the Hb level is less than 8 g% record this and the registration number in the report register in a different coloured ink (green) • Inform the health assistant who should form a link with an MHW of the village for follow up and auditing
Moderate anaemia (pregnancy more than 8 months but less than 9 months)	Refer	Refer to a CHC	• Double the intake of iron tablets • Deworming • Malaria prophylaxis • Follow up at 2 weeks with an appointment • Non-medical health officers/ assistant should give feed-back to the health assistant in charge of the patient's locality for home visits to check compliance and for auditing • If the compliance or tolerance is poor, give iron injections • If there is no improvement, refer	• Double the intake of iron tablets • Deworming • Malaria prophylaxis • Follow up at 2 weeks with an appointment • Non-medical health officers/ assistant should give feed-back to the health assistant in charge of the patient's locality for home visits to check compliance and for auditing • If the compliance or tolerance is poor, give iron injections • If there is no improvement, refer
Moderate anaemia (pregnancy more than 9 months)	Refer	Refer to a district hospital	Refer to a district hospital	• Admit • Give treatment as prescribed above
Patient with moderate anaemia in labour	Refer	Refer	Refer	• Active management of the third stage of labour • Blood should be cross-matched and kept ready
Severe anaemia (pregnancy less than 8 months)	Refer	Refer	Refer	• If the patient is not in failure then admit • Investigate the cause • Give double the dose of iron therapy, change to injectable iron if there is still no improvement • Refer if the patient is in failure

(Cont.)

Table 7.4 (*cont.*) Protocol for management of anaemia in pregnancy

Protocol for management	Village level/subcentre	PHC	CHC	District hospital
Severe anaemia (pregnancy more than 8 months)	Refer	Refer to a district hospital	Refer	• Investigation of anaemia • Blood transfusion (PCV) under diuretic cover • If the patient is in failure, refer to a higher centre
Severe anaemia in failure	Refer	Refer	Refer	Refer
Patient with severe anaemia in labour	Refer	Doctor/nurse/midwife to attend • Establish an IV line • Do a per vaginal examination Patient in early labour • Refer to the nearest state medical hospital/tertiary health centre • Trained midwife/nurse to accompany who can conduct the delivery • Carry adequate oxytocics and methergin Patient in advanced labour • Doctor to be present • Prop up the patient • Give oxygen inhalation • Avoid fluid overload • Keep injections of methergin, oxytocics and misoprostol tablets ready • Preferable: vacuum or outlet forceps delivery by a doctor or trained midwife • Active management of the third stage of labour • Inj. methergin or Tab. misoprostol 800 μg per rectal insertion in case of increased bleeding • Transfer to a district hospital where facility for transfusion is available, with trained personnel	Specialist to attend • Establish an IV line • Do a per vaginal examination Patient in early labour • Refer to the nearest state medical hospital/tertiary health centre • Trained midwife/nurse to accompany who can conduct the delivery • Carry adequate oxytocics and methergin Patient in advanced labour • Specialist to be present • Doctor to be present • Prop up the patient • Give oxygen inhalation • Avoid fluid overload • Keep injections of methergin, oxytocics and misoprostol tablets ready • Preferable: vacuum or outlet forceps delivery by a doctor or trained midwife • Active management of the third stage of labour • Inj. methergin or Tab. misoprostol 800 μg per rectal insertion in case of increased bleeding • Transfer to a district hospital where facility for transfusion is available, with trained personnel	Specialist to attend • Establish an IV line • Do a per vaginal examination Patient in early labour • Refer to the nearest state medical hospital/tertiary health centre • Trained midwife/nurse to accompany who can conduct the delivery • Carry adequate oxytocics and methergin Patient in advanced labour • Specialist to be present • Doctor to be present • Prop up the patient • Give oxygen inhalation • Avoid fluid overload • Keep injections of methergin, oxytocics and misoprostol tablets ready • Preferable: vacuum or outlet forceps delivery by a doctor or trained midwife • Active management of the third stage of labour • Inj. methergin or Tab. misoprostol 800 μg per rectal insertion in case of increased bleeding • Transfer to a district hospital where facility for transfusion is available, with trained personnel • Give packed cell/blood transfusion under diuretic cover

PHC: primary health centre; CHC: community health centre; Hb: haemoglobin; TLC: total leucocyte count; DLC: differential leucocyte count; IV: intravenous; Tab.: tablet; IM: intramuscular; Inj: injection; AWW: *anganwadi* worker; MHW: multipurpose health worker

References

1. Aggarwal N, Kriplani A. Anaemia in pregnancy—a review. *Asian Journal of Obstetrics & Gynaecology Practise* 1999;**3:**10–18.
2. Broek NV. The aetiology of anaemia in pregnancy in West Africa. *Tropical Doctor* 1996;**26:**57.
3. Alsi Y. Diagnosis of anaemia. Conquering anaemia. In: Nanavati MS (ed). *FOGSI Focus.* Mumbai: FOGSI Office; 2002:8–9.
4. World Health Organization. *Antenatal care standard 4: Management of anaemia in pregnancy. Standards Document, Vol. 1. Standards of Midwifery Practice for Safe Motherhood.* New Delhi: Regional Publication No. 38, SEARO; 1999:41.
5. Nawani M. Treatment of anaemia. Conquering anaemia. *FOGSI Focus.* Mumbai: FOGSI Office; 2002:10–11.
6. Hematological disorders (Chapter 49). In: *William Obstetrics, Vol. 21.* International edition. McGraw-Hill Companies, Inc.; 2003:1309.

8. Premature rupture of membranes

Table 8.1 Causes of preterm rupture of membranes (PROM)[1,2]

Direct causes	• Genital tract infection • Occult amniotic fluid infection • Multiple foetuses • Abruptio placentae • Polyhydramnios • Cervical incompetence
Indirect causes	Smoking, previous history of preterm delivery
Distant casues	

Table 8.2 Manifestations of and interventions for premature rupture of membranes

Manifestations	Medical interventions	Non-medical interventions
• Preterm delivery • Chorioamnionitis • Puerperal sepsis • Neonatal prematurity • Neonatal sepsis	• Corticosteroids to the mother to induce maturity of the foetal lung • Antibiotics to the mother • Care of the pre-mature newborn • Antibiotic therapy to the newborn	• Availability of anibiotics • Availability of a neonatologist • Transportation to a hospital where the above facilities are available • Finances for care of the neonate

Table 8.3 Management protocol for preterm rupture of membranes at various levels of health care

Management protocol	Village/subcentre	PHC	CHC	District hospital
Personnel	Midwife/nurse/MHW	Doctor/midwife	Specialist	Specialist
History	• Watery discharge per vagina, duration of discharge, h/o fever, h/o dirty discharge • Refer to a PHC	• Watery discharge per vagina, duration of discharge, h/o fever, h/o dirty discharge	• Watery discharge per vagina, duration of discharge, h/o fever, h/o dirty discharge	• Watery discharge per vagina, duration of discharge, h/o fever, h/o dirty discharge
Physical examination	• Record the pulse, temperature and BP • Examine the respiratory and cardiovascular systems	• Record the pulse, temperature and BP • Examine the respiratory and cardiovascular systems	• Record the pulse, temperature and BP • Examine the respiratory and cardiovascular systems	• Record the pulse, temperature and BP • Examine the respiratory and cardiovascular systems
P/A examination	• Fundal height • Presentation, contractions • Foetal heart sounds	• Fundal height • Presentation, contractions • Foetal heart sounds	• Fundal height • Presentation, contractions • Foetal heart sounds	• Fundal height • Presentation, contractions • Foetal heart sounds
P/S examination	• Confirm leakage by the presence of liquor • Smell of the discharge • Do not do a vaginal examination	• Confirm leakage by the presence of liquor • Smell the discharge • Do not do a vaginal examination	• Confirm leakage by the presence of liquor • Smell the discharge Do not do a vaginal examination	• Confirm leakage by the presence of liquor • Smell the discharge Do not do a vaginal examination
Laboratory investigations		• TLC and DLC	• TLC and DLC • Fern test	• TLC and DLC • Fern test • Ultrasound examination
Between 24 and 28 weeks	Refer to a district hospital or the nearest CHC having a gynaecologist	Refer to a district hospital or the nearest CHC having a gynaecologist	<u>If willing</u> • Terminate the pregnancy <u>If not willing</u> • Refer to a higher centre • Give betamethasone 12 mg 2 doses 24 hours apart	<u>If willing</u> • Terminate the pregnancy <u>If not willing</u> • Give betamethasone 12 mg 2 doses 24 hours apart • Do a USG to look for the amount of liquor • Terminate the pregnancy —If the mother has fever, tachycardia, foul-smelling liquor

(Cont.)

Table 8.3 (*cont.*) Management protocol for preterm rupture of membranes at various levels of health care

Management protocol	Village/subcentre	PHC	CHC	District hospital
				—If the TLC is raised, suggestive of infection —If on USG there is minimal or absent ammiotic fluid, add aminoglycoside 1.2 mg/kg 8-hourly • Refer to a higher centre if termination is not planned
Between 28 and 34 weeks			• Start antibiotics if the leakage is for <18 hours —Give Tab. erythromycin 250 mg 6-hourly • If the leakage is for >18 hours —Give erythromycin + Tab. metronidazole 400 mg 8-hourly —Give corticosteroids Inj. betamethasone 12 mg 2 doses 24 hours apart <u>If in labour</u> • Refer to a higher centre <u>If not in labour</u> • Start antibiotics • Refer to a higher centre	• Start antibiotics if the leakage is for <18 hours —Give Tab. erythromycin 250 mg 6-hourly • If the leakage is for >18 hours —Give erythromycin + Tab. metronidazole 400 mg 8-hourly —Give corticosteroids Inj. betamethasone 12 mg 2 doses 24 hours apart • <u>If in labour</u> —Give parenteral antibiotics: ampicillin alone if the leakage is not prolonged, and ampicillin + metronidazole and/or gentamicin 1.5 mg/kg 8-hourly • If prolonged leakage or chorioamnionitis is present, a neonatologist must be present at the time of delivery • <u>If not in labour</u> —Keep the patient admitted and ensure bed rest —Keep a record of the temperature and pulse —Start antibiotics and corticosteroids • Do a USG to see the amount of amniotic fluid —if the liquor is adequate, monitor every alternate day for the volume of liquor —Watch for signs of infection • Refer to a higher centre if neonatal facilities are not available and —if the patient needs termination of pregnancy —if the patient is in labour • Termination of pregnancy is needed —If the amount of liquor has reduced —If the mother has fever, tachycardia, uterine tenderness

(Cont.)

Table 8.3 (*cont.*) Management protocol for preterm rupture of membranes at various levels of health care

Management protocol	Village/subcentre	PHC	CHC	District hospital
				—If previous P/V examinations have been done —If the discharge is foul-smelling • Start antibiotics
Between 34 and 37 weeks	Refer to the district hospital	Start antibiotics and refer to the district hospital	Start antibiotics and refer to a higher centre	• If leakage is confirmed —Terminate the pregnancy —A paediatrician should attend the delivery —Continue antibiotics post-partum • If leakage is doubtful/not confirmed on P/S examination, admit the patient. If there is no leakage seen on the pad, a USG shows adequate liquor —Advise bed rest —Advise monitoring of foetal movements daily —Keep a record of the temperature and pulse —Watch for leakage or discharge on the pad —Repeat a USG after 48 hours —Ask the patient to contact if leakage restarts or if foetal movements are decreased • If leakage is confirmed or the liquor has reduced then terminate the pregnancy, otherwise discharge —Follow up the patient
Beyond 37 weeks, i.e. term rupture of membranes	Refer to the nearest health facility having a doctor	• Start antibiotics • If the patient is not in labour, induce labour	• Start antibiotics • If the patient is not in labour, induce labour	• Start antibiotics • Induce labour • Ensure that a paediatrician attends the delivery

PHC: primary health centre; CHC: community health centre; MHW: multipurpose health worker; BP: blood pressure; P/A: per abdomen; P/S: per speculum; TLC: total leucocyte count; DLC: differential leucocyte count; USG: ultrasonography; Tab.: tablet; Inj.: injection; Cap.: capsule

Table 8.4 Requirement of personnel, investigations and drugs at different levels of health care[1,3]

	Personnel	Tests	Drugs/equipment/supplies	Inpatient stay
Subcentre	• MHW • Nurse • Midwife		• Cap. ampicillin 500 mg 6-hourly • Cap. erythromycin 250 mg 6-hourly • Tab. metronidazole 400 mg 6-hourly	2 days
PHC	• Doctor • Midwife • Driver • Pharmacist		• Speculum for vaginal examination • Gloves • Thermometer	
CHC	• Obstetrician • Laboratory technician • Pharmacist • Nurse • Driver	TLC and DLC	• Cap. and Inj. ampicillin 500 mg 6-hourly • Inj. and Tab. metronidazole 400–500 mg 8-hourly • Inj. gentamicin 80 mg • Tab. erythromycin 250 mg 6-hourly • IV set, IV cannula	2–3 days to 2–3 weeks
District hospital	• Obstetrician • Pathologist • Radiologist	• TLC and DLC • USG	• Cap. and Inj. ampicillin 500 mg 6-hourly • Inj. and Tab. metronidazole 400–500 mg 8-hourly • Inj. gentamicin 80 mg • Tab. erythromycin 250 mg 6-hourly • IV set • IV cannula	

PHC: primary health centre; CHC: community health centre; Cap.: capsule; Tab.: tablet; Inj.: injection; TLC: total leucocyte count; DLC: differential leucocyte count; USG: ultrasonography; MHW: multipurpose health worker

References

1. Preterm birth (Chapter 27). In: *Williams Obstetrics, Vol 21*. International edition. Singapore: McGraw-Hill Companies, Inc.; 2003:699.
2. Chua S, Arul Kumaran S. Prelabour rupture of membranes. In: *The management of labor.* Chennai: Orient Longman; 1996:228.

9. Complications of the third stage of labour

Table 9.1 Causes of complications of the third stage of labour[1,2]

Condition	Direct causes	Indirect causes	Distant causes
Uterine inversion	• Strong traction to the umbilical cord attached at the fundus • Adherent placenta	Delivery by unskilled personnel	• Home delivery • Delay in transportation • Specialist available at a distance (all the above add to mortality)
Retained placenta	• Tearing of the cord • Placenta accreta • Chorioamnionitis	Delivery by unskilled personnel	• Home delivery • Delay in transportation • Lack of blood bank facilities • Specialist available at a distance (all the above add to mortality)

Table 9.2 Manifestations and interventions for complications of the third stage of labour[1,2]

Manifestation	Medical interventions	Non-medical interventions
Uterine inversion	• Reposition immediately followed by manual removal of the placenta • Reposition under anaesthesia • Do abdominal correction with laparotomy • Delivery by trained personnel	Arrange for transportation
Retained placenta	• Immediate manual removal of the placenta • Remove the placenta under anaesthesia • Perform laparotomy/hysterectomy	• Delivery by trained personnel • Arrange for transportation • Provide blood bank facilities

Table 9.3 Personnel, investigations and drugs required to manage complications of the third stage of labour[1,2]

Complications of the third stage	Personnel	Tests	Drugs	Inpatient stay
Uterine inversion	• Skilled birth attendants • Trained midwives • Nurse • Doctor • Specialist • Anaesthetist	Haemoglobin estimation	• Inj. ampicillin 500 mg 6-hourly • Inj. metronidazole 400 mg 8-hourly • Inj. gentamicin 1.5 mg/kg body weight 8-hourly • Inj. oxytocin • Inj. methergin • Tab. misoprostol 200 µg • IV fluids	2–3 days
Retained placenta	• Skilled birth attendants • Trained midwives • Nurse • Doctor • Specialist • Anaesthetist	• Haemoglobin estimation • Total and differential leucocyte count	• Inj. ampicillin 500 mg 6-hourly • Inj. metronidazole 400 mg 8-hourly • Inj. gentamicin 1.5 mg/kg body weight 8-hourly • Inj. oxytocin • Inj. methergin • Tab. misoprostol • IV fluids	2–3 days

Table 9.4 Management protocol for uterine inversion at various levels of health care[1–3]

Village/subcentre	PHC	CHC	District hospital
• All deliveries to be conducted by midwives/ nurses/skilled attendants • Refer to a PHC or nearest health facility having a doctor	• Doctor to be present • If uterine inversion occurs then immediate repositioning should be attempted • If repositioning is successful, manual removal of the placenta with support to the fundus should be done • Secure an IV line • Give ampicillin 500 mg 6-hourly + Inj. metronidazole 500 mg 8-hourly + Inj. gentamicin 1.5 mg/kg body weight • Give oxytocics after correction • If the initial attempt at repositioning fails, quickly transfer patient to a higher centre • If an initial attempt has been already made or there has been a delay since delivery —No further attempt should be made and the patient referred immediately after resuscitation	• Specialist to attend • If uterine inversion occurs then immediate repositioning should be attempted • If repositioning is successful, manual removal of the placenta with support to the fundus should be done • Secure an IV line • Give ampicillin 500 mg 6-hourly + Inj. metronidazole 500 mg 8-hourly + Inj. gentamicin 1.5 mg/kg body weight • Give oxytocics after correction • If the initial attempt at repositioning fails, quickly transfer patient to a higher centre • If an initial attempt has been already made or there has been a delay since delivery —No further attempt should be made and the patient referred immediately after resuscitation	• Specialist to attend • If uterine inversion occurs then immediate repositioning should be attempted • If repositioning is successful, manual removal of the placenta with support to the fundus should be done • Secure an IV line • Give ampicillin 500 mg 6-hourly + Inj. metronidazole 500 mg 8-hourly + Inj. gentamicin 1.5 mg/kg body weight • Give oxytocics after correction • If the initial attempt at repositioning fails, the patient needs correction under anaesthesia • Blood to be cross-matched and transfused as required • IV fluids should be given rapidly • Broad-spectrum antibiotic cover should be given • Under anaesthesia —Reposition the uterus —Then do manual removal of the placenta —Give oxytocics

Inj.: injection; Tab.: tablet; Cap.: capsule; IV: intravenous; PHC: primary health centre; CHC: community health centre

Table 9.5 Management of retained placenta

Subcentre	PHC	CHC	District level
• Delivery by midwife/nurse/ skilled birth attendant • Refer to a PHC or nearest health facility having a doctor • Transfer the patient to a PHC if the attempt fails • Give oral antibiotics	• Doctor/nurse to attend • If the placenta is retained, using asepsis and high gloves immediately remove the placenta manually • Secure an IV line • After removal, check that the placenta is complete • Give antibiotics —Inj. ampicillin 500 mg 6-hourly —Inj. gentamicin 1.5 mg/kg body weight 8-hourly —Tab. metronidazole 400 mg 8-hourly • Give oxytocics • If removal is not possible —Secure an IV line —Give oxytocin if there is bleeding —If there is no bleeding then give IV fluids rapidly —Give antibiotics ampicillin 500 mg 6-hourly + Inj. metronidazole 400 mg 8-hourly + Inj. gentamicin 1.5 mg/kg body weight —Transfer without delay with nurse/ midwife • If the patient needs blood transfusion after manual removal, transfer her to a district hospital	• Specialist to attend • If the placenta is retained, using asepsis and high gloves immediately remove the placenta manually • Secure an IV line • After removal, check that the placenta is complete • Give antibiotics —Inj. ampicillin 500 mg 6-hourly —Inj. gentamicin 1.5 mg/kg body weight 8-hourly —Tab. metronidazole 400 mg 8-hourly • Give oxytocics • If removal is not possible —Secure an IV line —Give oxytocin if there is bleeding —If there is no bleeding then give IV fluids rapidly —Give antibiotics ampicillin 500 mg 6-hourly + Inj. metronidazole 400 mg 8-hourly + Inj. gentamicin 1.5 mg/kg body weight —Transfer without delay with nurse/ midwife • If the patient needs blood transfusion after manual removal, transfer her to a district hospital • If the patient has a history of previous caesarean section, transfer her to a medical college	• Specialist to attend • If the placenta is retained, using asepsis and high gloves immediately remove the placenta manually • Secure an IV line • After removal, check that the placenta is complete • Give antibiotics —Inj. ampicillin 500 mg 6-hourly —Inj. gentamicin 1.5 mg/kg body weight 8-hourly —Tab. metronidazole 400 mg 8-hourly • Give oxytocics • If removal is not possible —Patient requires manual removal under anaesthesia —Cross-match blood —Catheterize the bladder —Start IV antibiotics ampicillin/ gentamicin/metronidazole • To perform manual removal of the placenta under anaesthesia —Give oxytocin, Inj. metronidazole —Continue uterine massage —Replace blood loss • Refer —If there is a history of previous caesarean section —If placenta accreta is suspected —If an attempt at manual removal has failed • A nurse should accompany the patient

Inj.: injection; Tab.: tablet; Cap.: capsule; IV: intravenous; PHC: primary health centre; CHC: community health centre

References

1. Singh K, Arulkumaran S. The third stage of labour. In: *The management of labour.* Chennai: Orient Longman; 1996:170.
2. Obstetrical hemorrhage (Chapter 25). In: *Williams Obstetrics, Vol. 21*. International edition. McGraw-Hill Companies, Inc.; 2003:640.
3. Inverted uterus, retained placenta. In: *Integrated Management of Pregnancy & Child birth*. WHO/SEARO/ Dec 2002:S-27.

10. Menstrual disorders

Table 10.1 Causes of abnormal menstrual cycle[1,2]

Direct causes
• Anovulation
• Fibroid uterus
• Hormonal disturbances
• Malignancy
Indirect causes
• Chronic diseases
• Leukaemias
• Thrombocytopenias
• Bleeding disorders
• Underlying anaemias

Table 10.2 Manifestations and treatment for abnormal uterine bleeding[1,2]

Manifestations	Treatment
Amenorrhoea (primary or secondary)	• Investigation • Refer to a higher centre
Dysfunctional uterine bleeding	• Investigation • Endometrial evaluation as per age group/history • Hormonal/surgical management • Refer if required
Fibroid uterus	• Investigation • Hormonal/surgical management • Refer to a higher centre
Postmenopausal bleeding	• Refer to a higher centre

Table 10.3 Management of menstrual disorders at various levels of care

	Village/subcentre	PHC	CHC	District hospital
Primary amenorrhoea[3]	Any girl beyond the age of 16 years who has not attained menarche OR Any girl who has not attained menarche and has cyclical abdominal pain • Refer to a PHC	Any girl beyond the age of 16 years who has not attained menarche OR Any girl who has not attained menarche and has cyclical abdominal pain • Doctor to examine and refer to a district hospital	Any girl beyond the age of 16 years who has not attained menarche OR Any girl who has not attained menarche and has cyclical abdominal pain • Specialist to examine the girl and refer to the district hospital if the diagnosis is cryptomenorrhoea, manage or refer to a medical college	Any girl beyond the age of 16 years who has not attained menarche OR Any girl who has not attained menarche and has cyclical abdominal pain • Specialist to examine the girl —If the diagnosis is imperforate hymen, surgical management is needed —Refer other cases to a medical college/tertiary centre
Secondary amenorrhoea	Refer to a PHC	• Doctor to review history • Examination —P/A —P/S —P/V • Refer to a medical college	• Specialist to review history • Examination —P/A —P/S —P/V • Refer to medical college if required	• Specialist to review history • Examination —P/A —P/S —P/V —USG of pelvic organs • Investigation —Hormonal profile (refer) —Give progesterone withdrawal/treat as per clinical presentation
Dysfunctional uterine bleeding	Woman with abnormal or excessive bleeding • Refer to a PHC	• Doctor to attend • History: Age, amount and duration of bleeding, whether there is passage of clots, whether bleeding is preceded by amenorrhoea, length of the menstrual cycle, any post-coital bleeding,	• Specialist to treat • History: Age, amount and duration of bleeding, whether there is passage of clots, whether bleeding is preceded by amenorrhoea, length of the menstrual cycle, any post-coital bleeding,	• Specialist to treat • History: Age, amount and duration of bleeding, whether there is passage of clots, whether bleeding is preceded by amenorrhoea, length of the menstrual cycle, any post-coital bleeding,

(Cont.)

Table 10.3 (*cont.*) Management of menstrual disorders at various levels of care

	Village/subcentre	PHC	CHC	District hospital
		underlying medical disorder • Examination —General —P/A —P/S: condition of the cervix —P/V: size of the uterus, presence of any adnexal mass • Management —Refer to a district hospital	underlying medical disorder • Examination —General —P/A —P/S: condition of the cervix —P/V: size of the uterus, presence of any adnexal mass • Management —Refer to a district hospital —Depends on the age group • Pubertal group —Investigations: Haemogram, coagulogram, RFT —Refer to a district hospital • 30–40 years age group —Investigations: same as above to rule out secondary causes —If required, conduct endometrial evaluation with histopathology —Refer to a district/tertiary hospital • 40 years and above —Refer to a district/tertiary hospital	underlying medical disorder • Examination —General —P/A —P/S: condition of the cervix —P/V: size of the uterus, presence of any adnexal mass • Management —Depends on the age group • Pubertal group —USG of pelvic organs —Investigations: Haemogram, coagulogram, RFT, hormonal profile —Ascertain the cause and treat accordingly —Oral contraceptive pills can be a treatment option in case of anovulation • 30–40 years age group —USG of pelvic organs —Investigations: same as above to rule out secondary causes —If required, conduct endometrial evaluation with histopathology —Treatment: Oral contraceptive pills/hormonal treatment as per clinical evaluation • 40 years and above —USG of pelvic organs —Investigation: Endometrial evaluation with histopathology —Hormonal or surgical management as per clinical evaluation
Fibroid uterus	Woman with abnormal or excessive bleeding • Refer to a PHC	If examination is suggestive of fibroid uterus, refer to a district hospital	If examination is suggestive of fibroid uterus, refer to a district hospital	• If examination is suggestive of fibroid uterus, confirm by USG • Management —Myomectomy to be done if fibroid is the cause of infertility or is symptomatic in a young patient —Depends on the surgical skill of the gynaecologist, size of the fibroid and availability of blood; refer otherwise —In case of sympotomatic fibroid in perimenopausal women, either do

(Cont.)

Table 10.3 (*cont.*) Management of menstrual disorders at various levels of care

	Village/subcentre	PHC	CHC	District hospital
				hysterectomy as per clinical presentation OR Refer to a higher centre
Post-menopausal bleeding/malignancies (endometrial/cervical)				Refer to a higher centre

PHC: primary health centre; CHC: community health centre; P/A: per abdomen; P/S: per speculum; P/V: per vaginam; RFT: renal function tests; USG: ultrasonography

References

1. Davey DA. Dysfunctional uterine bleeding. In: *Dewhertz textbook of gynaecology*. Butterworth-Heinemann Ltd.; p. 624.
2. Nelson L, Rybo G. Treatment of menorrhagia. *Am J Obstet Gynecol* 1971;**110:**713.
3. Sparoff L. Normal and abnormal sexual development. In: *Clinical endocrinology*. Part II. *Clinical gynaecologic endocrinology and infertility*. USA: Lippincott Williams & Wilkins; 2005:319.

11. Vaginal discharge

Table 11.1 Causes of vaginal discharge[1–3]

	Direct causes	Indirect causes	Distant causes
Main causes	• Cervicitis —Gonorrhoea —Chlamydial infection • Vaginitis —Bacterial vaginosis —Trichomoniasis —Candidiasis • Physiological • Malignancy	• Multiple sexual partners • Untreated male partner • Incomplete treatment	
Associated factors	Sexually transmitted diseases		

Table 11.2 Personnel, drugs and equipment required for the management of vaginal discharge

Level of health care	Personnel	Drugs and equipment	Inpatient stay
Subcentre	• Health workers		
Primary health centre (PHC)	• Doctor • Pharmacist	• Gloves • Sim or Cusco speculum • Tab. clotrimazole 150 mg • Tab. metronidazole 400 mg • Tab. ciprofloxacin 500 mg • Cap. doxycycline 100 mg • Tab. azithromycin	
Community health centre (CHC)	• Specialist • Pharmacist • Laboratory technician	Same as PHC	
District hospital	• Specialist • Pathologist	• IV antibiotics • IV sets • IV cannula • Disposable syringes	2–3 days

Tab.: tablet; Cap.: capsule; IV: intravenous

Table 11.3 Treatment of various causes of vaginal discharge[1,2]

Causes	Treatment	Advice
Candidiasis	Tab. clotrimazole 200 mg vaginally for 3 days OR Tab. miconazole same dose OR Tab. fluconazole 150 mg single dose	Treatment of sexual partner
Trichomoniasis	Tab. metronidazole 400 mg BD × 7 days	Treatment of sexual partner
Bacterial vaginosis	Tab. metronidazole 400 mg BD × 7 days	Treatment of sexual partner
Gonorrhoea and chlamydial infection	Tab. ciprofloxacin 500 mg single dose and Cap. doxycycline 100 mg BD × 7 days • Pregnant woman Azithromycin 2 g single dose OR Inj. ceftriaxone 250 mg IM single dose OR Tab. erythromycin stearate 500 mg 4 times for 7 days	• Treatment of sexual partner • Education and counselling • Condoms for protection and prevention of STD
Suspected cervical malignancy	Refer to a tertiary centre	Refer to a tertiary centre

Tab.: tablet; Cap.: capsule; IM: intramuscular; STD: sexually transmitted disease

Table 11.4 Management of vaginal discharge at various levels of health care

Village/subcentre	PHC	CHC	District hospital
Woman complains of vaginal discharge • Refer to a PHC	Woman complains of vaginal discharge • History: Type and colour of discharge, smell, whether associated with itching, whether partner is symptomatic, risk assessment for STD • Per speculum examination 1. Profuse discharge: Treatment for trichomoniasis and bacterial vaginosis 2. Clumped discharge: Treatment for candidiasis 3. Mucopus from the cervix: Treatment for gonorrhoea and chlamydial infections • When speculum is not available —Give syndromic treatment • When risk assessment is positive —Treatment for gonorrhoea and chlamydial infections irrespective of findings • Advice —Treatment for sexual partner —Education and counselling —Condoms for protection and prevention of STD • Refer in case of —Blood-stained vaginal discharge —Suspicious cervix —Malignancy —Recurrent infections —Assosciated genital ulcer or lymphadenopathy	Woman complains of vaginal discharge • History: Type and colour of discharge, smell, whether associated with itching, whether partner is symptomatic, risk assessment for STD • Per speculum examination 1. Profuse discharge: Treatment for trichomoniasis and bacterial vaginosis 2. Clumped discharge: Treatment for candidiasis 3. Mucopus from the cervix: Treatment for gonorrhoea and chlamydial infections • When speculum is not available —Give syndromic treatment • When risk assessment is positive —Treatment for gonorrhoea and chlamydial infections irrespective of findings • Advice —Treatment of sexual partner —Education and counselling —Condoms for protection and prevention of STD • Refer in case of —Blood-stained vaginal discharge —Suspicious cervix —Malignancy —Recurrent infections —Assosciated genital ulcer or lymphadenopathy	Woman complains of vaginal discharge • History: Type and colour of discharge, smell, whether associated with itching, whether partner is symptomatic, risk assessment for STD • Per speculum examination 1. Profuse discharge: Treatment for trichomoniasis and bacterial vaginosis 2. Clumped discharge: Treatment for candidiasis 3. Mucopus from the cervix: Treatment for gonorrhoea and chlamydial infections • When speculum is not available —Give syndromic treatment • When risk assessment is positive —Treatment for gonorrhoea and chlamydial infections irrespective of findings • Advice —Treatment of sexual partner —Education and counselling —Condoms for protection and prevention of STD • Refer in case of —Blood-stained vaginal discharge —Suspicious cervix —Malignancy —Recurrent infections —Assosciated genital ulcer or lymphadenopathy

PHC: primary health centre; CHC: community health centre; STD: sexually transmitted disease

References

1. Diseases characterised by vaginal discharge. *MMWR*. 2002; Vol. 51, No. RR-6; 42.
2. *National AIDS Control Programme*. Simplified STI and RTI Treatment Guidelines. Flowcharts distributed under NACO programme. New Delhi: NACO, Ministry of Health & Family Welfare, Government of India; p. 5.
3. World Health Organization. Vaginal discharge. *Management of sexually transmitted diseases at district and PHC levels*. WHO Regional Publication, SEARO, No. 25; 1998:14.

12. Pelvic inflammatory disease

Table 12.1 Causes of pelvic inflammatory disease[1,2]

Direct causes	Indirect causes	Distant causes
• Gonococcal infection • Chlamydial infection • Anaerobic infection • Tuberculosis	• Multiple sexual partners • Recent abortion or D&C • Following unclean per vaginal examination or delivery	• Untreated male partner • Incomplete treatment

D&C: dilatation and curettage

Table 12.2 Management of pelvic inflammatory disease at various levels of care[3]

Subcentre	PHC	CHC	District hospital
Any woman complaining of lower abdominal pain • Refer to a PHC	Any woman complaining of lower abdominal pain • A doctor to examine • History —H/o of any menstrual disturbance, abortion/delivery —Note the duration of symptoms • Examination —General —P/A —P/S —P/V • Treatment —Tab. ciprofloxacin 500 mg single dose + Cap. doxycycline 100 mg BD for 14 days + Tab. metronidazole 400 mg BD for 7 days —Same treatment to the male partner • Advise —to complete the treatment —return if symptoms worsen • Counsel and educate about STDs and condoms • Refer to a district hospital if: —the temperature is more than 38 ^{0}C —the symptoms are acute —the patient is sick	Any woman complaining of lower abdominal pain • A specialist to attend • History —H/o of any menstrual disturbance, abortion/delivery —Note the duration of symptoms • Examination —General —P/A —P/S —P/V • Treatment —Tab. ciprofloxacin 500 mg single dose + Cap. doxycycline 100 mg BD for 14 days + Tab. metronidazole 400 mg BD for 7 days —Same treatment to the male partner • Advise —to complete the treatment —return if symptoms worsen • Counsel and educate about STDs and condoms • Refer to a district hospital if: —the temperature is more than 38 ^{0}C —the symptoms are acute —the patient is sick	Any woman complaining of lower abdominal pain • A specialist to attend • History —H/o of any menstrual disturbance, abortion/delivery —Note the duration of symptoms • Examination —General —P/A —P/S —P/V • Treatment —Tab. ciprofloxacin 500 mg single dose + Cap. doxycycline 100 mg BD for 14 days + Tab. metronidazole 400 mg BD for 7 days —Same treatment to the male partner • Advise —to complete the treatment —return if symptoms worsen • Counsel and educate about STDs and condoms • If: —the temperature is more than 38 ^{0}C —the symptoms are acute —the patient is sick Treatment —Admit the patient

(Cont.)

Table 12.2 (*cont.*) Management of pelvic inflammatory disease at various levels of care

Subcentre	PHC	CHC	District hospital
			—Conduct an ultrasonography to rule out pelvic abscess or pus collection, or presence of a foreign body —Give IV antibiotics: Inj. cefotaxime 1 g 8-hourly + Inj. gentamicin 1.2 mg/kg body weight 8-hourly + Inj. metronidazole 500 mg 8-hourly —Change antibiotics as per culture reports —Refer to a higher centre if there is no improvement in 48 hours

PHC: primary health centre; CHC: community health centre; h/o: history of; Tab.: tablet; Cap.: capsule; STD: sexually transmitted disease; IV: intravenous; Inj.: injection; P/A: per abdomen; P/S: per speculum; P/V: per vaginam

References

1. WHO. Lower abdominal pain. In: *Management of sexually transmitted diseases at district and PHC levels*. New Delhi: WHO/SEARO Regional Publication, No. 25; 1998:22.
2. National AIDS Control Programme. *Treatment guidelines*. Flowcharts distributed by the Ministry of Health and Family Welfare; 1999.
3. Pelvic inflammatory disease. *MMWR sexually transmitted diseases treatment guidelines* 2002, Vol. 51/No.RR-6. p. 45.

13. Pelvic or abdominopelvic mass

Table 13.1 Differential diagnosis of pelvic or abdominopelvic mass

- Pregnancy
- Benign ovarian cysts
- Endometriosis
- Fibroids
- Chronic ectopic pregnancy
- Malignant ovarian tumours
- Metastatic tumours

Table 13.2 Management of pelvic or abdominopelvic mass at various levels of care

Subcentre	PHC	CHC	District hospital
Any woman having abdominal distension or feeling of swelling or mass in the abdomen • An MHW, nurse, skilled birth attendant or dai should refer the woman to a PHC	Any woman having abdominal distension or feeling of swelling or mass in the abdomen • A doctor to attend • Review history of the duration of symptoms of menstrual disturbances, infertility, pain (whether dull or acute), h/o weight loss, h/o abortion • Examination —General —Abdominal: Presence of any free fluid, size of mass, mobility, consistency, position, tenderness	Any woman having abdominal distension or feeling of swelling or mass in the abdomen • A specialist to attend • Review history of the duration of symptoms of menstrual disturbances, infertility, pain (whether dull or acute), h/o weight loss, h/o abortion • Examination —General —Abdominal: Presence of any free fluid, size of mass, mobility, consistency, position, tenderness	Any woman having abdominal distension or feeling of swelling or mass in the abdomen • A specialist to attend • Review history of the duration of symptoms of menstrual disturbances, infertility, pain (whether dull or acute), h/o weight loss, h/o abortion • Examination —General —Abdominal: Presence of any free fluid, size of mass, mobility,

(Cont.)

Table 13.2 (*cont.*) Management of pelvic or abdominopelvic mass at various levels of care

Subcentre	PHC	CHC	District hospital
	—P/S: condition of the cervix —P/V: site of mass, relation to the uterus, fixity, unilateral/bilateral, presence of any free fluid, tenderness • Management Refer to a tertiary centre	—P/S: Condition of the cervix —P/V: Site of mass, relation to the uterus, fixity, unilateral/bilateral, presence of any free fluid, tenderness • Management —Investigation: Haemogram, RFT, chest X-ray —Refer to a tertiary centre	consistency, position, tenderness —P/S: Condition of the cervix —P/V: Site of mass, relation to the uterus, fixity, unilateral/bilateral, presence of any free fluid, tenderness • Management —Investigations: Haemogram, RFT, chest X-ray —Ultrasonography of pelvic organs —If the patient has acute symptoms, rule out ectopic pregnancy or torsion which requires emergency management —Manage or refer to a tertiary centre with referral details —Refer other cases of pelvic masses, i.e. suspected malignancies

PHC: primary health centre; CHC: community health centre; MHW: multipurpose health worker; h/o history of; P/S: per speculum; P/V: per vaginam; RFT: renal function tests

Components of under-five mortality trends, current stagnation and future forecasting levels

ARVIND PANDEY*, B.N. BHATTACHARYA†, D. SAHU*, REHENA SULTANA*

The infant mortality rate (IMR)—probability of dying before one year of age expressed per 1000 live-births—and under-five mortality rate (U5MR)—probability of dying between birth and age 5 expressed per 1000 live-births—have been used as measures of children's well-being for many years. The International Conference on Primary Health Care held in Alma Ata in 1978 was the first global forum to consider how child mortality could be reduced by systematic development of a primary health care system. Since then, the United Nations has been actively involved in reducing IMR and U5MR in developing countries. To this end, the plan of action adopted at the International Conference on Population and Development (ICPD) held in Cairo in 1994 incorporates the reduction of maternal and child mortality.

In India, during 1968–70, the level of IMR was stable at 130 deaths per 1000 live-births. Following the Alma Ata declaration of 1978, the Government of India envisaged a national goal for the attainment of an IMR of 60 by the year 2000. Since then, substantial resources have been put into the child survival programmes over the past 25 years. The Sixth and Seventh Five-Year Plans had aimed at nationwide programmes to realize this goal. The twenty-point programme included, as a key component, rapid improvement in the conditions of women and children. In 1979, the Expanded Programme of Immunization (EPI) was established to provide tetanus toxoid (TT) vaccine to pregnant women, and BCG, DPT, polio and measles vaccine to children. The Universal Immunization Programme (UIP) and oral rehydration therapy (ORT) were both launched in 1985 and the Safe Motherhood Programme initiated during the Eighth Plan were prominent components of the Family Welfare Programme. In the early 1990s, these programmes were integrated and further strengthened to shape the Child Survival and Safe Motherhood (CSSM) Programme. In 1994, the CSSM Programme was further expanded to the Reproductive and Child Health (RCH) services. These programmes had the desired effect of reducing child mortality and improving child health as evidenced from the child mortality statistics of 1978–2002. The National Population Policy (2000) and National Health Policy (2002) addressed the issues of child survival and maternal health, and increased the outreach and coverage of the comprehensive package of RCH services through the government as well as the voluntary non–government sector together in partnership.

The U5MR, including infant, neonatal and child mortality rates, started declining since the late 1970s and until 1993 the rate of decline was substantial. The decline was, however, slow during 1993–98 (Fig. 1). The country's goal to achieve a U5MR of less than 100 per 1000 live-births and reducing the IMR to less than 60 per 1000 live-births by the year 2000 could not be achieved despite improved interventions and an increase in the overall resources. In the present scenario of IMR (2002), 25 per 1000 newborns died within the first week of birth; 40 per 1000 newborns died before reaching the age of 1 year and 85 per 1000 newborns died before reaching the age of 5 years. The major uncertainty seems to be whether the IMR is approaching a limiting value. This value does not have an ultimate cap that will hold forever. Perhaps the progress in reducing mortality in early infancy is possible with innovative interventions for newborn care. Nevertheless, the IMR and U5MR have become increasingly important indicators that need to be monitored.

The present investigation aimed to study the changes in each of the components of under-five mortality during the period 1978–2002; to analyse the factors associated with the apparent stagnation of the child mortality rate in India; and to develop projection scenarios of the IMR and U5MR

Fig. 1 Trends in the infant mortality rate for 1910–2002, India

*Institute for Research in Medical Statistics, ICMR, New Delhi

†Indian Statistical Institute, Kolkata
e-mail: arvindpandey@vsnl.net

by States by the year 2016. The study examines the impact of utilization of antenatal and natal services on neonatal mortality. It also looks into the levels of IMR and U5MR among socially and economically disadvantaged groups. Besides, the study tries to examine the reasons for the slowing down of the rate of decline in child mortality in recent years by analysing the prevalence of high-risk births among advantaged/disadvantaged groups and relating the same to differentials in the utilization of health care services.

Data

Primarily, two sources of data have been used for the analysis. The Sample Registration System (SRS) under the Registrar General of India provides the estimates of births and deaths at State/national level. The estimates of mortality indicators are used to study the levels and trends in child mortality prevailing during 1978–2002 and also to predict the IMR and U5MR. The study has also used data from two rounds of the National Family Health Surveys (NFHS) conducted during 1992–93 (NFHS-1) and 1998–1999 (NFHS-2). The data in NFHS-1 were collected from a probability sample of 89,777 ever-married women aged 13–49 years from 24 States including the National Capital Territory of Delhi. In NFHS-2, information was collected from a sample of 89,199 ever-married woman aged 15–49 years from all 26 States including Chhattisgarh, Jharkhand and Uttaranchal. Both the surveys included women who were the usual residents of the sample households or visitors who stayed in the sample households the night before the interview, and these women were referred to as eligible women.

Methods

Estimation of trends in components of child mortality

Trends in infant mortality were estimated by fitting a regression line to the relation between observations of IMR and time for each State and for India, allowing the rate of change of mortality to vary according to the number of independent observations available. The methodology is described in detail in Hill *et al.* 1997. The regression line shown below includes an underlying date variable, i.e. calendar year and additional variables measuring time since a series of knot dates (in the present analysis, 1982, 1987, 1992 and 1997). The definition of a knot is based on the idea that each five-year period of vital registration defines a particular slope. The rate of change of IMR can change at each knot. The equation is:

$$\ln({}_nq_0)_i = b_0 + b_1 * \text{date} + b_2 * \text{postk}_1 + b_3 * \text{postk}_2 + \ldots + e_i$$

where

$$\text{postk}_n = \begin{cases} \text{date} - k_n, \text{if date} > k_n \\ 0, \text{otherwise} \end{cases}$$

k_n = 1982, 1987, 1992 and 1997

*indicates multiplication

The rate of change is b_1 during 1977–82, $(b_1 + b_2)$ during 1982–87, $(b_1 + b_2 + b_3)$ during 1987–92, $(b_1 + b_2 + b_3 + b_4)$ during 1992–97and $(b_1 + b_2 + b_3 + b_4 + b_5)$ during 1997–2002.

The average annual rate of change in IMR is:

$$(1/n)\ ((\ln(({}_1q_{0\,[n]}) / ({}_1q_{0\,[n-1]})))$$

where [n] and [n–1] refer to the nth five-year period and the five-year period immediately preceeding it, respectively. A similar procedure is used to obtain the average annual rate of change in early neonatal, neonatal and U5MR.

Absolute changes in early neonatal, neonatal, IMR and U5MR are calculated as ${}_{7\,days}q_{0[n]} - {}_{7\,days}q_{0[n-1]}$, ${}_{28\,days}q_{0[n]} - {}_{28\,days}q_{0[n-1]}$, ${}_1q_{0[n]} - {}_1q_{0[n-1]}$ and ${}_5q_{0[n]} - {}_5q_{0[n-1]}$.

Early neonatal mortality rate (Early NMR) and U5MR are computed as follows:

$$\text{Early NMR} = \frac{\text{Perinatal mortality rate} - \text{stillbirth rate}}{1 - (\text{Stillbirth rate}/1000)}$$

According to the Reed–Merrel formula the U5MR =

$$1000 * {}_5q_0 = 1000 * (1 - \text{Exp}(-5 * {}_5m_0 - 125 * {}_5m_0^2))$$

where a = 0.008.

Because of the non-availability of mortality data for the period 1970–1980 for West Bengal and Bihar, the above indicators could not be calculated for the same period. Similarly, due to non-availability of SRS data for J&K since 1990, J&K is excluded from the analysis.

Estimation of components of child mortality using NFHS-2 data

In the NFHS-2, all eligible women were asked to provide information on complete birth history, which included sex, month and year of birth, and survival status for each live-birth. The information on age at death was recorded in days for children who had died in the first month of life; in months for children who had died after the first month but before completion of their second birthday, and in years for children who had died at later ages. However, for children who had died after their second birthday, the imputed values of age at death in months were provided. Detailed information on antenatal, delivery and postnatal care were also obtained only for the two most recent births which occurred to eligible women during the three years preceding the survey.

The present study excluded births that occurred during the month of survey from the analysis (time 0 refers to the last day of the month preceding the survey, referred to hereafter as *the reference date*). A child born on or before the reference date and who died during the month of interview was considered alive as on the reference date. The number of children exposed to the risk of death in the age segment (0, 60) months during the ten years preceding the reference date were those who were born during the ten years preceding the reference date or who were born during 10–14 years preceding the reference date and were alive at the beginning

of the 120th month, measured from the reference date.

The levels of neonatal, postneonatal and child mortality across categories of selected predictor variables are based on birth histories of eligible women and their records mentioned above. Children of eligible women who were ever exposed to the risk of death between age 0 and 60 months during the ten years preceding the reference date are considered. The study also examined the effects of health care received by mothers during pregnancy and at delivery on neonatal and postneonatal mortality using information relating to the two most recent births that occurred to eligible women during the thirty-five months preceding the survey date. For each age segment (0, 1), [1, 12) and [12, 60) months, a file of children who were ever exposed to the risk of death during the past ten years in the corresponding age segments was created from the birth histories. In each file, the record of each child included age at entry into the age interval, age at exit from the age interval, which was either due to death or censoring (an outcome associated with exit time wherein 0 means censored and 1 means death), and selected characteristics of the child and his/her mother, and the household.

For each category of predictor variables and for each age-segment (a_1, a_2) months, i.e. (0, 1), [1, 12) and [12, 60) months, the probability of surviving over the interval, given that the child survived at least first a_1 months adjusted for a specified number of covariates at their mean values, are obtained. The adjustment is made by estimating a stratified-on-group Cox-regression model. In stratified Cox regression, the hazard (in deviation form) at age 'a' for a child in the ith category of a factor, which has q categories, is given by:

$$h_i(a) = h^*_{0i}(a)\exp\{\beta_1(z_1 - z_1^*) + \beta_2(z_2 - z_2^*) + \ldots + \beta_k(z_k - z_k^*)\};\ i = 1, 2, \ldots, q$$

where, z_1, z_2, …, z_k are covariates for which the above relation is descriptive, are z_1^*, z_2^*, ..., z_k^*, respectively averages of z_1, z_2, …, z_k of children born at a particular moment.

$h_{0i}^*(a) = h_{0i}(a)\exp\{\beta_1 z_1^* + \beta_2 z_2^* + \ldots + \beta_k z_k^*\}$, and $h_{0i}(a)$ is the baseline hazard.

The coefficients $\beta_1, \beta_2, \ldots \beta_k$ are assumed to be the same regardless of category but the baseline hazard h_{0i} is allowed to be category-specific. $h^*_{0i}(t)$ is the baseline hazard for children belonging to ith category of the predictor variable for whom $z_j = z_j^*$ (j= 1, 2…, k).

For a child belonging the ith category of the factor, the probability of surviving over the age interval $[a_1, a_2)$ is:

$$S^*_i[(a_1, a_2)] = \exp\{-\int_{a_1}^{a_2} h^*_{0i}(t)\},\ i = 1, 2, \ldots, q$$

The averages of covariates for children born at a particular time are taken as averages of covariates for children born to eligible women during the 35 months preceding the reference date. The unadjusted conditional survival probabilities over different age intervals for each category of a predictor variable are obtained separately by using the Kaplan–Meier method. For each child at the country level, a weight was used for tabulation and hazard model (Cox regression) analysis.

Projection

A time series structure analysis is carried out by applying the autoregressive integrated moving averages (ARIMA) model to the IMR and U5MR to forecast beyond the series up to 2016. This technique allows for description of the degree of auto-explanation between observations based on the parameter p associated to auto-regression[1]; d specifies the number of times the series is to be differentiated in order to become stationary; and q indicates the number of moving average terms.

Time series IMR_t ($U5MR_t$), t=1977, 2002 which is non-stationary, is converted into a stationary series through a two-step transformation process. First, the series is transformed by taking its natural logarithm; the second step involves successive differencing of the transformed series. If we define the observed series IMR_t ($U5MR_t$) at time t as Y_t and define another transformed series $Z_t = \ln(Y_t)$, the series Z_t is an ARIMA (p,d,q) , and it may be expressed as:

$$\phi(B)(1 - B)^d Z_t = \delta + \theta(B)\,\varepsilon_t$$

where

$$\phi(B) = 1 - \phi_1 B - \phi_2 B^2 - \ldots \phi_p B^p$$

$$\theta(B) = 1 - \theta_1 B - \theta_2 B^2 - \ldots \theta_q B^q$$

and

$$BZ_{t+1} = Z_t$$

The estimated future values of Z_t are converted into those of Y_t to obtain the predicted values.

Results and discussion

Trends in child mortality

The level of IMR declined from about 220 deaths per 1000 live-births per year at the time of India's Independence to about 130 during the 1960s and remained stable at the same level for a decade. It started declining further after 1978 (Fig. 2). The momentum of decline continued until 1993 and the rate was halved during 1978–1993. The lowest decline was noticed in 1992–97. The IMR was found to be stagnating at around 72–74 per 1000 live-births during 1992–97 and this was followed by a slow pace of decline. Thus, India could not achieve the goal of reducing the IMR to less than 60 per 1000 live-births by the year 2000.

The State-wise average annual rates of change and absolute change in early neonatal, neonatal, infant and under-five mortality rates over the 25-year period 1977–

[1] The parameter provides information concerning the order of structural dependence existent between adjacent observations, indicating the existence of autocorrelation

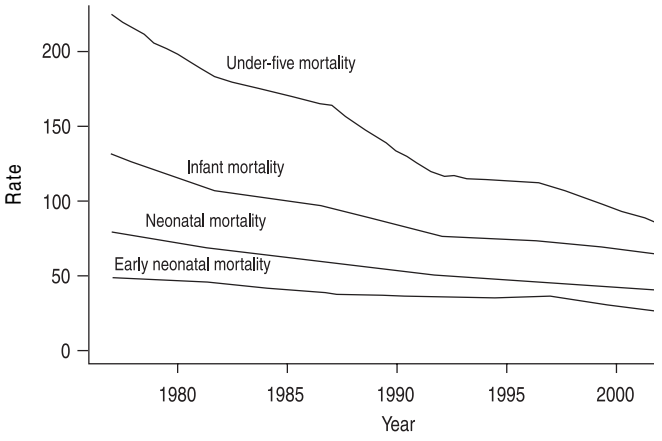

Fig. 2 Fitted trends in early neonatal, neonatal, infant and under-five mortality rates, India 1977–2002

Sources:
1. Under-five mortality rate: Sample Registration System, 1977–2002
2. Infant mortality rate: Sample Registration System, 1977–2002
3. Neonatal mortality rate: Sample Registration System, 1977–2002
4. Early neonatal mortality rate: Sample Registration System, 1977–2002

2002 are provided in Tables 1a–1d while the levels of still-birth rates, early neonatal mortality rates, neonatal mortality rates and post-neonatal mortality rates are given in Appendix 1, Table A1.1 and the levels of IMR, child mortality rates ($_4q_1$) and U5MR are provided in Appendix 1, Table A1.2. The levels, rates of change and absolute change in IMR and U5MR over the 25-year period from 1977 to 2002 are summarized as box plots for major States of India (Figs 3a–3c). It may be observed that the IMR was continuously declining in every five-year period between 1977 and 2002; it declined from 130 infant deaths per 1000 live-births in 1977 to 63 infant deaths per 1000 live births in 2002, with the sharpest decline of 4.3% during 1977–1982 and 4.5% during 1987–1992. The average annual rates of change and absolute change corroborate these findings. The absolute decline in IMR was, however, not uniform during 1977–2002. The highest decline was 25 infant deaths per 1000 in 1977–82 followed by 19 infant deaths per 1000 during 1987–92. The lowest decline of only 4 infant deaths per 1000 was observed during 1992–97. Within India, there are large differences in IMR among States; States have shown a decline in the levels of IMR over time but the pace of decline varies from one State to the other. Kerala had the lowest IMR in 1977, at 47 infant deaths per 1000 live-births, which fell to 10 infant deaths per 1000 live births in 2002, again the lowest among the States. UP had the highest level in 1977 (at about 177 per 1000 live-births), which fell to 79 per 1000 in 2002, again the second-highest among the States. The most dramatic decline in absolute values occurred in Gujarat, Tamil Nadu and Punjab—the level of IMR in these States was about 138, 103 and 105, respectively in 1977 and fell to 60, 44 and 51, respectively in 2002. The absolute decline in IMR was between 59 and 69 in Madhya Pradesh, Andhra Pradesh, Orissa, Punjab, Bihar, Gujarat and Tamil Nadu, around 50 in Haryana, Himachal Pradesh and Assam, around and below 40 in Maharashtra, J&K, West Bengal and Karnataka (Tables 1c–d).

Out of 16 major States, 9 experienced systematic decline in IMR during 1977–2002 (1982–2002 for West Bengal) (Table 1d). Of the 7 remaining States, the average annual rate of change during 1982–87 was positive (about 1%) in three States (AP, Assam and Rajasthan); during 1992–97 it was a little above zero (0.3%) in Assam, Bihar and Haryana, and about 1% in Rajasthan. If we ignore the small amount of positive value of the average annual rate of change in these States during the aforesaid period (which could be due to one or more reasons of underreporting of early infant deaths, sampling fluctuations, stagnation rates during these periods), we could say that 14 out of 16 States showed more or less declining trends in IMR over the past 25 years. Himachal Pradesh and Karnataka showed a declining trend since 1987, Karnataka experienced the highest decline in the average annual rate of change of 6% while the figure for India was 1.2% during the period 1992–97.

In summary, in the past 25 years, the most precipitous decline in IMR occurred during the period 1977–82 (a drop of 25 points) and during 1987–92 (a drop of 16 points). The smallest decline was seen during 1992–97 (8 points) (Appendix 1, Table A1.2). The highest decline in the absolute value of IMR occurred in Andhra Pradesh, Himachal Pradesh, Maharashtra, Punjab and Rajasthan during 1977–82; Assam, Bihar, Gujarat and Uttar Pradesh during 1987–92; Orissa during 1992–97; Haryana, Kerala, Tamil Nadu during 1977–82 and 1987–92; and Karnataka during 1977–82 and 1992–97.

In Madhya Pradesh, the decline in the absolute value of IMR and the average annual rate of change in IMR during the periods 1977–1982 to 1997–2002 was small. In Uttar Pradesh, a rapid decline in the absolute value as well as average annual rate of change of IMR was observed during the consecutive five-year period from 1977–82 to 1987–92 (Tables 1b and 1d). The average annual rate of change in U5MR and IMR were more or less the same during each five-year period except during 1987–92 and 1997–2002, which indicated a faster decline in child mortality compared to IMR during the three periods (Table 1b). Also, during 1982–87 and 1997–2002, the annual rate of decline in neonatal mortality rate (NMR) was faster than the decline in IMR (Tables 1a and 1b) and the average rate of decline in early neonatal mortality was faster than the neonatal mortality rate, indicating relatively greater improvement in early neonatal mortality as compared to late neonatal mortality, and in neonatal mortality than post-neonatal mortality during the said periods. All the States experienced a substantial decline in infant and child mortality during 1978–93. Orissa had the highest points and Kerala the lowest. Orissa, Rajasthan and Uttar Pradesh exhibited declines in child mortality of at least 100 points. The IMR in India

Table 1a. Average annual rates of change in mortality according to five-year periods by States and India

	Percentage decline in									
	Early neonatal mortality					Neonatal mortality				
State/India	1977–82	1982–87	1987–92	1992–97	1997–2002	1977–82	1982–87	1987–92	1992–97	1997–2002
Andhra Pradesh	–11.1	3.2	–2.0	0.1	–5.9	–9.3	0.8	–3.1	–0.2	–3.4
Assam	–2.5	7.6	–13.5	6.1	–3.6	–2.5	2.3	–8.2	0.9	–2.1
Bihar	NA	–4.2	–2.4	1.1	–4.4	NA	–2.1	–6.3	0.9	–3.6
Gujarat	–0.3	–3.2	–6.1	0.5	0.2	–1.4	–3.0	–7.1	–0.4	–0.1
Haryana	–1.4	–2.0	–2.1	3.6	–7.6	–3.5	–3.6	–3.3	1.2	–3.8
Himachal Pradesh	–6.6	3.6	–5.9	–1.3	–3.0	–6.6	3.6	–5.9	–1.3	–3.0
Karnataka	–2.6	4.8	1.0	–4.3	–4.0	–4.1	3.6	–0.4	–5.9	0.4
Kerala	1.0	–6.2	–7.8	–3.1	–7.0	–4.2	–4.9	–11.4	–0.5	–4.6
Madhya Pradesh	3.4	–5.5	2.4	0.6	–6.3	–0.1	–3.0	–0.7	–0.6	–4.1
Maharashtra	–3.7	–2.9	–1.4	–2.3	–4.9	–4.0	–2.5	–2.2	–4.4	–2.4
Orissa	0.9	–0.4	0.6	–1.2	–2.6	1.1	–0.7	–1.0	–3.6	–2.1
Punjab	–3.6	–3.6	–3.6	4.4	–2.6	–6.9	–3.9	–3.3	–0.5	1.6
Rajasthan	–4.2	2.2	–2.2	2.3	–6.9	–4.2	1.7	–5.1	1.3	–3.4
Tamil Nadu	–3.4	0.7	–3.7	–1.4	–9.6	–4.6	–2.0	–3.8	–2.4	–4.3
Uttar Pradesh	4.4	–9.4	–1.3	–0.3	–3.9	1.9	–5.9	–5.0	–2.7	–1.0
West Bengal	NA	–10.1	3.4	–2.1	–5.8	NA	–7.0	0.3	–3.2	–4.0
India	–1.5	–3.8	–1.2	0.6	–6.3	–3.2	–2.7	–3.5	–1.1	–3.0

Table 1b. Average annual rates of change in mortality according to five-year periods by States and India

	Percentage decline in									
	Infant mortality					Under-five mortality				
State/India	1977–82	1982–87	1987–92	1992–97	1997–2002	1977–82	1982–87	1987–92	1992–97	1997–2002
Andhra Pradesh	–9.8	1.3	–3.8	–1.2	0.0	–9.7	0.4	–7.9	–1.1	–2.4
Assam	–3.1	1.0	–6.4	0.3	–1.5	1.3	–1.2	–4.3	–2.9	–2.0
Bihar	NA	–0.7	–7.6	0.4	–3.3	NA	–1.7	–10.1	2.0	–8.8
Gujarat	–3.9	–1.9	–9.2	–0.1	–0.6	–5.1	–2.5	–8.4	–1.7	–1.8
Haryana	–3.2	–1.8	–4.8	0.3	–1.8	–7.0	–1.4	–5.8	1.4	–5.2
Himachal Pradesh	–6.6	3.6	–5.9	–1.3	–3.0	–5.8	0.8	–7.7	–0.7	–6.9
Karnataka	–4.9	2.4	–0.6	–6.0	1.2	–4.5	0.1	–3.1	–5.5	–1.2
Kerala	–7.4	–3.2	–12.7	–0.3	–5.7	–5.6	–6.7	–15.7	0.8	–8.8
Madhya Pradesh	–2.4	–2.1	–2.0	–2.6	–2.3	–4.3	–1.0	–4.9	–3.1	–4.0
Maharashtra	–5.2	–2.2	–3.1	–3.0	–1.3	–5.3	–3.1	–6.6	–3.8	–3.2
Orissa	–1.8	–1.2	–1.6	–3.7	–1.4	–2.2	0.8	–4.9	–2.8	–3.8
Punjab	–7.5	–3.4	–3.2	–1.1	–0.2	–9.3	–0.7	–6.5	–1.6	–0.8
Rajasthan	–5.6	0.9	–5.5	0.9	–2.1	–3.1	–0.7	–8.0	0.4	–5.2
Tamil Nadu	–4.3	–2.3	–6.3	–0.5	–3.3	–4.7	–6.0	–9.2	–1.7	–3.5
Uttar Pradesh	–2.2	–3.3	–6.7	–2.1	–0.7	–5.0	–1.8	–7.6	–1.8	–4.0
West Bengal	NA	–3.8	–1.7	–3.9	–1.5	NA	–6.5	–4.7	–1.9	–7.3
India	–4.3	–1.9	–4.5	–1.2	–2.3	–4.3	–1.9	–6.9	–1.0	–5.1

declined by 63 points (51%) from 129 in 1976 to 63 in 2002 (SRS) (Appendix 1, Table A1.3). However, during the same period, the post-neonatal component declined (54%) slightly more than the NMR (Appendix 1, Table A1.4), which declined by 48%, resulting in an increase in the share of the NMR in the IMR. The trend suggested that with the progressive decline in IMR, the share of the NMR would increase. The child mortality declined by 76 points (78 in rural) from 98 in 1976 to 22 in 2002 (Appendix 1, Table A1.2). Much of the decline in IMR, which occurred due to faster decline in post-neonatal mortality, is attributed to improvements in general nutrition, environmental sanitation and immunization coverage.

Some determinants of child health: An evaluation of their effects

The complex setting and the interaction of social, economic, biological and demographic factors in developing countries often act as detriments to maternal, infant and child survival in the population (Govindasamy and Ramesh 1997; Govindasamy *et al.* 1993). Mosley and Chen (1984) have

Table 1c. Absolute change in components of under-five mortality rate according to five-year periods by States and India

	Absolute change in									
	Early neonatal mortality					Neonatal mortality				
State/India	1977–82	1982–87	1987–92	1992–97	1997–2002	1977–82	1982–87	1987–92	1992–97	1997–2002
Andhra Pradesh	–27.0	6.4	–4.0	0.1	–9.9	–32.3	2.3	–8.2	–0.4	–7.5
Assam	–4.9	17.2	–26.9	10.0	–6.3	–8.9	7.8	–24.6	2.2	–5.2
Bihar	NA	–8.2	–4.0	1.7	–6.4	NA	–6.5	–16.0	2.0	–7.4
Gujarat	–0.8	–7.4	–11.4	0.8	0.3	–5.3	–10.0	–18.2	–0.9	–0.3
Haryana	–2.4	–3.0	–2.9	5.2	–10.0	–10.8	–9.3	–7.1	2.5	–7.2
Himachal Pradesh	–29.0	–5.6	–2.5	–4.2	–8.5	–29.0	14.4	–22.5	–4.2	–8.5
Karnataka	–4.4	8.4	2.0	–2.1	–12.1	–10.3	8.8	–1.0	–13.5	0.8
Kerala	0.9	–4.9	–4.3	–1.3	–2.3	–5.6	–5.3	–8.2	–0.2	–2.1
Madhya Pradesh	8.4	–12.9	5.2	1.4	–13.0	–0.2	–10.8	–2.3	–1.9	–11.8
Maharashtra	–7.3	–4.9	–2.1	–3.2	–5.6	–11.4	–6.0	–4.7	–8.0	–3.6
Orissa	2.1	–1.0	1.4	–2.9	–5.6	4.5	–2.9	–3.7	–12.5	–6.3
Punjab	–4.5	–5.5	–3.9	4.7	–3.0	–18.2	–7.8	–5.5	–0.7	2.5
Rajasthan	–9.1	4.5	–4.4	4.7	–12.7	–14.2	5.5	–14.8	3.5	–8.6
Tamil Nadu	–7.7	1.4	–7.3	–2.3	–12.6	–14.9	–5.4	–9.1	–5.0	–7.4
Uttar Pradesh	13.1	–25.0	–2.0	–2.5	–1.2	9.0	–25.8	–16.1	–8.9	47.2
West Bengal	NA	15.8	4.4	–2.9	–5.3	NA	–16.9	0.5	–6.0	–5.1
India	–3.6	–7.7	–2.2	1.1	–9.7	–11.6	–8.6	–9.4	–2.6	–6.4

Table 1d. Absolute change in components of under-five mortality rate according to five-year periods by States and India

	Absolute change in									
	Infant mortality					Under-five mortality				
State/India	1977–82	1982–87	1987–92	1992–97	1997–2002	1977–82	1982–87	1987–92	1992–97	1997–2002
Andhra Pradesh	–49.2	5.3	–14.5	–3.9	–0.1	–80.4	2.4	–42.9	–4.6	–9.4
Assam	–16.7	5.0	–28.4	1.2	–5.5	11.4	–10.3	–33.5	–18.5	–11.4
Bihar	NA	–3.6	–31.8	1.3	–10.7	NA	–16.8	–74.6	12.0	–44.4
Gujarat	–23.4	–9.8	–36.4	–0.3	–1.9	–52.6	–21.3	–54.6	–8.7	–8.5
Haryana	–16.6	–8.1	–18.6	1.0	–5.9	–60.8	–9.6	–33.7	7.2	–24.8
Himachal Pradesh	–29.0	14.5	–22.5	–4.2	–8.5	–38.8	4.9	–38.4	–2.9	–22.9
Karnataka	–18.5	8.7	–2.3	–19.1	3.5	–29.8	0.4	–17.1	–24.5	–4.5
Kerala	–14.8	–4.9	–13.2	–0.3	–3.5	–17.9	–15.7	–21.6	0.8	–6.7
Madhya Pradesh	–16.9	–13.3	–11.1	–13.0	–10.4	–56.5	–11.4	–48.9	–24.9	–27.0
Maharashtra	–22.1	–7.6	–9.5	–7.9	–3.2	–36.3	–17.3	–29.2	–12.7	–9.1
Orissa	–12.7	–7.6	–9.7	–19.3	–6.4	–22.9	7.5	–44.1	–20.8	–23.7
Punjab	–35.0	–12.2	–9.4	–3.0	–0.6	–67.6	–4.0	–30.3	–6.1	–2.7
Rajasthan	–33.5	4.6	–25.9	3.9	–8.3	–34.8	–7.0	–66.2	2.6	–31.1
Tamil Nadu	–20.8	–9.3	–20.6	–1.5	–8.2	–39.7	–39.2	–40.9	–5.6	–10.4
Uttar Pradesh	–17.7	–23.3	–37.2	–9.2	–4.0	–70.9	–21.9	–72.7	–3.4	–42.4
West Bengal	NA	–12.0	–7.5	–10.5	–5.3	NA	–36.1	–27.0	–10.9	–22.3
India	–25.2	–9.7	–19.4	–4.4	–7.8	–43.6	–16.8	–42.7	–5.9	–24.7

identified a set of 14 intermediate variables which directly influence the risk of morbidity and mortality. They grouped 14 variables into five factors: maternal factors, environmental contamination with infectious agents, nutrient deficiency, injury and personal illness. The effects of social, economic, cultural and geographical variables are said to operate through these biomedical factors to exert an impact on mortality. Maternal factors include mother's age, parity and birth interval. First-born children, children born to very young or very old mothers and those from closely spaced pregnancies generally have a higher mortality rate than others (Boerma 1987; Boerma and Bicego 1992; Fauveau *et al.* 1988; Hobcraft 1987; Pandey *et al.* 1998). Low birthweight babies are subjected to a higher risk of dying than babies of normal birth weight. However, survival of such children could be affected through the quality of care—both prenatal and postnatal.

It is well documented that children belonging to a disadvantaged group, i.e. born in families or to women with low socioeconomic status are at greater risk of mortality

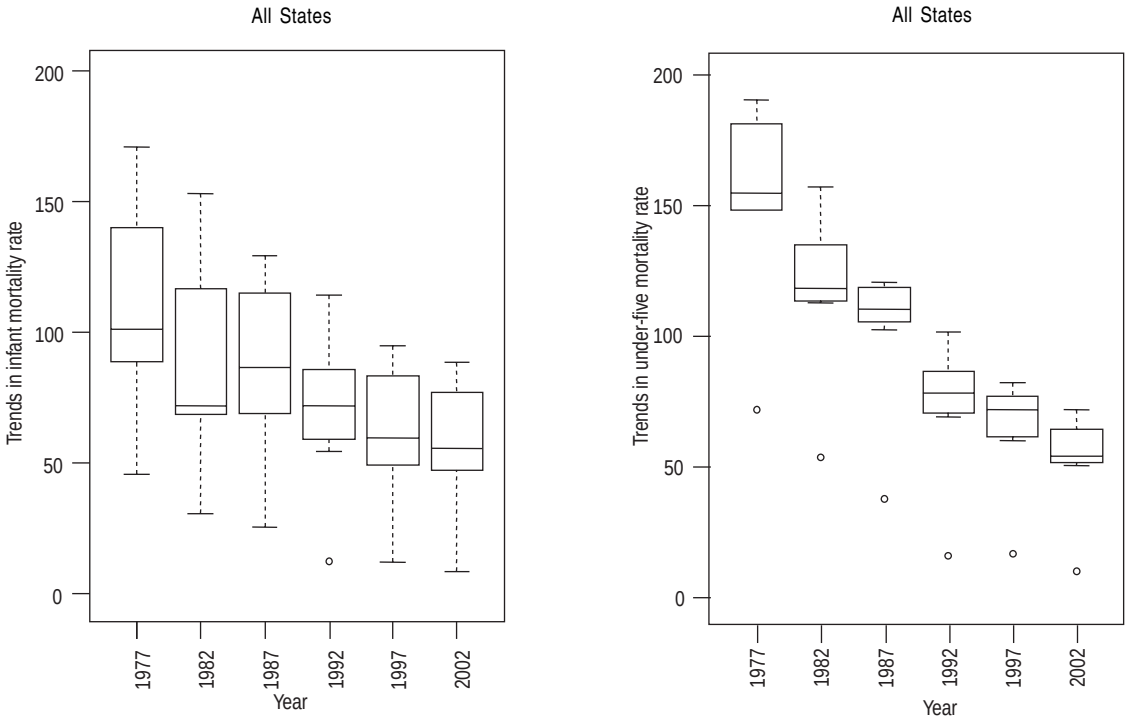

Fig. 3a Infant and under-five mortality rates by five-year periods, 1977–2002

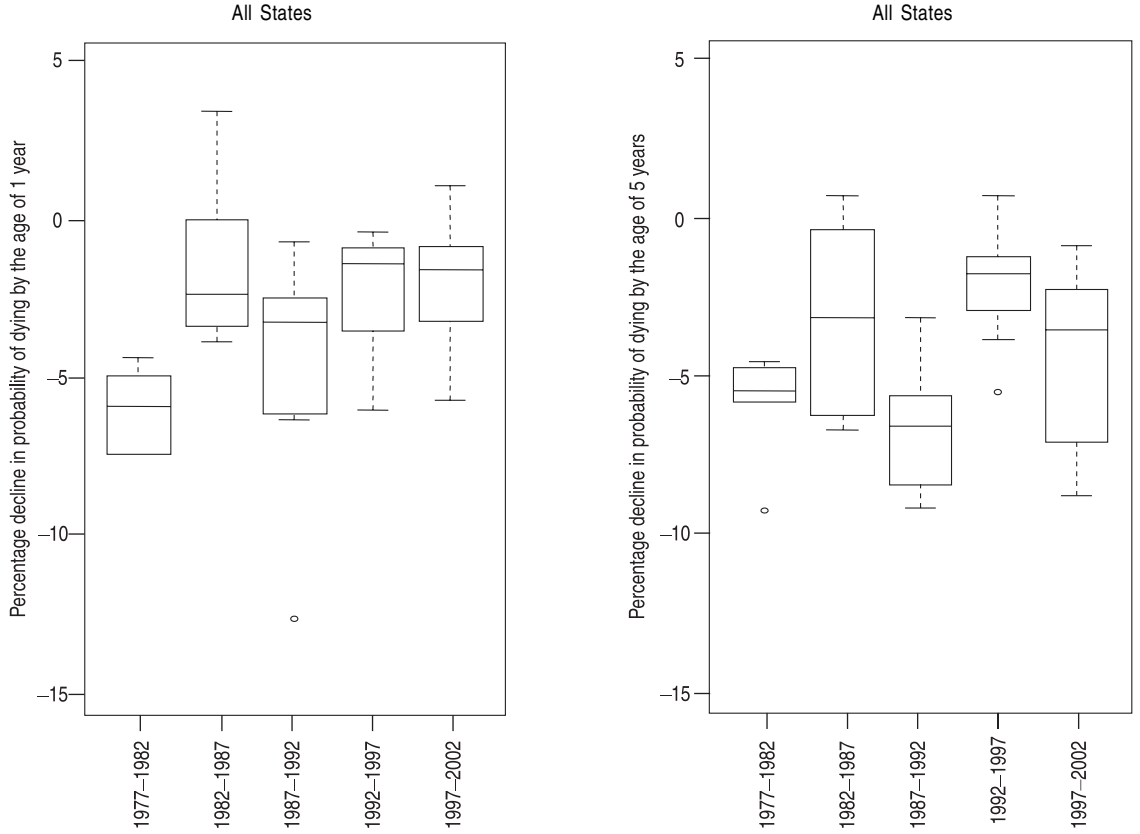

Fig. 3b Average annual rate of change in infant and under-five mortality rate by five-year periods, 1977–2002

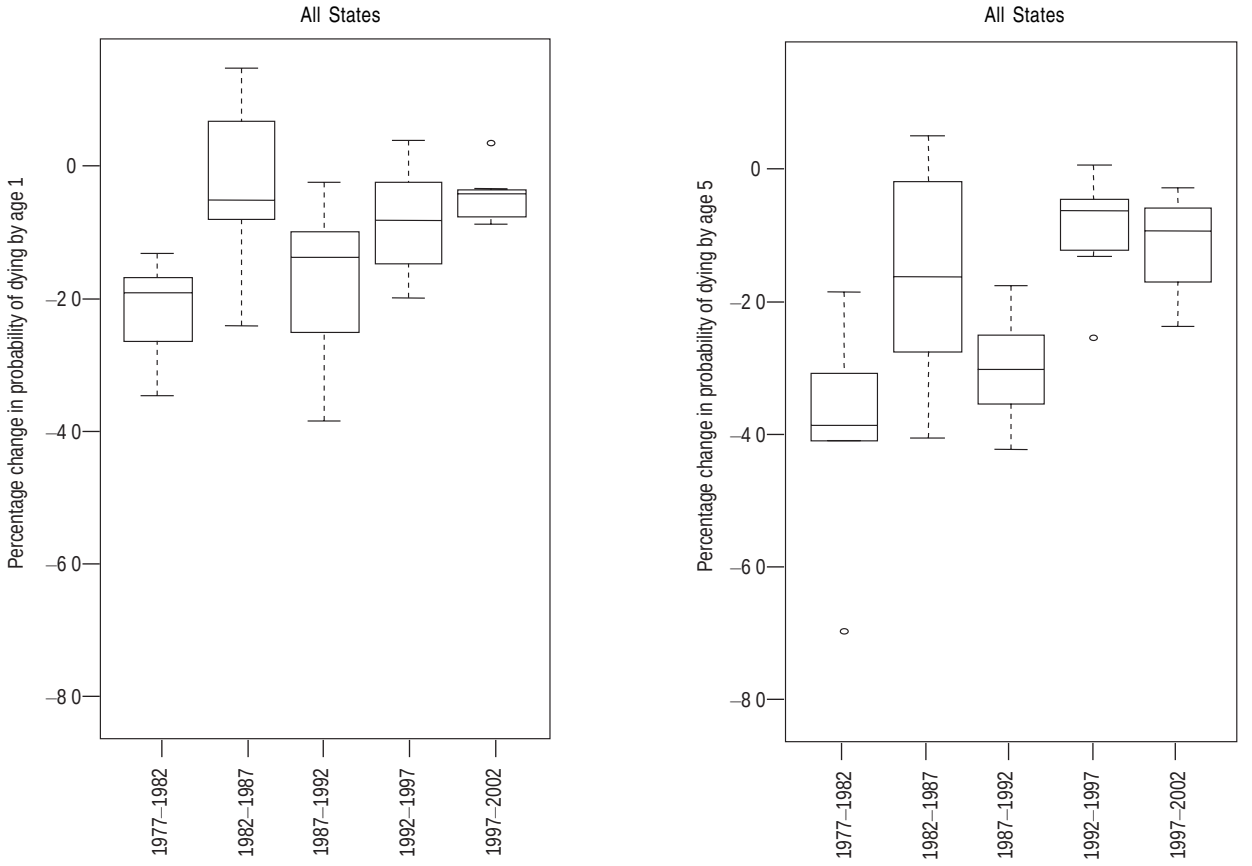

Fig. 3c Absolute change in infant and under-five mortality rate by five-year periods, 1977–2002

than those born in an advantaged group. Low socioeconomic status through intermediate variables leads to the proximal causes of death (nearer in time to the terminal event) such as undernutrition, infectious diseases and injury (Black *et al.* 2003). We focus here on the following:

- Levels of excess of child mortality among high-risk births
- Effect of socioeconomic status on components of child mortality. Four indicators—place of residence, caste, mother's education and standard of living index are considered to represent the social and economic status of a child. Place of residence is considered as an indicator because health services are more easily accessible in urban rather than in rural areas.
- Effects of breastfeeding practices on post-neonatal mortality
- Effects of the use of antenatal care services, immunization with tetanus toxoid and consumption of iron–folic acid during pregnancy on infant mortality.

The NFHS-2 data have been used to study the effects of high-risk births, social status and utilization of MCH on child mortality. The NFHS-1 data are used to study the effect of breastfeeding on post-neonatal mortality.

High-risk births and child mortality

As mentioned above, age of the mother at the time of birth has an important bearing on the survival of the child. Those mothers who give birth or become pregnant before they attain full physical growth tend to have a greater risk of complications during pregnancy or childbirth. Children born to mothers who were under the age of 20 years or 35 years and above are likely to have a higher risk of mortality. An analysis of the NFHS-2 estimates of the level of neonatal, post-neonatal and infant mortality by age of mothers at the time of birth has shown that too early or too late child-bearing has an effect on child mortality. Age of the mother at birth and birth order may have independent effects on neonatal and post-neonatal mortality. Children of birth order 2–3 have the lowest neonatal mortality rate whereas those of birth order 1 have the lowest post-neonatal and child mortality rates. Children of birth order 4 and above have the highest neonatal as well as post-neonatal and child mortality rates. The effect of length of preceding birth interval on infant survival is substantial—neonatal, post-neonatal and child mortality are almost double among children born after a birth interval of less than 2 years as compared to those born after two years (Pandey *et al.* 2004). Low birth-weight babies (less than 2500 g) are subjected to a higher risk of dying than babies of normal birth weight. In India, a large proportion (about 70%) of births is conducted at home and babies are not likely to be weighed. We have therefore taken the perceived size of the baby (large, average,

small and very small) as a proxy for birth weight and estimated the neonatal and post-neonatal mortality according to the size of the baby. By and large, neonatal mortality among babies of small and very small sizes at birth is almost twice as high as that among babies whose size at birth was average. Estimated neonatal mortality by the size of the baby after adjusting for the sociodemographic and antenatal health care factors also shows the same pattern (Table 2).

Income/standard of living index

Income is considered to serve as an indicator of children's consumption of calories and nutrients, use of medical systems and adult supervision, all of which affect their health. WHO has acknowledged that the health status of an individual is influenced by social and economic circumstances, over which individuals have little control (Victora *et al.* 2003). Measham *et al.* (1999) have documented an inverse relationship between per capita income and IMR. Non-income factors are found to play a significant role in lowering the IMR, i.e. the effect of technological progress (including access to preventive and curative health services) on lowering the IMR has been found to be the strongest with the magnitude of decline being 20% between 1975 and 1990.

The NFHS did not collect information on income; instead, it collected information on a household's level of wealth, mainly in the form of a stock of assets of a particular type. The standard of living index (SLI) of households —a measure of socioeconomic status— is constructed based on the scores assigned to type of house, type of toilet facility, source of lighting, main fuel for cooking, source of drinking water, separate room for cooking, ownership of house, ownership of agricultural land, ownership of irrigated land, ownership of livestock and ownership of durable goods (IIPS and ORC Macro 2000). Households are classified into three categories according to the standard of living index score as low, medium and high. Children born in households belonging to a low SLI are more likely to have higher exposure to diseases than those born in households with a high standard of living. The level of neonatal mortality in households with low SLI is 19% higher than that in households with medium SLI, and it is 90% higher than that in households with high SLI (Table 2).

Post-neonatal mortality in low SLI households was 38% higher than that of medium SLI households and 175% higher than that of high SLI households. Similarly, child mortality in households with low SLI was 74% and 400% higher than that in medium and high SLI households. It may however be mentioned that SLI affects mortality through a number of intermediate variables and by including them in the hazard model will ultimately reduce the impact of SLI. The unadjusted mortality rates show a rapid decline across the three groups of SLI. However, the adjusted effects of SLI are much smaller: negligible in the case of neonatal mortality.

Place of residence

Research on child mortality in India has shown that mortality is lower in urban areas. One of the frequently mentioned possible causes is the relatively greater availability of medical services and their quality. Large differences in neonatal, post-neonatal and child mortality between rural and urban areas may be due to factors closely related to certain development activities and services available in urban and not in rural areas (Fig. 4). Urban populations benefit more from better health resources, but they also have a higher standard of living, more knowledge about seeking help when it is needed, and are better educated than rural dwellers. Also, the proportion of socially disadvantaged groups (SC/ST population) and high-risk births are lower in urban areas. When mother's education and mother's age at childbirth, standard of living of households, child's sex, preceding birth interval, mother's exposure to media and caste are controlled, the gap between urban and rural mortality is considerably reduced.

Mother's education

In developing countries including India, mother's education has been considered to have a strong effect on the mortality of young children (Das and Dey 2003; Khasakhala 2003; Rama Rao *et al.* 1997). Educated mothers are more likely than non-literate mothers to ensure a healthy environment, nutritious food, and have better knowledge about reproductive health at conception and health care facilities for their children. As a result, literate mothers give birth to healthier babies because they themselves tend to be healthier and are likely to experience lower mortality among their children at all ages. The level of mother's education is inversely related to the level of child mortality. The higher the educational level of mother the lower the level of neonatal, post-neonatal and child morality. The IMR and the child mortality rate (CMR) are higher (44% and 112%, respectively) for children born to illiterate mothers than for children born to just-literate ones (who are literate but less than middle school completed) (Table 2). It is almost one-and-a-half times higher in case of IMR and five times higher in case of CMR among children born to illiterate mothers as compared to children born to mothers who have completed middle school and above. The differences in adjusted mortality rates between children born to mothers belonging to two consecutive educational levels remain high. Thus, it can be summarized that mother's education emerges as an important factor associated with U5MR and also has an effect on post-neonatal mortality rate (PNMR).

Caste

Membership of the head of the household to an SC/ST is known to affect many aspects of the life of their families, particularly survival of the newborn. Such effects reflect

Table 2. Per cent distribution of births during the 35 months preceding the reference date, and unadjusted and adjusted neonatal, post-neonatal, infant, and child mortality rates for the ten-year period preceding the reference date by selected background characteristics of mother, India

Characteristic	Percentage of births during the 35 months preceding the reference date	Unadjusted					Adjusted				
		NMR	PNMR	IMR	CMR	U5MR	NMR	PNMR	IMR	CMR	U5MR
Place of residence											
Rural	77.7	52	28	81	31	107	41	21	62	20	80
Urban	22.3	33	15	49	16	63	34	18	42	17	68
Mother's education (completed years of education)											
0	54.4	57	32	89	36	120	49	28	76	29	102
1–7	22.2	42	20	62	17	77	38	19	56	18	73
8+	23.4	27	11	37	6	43	28	15	43	9	51
Caste											
SC/ST	30.2	54	31	85	23	104	43	21	62	19	81
OBC/Others	69.8	46	24	69	37	103	39	10	59	21	69
Standard of living index of the households											
Low	36.3	57	33	90	40	125	42	25	66	28	92
Medium	47.5	48	24	72	23	92	40	21	60	18	77
High	16.2	30	12	42	8	49	38	17	55	11	65
Sex of the child											
Female	48.1	45	27	72	33	101	37	22	57	22	79
Male	51.9	51	24	75	22	94	43	20	62	17	78
Preceding birth interval for birth order two or more											
<24 months	24.4	67	37	104	39	144	63	35	95	34	126
24 months+	75.6	32	21	53	24	76	31	20	50	21	70
Birth order of the child											
1	29.2	53	22	75	17	90	46	17	63	11	73
2–3	43.5	42	23	64	26	91	36	20	55	21	75
4–5	17.4	47	28	75	35	110	37	24	60	26	84
6+	9.9	65	40	105	43	148	54	37	89	38	123
Mother's age at the time of first birth											
<20	51.7	62	29	91	23	110	49	21	68	13	81
20–34	48.1	40	14	54	11	64	40	14	53	9	62
35+	0.2	50	NC	NC	NC	NC	7	NC	NC	NC	NC
Mother's age at birth order two or more											
<20	12.0	66	32	98	37	129	35	28	62	24	85
20–34	82.4	42	25	67	29	93	38	22	59	23	81
35+	5.6	63	38	101	40	135	55	22	76	38	124
Mother's exposure to the media											
No	45.5	56	32	88	38	121	39	25	62	26	87
Yes	54.5	41	19	60	17	75	40	20	59	16	74

NMR: neonatal mortality rate; PNMR: post-neonatal mortality rate; IMR: infant mortality rate; CMR: child mortality rate; U5MR: under-5 mortality rate; NC: Calculation not done because of small number of deaths

differences in lifestyle based on traditions or beliefs and practices related to childbearing, childbirth, childfeeding and health care. These may affect the child's health and accessibility of health facilities and services. Each of the components of under-five mortality is higher among SC/ST families than among families which belong to OBC or other castes. However, between caste groups, the differences in adjusted mortality rates are much smaller than the difference between the unadjusted mortality rates.

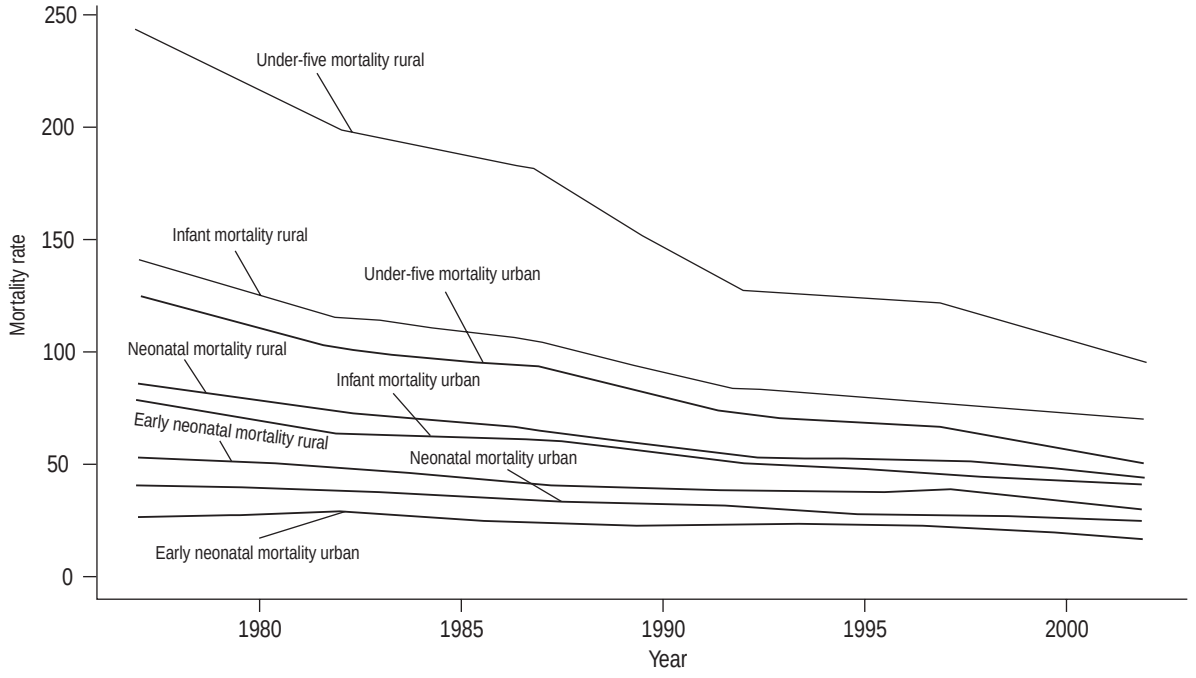

Fig. 4 Trends in early neonatal, neonatal, infant and under-five mortality rates by residence, India, 1977–2002

Mother's exposure to the media

Mother's exposure to the media should tend to reduce the mortality of children because such women are more likely to have access to information on ways of enhancing maternal and child health care. Exposure to the mass media is an indicator of the economic status of the household as well. The level of neonatal mortality exhibits the expected relationship; it is higher for children of mothers who are not exposed to the mass media. However, when the effects of other variables are controlled, the effect of the mass media is negligible. In fact, the neonatal mortality is likely to be affected by endogenous variables as well as biological factors. As expected, post-neonatal and child mortality is consistently higher for children whose mothers are not exposed to the mass media even after controlling for the effects of other variables.

Child's sex

Mortality during the neonatal period is expected to be higher among male children than among female children as during the neonatal stage males are more prone to death than females due to their biological constitution. The NMR is higher among male than among female children. Son preference in a patriarchal society like India affects it through parental care during infancy through postnatal care. The post-neonatal and child mortality rates among female children are 13% and 50% higher, respectively, than among male children (Table 2).

SRS data show that the ratio of female to male IMR for India was less than unity during 1981 (0.981) and 1991 (0.988), whereas during 1998 it exceeded unity. In other words, during 1981 and 1991 more male than female infants died whereas the reverse was true for the year 1998. The ratios of female to male mortality in India have not only been invariably in favour of males but they have also been larger than what was observed during infancy. Over time, these gaps have further widened. During the early 1980s, an additional 366 female children 1–4 years of age died for every 1000 deaths among male children. The excess of female child deaths increased by 405 in the next five years and widened further to 463 during 1993–97 (Usha 2004).

Prenatal care and child mortality reduction

Further, we examine the effect on neonatal and post-neonatal mortality of programmatic factors such as antenatal and delivery care, especially the number of antenatal check-ups during pregnancy, immunization of women against tetanus during pregnancy, full consumption of iron–folic acid (IFA) tablets or syrups during pregnancy, and delivery in a medical facility. Ideally, antenatal care (ANC) should monitor a pregnancy for signs of complications, detect and treat pre-existing and concurrent problems of pregnancy, and provide advice and counselling on preventive care, diet during pregnancy, delivery care, postnatal care and related issues (IIPS & ORC Macro 2000). Infant mortality is lower (48%) among children whose mothers received 3 or more ANC check-ups as compared to those children born to mothers who either did not receive any ANC check-up or received less than 3 (Table 3). The effect of ANC visits on survival during the post-neonatal period is more pronounced than during the neonatal period. The adjusted effect is a little

Table 3. Per cent distribution of births during the 35 months preceding the reference date, and unadjusted and adjusted neonatal, post-neonatal and infant mortality rates for the three-year period preceding the reference date by selected characteristics, India

Characteristic	Percentage of births during the 35 months preceding the reference date	Unadjusted			Adjusted		
		NMR	PNMR	IMR	NMR	PNMR	IMR
Antenatal care visits made by mothers during pregnancy							
0–2	55.3	45	29	73	35	26	60
3+	44.7	25	13	38	28	13	41
Full consumption of iron–folic acid tablets or syrups							
No	20.2	32	25	56	28	21	48
Yes	79.8	26	14	40	27	13	40
Mother received at least two injections during pregnancy							
No	31.9	52	33	83	49	20	68
Yes	68.1	26	16	42	27	15	42
Place of delivery							
Institutional	33.9	39	14	52	38	16	53
Home or others	66.1	31	25	55	32	19	50
Size of the baby at birth							
Large/very large	14.0	34	16	49	33	14	47
Average	61.3	27	20	46	26	17	43
Small/very small	24.6	54	28	80	50	18	67

lower than the unadjusted effect. Because utilization of ANC services is correlated with receipt of tetanus toxoid (TT) injections, consumption of IFA tablets or syrups and the socioeconomic background characteristics, these are included in the model for estimating adjusted effects (Table 3).

Tetanus is a major cause of neonatal mortality in developing countries. Pregnant women require two injections of TT to protect their newborns. It is observed that if the mother is immunized with TT during pregnancy, the likelihood of both neonatal and post-neonatal death is reduced to half. Even after controlling for sociodemographic characteristics and other ANC variables, the adjusted effect of TT immunization to pregnant women on neonatal mortality remained more or less the same. The finding reinforces the importance of TT immunization for pregnant women as this reduces infant and under-five mortality in general and neonatal mortality in particular.

Iron deficiency anaemia among mothers is a major threat to safe motherhood and to the health and survival of the infant because it contributes to low birth weight, lowered resistance to infection and impaired development. Supplementary iron in the form of tablets or syrup is the simplest method of preventing iron deficiency when the requirement is higher over a relative short period of time as in pregnancy and lactation. In India the provision of IFA tablets to pregnant and lactating mothers to prevent anaemia forms an integral part of the RCH Programme. Though the effect of consumption of IFA tablets or syrups is observed to be small during the neonatal period, it is quite high during the post-neonatal period. The adjusted post-neonatal mortality is 40% lower among children whose mothers consumed IFA tablets or syrups than those children whose mothers did not.

It is assumed that children born in institutions are likely to have a better sanitary environment and health care assistance, and thereby have lower neonatal, post-neonatal and infant mortality rates. In India, two-thirds of deliveries take place at home. Some States in India have a high percentage of institutional deliveries (93% in Kerala, 79% in Tamil Nadu and around 50% in Gujarat, Maharashtra, Andhra Pradesh and Karnataka). The effect of place of delivery on neonatal mortality is observed in States where institutional deliveries are higher. In States where facilities for institutional deliveries are lacking, women delivering in institutions are mostly from a high socioeconomic status.

Breastfeeding practices and child mortality

An analysis of the NFHS-1 (1992–93) was carried out to estimate the effect of breastfeeding practices on mortality over the period of infancy (IRMS 2004). It found that the risk of dying during the age segment (1, 6) months was lowest for children breastfeeding and having plain water only compared to the children who were given other types of breastfeeding (Table 4). In the age segment (1, 6) months breastfeeding and plain water only appear to be more

Table 4. Death rates (per 1000 person-years) in different age segments of the first year of life according to type of breastfeeding exposure by selected characteristics irrespective of the level of PNMR*, NFHS-1

Characteristics/ category	Age (in months)	Period of exposure (no. of months preceding the survey)	Death rate (per 1000 person-years) during the period child was under						
			Exclusive breast-feeding	Breast-feeding + water only	Full breast-feeding	Breast-feeding + supplements	Non-exclusive breast-feeding	No breast-feeding	Total
All	1–6	0–12	65.1	13.5	39.2	35.6	23.2	177.4	42.2
	6–12	0–24	114.4	14.9	35.5	11.6	12.4	44.7	21.2
Standard of living index									
Low	1–6	0–12	62.9	11.0	39.6	58.4	29.4	317.0	48.7
	6–12	0–24	87.6	16.1	33.5	13.9	14.5	81.1	24.9
Medium / high	1–6	0–12	70.1	15.6	40.5	25.3	20.2	114.2	38.4
	6–12	0–24	145.1	14.5	36.9	10.7	11.5	31.8	18.8
Residence									
Urban	1–6	0–12	53.8	3.8	23.5	25.4	13.8	81.7	27.3
	6–12	0–24	100.5	11.6	27.6	12.1	12.0	14.0	15.2
Rural	1–6	0–12	67.4	16.7	43.3	39.4	26.5	253.3	46.7
	6–12	0–24	116.6	15.6	37.0	11.5	12.5	64.1	23.0
Mother's education									
Illiterate	1–6	0–12	69.1	16.6	44.2	49.3	29.4	350.2	51.3
	6–12	0–24	126.5	18.7	42.5	12.8	14.5	69.4	26.2
Literate, < middle school	1–6	0–12	52.3	2.3	24.6	11.0	6.6	136.3	23.6
complete	6–12	0–24	61.1	2.3	11.2	10.2	8.7	32.7	13.0
Middle school complete	1–6	0–12	54.8	12.1	29.6	25.4	19.3	5.2	26.4
and above	6–12	0–24	29.6	0.0	4.8	9.1	8.0	18.4	10.5
Caste									
Scheduled caste	1–6	0–12	61.8	15.2	38.1	29.9	22.0	154.0	39.3
	6–12	0–24	109.6	12.2	31.9	10.8	11.1	37.4	18.7
Scheduled tribe	1–6	0–12	55.2	6.2	30.5	29.9	12.3	431.7	37.2
	6–12	0–24	88.3	17.9	33.1	6.6	10.3	110.3	23.9
Others	1–6	0–12	88.9	9.7	51.0	81.2	37.7	243.7	62.3
	6–12	0–24	154.0	25.5	54.2	20.3	21.8	63.9	34.2

*North-eastern States are excluded

beneficial than breastfeeding with supplements, which contradicts the concluding remarks of Anandiah and Choe (2000). The results show that any type of breastfeeding during [1, 6) months and non-exclusive breastfeeding[1] during [6, 12) months reduces the instantaneous risk of mortality. However, during the age segment [1, 6) months the risk of death of children receiving breast milk with plain water only was lower compared to children receiving only breast milk, as well as children receiving breast milk with supplementary food.

A large number of factors other than breastfeeding may affect the post-neonatal mortality rate. The multivariate hazard model with provision of time-dependent covariates was used to compare the effects of exclusive breastfeeding, breastfeeding and water only, and not breastfeeding with respect to breastfeeding with supplements after controlling for the effects on mortality of potentially confounding variables. The results of the hazard analysis revealed that though the relative risk of death of children currently receiving only breast milk at any given age 'a' ($1 \leq a < 6$ months) compared to children currently receiving breast milk plus food supplements at that age is higher, the relative risk of death of children currently receiving breast milk plus plain water only at any given age between 1 and 6 months was much lower compared to children currently receiving breast milk and food supplements (Table 5).

UNICEF and WHO recommend that children should be exclusively breastfed for about 6 months, and complementary foods should begin at around 6 months of age, and that breastfeeding should continue well into the second year of life and beyond. Analysis of the NFHS-1 data revealed that during the early post-neonatal period, if breastfeeding was supplemented with plain water then the mortality was lowest compared to other types of breastfeeding practices. One of

[1]A breastfeeding child given plain water only was also considered as non-exclusively breastfeeding.

Table 5. Proportional hazards analysis of different types of breastfeeding practices on mortality for the age groups 1–4 and 4–6 months for births with an exposure in the aforesaid age group during 12 months and for age groups 6–9, 9–12 months for births with an exposure in the aforesaid age group during 24 months preceding the survey by different background characteristics, NFHS-1

			Children belonging to the age group (in months) Hazard ratio			
Variable	Reference category	Category	1–4	4–6	6–9	9–12
Type of breastfeeding	Breastfeeding + supplements	Exclusive breastfeeding indicator	0.90	1.65	4.78*	10.80*
		(Breastfeeding + water) indicator	0.29*	0.64	0.69	1.33
		Not breastfeeding indicator	5.54*	3.94*	6.36*	3.82*
Place of residence	Rural	Urban	1.14	0.58	0.85	1.19
Mother's age at birth	20–34 years	Less than 20 years	1.58*	0.48	0.79	0.91
		35 years or more	1.49	1.39	1.95*	2.37*
Mother's education	Illiterate	Literate, <middle school complete	0.50*	0.41**	0.66	0.95
		Middle school complete and above	0.35*	0.57	0.38*	0.62
Mother's occupation	Non-gainful occupation	Gainful occupation	1.72*	0.96	1.14	1.51
Interval since last birth	First birth/preceding birth interval 2 years or more	Preceding birth interval less than 2 years	2.01*	1.60	1.69*	1.59†
Antenatal and natal care	No	Yes	0.63*	0.73	0.49*	0.33*
Size of the baby	Medium/large	Small	1.92*	2.54*	1.65*	1.33
Standard of living index	Low	Medium/high	1.05	1.95†	1.02	0.99

Note: Statistical significance: *p<0.05, **0.05<p<0.10, unmarked coefficients are not significant

the possible reasons could be that children on exclusive breastfeeding were possibly not getting the minimum required water that is essential for maintaining body fluids and they might get dehydrated thus leading to death whereas children on water supplements were able to maintain the fluid level leading to better survival. Mortality among young post-neonates receiving food supplements along with breast milk was higher than those who were exclusively breastfed. This reflects the fact that supplementary food given to children is neither sufficient in quantity nor nutritive enough in relation to the requirement. Studies conducted in different parts of the country reported that dehydration following diarrhoea was a major cause of post-neonatal mortality. Transmission of bacilli causing diarrhoea and related illnesses is primarily through food and water. Solid foods given during the second half of the post-neonatal age become a major cause of diarrhoea.

Inequalities in utilization of RCH services

Evaluating the impact of the RCH Programme is a daunting task. With increasing resources being spent to hasten improvement in maternal and child health conditions, there is a need to evaluate these efforts on a regular basis. The most common approach to the evaluation of MCH programmes is to measure the services reported by these programmes and their complementary activities—number of pregnant women who received ANC check-ups, were immunized against TT and received an adequate quantity of iron; number of children immunized, age at immunizations, awareness among mothers about the causes and treatment of diarrhoea, knowledge of mothers about the symptoms of ARI, place of treatment, etc. Such knowledge is essential to reformulate the intervention strategies and their implementation, identify new thrust areas and introduce new approaches to achieve optimum results. Service statistics generate data on a yearly basis on programme inputs and outputs such as immunizations, institutional deliveries, care-seeking indicators, etc. These data are generated from institutions which are covered under the Programme and do not include the inputs and outputs, and the outcomes of the private and voluntary sectors. Thus, most of the published indicators have limited value in assessing outputs and outcomes. Furthermore, outcomes and outputs are sometimes overstated. For example, throughout the 1990s, the reported DPT3 rate for India as a whole was around 90% (Claeson *et al.* 1999), but the DPT3 coverage was found to be only 52% in the NFHS-1 and NFHS-2 (IIPS 1995, IIPS & ORC Macro 2000). Similarly, the Multi-indicator Cluster Survey in 2000 (MICS 2000) gave the DPT3 coverage as 47%.

In the absence of reliable service statistics on the utilization and quality of health care services, it was somewhat difficult to attribute the trends in improvement in child health conditions and reduction in child mortality to programme inputs. Two rounds of the NFHS conducted in 1992–93 (NFHS-1) and 1998–99 (NFHS-2) created a landmark in the field of data collection on various aspects of health and family welfare. The main objective was to provide reliable and high-quality data on a number of issues required for the development, monitoring and evaluation of programmes. To explore the possible reasons for the slowing down of the decline in child mortality we have analysed the extent of utilization of antenatal, natal and postnatal care services among children who were born during the three years preceding the survey for NFHS-2 and four years preceding the survey for NFHS-1. We have also analysed the differentials in the nutritional status, family planning acceptance and reproductive health problems among women

in the study population. The effect of programmatic factors, such as antenatal and delivery care, immunization of pregnant women against tetanus, full consumption of IFA tablets or syrups and delivery in a medical facility on neonatal mortality have been estimated using the NFHS-2 data. Table 6 presents the differentials of prenatal, natal and postnatal care, nutritional status, knowledge of mothers about treatment of diarrhoea, etc. Table 7 presents the status of fertility, family planning and nutrition, and the reproductive health problems of mothers, etc.

ANC check-ups

According to NFHS-2, in India only about two-thirds of mothers received ANC during pregnancy, and only 44% received 3 or more check-ups. The above figure has not undergone any change since NFHS-1. Women who did not receive antenatal check-ups are mostly older women, those having high parity, from rural areas, ST, illiterate and poor women. Check-ups during the first trimester were about twice as common in urban areas as in rural areas (IIPS, 1995; IIPS & ORC Macro 2000). The effectiveness of antenatal check-ups in ensuring safe motherhood depends in part on the tests and measurements done and the advice given during the check-ups. The NFHS-2 results show that most of these tests were performed at least 1.5 times more frequently for mothers living in urban areas than for those living in rural areas.

Tetanus toxoid

The proportion of mothers who received two or more TT injections during their pregnancy rose from 55% to 67% between the NFHS-1 to NFHS-2. Coverage (two or more injections) was found to be lower for births to women 35 years of age and above than to younger women; varying inversely by birth order, literacy level of the mother, standard of living; rural and urban areas, and for births to SC mothers.

Institutional deliveries

Deliveries in health facilities in India have increased from 26% during NFHS-1 to 34% during NFHS-2. It is largely assumed that children born in health care institutions are more likely to get a better sanitary environment and receive the required health care assistance, and thereby tend to have lower neonatal mortality than those born at home.

Iron and folic acid supplements

For India as a whole, iron–folic acid (IFA) coverage improved slightly from 52% in NFHS-1 to 58% in NFHS-2. Some of this improvement may be due to the fact that IFA syrup was included in the measurement of IFA coverage in NFHS-2 but not in NFHS-1. Only 66% of mothers received an adequate supply and consumed all of it, 16% received but did not consume all that was supplied.

Vaccinations

The percentage of children fully vaccinated in the age group of 12–23 months in NFHS-1 and NFHS-2 were 35.4% and 42.0%, respectively. The percentage of those vaccinated was higher among urban than rural children. A strong positive relationship of children's immunization coverage was observed with mother's education and standard of living. ST children were less likely to be fully vaccinated than SC children and those of other castes.

Diarrhoea

Deaths from acute diarrhoea are most often due to dehydration and loss of water and electrolytes. Nearly all dehydration-related deaths can be prevented by prompt administration of rehydration solutions. One major goal of the 'Oral Rehydration Therapy Programme' is to increase awareness among mothers and communities about the cause and treatment of diarrhoea. The percentage of children under 3 years of age who suffered from diarrhoea in the two-week period before the survey was 10% in the NFHS-1 and 19% in NFHS-2. However, because of seasonal variations in the prevalence of diarrhoea, the NFHS-1 and NFHS-2 rates cannot be compared. The prevalence among children of mothers with high school or higher education and children living in households with a high standard of living (NFHS-2) was lesser than that among other children. Sixty-two per cent of mothers who had given birth during the three years preceding the survey (NFHS-2) knew about ORS packets; against only 43% in NFHS-1. Knowledge was considerably low among rural, illiterate and ST mothers. However, ORT is a stop-gap measure that does not always manage to compensate for the lack of safe drinking water and clean living conditions, which in many places appear to remain unattainable goals. To assess the possible impact of ORT in reducing deaths due to diarrhoea, it is equally important to know the practices with regard to the food and fluids usually given to children during diarrhoea. Rural, illiterate and ST mothers were much less likely to report correctly (NFHS-2) that children with diarrhoea should be given more to drink. Among children who suffered from diarrhoea during the two weeks preceding the survey, 61.2% in NFHS-1 and 63.2% in NFHS-2 were taken to a health facility. Again, the percentage taken to a health facility was much higher for urban than rural children, and children of more educated mothers. The percentage was particularly low for ST children and for children living in households with a low SLI.

Table 6. Description of ANC care, natal and PNC, prevalance of ARI and diarrhoea and their treatment, India

Characteristic	NFHS round	Place of residence			Mother's education				Caste			Standard of living index		
		All	Urban	Rural	Illiterate	Literate, <Middle school complete	Middle school complete	High school complete and above	SC	ST	Other than SC, ST	Low	Medium	High
Antenatal check-ups														
Percentage of mothers who received three or more antenatal check-ups during pregnancy	1	43.6	66.0	37.0	29.0	57.0	79.0		32.0	32.0	48.0	28.0	66.0	69.0
	2	43.8	69.0	37.0	26.0	55.0	77.0		35.0	35.0	48.0	31.0	45.0	71.0
Percentage of mothers who received no antenatal check-ups during pregnancy	1	36.8	17.8	42.4	48.8	19.9	9.8	4.2	42.2	52.3	34.0			
	2	34.0	13.6	39.8	48.4	19.3	13.5	5.8	38.2	43.1	31.1	45.1	32.8	12.4
Tetanus toxoid vaccinations														
Percentage of mothers who received two or more TT injections during pregnancy	1	53.8	74.4	47.7	40.3	71.9	84.3	91.5	47.4	34.1	57.3	39.0	75.0	78.0
	2	66.8	81.9	62.5	54.7	78.4	84.2	91.2	64.8	46.4	70.5	55.4	68.7	87.5
Percentage of mothers who received no TT injections during pregnancy	1	39.0	19.4	44.7	51.7	20.9	10.0	4.4	45.4	56.2	35.7			
	2	24.1	9.9	28.2	35.3	12.5	7.5	3.0	25.8	38.7	21.2	34.1	22.3	6.4
Place of delivery and birth attendant at time of delivery														
Percentage of institutional deliveries	1	35.5	17.0	56.0	11.8	37.8	55.4	75.3	16.0	9.1	29.2	11.0	48.0	23.0
	2	33.6	65.0	25.0	17.4	43.4	55.1	75.0	26.8	17.1	38.0	18.5	34.9	64.6
Percentage of home deliveries attended by trained birth attendent	1	35.2	22.0	39.1	41.8	30.2	20.4	10.5	39.5	41.8	33.7	—	—	—
	2	35.0	18.8	39.6	44.7	28.4	21.0	11.8	37.7	44.4	33.0	43.5	34.3	17.5
Consumption of iron–folic acid tablets or syrups														
Percentage of mothers given IFA tablets during pregnancy														
During four years preceding the survey	1	50.5	68.7	45.1	38.3	66.6	77.2	85.6	44.2	40.2	52.8	51.0	49.0	59.0
During three years preceding the survey	2	57.6	75.7	52.5	43.6	70.4	78.5	86.5	54.6	48.6	60.2	46.0	59.4	79.2
Among those who received IFA during pregnancy percentage who consumed all the supply	2	80.5	83.2	79.4	76.3	80.7	81.5	88.7	76.2	82.0	81.5	77.1	80.2	86.1
Percentage of children age 12–23 months who received all vaccinations														
All*	1	35.4	50.7	30.9	24.0	46.9	60.3	70.0	26.8	24.8	38.2	—	—	—
	2	42.0	60.5	36.6	27.8	52.3	62.7	72.7	40.2	26.4	45.1	30.4	43.2	64.7
None	1	30.0	16.4	34.0	40.1	16.9	8.2	4.2	36.9	41.8	27.4			
	2	14.4	6.40	16.7	21.2	8.0	4.6	1.4	15.1	24.2	12.5	20.8	13.1	4.0
Percentage of children who were ill with diarrhoea† during the two weeks preceding the survey														
Among all children under four years of age	1	10.0	8.80	10.4	10.3	10.4	9.6	8.3	11.4	9.9	9.8	—	—	—
Among all children under three years of age	2	19.2	19.6	19.0	20.1	19.8	18.6	15.0	19.8	21.1	18.7	19.9	19.7	16.1
Percentage of diarrhoea cases taken to a health facility or provider														
Among all children under four years of age	1	61.2	68.7	59.3	58.0	66.0	67.5	70.9	61.2	51.5	62.4	—	—	—
Among all children under three years of age	2	63.4	75.2	59.9	58.5	65.2	74.2	78.2	54.6	52.2	65.1	55.5	65.1	77.2
Percentage of children who were ill with ARI during the two weeks preceding the survey														
Among all children under four years of age	1	6.5	5.1	6.9	6.5	7.7	5.9	4.6	6.8	6.1	6.5	—	—	—

(Cont.)

Table 6 *(cont.)*. Description of ANC care, natal and PNC, prevalance of ARI and diarrhoea and their treatment, India

Characteristic	NFHS round	All	Place of residence		Mother's education				Caste			Standard of living index		
			Urban	Rural	Illiterate	Literate, <Middle school complete	Middle school complete	High school complete and above	SC	ST	Other than SC, ST	Low	Medium	High
Among all children under three years of age	2	19.3	16.2	20.3	20.6	20.3	18.8	13.8	19.6	22.4	18.9	21.0	19.4	15.7
Percentage with ARI taken to a health facility or provider														
Among all children under four years of age	1	66.3	77.1	63.9	62.4	70.4	72.0	84.9	64.0	59.1	67.6	—	—	—
Among all children under three years of age	2	64.0	75.1	61.4	58.3	69.5	76.3	77.0	60.3	50.4	67.5	55.1	67.4	76.9
Percentage of children who were classified as undernourished according weight for age														
Weight - for - height														
Among children under under four years of age														
Percentage below –3 SD ***	1	20.6	14.8	22.4	24.7	16.7	12.4	7.8	23.7	25.3	19.5	—	—	—
Among children under under three years of age														
Percentage below –3 SD ***	2	18.0	11.6	19.9	24.1	13.1	10.8	5.8	21.2	26.0	15.6	25.3	16.5	6.7
Percentage of having iron-deficiency anaemia by degree of anaemia														
Children age 6–35 months with any anaemia	2	74.3	70.8	75.3	78.2	74.6	69.7	61.9	78.3	79.8	72.4	78.7	73.5	67.3
Percentage of births during three years preceding the survey														
Birth order														
4+	1	31.0	24.1	32.9	38.5	23.6	12.4	7.4	—	—	—	—	—	—
	2	27.5	19.2	29.9	38.2	19.6	9.3	4.6	31.5	34.0	25.3	36.7	25.7	11.7
Preceding birth interval														
Less than 24 months	1	26.9	28.8	26.3	25.8	28.7	30.6	30.3	27.2	26.5	—	—	—	—
	2	28.3	29.4	27.9	27.5	30.0	31.3	27.8	27.3	29.3	28.4	27.2	28.9	29.3

Note:

*Children who were fully vaccinated, i.e. those who received BCG, measles and three doses of DPT and polio vaccine (excluding polio 0)

†Diarrhoea includes blood also

***Includes children who were below –3 SD from the International Reference Population Median.

Sources: International Institute for Population Sciences (IIPS). National Family Health Survey India, 1992–93 (NFHS-1), International Institute for Population Sciences, Mumbai, 1995
International Institute for Population Sciences (IIPS) and ORC Macro. National Family Health Survey (NFHS-2), India, 1998–99. Mumbai: IIPS; and Maryland, USA: ORC Macro; 2000

Table 7. Description of various characteristics of fertlity, family planning, nuitritional and reproductive health problems among mothers, India

Characteristic	NFHS round	All	Place of residence		Mother's education				Caste			Standard of living index		
			Urban	Rural	Illiterate	Literate, <middle school complete	Middle school complete	High school complete and above	SC	ST	OBC/ others	Low	Medium	High
Median age at first marriage among women age 20–49 years														
Current age														
20–49 years	1	16.1	18.1	15.5	15.0	16.8	18.4	21.3	15.0	15.8	16.3	—	—	—
	2	16.4	18.4	15.8	—	—	—	—	—	—	—	—	—	—
Total fertility rate														
During three years preceding the survey	1	3.4	2.7	3.7	4.0	3.0	2.5	2.2	3.9	3.6	3.3	—	—	—
	2	2.9	2.3	3.1	3.5	2.6	2.3	2.0	3.2	3.1	—	3.4	2.9	2.1
Contraceptive method														
Percentage of currently married women	1	36.3	45.3	33.1	31.5	44.8	42.4	45.0	31.7	30.8	37.6	—	—	—
using any modern method	2	42.8	51.2	39.9	39.2	49.7	44.6	47.1	40.1	35.2	44.7	35.5	43.35	3.1
Percentage of ever married women with BMI below 18.5 kg/m²														
Among ever married women[2]	2	35.8	22.6	40.6	42.6	32.6	28.0	17.8	42.1	46.3	32.9	48.1	35.6	17.3
Percentage of having iron-deficiency anaemia by degree of anaemia														
Percentage of ever married women with any anaemia	2	51.8	45.7	53.9	55.8	50.1	48.0	40.3	56.0	64.9	49.0	60.2	50.3	41.9
Symptoms of reproductive health problem														
Percentage of ever married women reporting any abnormal vaginal dischage or symptoms of urinary tract infection[1]	2	35.5	33.1	36.4	37.3	36.2	34.3	28.0	36.1	39.2	35.1	37.4	36.3	30.6
Percentage of currently married women reporting any reproductive health problem	2	39.2	36.7	40.1	40.8	39.9	38.6	32.4	39.9	42.0	38.7	41.3	40.1	34.0
Home visits by a health or famiy planning worker														
Percentage of ever married women who had at least one visit in the 12 months preceding the survey	2	13.0	10.0	14.0	11.5	15.9	17.0	12.6	13.4	17.9	12.4	14.2	13.3	10.3
*Percentage who know two or more signs for medical treatment of diarhoea**														
Among mothers with births during the three years preceding the survey	2	37.1	37.1	37.1	34.9	38.3	42.1	41.4	37.5	35.1	37.2	—	—	—
Percentage of mothers with births who know about ORS packets														
At least one birth during the four years preceding the survey	1	42.7	55.6	38.9	31.8	56.4	62.7	75.4	35.3	26.8	45.9	—	—	—
At least one birth during the three years preceding the survey	2	62.4	75.8	58.6	51.2	72.3	77.2	86.5	59.3	51.3	64.8	—	—	—
Reported quantity (of ORS) to be given to drink during diarrhoea														
More amount	2	29.4	36.8	27.3	22.2	32.0	51.1	26.5	22.0	31.3	—	—	—	—

*Diarrhoea includes blood also

[1]Includes pain or burning while urinating or more frequent or difficult urination. [2] Excluded women who were pregnant and women with a birth in the preceding two months

Sources: International Institute for Population Sciences (IIPS). National Family Health Survey India, 1992–93 (NFHS-1), International Institute for Population Sciences, Mumbai, 1995
International Institute for Population Sciences (IIPS) and ORC Macro. National Family Health Survey (NFHS-2), India, 1998–99. Mumbai: IIPS; and Maryland, USA: ORC Macro; 2000

Acute respiratory infection

Small variations in the prevalence of ARI by most of the background characteristics was observed. ARI was somewhat more common among children living in rural areas than those living in urban areas. Children of mothers with at least middle school education seemed to have a lower occurrence of ARI than children of those educated below middle school. The prevalence of ARI was higher among children in ST households and those having a low standard of living. The percentage of children with ARI taken to a health facility or provider has a strong positive relationship with the mother's educational attainment and household standard of living. By caste/tribe, this percentage is lower for SC and ST than other backward classes or 'others'. By place of residence, urban children were taken more often to a health facility or provider than rural children.

Low birth weight

The NFHS-1 and NFHS-2 had taken the perceived size of the baby (large/very large, average and small/very small), as the proxy for birth weight, and provided estimates of NMR, PNMR and IMR according to the size of the baby. The percentage of children with a small size or who were very small at birth was found to be higher among mothers belonging to socioeconomically disadvantaged households than among socioeconomically advantaged ones.

Nutritional status of the children

Nutrition has an important bearing on maternal and child health. Inadequate or unbalanced diets and chronic illness are associated with poor nutrition among children. Diarrhoea and dehydration may be underlying causes of death among children who have low levels of nutrition than among better-nourished children. Malnutrition is precipitated and aggravated by diarrhoea through inadequate absorption of nutrients from the food. The proportion of children under 4 years of age in NFHS-1 and under 3 years of age in NFHS-2 who were severely undernourished was 20.6% (according to weight for age) and 18.0%, respectively. Further, anaemia among young children can result in impaired cognitive performance, behavioural and motor development and scholastic achievement, as well as increased morbidity from infectious diseases (Seshadri 1997). According to NFHS-2, among children 6–24 months of age, 74% had some level of anaemia; 23% were mildly anaemic, 46% were moderately anaemic and 5% severely anaemic, confirming the findings of Stoltzfus and Deyfuss (1998) that one of the most vulnerable groups among children is the age group of 6–24 months. Undernutrition and a high level of anaemia were found to be higher among rural children, children whose mothers were illiterate, children belonging to the SC and ST and poor children.

Family planning

The reduction in maternal and child mortality can be achieved by promoting family planning spacing methods, which help in reducing high-risk pregnancies (pregnancy at younger ages and closely spaced pregnancies), while terminal methods, which greatly help in reducing pregnancies of a higher order and in older women. Comparison of the results of the NFHS-2 with NFHS-1 for current contraceptive use revealed an 18% increase in the use of modern methods of contraceptives in 6–7 years' time. A low rate of increase between the two surveys could be due to a shift from traditional target systems of family planning to a target-free approach and integrating such services into health programmes. The percentage of couples currently using contraceptives was found to increase with education and standard of living; it was lowest among women from the SC and highest among women who did not belong to the SC and ST; and higher among urban women as compared to rural women.

Nutritional status of women

The nutritional status of and level anaemia in children were strongly related to the nutritional status of and level of anaemia in the mother. The NFHS-2 provided information on body mass index (BMI), a measure of nutritional status, and prevalence of anaemia among ever-married women 15–49 years of age. The BMI, which is used to assess both thinness and obesity (defined as the weight in kilograms divided by the height in meters squared [kg/m^2]), was obtained for ever-married women who were non-pregnant at the time of the survey and those who had not given birth during the two months preceding the survey. Chronic energy deficiency is usually indicated by a BMI of less than 18.5. Thirty-six per cent of women had a BMI below 18.5, indicating a high prevalence of nutritional deficiency.

Anaemia among women may have detrimental effects on their health and that of their children, and may become an underlying cause of maternal and perinatal mortality. Anaemia results in an increased risk of premature delivery and low birth weight. The overall prevalence of anaemia among women, as reported in NFHS-2, was 52%. The prevalence of mild, moderate and severe anaemia was, respectively, 35%, 15% and 2%. The prevalence of anaemia and low BMI were both considerably higher for rural women, for SC/ST women and inversely related to educational level and SLI. Though differences in the prevalence of anaemia by background characteristics was observed, it was substantial for every population group.

Disadvantaged and advantaged groups

Inequalities in child health are well documented in NFHS-1 and NFHS-2. Data reveal that disadvantaged/vulnerable groups are less likely to receive child survival interventions

that can prevent the most common diseases. It is well-documented (Victora *et al*. 2003) that children from poor households are more likely to be exposed to many disease agents and have lower resistance to those risks and become sick vis-à-vis children from richer households.

The disadvantaged group consists of people belonging to the lowest socioeconomic rungs of society. They have low levels of literacy, a low standard of living, are less exposed to media, and thus have low concern about health care. This gets reflected in under-usage of proper medical services, low nutritional imbalance and poor personal hygiene. Thus higher rate of mortality among the disadvantaged may be due to inequalities in coverage of preventive interventions. These reasons may also be responsible for the slowing down/stagnation of the decline in infant and child mortality. The differentials in the level of child mortality within and between States may be due to varying proportions of disadvantaged groups. Therefore, further reduction in IMR and CMR can be realized by improving the delivery of RCH services in communities with a relatively high IMR. A healthy child requires many coordinated preventive and therapeutic interventions and these demand renewed action.

Projection

The National Population Policy (2000) has set the goal to reduce the IMR to less than 30 by the year 2010. This necessitates projecting the IMR and U5MR for India up to 2016. Assuming that in the immediate future, the mortality will continue to follow the prevailing trend, the projected figures and values of IMR and U5MR up to 2016 for India are presented in Figs 5 and 6 (Tables 8 and 9), respectively. It appears that without further intervention, India will not be able to achieve the set target of an IMR of less than 30 by 2010. However, despite the inherent limitations of SRS data, the declining trend observed for IMR in the present study can be considered valid.

Mortality forecasts largely depend on the factors associated with child mortality. Therefore, one way to improve the mortality forecasts is to gain insight into the causes and predictors of mortality. If the risk profile of the current cohort compared to previous cohorts is determined, then the forecasts will improve. Mortality projections based on several covariate alternatives should be obtained. These alternatives should cover a realistic future-related range of variation in programme-related aspects. However, the number of alternatives should not be so large that it creates confusion and complications in using the mortality projections.

Conclusion

The foregoing analysis shows that the under-five mortality in India and its States has declined during the period 1978–2002. However, the pace of decline has not been constant, it has sometimes been slow and stagnated, and at times even increased. Such a scenario generates curiosity to unearth the factors that might have played a role in the decline of child mortality in the country and in some of the major States. Disparities observed in child mortality might be explained, to some extent, by the differences in the socioeconomic status of mothers and households. Analysis of two rounds of the NFHS-1 and NFHS-2 depicted an inverse relationship between child mortality and socioeconomic factors including education of mother and standard of living of the household. However, a keen observation of child mortality data revealed that the differences in child mortality continued even after controlling for the effect of socioeconomic status of the household. Such a scenario necessitates examination of the differences in child mortality among the socially and economically vulnerable/disadvantaged groups. We can attribute the differences in child mortality to complex set of social, economical and biological factors affecting the same. In addition, the decline in child mortality could also be a result of programme factors such as public health

Fig. 5 Forecast of infant mortality rate of India for 2003–2016

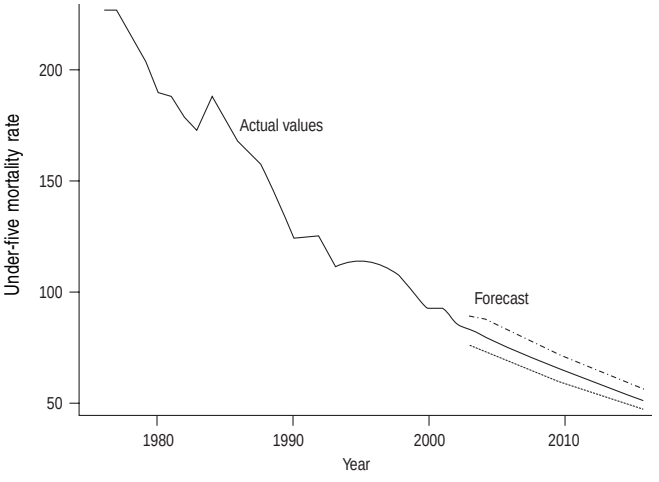

Fig. 6 Forecast of under-five mortality rate of India for 2003–2016

Table 8. Predicted values of infant mortality rates and associated 95% confidence intervals using the ARIMA model by States and India, 2003–2016

State	Fitted ARIMA model	IMR Values	Years													
			2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016
Andhra Pradesh	(0,1,1)	Predicted	59	58	56	55	53	51	50	49	47	46	45	43	42	41
		LCL	53	48	45	42	39	37	35	33	31	30	28	27	25	24
		UCL	66	69	70	71	72	72	72	72	71	71	70	70	9	69
Assam	(0,1,1)	Predicted	69	67	66	65	64	62	61	60	59	57	56	55	54	53
		LCL	61	57	55	52	50	48	46	44	42	41	39	38	37	36
		UCL	78	79	80	81	81	81	1	81	81	81	0	80	80	9
Bihar	(0,1,1)	Predicted	57	56	54	53	51	50	49	47	46	45	44	43	41	40
		LCL	50	46	43	40	38	35	34	32	30	29	27	26	25	24
		UCL	65	67	69	70	70	70	70	70	70	70	70	69	69	68
Gujarat	(0,1,1)	Predicted	58	56	54	52	50	49	47	46	44	43	41	40	39	37
		LCL	51	48	45	42	40	38	36	34	32	31	29	28	27	25
		UCL	65	65	65	65	64	63	0	61	60	9	58	57	6	55
Haryana	(0,1,1)	Predicted	61	60	59	57	56	55	53	52	51	50	49	48	47	46
		LCL	54	53	51	49	48	47	45	44	43	41	40	39	38	37
		UCL	69	68	67	66	65	64	63	62	61	60	59	58	57	56
Himachal Pradesh	(0,1,1)	Predicted	51	49	48	47	45	44	43	42	41	40	39	37	36	35
		LCL	42	39	36	34	32	30	28	27	25	24	23	22	21	20
		UCL	61	63	64	64	65	65	65	65	65	65	65	65	64	64
Karnataka	(0,1,1)	Predicted	54	54	53	52	51	50	49	48	48	47	46	45	45	44
		LCL	47	45	43	41	39	38	36	35	34	33	32	31	30	29
		UCL	62	64	65	66	66	67	67	67	67	67	67	67	67	67
Kerala	(0,1,1)	Predicted	10	9	9	8	8	7	7	6	6	6	5	5	5	4
		LCL	8	7	6	6	5	5	4	4	4	3	3	3	3	3
		UCL	13	12	12	12	11	11	10	10	10	9	9	8	8	8
Madhya Pradesh	(0,1,1)	Predicted	81	79	77	75	74	72	70	69	67	66	64	63	62	60
		LCL	74	72	70	68	66	64	62	61	59	57	56	54	53	51
		UCL	87	86	85	83	82	81	80	78	77	76	74	73	72	71
Maharashtra	(0,1,1)	Predicted	42	41	40	38	37	36	35	34	33	32	32	31	30	29
		LCL	37	36	35	34	33	32	31	30	29	28	28	27	26	25
		UCL	48	47	45	44	43	42	40	39	38	37	36	35	34	33
Orissa	(0,1,1)	Predicted	86	85	84	82	80	78	77	75	74	72	70	69	68	66
		LCL	80	78	76	75	73	71	70	68	67	66	64	63	61	60
		UCL	94	93	92	90	88	86	84	83	81	79	77	76	74	73
Punjab	(0,1,1)	Predicted	49	47	46	45	43	42	41	40	38	37	36	35	34	33
		LCL	42	40	38	36	35	33	32	30	29	28	27	26	25	24
		UCL	56	56	55	55	54	53	52	51	51	50	49	48	47	46
Rajasthan	(0,1,1)	Predicted	76	74	72	71	69	67	66	64	63	61	60	58	57	56
		LCL	65	61	57	54	52	49	47	45	43	41	39	38	36	35
		UCL	89	90	91	92	92	92	92	92	92	91	91	90	90	89

(Cont.)

Table 8 (*cont.*). Predicted values of infant mortality rates and associated 95% confidence intervals using the ARIMA model by States and India, 2003–2016

State	Fitted ARIMA model	IMR Values	Years													
			2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016
Tamil Nadu	(0,1,1)	Predicted	43	42	41	39	38	37	36	34	33	32	31	30	29	28
		LCL	39	37	35	34	32	31	29	28	27	26	24	23	22	22
		UCL	48	47	47	46	45	44	43	42	41	40	39	38	38	37
Uttar Pradesh	(0,1,1)	Predicted	77	74	72	70	68	66	64	62	60	58	57	55	53	52
		LCL	70	66	62	59	56	53	51	48	46	44	42	41	39	37
		UCL	84	84	84	83	82	81	80	79	78	7	75	74	73	71
West Bengal	(0,1,1)	Predicted	47	46	44	43	42	41	40	39	37	36	35	34	34	33
		LCL	43	42	40	39	38	37	36	35	34	33	32	31	31	30
		UCL	51	50	48	47	46	45	43	42	41	40	39	38	37	36
India	(0,1,1)	Predicted	61	60	58	56	55	53	52	50	49	48	46	45	44	42
		LCL	58	55	53	51	49	47	45	44	42	41	39	38	37	35
		UCL	65	64	63	62	61	60	59	58	57	55	54	53	52	51

ARIMA: autoregressive integrated moving averages; LCL: lower confidence level; UCL: upper confidence level

Table 9. Predicted values of under-five mortality rates and associated 95% confidence intervals using the ARIMA model, India, 2003–2016

Fitted ARIMA model	U5MR values	Year													
		2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016
(0,1,2)	Predicted	82	81	78	75	72	69	67	64	62	60	58	55	53	51
	LCL	76	74	71	68	66	64	61	59	57	55	53	51	49	47
	UCL	89	88	85	82	79	76	73	70	68	65	63	61	58	56

ARIMA: autoregressive integrated moving averages; LCL: lower confidence level; UCL: upper confidence level

reforms, access and quality of health care services and community mobilization. Several studies indicate that health care interventions are determinants of the variations in infant and child mortality.

The trend in IMR for the country over the past 25 years revealed that the share of neonatal morality has remained more or less constant but an appreciable decline is observed in case of post-neonatal mortality. Usually any intervention programme targeted at reducing child mortality shows its impact first on post-neonatal mortality and later on early and late neonatal mortality. This is expected as most of our programmes, such as the EPI (Expanded Immunization Programme) started in 1978 to combat infectious diseases such as polio, etc. among children were targeted at reducing child mortality during the post-neonatal stage. Thus, new intervention programmes need to focus more on lowering neonatal mortality. This could be done to a large extent by strengthening antenatal, natal and delivery care interventions. During the 1970s, 40% of neonatal deaths in rural areas and 25% in urban areas were due to tetanus. By focusing on strategies to increase institutional deliveries, births attended by trained birth attendants and coverage of two doses of TT immunization to pregnant mothers, deaths due to tetanus have declined from 14% in 1978 to less than 5% in the early 1990s and 1% in the late 1990s.

Further, a rapid decline observed in IMR and under-five mortality during 1980–90 was followed by a period of stagnation from 1993, as it was hovering around 72 per thousand live-births (GOI 2000). The stagnation during this period may indicate that the programmes addressing reduction in child mortality were not effective in reducing the IMR as a large proportion of infants were dying in the neonatal stage. Thus, programmes such as RCH Programme, immunization programme and ICDS were not really oriented towards capturing infants dying during the neonatal stage. The other reason for the stagnation in IMR could be the lack of access to health and other types of services by disadvantaged/vulnerable groups.

The IMR and child mortality projected up to 2016 show that India might not be able to achieve the set target of an IMR of 30 by 2010 without making concerted efforts to improve the content and quality of RCH services and concentrate on community mobilization strategies. In addition, economic and social reforms should be commensurate with programme interventions bringing about an appreciable reduction in IMR and child mortality in the near future.

References

Anandiah R, Choe MK. Are the WHO guidelines on breastfeeding appropriate for India. *National Family Health Survey Subject Reports No. 16*, Mumbai: International Institute for Population Sciences; Honolulu: East West Center; 2000.

Black RE, Morris SS, Bryce J. Child survival I: Where and why are 10 million children dying every year. *The Lancet* 2003;**36:**2226–34.

Boerma JT, Bicego GT. Preceding birth interval and child survival: Searching for pathways of influence. *Studies in Family Planning* 1992;**23:**243–56.

Burkhalter BR, Galway SO, Rustein SO, Smith SM. Incidence of high-risk births in seven developing countries, Rosslyn, Virginia: Center for International Health Information; 1991.

Claeson M, Bos ER, Mawji T, Pathmathan I. Reducing child mortality in India in the new millennium. *Bulletin of the World Health Organization* 2000;**78:**1192–9.

Claeson M, Bos E and Pathmanathan. *Reducing child mortality*. The International Bank for Reconstruction and Development, The World Bank, Washington 1999, ISBN 1-932126-27-9.

Cox DR. Regression models and life tables. *Journal of the Royal Statistical Society (Series B)* 1972;**34:**97–9.

Das NP, Dey D. Understanding the causative factors behind stalling of infant mortality in India during the recent period. *Demography India* 2003;**32:**249–74.

DWCS & UNICEF (2001), *Multiple Indicator Survey*, 2000, UNICEF, New Delhi.

Fauveau V, Koenig M, Chakraborty J, Chowdhury A. Causes of maternal mortality in rural Bangladesh: 1976–1985. *Bulletin of the World Health Organization* 1988;**66:**643–51.

Govindasamy P, Ramesh BM. Maternal education and the utilization of maternal and child health services in India. *National Family Health Survey Subject Report No.5*. Mumbai: International Institute for Population Sciences; and Calverton, Maryland: Macro International; 1997.

Govindasamy P, Stewart MK, Rutstein SO, Boerma JT, Sommerfelt AE. High-risk births and maternity care. DHS Comparative Studies, No 8. Columbia, Maryland: Macro International Inc:, 1993.

Government of India. *Family Welfare Programme in India, Year Book, 1996–97*. New Delhi: Ministry of Health and Family Welfare (MOHFW), Department of Family Welfare; 1998.

Government of India. *Newborn health: Key to child survival*. New Delhi: Child Health Division, Department of Family Welfare; Ministry of Health and Family Welfare; 2000.

GOI. Department of Women and Child Development United Nations Children's Fund. *Multiple Indicator Survey (MICS-2000)*. India Summary Report, December 2001.

Hill K, Pandey R, Jone G. *Trends in the infant and child mortality, 1960–65*. New York: UNICEF; 1997.

Hobcraft J. Does family planning save children's lives? Paper presented at the International Conference on Better Health for Women and children through Family Planning, Nairobi, Kenya, October: 5–9, 1987.

Institute for Research in Medical Statistics. An examination of WHO Guidelines for Exclusive Breastfeeding in relation to Child Survival in India, IRMS, ICMR, New Delhi, 2004.

International Institute for Population Sciences (IIPS). *National Family Health Survey India, 1992–93 (NFHS-1)*. Mumbai: International Institute for Population Sciences; 1995.

International Institute for Population Sciences (IIPS) and ORC Macro. *National Family Health Survey (NFHS-2)*, India, 1998–99. Mumbai: IIPS; and Maryland, USA: ORC Macro; 2000.

Khasakhala AA. Effect of maternal education on infant survival in rural Kenya. *Demography India* 2003;**30:**93–106.

Measham A, Rao KD, Jamison DT, Wang J, Singh A. *The performance of India and Indian states in reducing infant mortality and fertility, 1975–1990*. Report prepared for the World Bank. *Economic and Political Weekly* 1999;**34:**1359–67.

Mosley WH, Chen LC. An analytical framework for the study of child survival in developing countries. In: Mosley WH, Chen LC (eds). *Child survival strategies for research. Population and Development Review* 1984;**10:**24–5.

National Health Policy. Department of Family Welfare, Ministry of Health & Family Welfare, Government of India, New Delhi, 2002.

National Population Policy, Department of Family Welfare, Ministry of Health & Family Welfare, Government of India, New Delhi, 2000.

Pandey A, Choe MK, Luther NY, Sahu D, Chand J. *Infant and child mortality in India*. National Family Health Survey Subject Reports No. 11, International Institute for Population Sciences, Mumbai, India and East-West Center Program on Population, Honolulu, Hawaii, USA, 1998.

Pandey A, Bhattacharya BN, Sahu D, Sultana R. Are too early, too quickly and too many births the high risk births: An analysis of infant mortality in India using National Family Health Survey, *Demography India* 2004;**33:**127–56.

Rama Rao S, Pandey A, Shajy KI. Child mortality in Goa: A cross–sectional analysis. *Social Biology* 1997;**44:**101–10.

Registrar General of India. Sample Registration System. Annual Reports. New Delhi: Office of the Registrar General, 1977–2002.

Seshadri S. Nutritional anaemia in South Asia. In: Gillespie G (ed). *Malnutrition in South Asia: A regional profile.* Kathmandu: Regional Office for South Asia, UNICEF; 1997.

Stolfus RJ, Deyfuss ML. *Guidelines for the use of iron supplements to prevent and treat iron deficiency anaemia*. International Nutritional Anemia Consultative Group. Washington, DC: International Life Sciences Institute Press; 1998.

Usha R. Emerging issues from declining fertility in India. In: Roy TK, Guruswamy MK, Arokiasamy P (ed). *Population, health and development in India: Changing perspective*. New Delhi: Rawat Publisher; 2004:291–322.

Victora CG, Wagstaff A, Schellenberg JA, Gwatkin D, Claeson M, Habicht J. Applying an equity lens to child health and mortality: More of the same is not enough. *Lancet* 2003;**362:** 24–32.

Appendix 1

Table A1.1 Stillbirth rate and components of infant mortality rate, India, 1970–2002

	Stillbirth rate			Early neonatal mortality rate			Neonatal mortality rate			Postneonatal mortality rate		
Year	Total	Rural	Urban	Total	Rural	Urban	Total	Rural	Urban	Total	Rural	Urban
1970	NA	NA	NA	NA	NA	NA	69	72	46	61	64	45
1971	18	18	13	37	39	23	75	81	45	54	57	37
1972	18	19	14	33	35	23	72	77	45	68	73	40
1973	18	18	15	34	36	23	68	72	48	66	71	41
1974	17	18	13	36	38	24	70	76	41	56	60	33
1975	18	19	13	38	41	24	78	84	46	62	67	38
1976	18	19	11	50	59	33	77	83	49	52	56	31
1977	16	17	9	49	54	27	80	88	42	50	52	39
1978	15	16	10	48	53	23	77	85	38	50	52	36
1979	13	13	9	47	50	30	72	78	42	48	52	30
1980	11	12	8	45	48	28	69	76	39	45	48	26
1981	11	11	6	44	48	25	70	76	39	41	44	24
1982	9	10	5	45	48	28	67	73	39	38	41	26
1983	9	9	8	45	49	27	67	74	39	38	40	27
1984	10	11	8	44	48	28	66	72	40	38	41	26
1985	10	11	9	38	42	22	60	67	33	37	40	26
1986	10	11	9	38	42	24	60	66	36	37	39	26
1987	13	14	10	38	41	23	58	64	33	38	41	27
1988	14	14	12	37	40	23	57	62	35	38	40	28
1989	13	13	11	35	38	20	56	62	31	35	36	26
1990	12	12	11	37	40	23	53	57	31	27	29	20
1991	11	11	10	36	39	23	51	55	32	29	31	21
1992	12	12	10	36	39	24	50	54	33	29	31	20
1993	11	11	9	34	38	22	47	52	28	26	28	16
1994	9	7	15	34	36	24	48	52	33	26	28	20
1995	9	9	9	36	39	23	48	52	29	26	28	19
1996	9	9	9	35	37	23	47	50	28	25	27	17
1997	9	9	9	35	38	20	46	51	26	25	26	19
1998	9	9	8	34	37	22	45	49	27	27	28	18
1999	10	11	8	34	37	22	45	49	28	24	26	16
2000	8	9	7	32	35	19	44	49	27	23	25	16
2001	9	10	8	27	30	17	40	44	25	26	28	17
2002	9	9	7	25	29	16	40	44	24	24	26	16

Source: Sample Registration System, 1970–2002

Table A1.2 Selected indicators of under-five mortality by residence, India, 1970–2002

	Infant mortality rate			Child mortality rate ($1000*{}_4q_1$)			Under-five mortality rage ($1000*{}_5q_0$)		
Year	Total	Rural	Urban	Total	Rural	Urban	Total	Rural	Urban
1970	129	136	90	122	137	66	235	255	150
1971	129	138	82	117	127	74	231	247	150
1972	139	150	85	131	144	71	252	272	150
1973	134	143	89	113	124	63	232	250	146
1974	126	136	74	111	123	59	223	242	128
1975	140	151	84	119	132	69	243	263	147
1976	129	139	80	113	121	64	227	243	139
1977	130	140	81	111	124	50	227	247	127
1978	127	137	74	102	114	54	216	236	124
1979	120	130	72	98	110	44	206	226	113
1980	114	124	65	86	96	43	190	208	106
1981	110	119	63	87	98	37	188	205	97
1982	105	114	65	83	96	37	179	199	100
1983	105	114	66	76	86	39	173	190	102
1984	104	113	66	93	107	47	188	208	110
1985	97	107	59	87	100	42	176	196	99
1986	96	105	62	80	91	40	168	186	100
1987	95	104	61	74	86	28	162	181	87
1988	94	102	62	67	70	29	154	165	90
1989	91	98	58	53	62	25	140	154	81
1990	80	86	50	48	55	24	124	136	73
1991	80	87	53	49	54	25	125	136	77
1992	79	85	53	50	56	24	125	136	75
1993	74	82	45	41	47	21	112	125	65
1994	74	80	52	42	47	25	113	123	76
1995	74	80	48	44	49	26	114	125	72
1996	72	77	46	44	50	24	113	123	69
1997	71	77	45	42	47	19	110	121	64
1998	72	77	45	38	44	18	107	117	62
1999	70	75	44	29	36	14	97	109	57
2000	68	74	43	27	32	14	93	103	56
2001	66	72	42	28	33	13	93	102	54
2002	63	69	40	24	27	11	85	94	50

Source: Sample Registration System, 1970–2002

Table A1.3 Percentage of early neonatal deaths among neonates, percentage of neonatal deaths among infants and percentage of infant deaths among under-five deaths, India, 1970–2002

	Early neonatal mortality as a percentage of NMR			NMR as a percentage of IMR ($1000*{}_4q_1$)			IMR as a percentage of under-five mortality		
Year	Total	Rural	Urban	Total	Rural	Urban	Total	Rural	Urban
1970	NA	NA	NA	53.1	53.2	51.1	54.9	53.4	60.0
1971	48.6	48.5	50.7	58.3	58.4	55.4	55.9	55.8	54.8
1972	46.8	46.3	50.2	51.5	51.1	52.8	55.3	55.2	56.8
1973	49.5	49.9	47.7	50.9	50.4	53.4	57.7	57.3	60.9
1974	50.9	50.0	58.8	55.6	55.7	55.4	56.5	56.2	57.7
1975	48.9	48.6	51.7	55.9	55.8	55.0	57.7	57.4	57.0
1976	65.2	71.1	67.3	59.7	59.7	61.3	56.8	57.1	57.7
1977	61.0	60.9	64.1	61.7	62.9	51.9	57.3	56.7	63.6
1978	61.9	61.9	61.7	60.9	62.2	51.4	58.7	58.1	59.8
1979	65.5	65.0	70.5	59.8	59.9	58.7	58.3	57.5	63.8
1980	64.8	64.1	70.6	60.8	61.0	60.0	59.9	59.6	61.8
1981	63.6	63.4	66.1	63.3	63.5	61.6	58.9	58.1	64.2
1982	67.0	66.4	72.3	63.6	64.1	59.5	58.6	57.2	65.4
1983	66.5	66.2	69.3	64.1	64.7	59.7	60.8	59.9	64.6
1984	66.7	66.2	70.6	63.3	63.7	60.1	55.5	54.5	60.1
1985	63.4	63.1	65.1	61.8	62.5	56.5	55.3	54.3	59.7
1986	64.0	63.7	66.1	62.0	62.6	58.4	57.3	56.3	62.2
1987	65.3	65.0	68.5	60.7	61.2	54.6	58.5	57.4	69.9
1988	64.4	64.1	66.7	60.4	60.8	55.8	60.9	62.0	69.2
1989	62.0	61.7	63.8	62.0	63.4	54.1	65.2	63.7	71.3
1990	70.5	70.2	75.3	65.6	66.7	61.8	64.6	63.2	68.6
1991	69.8	69.7	72.4	63.9	63.7	60.8	64.1	63.9	68.7
1992	72.6	72.3	73.2	63.3	63.1	62.3	63.3	62.4	70.4
1993	72.3	71.7	77.1	63.6	63.8	63.1	65.9	65.5	69.3
1994	71.1	69.9	73.2	64.5	65.0	62.7	65.4	65.1	68.7
1995	74.4	74.3	77.9	64.9	65.0	60.4	64.6	64.1	66.2
1996	75.1	74.7	82.9	65.3	64.9	60.9	63.6	62.4	66.9
1997	75.7	74.6	78.0	64.8	66.2	57.8	64.8	63.8	70.8
1998	75.6	75.5	81.5	62.5	63.6	60.0	67.4	65.7	72.4
1999	75.6	75.5	78.6	64.3	65.3	63.6	71.9	69.0	77.3
2000	72.7	71.4	70.4	64.7	66.2	62.8	72.7	71.6	76.7
2001	67.5	68.2	68.0	60.6	61.1	59.5	71.3	70.4	77.2
2002	67.5	65.9	66.7	63.5	63.8	60.0	74.1	73.4	80.0

Calculated values

Table A1.4 Percentage distribution of mothers according to selected characteristics, NFHS-1 and NFHS-2

Characteristics	NFHS round	Place of residence		Mother's education			Caste		Standard of living index		
		Rural	Urban	Illiterate	Literate, <middle school complete	Middle school complete and above	SC/ST	OBC/ Others	Low	Medium	High
Percentage of mothers who received any iron–folic acid tablets or syrups during pregnancy	1	53	47	43	57	70	54	46	51	49	59
	2	53	76	42	71	83	54	60	47	60	79
Percentage of mothers who received 3 or more antenatal check-ups during pregnancy	1	37	66	29	57	79	32	48	28	66	69
	2	37	69	26	55	77	35	48	31	45	71
Percentage of babies having very small/small size at time of birth	1	22	20	22	23	26	21	21	22	20	25
	2	25	22	26	25	19	26	24	26	25	19
Percentage of mothers received 2 or more tetanus toxoid injections during pregnancy	1	48	73	40	68	86	41	59	39	75	78
	2	63	82	54	78	88	59	71	56	69	87
Percentage of mothers whose age was <20 years at time of birth	1	22	16	23	23	14	23	20	23	18	19
	2	25	18	24	29	17	25	23	25	25	15
Percentage of births of order two or more and the length of the preceding birth interval <24 months	1	38	35	40	35	30	39	36	40	33	32
	2	17	17	17	18	14	17	16	16	17	16
Percentage of births of order four or more	1	32	23	38	24	9	34	28	38	19	13
	2	30	19	39	20	7	32	25	36	26	12

Sources: International Institute for Population Sciences (IIPS). National Family Health Survey India, 1992–93 (NFHS-1), International Institute for Population Sciences, Mumbai, 1995

International Institute for Population Sciences (IIPS) and ORC Macro. National Family Health Survey (NFHS-2), India, 1998–99. Mumbai: IIPS; and Maryland, USA: ORC Macro; 2000

Newborn and child health in India: Problems and interventions

SIDDARTH RAMJI

Infant mortality showed an appreciable decline during the 1980s and the early part of the 1990s. Thereafter, its pace of decline has slackened considerably. Earlier declines in the infant mortality rate (IMR) have been largely due to reduction in post-neonatal mortality, with neonatal mortality rates (NMRs) not contributing as substantially. As a result, currently almost two-thirds of the IMR is being contributed by the NMR. Consequently, the focus of child health shifted to neonatal health. This was rightly so, but should not be at the cost of health interventions for children in the age group of 1 month to 5 years. We review the current proportion of child mortality between birth and 5 years in India. The mortality rate in the age group of 0–28 days is about 35/1000 live-births, 1–12 months about 30/1000 live-births and 1–5 years about 26/1000 live-births. Thus, the ratio of neonatal death rate to 1–5-year death rate is about 1.3. In contrast, in most developed countries the ratio is over 10. Thus, while efforts are under way to reduce neonatal mortality in India, it is equally important that the risk of mortality of a child who survives the neonatal period decreases substantially; else there will only be a shift in the burden of death from an early period of infancy to a later part of early childhood.

Why is the NMR still so high?

A review of ages at death during the first 28 days reveals that two-thirds of deaths occur in the first week of life and two-thirds of these within the first 2 days of life (Baseline surveys of Multi-centric Home based Intervention project of the Indian Council of Medical Research [ICMR]). Thus, almost 45% of neonatal deaths take place within 48 hours of birth. The major causes of death during this period are birth asphyxia and trauma, problems related to low birth weight (LBW) (such as hypothermia, respiratory problems, feeding and peripartum infections) and malformations. Most of these problems occur due to inadequate care during the antenatal period and during labour. Inadequate care immediately after birth and inadequate care of LBW infants within the first 48 hours contribute to the rest. Although a significant proportion of women would be categorized as high-risk and identified for institutional delivery, yet over 75% of all births take place in the community and mostly in the hands of unskilled birth attendants with little postpartum care to either the mother or the newborn. Clearly, the intervention package must focus not only on the newborn alone but treat the mother–baby dyad as one. (This would be in tandem with our efforts to reduce the MMR in India.)

What are our options?

We should either push for universalization of institutional delivery for all women, or use an at-risk approach to ensure institutional deliveries for high-risk women and provide a skilled birth attendant in the community for the remaining women. The first option needs to be weighed against the existing capacity of health care institutions (in terms of their numbers and quality of health care provided) both in the public and private sectors, the capacity of the user to pay for these services (especially given the disparity in costs incurred between the two sectors and the per capita income of the users), and the evidence as to the type of skilled birth attendant needed to assist the delivery of low-risk women. In the final analysis, the answer is that this is an intangible solution.

Can we push for the second option?

The answer is probably yes. It should be possible to enhance the capacity of the existing health delivery system to handle this load of deliveries (this may also include a public–private partnership). Besides, if the auxiliary nurse–midwife (ANM) (or the current health worker-female [HW-F]) was to be 'freed' of her non-mother and child health (MCH) activities (which could easily be shifted to other cadres of workers, especially the HW-M, who is currently underutilized and underworked), then she would more likely be able to significantly increase the proportion of births she attends and the postpartum care she offers to the mother and her newborn. In Integrated Child Development Scheme (ICDS)

Department of Paediatrics
Maulana Azad Medical College, New Delhi
e-mail: sumisid@vsnl.com

areas, the *anganwadi* worker (AWW) has it in her charter to provide some care to expectant and lactating mothers and their infants. It is another issue that she does not do so in most places. Implementation of their assigned jobs, with training in identifying and solving some newborn health problems, could augment through home visits the identification of a significant proportion of the 60% of neonates who die after 2 days of life in the community. The pilot Integrated Management of Neonatal and Childhood Illnesses (IMNCI) intervention by health workers in the Border District Cluster Strategy (BDCS) districts in collaboration with the UNICEF has demonstrated that AWWs do have the capacity to provide home-based newborn care and identify most sick newborns.

It is equally important to empower communities, families and the mother, so that they seek and demand care for the mother and her newborn. This stems from the observation that among communities in India women and newborn health is accorded a low priority, especially if the newborn is a female infant. Second, even if families opt to take sick newborns to hospitals, transportation, finances and the poor image of public health institutions hinder the sick neonate from receiving the care it requires.

The choice of the institution where a sick newborn needs to be referred has been a matter of frequent debate. During the Child Survival and Safe Motherhood (CSSM) intervention, operationalization of newborn care was initiated at district level. This included training in newborn care of medical officers (MOs) and nurses at the Primary Health Centre (PHC), first referral unit (FRU) and District Hospitals in 30 districts along with the supply of essential (indigenously manufactured) neonatal care equipment. This project was implemented by the National Neonatology Forum (NNF) with support from the Government of India (GOI). The project monitoring report revealed that at PHCs and FRUs the utilization of neonatal care equipment such as weighing machines, thermometers and warmers was a mere 50% (in some facilities the NNF review staff found the equipment still in their packing cases even after a year). Most of the trained medical staff had been transferred and the new incumbents were unaware of the use of the equipment or the principles of essential newborn care. The situation in District Hospitals was better but still well below optimum in spite of the presence of a specialist paediatrician. During the Reproductive and Child Health (RCH)-I Programme, an essential newborn care package was incorporated for training at all levels. The training and supply of equipment was extended to more districts, the involvement of nurses was augmented and obstetricians were also included in a three-day training package at the District Hospital. Unfortunately, no evaluation of its implementation during RCH-I is available. However, the experience of neonatal care specialists in India, who have been involved with these interventions for almost a decade, suggests that the facility must have a paediatrician and nurses, as well as minimal laboratory facilities if moderately sick newborns are to be treated. At present, in most part of the country, it is either a District Hospital or a similar facility, and in some regions it could be an FRU or a small nursing home/hospital.

There is also a need for some tertiary care facilities which can cater to the needs of very sick newborns. In the governmental health system, medical colleges are expected to play this role. Unfortunately, the state of these institutions is dismal, with respect to availability of manpower (both teaching and paramedical), equipment and other support infrastructure. Even the basic newborn care equipment that is being supplied to District Hospitals/FRUs is not available here. This disparity stems from the fact that central funding from the Department of Family Welfare supports non-teaching health institutions in the State through its national programmes, while the finance-drained State Directorates of Medical Education are expected to fund medical colleges. There is an urgent need to ensure that this anomaly is corrected. This becomes even more urgent now that child health is an independent subject for training and examination at the MB,BS level and 25% of this training is meant for newborn health. If medical colleges are poorly equipped and starved of quality care, the quality and competence of our medical graduate students is bound to be poor and reflect ultimately in the health care provided, and the morbidity and mortality statistics of the nation.

Child health interventions

While a number of vertical programmes have been initiated to address the issue of child health and mortality, it is evident that the success of these programmes has been partial.The immunization programme has been successful in reducing the proportion of vaccine-preventable diseases (VPDs), but the diarrhoea control programme has been only partially effective in reducing the proportion of under-5 mortality. Estimations of the burden of diarrhoeal diseases in India by the National Institute of Cholera and Enteric Diseases (NICED) indicate that diarrhoeal diseases contribute to about 9.1% of deaths in the age group of 0–6 years. It has been further estimated that the years of life lost (YLL) due to diarrhoeal diseases in the 0-6 years' population presently contribute to about 98% of disability-adjusted life-years (DALYs) and would probably remain unchanged over the next decade till 2016. It clearly indicates the need to shift gears to address the projected stagnation in diarrhoea-related mortality among children. However, pneumonia-related deaths, childhood tuberculosis and undernutrition still remain major problems. This reflects the problems in the Acute Respiratory tract Infections (ARI) Control Programme, the Revised National Tuberculosis Control Programme (RNTCP) vis-à-vis childhood tuberculosis (the new RNTCP guidelines for childhood TB have been published only this year and it is hoped that these will fill the existing void) and numerous nutrition-related programmes. Most importantly, all these vertical single-

disease interventions have not taken cognizance of the one fact that sick children (especially those under 5 years of age) invariably suffer from more than one illness and are also often undernourished and unimmunized. There is a need to look at sick children holistically both when they come as outpatients and while treating them as inpatients. The IMNCI package of the WHO attempts to fill this gap and also provides modules for inpatient treatment of childhood illnesses (this component has not been evaluated nor adapted for India). However, one of the major hurdles in its implementation is the training schedule. In the RCH-II, the IMNCI has been incorporated as a major package for intervention. There is a need to evaluate the capacity of the health system to train the enormous cadre of health care providers required. One needs to put into place a full-time nodal agency to oversee the training, follow-up and monitoring of its implementation. The implementation also requires health systems' augmentation; else this effort will also peter out as with all other programmes.

Estimation of the burden of diarrhoeal diseases in India

NATIONAL INSTITUTE OF CHOLERA AND ENTERIC DISEASES, KOLKATA

Precise information about diarrhoea and its incidence, causation, consequences and trend is necessary for informed policy-making. As well informed people demand more health services and intervention than available resources can finance, decision-makers at all levels are increasingly required to identify diseases/health conditions based on sound and uniform methodologies for setting priorities in biomedical research and for rational allocation of limited resources to combat health menaces.

In 1993, the Harvard School of Public Health in collaboration with the World Bank and WHO assessed the global burden of disease (GBD). Aside from generating the most comprehensive and consistent set of estimates of mortality and morbidity by age, sex and region ever produced, GBD also introduced a new metric—disability-adjusted life-year (DALY)—to quantify the burden of disease. The use of DALY allows researchers to combine years of life lost (YLL) from premature death and years of life lived with disabilities into a single indicator.

DALYs for a disease or health condition are calculated as the sum of the YLL due to premature mortality in the population and the equivalent 'healthy' years lost due to disability (YLD) for incident cases of the condition.

To use time as a common currency for non-fatal health states and for YLL due to mortality, we must define, measure and numerically value time lived in the non-fatal health state. The 'valuation' of time lived in the non-fatal health state formalizes and quantifies social preferences for different health conditions as health state weights. Most such weights are measured on a scale of 0 to 1. Because the DALY measures loss of health, the weights are inverted for DALY calculation with 0 representing a state of optimal health (no loss) and 1 representing a state equivalent to death. The disability weights used for calculating DALYs quantify societal preferences for different health states. These weights do not represent the lived experience of any disability or health state, or imply any societal value of the person in a disability or health state. Rather, they quantify societal preferences for health states in relation to the societal 'ideal' of optimal health.

The DALY measures the future stream of healthy YLL due to each incident case of disease or injury. It is thus an incidence-based measure rather than a prevalence-based measure. The GBD applied a 3% time discount rate to YLL in the future to estimate the net present value of years of life lost. With this discount rate, a year of healthy life gained in 10 years' time is worth 24% less than one gained now.

Discounting of future benefits is a standard practice in economic analysis and there are some specific arguments for applying discounting to the DALY in measuring population health.

YLD are the disability component of DALYs. Estimating YLD is the most difficult component of a GBD study. It will frequently require an in-depth understanding of the epidemiology of particular diseases to identify alternative estimation methods and will involve the use of judgement and creativity. The data required to estimate YLD are: incidence of disability, duration of disability, age of onset, and distribution by severity class, all of which must be disaggregated by age and sex. These, in turn, require estimates of incidence, remission, case-fatality rates or relative risks, by age and sex. With zero discounting and uniform age weights, the basic formula for calculating YLD is:

$$YLD = I \times DW \times L$$

where I is the number of incident cases in the reference period, DW is the disability weight (in the range of 0–1) and L is the average duration of disability (in years).

Data needed for estimation of the disease burden are:

(i) General demographic estimates
(ii) Cause-specific mortality proportion (CSMP)
(iii) Descriptive epidemiological information
(iv) Health state valuation in the community

Data sources for estimation of the diseases burden

Demographic data are available from Census of India 2001 and Sample Registration System (SRS). Causes of death are available from Survey of Causes of Death for rural areas (the procedure has been changed at present) and Medical Certification of Causes of Death (MCCD) for urban areas. Descriptive epidemiological data, i.e. incidence/

Table 1. Data used for estimation of the disease burden in India

	0–6 years		>6 years		Total	
	Rural	Urban	Rural	Urban	Rural	Urban
	122,336,460 (16.5% of rural)	35,493,735 (12.4% of urban)	619,323,833 (83.5% of rural)	249,861,219 (87.6% of urban)	741,660,293 (72.2%)	285,354,954 (27.7%)
Total	157,830,195 (15.4% of the total population)		869,185,052 (84.6% of the total population)		1,027,015,247	

Source: Census of India 2001

Table 2. Data used for estimation of burden due to diarrhoea in India

		Projected values		
Indices	Current values (2001)	2001–06	2006–11	2011–16
Total population (in crore)	102.7	109.41 (2006)	117.89 (2011)	126.35 (2016)
Life expectancy at birth (years)				
Male	62.30 (projected)	63.87	65.65	67.04
Female	65.27 (projected)	66.91	67.67	69.18

Source: Registrar General of India 1996

prevalence can be obtained from published/unpublished papers and reports. Summary measure of the disease burden provides an estimate of the disease burden in terms of YLL due to premature mortality, and also YLD due to the disease.

The general demographic data that have been used in the present estimation process (including the estimation of projected values for future years) are presented in Tables 1 and 2.

Data on morbidity, mortality and disability from diarrhoeal diseases

For estimation of the disease burden, incidence rather than prevalence data are useful. However, data on incidence are not easy to obtain, and thus, sometimes we have to depend on prevalence data. Of course, a number of community-based longitudinal studies from different parts of India are available, which show varied incidence data depending on situations in which the estimates were made. In the present estimation, the average value for such incidence data was used.

Data on mortality are required to calculate YLL component of DALYs. Similar to the morbidity data, literature search revealed wide variations in mortality estimates too in different studies and a declining trend over the years. We estimate deaths due to diarrhoea from reported values for age group-specific crude death rate (CDR) and proportionate mortality due to diarrhoea (Table 3).

To assess disability due to diarrhoea, we used the average of the common values reported for the duration of diarrhoea. Compared to morbidity and mortality, fewer studies reported the duration of diarrhoea. Most data on duration were obtained from hospital-based clinical trials, which were conducted under stringent experimental conditions, and thus, the duration is likely to be less than that might occur under normal field conditions. The disability weight was obtained from the GBD estimation sources.

Estimation of YLL due to diarrhoea

YLL = (Number of deaths) × (Life expectancy at age X)

Table 3. Estimation of mortality due to diarrhoea in India

Crude death rate (India, rural)	=	9.3 per 1000 population
Total number of deaths	=	6,897,441
Total deaths in 0–6 years	=	1,517,437 (22% of total rural deaths)
Total deaths in >6 years	=	5,380,004 (78% of total rural deaths)
Crude death rate (India, urban)	=	6.3 per 1000 population
Total number of deaths	=	1,797,736
Total deaths in 0–6 years	=	221,122 (12.3% of total urban deaths)
Total deaths in >6 years	=	1,576,614 (87.7% of total urban deaths)
Total deaths (all ages; rural + urban)	=	8,695,177
Total 0–6 years deaths (rural + urban)	=	1,738,559
Proportionate mortality due to diarrhoea (all ages)	=	5.23% [SBHI, 2002]
Total diarrhoeal deaths (all ages)	=	454,758
Proportionate mortality due to diarrhoea (0–6 years)	=	9.1% [SBHI, 2002]
Total diarrhoeal deaths among 0–6 years	=	158,209
Total diarrhoeal deaths among 6+ years	=	296,549

Sources: Crude death rates. *Sample Registration System Bulletin*. 2001; 32. Age-specific death rates—Sample Registration System, 1998.
Note: The estimated total deaths due to diarrhoea are less than the estimation of 576,480 deaths by Zaidi *et al.* (2004)

Table 4. Life expectancy, India [SRS-based abridged life tables, 1988–92]

Age (years)	Life expectancy (years)
0 (at birth)	61.4
1	64.9
5	62.9
10	58.6
20	49.4
30	40.4
40	31.5
50	23.0
60	15.8
70	10.3

Table 5. Estimation of YLL due to diarrhoea

Age group (years)	Number of deaths due to diarrhoea	Life expectancy (years)	YLL (years)
0–6	158,209	62.9	9,951,346.1
>6	296,549	40.4 (at age 30)	11,980,579.6
Total			21,931,925.7

YLL = (Number of deaths) x (Life expectancy at age X)

Estimation of YLD due to diarrhoea

YLD = (Total number of episodes) × (Duration of each episode) × (Disability weight)

Table 6. Estimation of total diarrhoeal episodes

Area	Age group (year)	Population (2001)	Average estimated incidence (episodes/ person/year)	Total number of episodes (per year)
Rural	0–6	122,336,460	1.71	209,195,347
	>6	619,323,833	0.63	390,174,015
Urban	0–6	35,493,735	1.09	38,688,171
	>6	249,861,219	0.33	82,454,202

Note: The average estimated incidences of diarrhoea in different populations and the average duration of each episode have been obtained by a review of incidence data from published and unpublished literatures (as listed in the Bibliography section) on diarrhoeal morbidity from different States of the country.

Duration of diarrhoeal episodes: 4 days (average) = 0.01096 years

Disability weight for diarrhoea: 0.02–0.12 (average 0.05) [from GBD estimates]

Table 7. Estimation of YLD due to diarrhoea

Area	Age group (years)	Total no. of episodes (per year)	Average duration of episodes (years)	Average disability weight	Estimated YLD (years)
Rural	0–6	209,195,347	0.01096	0.05	114,639.05
	>6	390,174,015	0.01096	0.05	213,815.36
Urban	0–6	38,688,171	0.01096	0.05	21,201.12
	>6	82,454,202	0.01096	0.05	45,184.90

Estimation of DALY due to diarrhoea

DALY = YLL + YLD

Table 8. Estimation of DALY

Age group	YLL (years)	YLD (years)	DALY (years)
0–6 years	9,951,346.1	135,840.2	10,087,186.3
>6 years	11,980,579.6	259,000.3	12,239,579.9
Total	21,931,925.7	394,840.5	22,326,766.2

From the estimated DALYs, it is observed that the YLL and YLD components account for 98.2% and 1.8% of total DALYs, respectively. According to Murray and Lopez (1996), more than 84% of the disease burden (from all diseases) among children in India was estimated to be due to premature mortality.

Projections

Table 9. DALYs lost due to diarrhoea: Projected values*

Year	Total population	0–6 years' population	>6 years' population
2006	1,094,100,000	168,491,400	925,608,600
2011	1,178,900,000	181,550,600	997,349,400
2016	1,263,500,000	194,579,000	1,068,921,000

*Based on the population projections in Table 2

Note: We assume similar age distribution (as shown for 2001) of projected populations.

Table 10. Estimation of projected YLD

Year	No. of episodes in 0–6 years	No. of episodes in 6+ years	Average duration (years)	Average disability weight	YLD in 0–6 years	YLD in 6+ years	Total YLD
2006	264,531,498	499,828,644	0.01096	0.05	144,963.3	273,906.1	418,869.4
2011	285,034,442	538,568,676	0.01096	0.05	156,198.9	295,135.6	451,334.5
2016	305,489,030	577,217,340	0.01096	0.05	167,408.0	316,315.1	483,723.1

Table 11. Estimation of projected YLL

Year	Diarrhoea deaths in 0–6 years	Diarrhoea deaths in 6+ years	Life expectency at 6 years	Life expectency at 30 years	YLL in 0–6 years	YLL in 6+ years	Total YLL
2006	168,896	315,818	62.9	40.4	10,623,544.5	12,759,033.2	23,382,577.8
2011	181,986	340,296	62.9	40.4	11,446,939.6	13,747,942.9	25,194,882.5
2016	195,046	364,716	62.9	40.4	12,268,392.7	14,734,520.1	27,002,912.9

Table 12. Estimation of projected DALY

Year	YLL	YLD	DALY
2006	23,382,577.8	418,869.4	23,801,447.1
2011	25,194,882.5	451,334.5	25,646,217.0
2016	27,002,912.9	483,723.1	27,486,636.0

DALY: disability-adjusted life-year; YLL: years of life lost; YLD: years lost due to disability

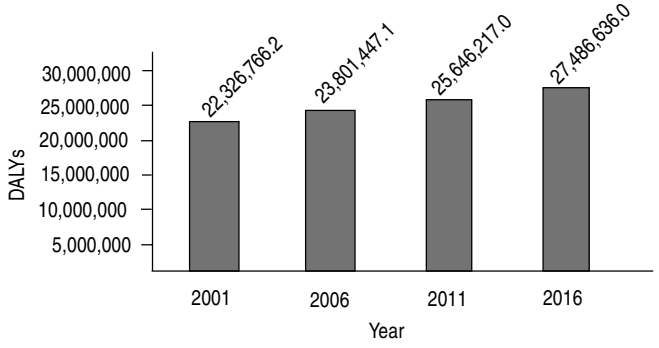

Fig. 1 Diarrhoeal diseases in India: Estimated DALYs lost (current and projected)

DALY: disability-adjusted life-year

References

Census of India, 2001. Available from URL: www.censusindia.net.

Murray CJL, Lopez AD (eds). *The global burden of disease: A comprehensive assessment of mortality and disability from diseases, injuries and risk factors in 1990 and projected to 2020*. Harvard School of Public Health, Harvard University Press; 1996.

Registrar General of India. Population projections for India and states 1996–2016. Report of the Technical Group on Population Projections constituted by the Planning Commission. New Delhi: Government of India; August 1996.

State Bureau of Health Intelligence, 2002. Government of West Bengal.

Zaidi AKM, Awasthi S, deSilva HJ. Burden of infectious diseases in South Asia. *BMJ* 2004;**328:**811–15.

Bibliography

Ali A. The present health scenario in India. *Health Millions* 2000;**26:**4–5.

Anand K, Sundaram KR, Lobo J, Kapoor SK. Are diarrheal incidence and malnutrition related in under five children? A longitudinal study in an area of poor sanitary conditions. *Indian Pediatr* 1994;**31:**943–8.

Awasthi S, Pande VK. Cause-specific mortality in under fives in the urban slums of Lucknow, north India. *J Trop Pediatr* 1998;**44:**358–61.

Ayyagari A, Bhargava A, Agarwal R, Mishra SK, Mishra AK, Das SR, *et al*. Use of telemedicine in evading cholera outbreak in Mahakumbh Mela, Prayag, UP, India: An encouraging experience. *Telemed J E Health* 2003;**9:**89–94.

Badari S, Gopal YS, Devaramani SC. Infant mortality, its components and correlates: Findings from a longitudinal study in rural Karnataka, India. *Genus* 1991;**47:**89–108.

Basu S, Sengupta B, Paladhi PK. Single megadose vitamin A supplementation of Indian mothers and morbidity in breastfed young infants. *Postgrad Med J* 2003;**79:**397–402.

Bhan MK, Bhandari N, Bhatnagar S, Bahl R. Epidemiology and management of persistent diarrhoea in children of developing countries. *Indian J Med Res* 1996;**104:**103–14.

Bhandari N, Bahl R, Mazumdar S, Martines J, Black RE, Bhan MK. Infant Feeding Study Group. Effect of community-based promotion of exclusive breastfeeding on diarrhoeal illness and growth: A cluster randomised controlled trial. *Lancet* 2003;**361:**1418–23.

Bhandari N, Bahl R, Taneja S, Strand T, Molbak K, Ulvik RJ, *et al*. Substantial reduction in severe diarrheal morbidity by daily zinc supplementation in young north Indian children. *Pediatrics* 2002; **109:**e86.

Bhandari N, Bhan MK, Sazawal S. Impact of massive dose of vitamin A given to preschool children with acute diarrhoea on subsequent respiratory and diarrhoeal morbidity. *BMJ* 1994;**309:**1404–7.

Bhandari N, Bhan MK, Sazawal S. Mortality associated with acute watery diarrhea, dysentery and persistent diarrhea in rural north India. *Acta Paediatr* 1992;**381:**111–16.

Bhargava I. Review of mother and child health programmes and targets in light of the national health policy. *ICCW News Bull* 1987;**35:**2–6.

Bhatia V, Swami HM, Bhatia M, Bhatia SP. Attitude and practices regarding diarrhoea in rural community in Chandigarh. *Indian J Pediatr* 1999;**66:**499–503.

Bhattacharya MK, Bhattacharya SK, Dutta D, Deb AK, Deb M, Dutta A, *et al*. Efficacy of oral hypomolar glucose-based and rice-based oral rehydration salt solutions in the treatment of cholera in adults. *Scand J Gastroenterol* 1998;**33:**159–63.

Bhattacharya MK, Ghosh S, Mukhopadhyay AK, Deb A, Bhattacharya SK. Outbreak of cholera caused by *Vibrio cholerae* 01 intermediately resistant to norfloxacin at Malda, West Bengal. *J Indian Med Assoc* 2000;**98:**389–90.

Bhattacharya SK, Bhattacharya MK, Manna B, Dutta D, Deb A, Dutta P, *et al*. Risk factors for development of dehydration in young children with acute watery diarrhoea: A case–control study. *Acta Paediatr* 1995;**84:**160–4.

Bhattacharya SK, Bhattacharya MK, Nair GB, Dutta D, Deb A, Ramamurthy T, *et al*. Clinical profile of acute diarrhoea cases infected with the new epidemic strain of *Vibrio cholerae* O139: Designation of the disease as cholera. *J Infect Dis* 1993;**27:**11–5.

Bhattacharya SK, Goswami AG, Bhattacharya MK, Dutta D, Deb A, Deb M, *et al*. Epidemic of *Vibrio cholerae* 0139 in Calcutta. *Indian J Med Res* 1994;**100:**213–16.

Bhattacharya SK. Progress in the prevention and control of diarrhoeal diseases since Independence. *Natl Med J India* 2003;**16** (Suppl. 2):15–19.

Biswas AB, Basu M, Das KK, Biswas R. (Dr P. C. Sen Award on rural health practice). Infant and early childhood mortality in some rural ICDS blocks of west Bengal. *Indian J Public Health* 1993;**37:**81–6.

Cause of Death. Institute of Health Systems, Hyderabad, India. Available from URL: www.ihsnet.org.in.

Choudhary SR, Jayaswal ON. Infant and early childhood mortality in urban slums under ICDS scheme—a prospective study. *Indian Pediatr* 1989;**26:**544–9.

Crook N, Malaker CR. Child mortality in new industrial localities and opportunities for change: A survey in an Indian steel town. *Health Transit Rev* 1992;**2:**165–76.

Diarrhoeal Diseases Control Programme. *Diarrhoea Dialogue* 1980;**1:**6.

Dutta D, Bhattacharya MK, Deb A, Chowdhury AS, Nair GB, Ramakrishna BS, *et al*. Uncooked rice powder in oral rehydration solution: An alternative to glucose or cooked rice powder. *Indian J Med Res* 1998;**107:**257–62.

Dutta D, Bhattacharya MK, Deb AK, Sarkar D, Chatterjee A, Biswas AB, *et al*. Evaluation of oral hypo-osmolar glucose-based and rice-based oral rehydration solutions in the treatment of cholera in children. *Acta Paediatr* 2000;**89:**787–90.

Dutta P, Mitra U, Rasaily R, Bhattacharya SK, Bhattacharya MK, Manna B, *et al*. Assessing the cause of in-patients pediatric diarrheal deaths: An analysis of hospital records. *Indian Pediatr* 1995;**32:**313–21.

Ghosh S, Sengupta PG, Gupta DN, Mondal SK, Goswami M, Bhattacharya SK, *et al*. Maternal knowledge on risk behavioural practices and its association with diarrhoea in a rural community of West Bengal, India. *J Commun Dis* 1998;**30:**251–5.

Gupta DN, Mondal SK, Ghosh S, Rajendran K, Sur D, Manna B. Impact of zinc supplementation on diarrhoeal morbidity in rural children of West Bengal, India. *Acta Paediatr* 2003;**92:**531–6.

Gupta DN, Sircar BK, Sengupta PG, Ghosh S, Banu MK, Mondal SK, *et al*. Epidemiological and clinical profiles of acute invasive diarrhoea with special reference to mucoid episodes: A rural community-based longitudinal study. *Trans R Soc Trop Med Hyg* 1996;**90:**544–7.

Gupta P, Murali MV, Seth A. Epidemiology of diarrhea in urban slums. *Indian Pediatr* 1998;**35:**147–51.

Hirve S, Ganatra B. A prospective cohort study on the survival experience of under five children in rural western India. *Indian Pediatr* 1997;**34:**995–1001.

ICMR-IVI. Surveillance for cholera and typhoid fever in eastern Kolkata, India. ICMR-IVI Collaborative Project, Kolkata, West Bengal (unpublished).

Indrayan A, Wysocki MJ, Kumar R, Chawla A, Singh N. Estimates of the years-of-life-lost due to the top nine causes of death in rural areas of major states in India in 1995. *Natl Med J India* 2002; **15:**7–13.

Kang G, Ramakrishna BS, Daniel J, Mathan M, Mathan VI. Epidemiological and laboratory investigations of outbreaks of diarrhoea in rural South India: Implications for control of disease. *Epidemiol Infect* 2001;**127:**107–12.

Khalique N, Sinha SN, Yunus M, Malik A. Certain aspects of infant mortality—a prospective study in a rural community. *Indian J Matern Child Health* 1992;**3:**85–8.

Khalique N, Sinha SN, Yunus M, Malik A. Early childhood mortality—a rural study. *J R Soc Health* 1993;**113:**247–9.

Kothari G. Diarrhoea in urban slums: Bombay. *Dialogue Diarrhoea* 1987 Dec;**(**31):4–5.

Kumar Karn S, Harada H. Field survey on water supply, sanitation and associated health impacts in urban poor communities—a case from Mumbai city, India. *Water Sci Technol* 2002;**46:**269–75.

Kumar S, Debnath A, Goswami A. Some aspects of Diarrhoea Training and Treatment Unit in Infectious Diseases Hospital, Calcutta. *Indian J Public Health* 1994;**38:**81–6.

Lal P, Bansal AK, Aggarwal CS, Taneja DK, Gogia V. Incidence of diarrhoea and some related environmental and behavioural factors in *jhuggis* of Delhi. *Indian J Public Health* 1996;**40:**35–7.

Lal S. Surveillance of acute diarrhoeal diseases at village level for effective home management of diarrhoea. *Indian J Public Health* 1994;**38:**65–8.

Mahendraker AG, Dutta PK, Urmil AC, Moorthy TS. A study of medico social profile of under five children suffering from diarrhoeal diseases. *Indian J Matern Child Health* 1991;**2:**127–30.

Mondal NC, Biswas R, Manna A. Risk factors of diarrhoea among flood victims: A controlled epidemiological study. *Indian J Public Health* 2001;**45:**122–7.

Mondal SK, Gupta PG, Gupta DN, Ghosh S, Sikder SN, Rajendran K, *et al*. Occurrence of diarrhoeal diseases in relation to infant feeding practices in a rural community in West Bengal, India. *Acta Paediatr* 1996;**85:**1159–62.

Murray CJ, Acharya AK. Understanding DALYs. *J Health Economics* 1997;**16:**703–30.

National Burden of Disease Studies: A practical guide edition 2.0 October 2001.

National Health Policy, 2002. Available from URL: http://mohfw.nic.in/np2002.htm.

National Population Policy, 2000. National Commission on Population. Available from URL: http://populationcommission.nic.in.

Natinal Family Health Survey (NFHS)-2.

NICED. An operational study on effect of zinc supplementation on reduction of diarrhoeal morbidity among rural children.

NICED. Surveillance under DBT project at NICED, Kolkata.

Niyogi SK, Saha MR, De SP. Enteropathogens associated with acute diarrhoeal diseases. *Indian J Public Health* 1994;**38:**29–32.

Pandey A, Sengupta PG, Mondal SK, Gupta DN, Manna B, Ghosh S, *et al*. Gender differences in healthcare-seeking during common illnesses in a rural community of West Bengal, India. *J Health Popul Nutr* 2002;**20:**306–11.

Rahman MM, Vermund SH, Wahed MA, Fuchs GJ, Baqui AH, Alvarez JO. Simultaneous zinc and vitamin A supplementation in Bangladeshi children: Randomised double blind controlled trial. *BMJ* 2001;**323:**314–18**.**

Ramakrishnan R, Venkatarao T, Koya PK, Kamaraj P. Influence of recall period on estimates of diarrhoea morbidity in infants in rural Tamil Nadu. *Indian J Public Health* 1999;**43:**136–9.

Ray SK, Kumar S, Saha I, Dasgupta S. Utilisation of ORT during diarrhoea in three districts of West Bengal. *Indian J Public Health* 1994;**38:**73–6.

Ray SK, Roy P, Deysarkari S, Lahiri A, Mukhopadhaya BB. A cross sectional study of undernutrition in 0–5 years age group in an urban community. *Indian J Matern Child Health* 1990;**1:**61–2.

Saha DR, Gupta DN, Sengupta PG, Mondal SK, Ghosh S, Saha NC, *et al*. Intestinal parasitism: A childhood problem in rural Bengal. *J Commun Dis* 1995;**27:**170–4.

Sarkar K. Role of oral rehydration therapy in controlling epidemic of cholera and watery diarrhoea. *J Indian Med Assoc* 2003;**101:**379–80, 386.

Sazawal S, Black RE, Bhan MK, Bhandari N, Sinha A, Jalla S. Zinc Supplementation in young children with acute diarrhea in India. *N Engl J Med* 1995;**333:**839–44.

Sengupta B, Dasgupta S, Saha I, Mandal AK, Palodhi PK. Experience in running a Diarrhoeal Training cum Treatment Unit (DTTU) in a state teaching hospital in Calcutta. *J Indian Med Assoc* 1998;**96:** 104–5, 108.

Sengupta PG, Mandal S, Sen D, Das P, Deb BC, Pal SC. Multidrug resistant epidemic shigellosis in a village in West Bengal, 1984. *Indian J Public Health* 1990;**34:**15–19.

Shah SM, Yousafzai M, Lakhani NB, Chotani RA, Nowshad G. Prevalence and correlates of diarrhea. *Indian J Pediatr* 2003;**70:** 207–11.

Shah SM, Yousafzai M, Lakhani NB, Chotani RA, Nowshad G. Prevalence and correlates of diarrhea. *Indian J Pediatr* 2003;**70:**207–11.

Sharma A, Gupta S. Impact of ICDS on health and nutritional status of children. *Indian J Matern Child Health* 1993;**4:**27–30.

Singh J, Gowriswari D, Chavan BR, Patiat RA, Debnath AC, Jain DC, *et al.* Diarrhoeal diseases amongst children under five. A study in rural Alwar. *J Commun Dis* 1992;**24:**150–5.

Singh K, Kumar K. Mothers' concept of the ideal number, colour and consistency of stools of their infants. *Indian J Matern Child Health* 1993;**4:**62–3.

Singh SP, Reddy DC, Mohapatra SC, Gaur SD. Study of infant and childhood mortality in an ICDS block of eastern UP. *Indian J Public Health* 1993;**37:**61–5.

Sircar BK, Deb BC, Sengupta PG, Mondal S, Gupta DN, Sarkar S, *et al.* An operational study on implementation of oral rehydration therapy in a rural community of West Bengal, India. *Indian J Med Res* 1991;**93:**297–302.

Sircar BK, Ghosh S, Sengupta PG, Gupta DN, Mondal SK, Sur D, *et al.* Impact of vitamin A supplementation to rural children on morbidity due to diarrhoea. *Indian J Med Res* 2001;**113:**53–9.

Sircar BK, Saha MR, Deb BC, Singh PK, Pal SC. Effectiveness of oral rehydration salt solution (ORS) in reduction of death during cholera epidemic. *Indian J Public Health* 1990;**34:**68–70.

Srivastava RN. Programme for control of diarrhoeal diseases (editorial). *J Indian Assoc Commun Dis* 1982;**5:**48–9.

Sur D, Dutta P, Nair GB, Bhattacharya SK. Severe cholera outbreak following floods in a northern district of West Bengal. *Indian J Med Res* 2000;**112:**178–82.

Sur D, Gupta DN, Mondal SK, Ghosh S, Manna B, Rajendran K, *et al.* Impact of zinc supplementation on diarrheal morbidity and growth pattern of low birth weight infants in Kolkata, India: A randomized, double-blind, placebo-controlled, community-based study. *Pediatrics* 2003;**112**(6 Pt 1):1327–32.

Sur D, Mondal SK, Gupta DN, Ghosh S, Manna B, Sengupta PG. Impact of breastfeeding on weight gain and incidence of diarrhea among low birth weight infants of an urban slum of Calcutta. *Indian Pediatrics* 2001;**38:**381–4.

Sur D, Sengupta PG, Mondal SK, Dutta P, Gupta DN, Ghosh S, *et al.* A localised outbreak of *Vibrio cholerae* O139 in Kolkata, West Bengal. *Indian J Med Res* 2002;**115:**149–52.

Victora CG, Huttly SR, Fuchs SC, Barros FC, Garenne M, Leroy O, *et al.* International differences in clinical patterns of diarrhoeal deaths: A comparison of children from Brazil, Senegal, Bangladesh, and India. *J Diarrhoeal Dis Res* 1993;**11:**25–9.

World Health Organization (WHO). Division of Diarrhoeal and Acute Respiratory Disease Control. Rational management of diarrhoea in children. *Essent Drugs Monit* 1991;10–11.

WHO. Office of Information. Cholera: Ancient scourge on the rise. WHO announces global plan for cholera control. *WHO Feature* 1991; (154):1–3.

WHO. *Global Program on Evidence for Health Policy.* Geneva.

Zodpey SP, Deshpande SG, Ughade SN, Kulkarni SW, Shrikhande SN, Hinge AV. A prediction model for moderate or severe dehydration in children with diarrhoea. *J Diarrhoeal Dis Res* 1999;**17:**10–16.

Causal analysis and treatment protocols for childhood diseases

SIDDARTH RAMJI

1. Birth asphyxia

Table 1.1 Causes of birth asphyxia (by significance)[1–3]

	Direct	Indirect	Distant
Main causes	• LBW (preterm and IUGR) • Obstetric complications (APH, cord prolapse, PIH, etc.) • Foetal malformations	• Unskilled birth attendant (especially newborn resuscitation skills) • Poor maternal health (e.g. poor nutrition, medical illness) • Inadequate antenatal care • Maternal age (<18 or >35 years) • Poor/absent emergency obstetric and newborn care • Absence of credible referral system	• Poor maternal literacy • Lack of community awareness • Low socioeconomic status

LBW: low birth weight; IUGR: intrauterine growth retardation; APH: antepartum haemorrhage; PIH: pregnancy-induced hypertension

Table 1.2 Interventions (by significance) for the management of birth asphyxia[4–6]

Outcome	Medical interventions	Non-medical interventions/prevention
Establish cry at birth	• Skilled birth attendant • Strengthening EmOC services • Training of *dais* • Resuscitation • Developing an effective and functional referral system • Thermal control • Improving the quality of ANC (including monitoring of gain in weight of the mother)	• Improving maternal nutrition during pregnancy (role of ICDS) • Improving the condition of roads for faster transportation of the newborn to the nearest appropriate health facility • Preventing child marriage so that women conceive at the appropriate age

EmOC: emergency obstetric care; ICDS: Integrated Child Development Scheme; ANC: antenatal care

Table 1.3 Standard treatment protocols for birth asphyxia[7,8]

Personnel (units of time and type)	Drugs (dosage, type and time)	Inpatient stay	Equipment
District hospital • Paediatrician (1 hour/day) • Nurse (2 hours/day) • Obstetrician (for EmOC, management of complicated labour)	• Anticonvulsants (phenobarbitone and phenytoin 20 mg/kg stat and then 3–5 mg/kg/day bd) • Oxygen	Up to 5 days	• Radiant warmers • Oxygen hoods • Resuscitation bags
CHC • Paediatrician (1 hour/day) • Nurse (2 hours/day) • Obstetrician (for EmOC, management of complicated labour)	• Anticonvulsants (phenobarbitone and phenytoin 20 mg/kg stat and then 3–5 mg/kg/day bd) • Oxygen	1–2 days	• Radiant warmers • Oxygen hoods • Resuscitation bags
PHC • Medical officer • Auxiliary nurse–midwife		<1 day	Resuscitation bags

CHC: community health care: PHC: primary health centre; EmOC: emergency obstetric care; ICDS: Integrated Child Development Scheme

Department of Paediatrics, Maulana Azad Medical College, New Delhi; e-mail: sumisid@vsnl.com

2. Neonatal sepsis

Table 2.1 Causes of neonatal sepsis (by significance)

	Direct	Indirect	Distant
Main causes	• Low birth weight • Acute maternal intrapartum infection (including STIs) • Prolonged and preterm rupture of membranes • Delayed and non-exclusive breastfeeding • Inappropriate community practices (e.g. cord care practices, branding, etc.) • Inappropriate intrapartum interventions by untrained attendants	• Unskilled birth attendant • Unhygienic delivery and postnatal conditions • Delayed recognition and care-seeking • Increased biological risk in males	• Poor maternal literacy • Lack of community awareness • Low socioeconomic status

STI: sexually transmitted infection

Table 2.2 Interventions (by significance) for neonatal sepsis

Outcome	Medical interventions	Non-medical interventions/prevention
Survival	• Injectable antibiotics • Oxygen therapy • IV fluids	• Exclusive breastfeeding • Use of '5' cleans during delivery (includes neonatal tetanus) • Tetanus toxoid for mother (for neonatal tetanus)

Table 2.3 Standard treatment protocols for neonatal sepsis

Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay	Supportive care
District hospital • Paediatrician (1 hour/day) • Nurse (24 hours/day)	• Blood culture • CSF examination • Blood counts	• Inj. ampicillin 100 mg/kg/day x 7–10 days • Inj. gentamicin 7.5 mg/kg/day x 7–10 days	7 days	• IV fluids • Oxygen therapy • Breastfeeding support during recovery of infant
CHC • Paediatrician (1 hour/day) • Nurse (24 hours/day)	Blood counts	• Inj. ampicillin 100 mg/kg/day x 7–10 days • Inj. gentamicin 7.5 mg/kg/day x 7–10 days	3 days	• IV fluids • Oxygen therapy • Breastfeeding support during recovery of infant
PHC • Medical officer • Auxiliary nurse–midwife		• First dose of antibiotic —Inj. ampicillin 50 mg/kg (or oral co-trimoxazole) and Inj. gentamicin 5 mg/kg	Referral	

CHC: community health centre; CSF: cerebrospinal fluid; PHC: primary health centre

3. Low birth weight

Table 3.1 Causes of low birth weight (by significance)[9–12]

Direct	Indirect causes	Distant causes
• Poor maternal health • Obstetric complications • Medical illness (e.g. malaria) • Multiple pregnancy • Foetal malformations	• Inadequate antenatal care • Heavy maternal work and inadequate rest during pregnancy • Adolescent pregnancy • Maternal age <18 or >35 years • Previous childbirth <2 years ago	• Poor maternal literacy • Lack of community awareness • Low socioeconomic status

Table 3.2 Interventions (by significance) for low birth weight[6,13–15]

Outcome	Medical interventions	Non-medical interventions/prevention
Birth weight <1500 g	• Thermal control (warmers/incubators/KMC) • IV fluids/breastfeeding/vitamin–mineral supplements • Drugs, depending on complications	• Clean environment/hygienic practices • Adequate antenatal care • Improved maternal nutrition
Birth weight 1500–1800 g	• Thermal control (warmers/KMC) • Breastfeeding • Drugs, depending on complications	• Clean environment/hygienic practices • Adequate antenatal care • Improved maternal nutrition
Birth weight 1800–2500 g	• Thermal control (KMC, rooming-in) • Breastfeeding • Drugs, depending on complications	• Clean environment/hygienic practices • Adequate antenatal care • Improved maternal nutrition

KMC: kangaroo mother care

Table 3.3 Standard treatment protocols for the management of low birth weight[15]

Condition	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay
Birth weight <1500 g	• Paediatrician (4 hours/day) • Nurse (24 hours/day)	Blood sugar	Depends on complications	14–21 days
Birth weight 1500–1800 g	• Paediatrician (1/2 hour/day) • Nurse (3 hours)	—		3–5 days
Birth weight 1800–2500 g	• Paediatrician (1/2 hour/day) • Nurse (1 hour)			1–2 days

4. Diarrhoea

Table 4.1 Causes (by significance) of diarrhoea/dysentery

	Direct	Indirect	Distant
Main causes	• Non-exclusive breastfeeding (<6 months) • Contaminated water and food	• Low birth weight/malnutrition • Vitamin A deficiency	• Poor maternal literacy • Lack of community awareness about sanitation • Low socioeconomic status • Improved sanitation, safe water supply
Interaction with other causes	Urinary tract infection		

Table 4.2 Interventions (by significance) for diarrhoea/dysentery[15,16]

Disease/condition	Outcome	Medical interventions	Non-medical interventions/prevention
Diarrhoea	Severe dehydration	• IV fluids • ORS • Antibiotics in case of cholera or dysentery	• Adequate nutrition • Safe drinking water
	Some dehydration	ORS	• Adequate nutrition • Safe drinking water
	No dehydration	ORS	• Home available fluids • Adequate nutrition • Safe drinking water
Dysentery		• Oral antibiotics • ORS	• Adequate nutrition • Safe drinking water

ORS: oral rehydration salt

Table 4.3 Standard treatment protocols for diarrhoea/dysentery[15,16]

Condition	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay
Severe dehydration	*DH/CHC/PHC* • Paediatrician (1/2 hour/day) • Nurse (6–8 hours/day)	Stool examination	IV fluids—Ringer lactate 150 ml/kg x 8 hours	1 day
Some dehydration	*DH/CHC/PHC/SC* • Paediatrician/MO (1/2 hour/day) • Nurse/ANM (2 hours)	—	Oral rehydration salt 75 ml/kg x 4 hours	OPD observation for 4 hours
Dysentery	OPD consultation	Stool examination	Oral nalidixic acid 55 mg/kg/day x 5 days	Nil

DH: district hospital; CHC: community health centre; PHC: primary health centre; SC: subcentre; ANM: auxiliary nurse–midwife

5. Pneumonia

Table 5.1 Causes of pneumonia (by significance)

	Direct	Indirect	Distant
Main causes	• LRTI in the family • Use of biomass fuels	• Non-exclusive breastfeeding (<4 months) • LBW/severe malnutrition • Vitamin A deficiency • Inappropriate immunization for age • Passive smoking	• Lack of community awareness • Low socioeonomic status • Poor literacy • Male sex
Interaction with other causes	• Asthma • Heart disease • Gastroenteritis • Measles • Whooping cough		

LRTI: lower respiratory tract infection; LBW: low birth weight

Table 5.2 Interventions (by significance) for the management of pneumonia[15,16]

Outcome	Medical interventions	Non-medical interventions/prevention
Severe pneumonia	• Injectable antibiotics • Oxygen • IV fluids	• Cough remedies • Adequate nutrition • Protection from passive smoking
Pneumonia	Oral antibiotics (e.g. co-trimoxazole)	• Home-based cough remedies • Adequate nutrition • Protection from passive smoking
Cough/cold		Home-based cough remedies

Table 5.3 Standard treatment protocols for pneumonia[15,16]

Condition	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay	Supportive care
Severe pneumonia	• Paediatrician (2 hours/day) • Nurse (6–8 hours/day)	Chest X-ray	• Inj. chloramphenicol 100 mg/kg/day x 7–10 days • Inj. ampicillin 100 mg/kg/day x 7–10 days	5–7 days	• Oxygen • IV fluids
Pneumonia	• Paediatrician/MO (2 hours/day) • ANM (4–6 hours/day)		• Oral co-trimoxazole 6–8 mg/kg/day x 5 days • Oral amoxycillin 20–40 mg/kg/day x 5 days	Ambulatory	

MO: medical officer; ANM: auxiliary nurse–midwife

6. Malnutrition

Table 6.1 Causes of malnutrition (by significance)

	Direct	Indirect	Distant
Main causes	• Non-exclusive breastfeeding during the first 6 months • Inadequate quantity of weaning foods • Non-energy dense feeds • Recurrent infections	• Low birth weight • Poverty • Community feeding practices	Natural disasters (drought, floods, etc.)
Interaction with other causes	Measles		

Table 6.2 Interventions (by significance) for the management of malnutrition[15,16]

Outcome	Medical interventions	Non-medical interventions/prevention
Severe malnutrition	• Dietary rehabilitation • Micronutrient supplementation • Treatment of severe anaemia • Treatment of infections • Correcting dehydration	• Maternal education • Community IEC activities • Immunization • Food security • Preventing low birth weight (adequate antenatal care)
Mild–moderate malnutrition	• Nutritional counselling • Micronutrient supplementation	• Maternal education • Community IEC activities • Immunization • Food security

IEC: information, education and communication

Table 6.3 Standard treatment protocols for malnutrition[15,16]

Condition	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay
Severe malnutrition	*DH* • Paediatrician (1 hour/day) • Nurse (2 hours/day)	• Haemoglobin • Peripheral blood smear • Stool examination	• Micronutrients • Antibiotics (if infection is present) • ORS in case of diarrhoea	Up to 2 weeks
	CHC • Paediatrician (1 hour/day) • Nurse (2 hours/day)	• Haemoglobin • Peripheral blood smear • Stool examination	• Micronutrients • Antibiotics (if infection is present) • ORS in case of diarrhoea	Up to 2 weeks
	PHC • MO • ANM (30 minutes)			Immediate referral
Mild–moderate malnutrition	*DH/CHC/PHC* • Paediatrician • MO (30 minutes)		Micronutrients	Ambulatory

DH: district hopital; CHC: community health centre; PHC: primary health centre; ORS: oral rehydration salt; MO: medical officer

References

1. Chandrashekar S, Rao RSP, Chakladar BK, Krishnan L, Nair NS. Perinatal mortality in rural district of South India. *Indian Pediatr* 1998;**68:**709–15.
2. Misra PK, Thakur S, Kumar A, Tandon S. Perinatal mortality in rural India with special reference to high risk pregnancies. *J Trop Pediatr* 1993;**39:**41–4.
3. Shah U, Pratinidhi AK, Bhatiawande PV. Perinatal mortality in rural India: A strategy for reduction through primary care. *J Epidemiol Community Health* 1984;**38:**134–7.
4. Ramji S, Rasaily R, Mishra PK, Narang A, Jayam S, Kapoor AN, *et al.* Resuscitation of asphyxiated newborns with room air or 100% oxygen at birth: A multicentric clinical trial. *Indian Pediatr* 2003;**40:**510–17.
5. Kumar R. Effectiveness of training TBAs for management of asphyxia neonatorum using resuscitation equipment. *Prenat Neonat Med* 1998;**3:**25–6.
6. Bang AT, Bang RA, Baitule S, Reddy MH, Deshmukh M. Effect of home based neonatal care and management of sepsis on neonatal mortality: Field trial in rural India. *Lancet* 1999;**354:**1955–61.
7. Finegold JG, Mizrahi EM, Lee RT. The newborn nervous system.

In: Taeusch HW, Ballard RA (eds). *Avery's diseases of the newborn.* 7th ed. New Delhi: Harcourt Asia Pvt Ltd; 2000:839–92.
8. Ramji S. Treat and counsel. In: Kumar H, Kuba R (eds). *Care of newborn and young infant (ME-103).* New Delhi: Indira Gandhi Open University, School of Health Sciences; 2003:61–77.
9. Hirve SS, Ganatra BR. Determinants of low birth weight: A community based prospective cohort study. *Indian Pediatr* 1994;**31:**1221–5.
10. Agarwal KN, Agarwal DK, Agarwal A, Rai S, Prasad R, Agarwal S, *et al.* Impact of the ICDS on maternal nutrition and birth weight in rural Varanasi. *Indian Pediatr* 2000;**37:**1321–7.
11. Radhakrishnan T, Thankappan KR, Vasan RS, Sarma PS. Socio-economic and demographic factors associated with birth weight: A community based study in Kerala. *Indian Pediatr* 2000;**37:**872–6.
12. Deshmukh JS, Motghare DD, Zodpey SP, Wadhva SK. Low birth weight and associated maternal factors in an urban area. *Indian Pediatr* 1998;**35:**33–6.
13. Pratinidhi A, Shah U, Shotri A, Bodhani N. Risk approach strategy in neonatal care. *Bull World Health Organ* 1986;**64:**291–7.
14. Dayal RS. Problems and management of low birth weight babies in an improvised neonatal unit of a developing country. *Bull Int Pediatr Assoc* 1980;**3:**36.
15. Sharma S, Sethi GR, Gulati RK (eds). *Standard Treatment Guidelines*. Delhi: Delhi Society for Promotion of Rational Use of Drugs; 2002.
16. World Health Organization. *Management of the child with a serious infection or severe malnutrition. Guidelines for care at the first referral level in developing countries.* New Delhi: WHO; 2000.

Section II

Non-communicable diseases

Forecasting vascular disease cases and associated mortality in India

A. INDRAYAN

The mandate was to forecast cases with cardiovascular disease (CVD) and associated mortality in India through the year 2015 at 5-year intervals. A break-up of forecasts in age/gender/urban/rural categories was desirable and State-wise projections were expected. This exercise is based on the data gathered and supplied by Centre for Chronic Disease Control (CCDC), New Delhi. These voluminous data lack State-wise details, which rendered the ideal unattainable and even cross-classification by age, gender and area became difficult. Recent data were even more scanty. We made the best use of whatever data were supplied to us. Limitations of our estimates are stated at the end of this paper. However, we expect our estimates to be not far from reality.

Nobody doubts that cases of CVD would rapidly increase in India during the next few years. This increase is attributable to (i) sheer increase in the population size due to natural growth, (ii) ageing of the population which makes people more vulnerable to chronic diseases, and (iii) increased vulnerability due to lifestyle changes that promote CVD. The first would happen in any case, and the second would operate even if age–gender-specific prevalence rates remain the same. The third would manifest in terms of higher age–gender-specific rates if people tend to become more obese, consume more calories, eat more processed food, take more salt or a high carbohydrate diet which can increase cholesterol and blood pressure levels, adopt a more sedentary lifestyle, smoke more, etc. Many more would get diabetes (*see* Appendix 1) which in turn is a strong risk factor for CVD. One factor that is generally ignored is the stress level that acts as a twin-edged sword. Poverty and ignorance can make life difficult and stressful for the deprived, and development coupled with urbanization and vanishing family security can bring its own set of problems.

Methodology

The first approach to forecasting is to estimate the increase in risk due to apprehended changes in lifestyle and other factors, and impute this increase to forecast prevalence estimates. This requires a study of past trends in vulnerability factors. According to Singh and Sen (2003), the risk factors for coronary heart disease (CHD) are a formidable list: obesity, a sedentary lifestyle, smoking, hypertension, high low-density lipoprotein (LDL), low high-density lipoprotein (HDL), diabetes, insulin resistance, triglycerides, lipoprotein (a) (Lp[a]), homocysteine, fibrinogen, HbA1c, albumin, etc.

Although an equation linking the risk of CHD with some of its risk factors is available from the Framingham study (Wilson *et al.* 1998), this is for individuals and cannot be easily used for the present exercise. In fact, the risk factor approach to forecasting requires a study of past trends in various risk factors and an assumption that the same trend would continue. This also requires the presence of a relationship between disease prevalence and risk factors, which itself would be subject to much uncertainty. In addition, the data supplied to us on various risk factors were inadequate for this approach. Lack of data can be misleading and validity would suffer. Also, the foregoing list is restricted to the known factors. Many factors affecting the vulnerability to CHD are unknown. Therefore we adopted the second approach based on trends in prevalences to get more valid projections. This second approach is to use previous trends in age–gender–area-specific prevalence rates and project it to the future. This trend automatically takes care of the trend in the conglomerate of risk factors. This approach obviates the need to know the relationship between risk factors and prevalence. The projected age–gender–area-specific rates are used on the estimated age–gender–area-specific population to get the projected number of cases. This approach assumes that both vulnerability factors and preventive strategies would continue to rise in the same fashion as before. Thus, the decelerating effect of positive changes in lifestyle and other factors is also in-built. Boyle *et al.* (2001) used this approach to project cases of diabetes in the US through the year 2050.

Age–gender–area-wise population projection

India seems to be all set for a demographic transition. Life expectancy is increasing primarily due to a decline in infant mortality but the adult mortality is also declining as chronic

Division of Biostatistics and Medical Informatics
University College of Medical Sciences
Dilshad Garden, Delhi 110095
e-mail: aindrayan@hotmail.com

Why the Framingham equation cannot be easily used in this work

Wilson *et al.* (1998) developed the following equation that links the risk for coronary heart disease (CHD) with its factors:

$$P = 1 - S(t)^{\exp[f(x,M)]},$$

where $f(x,M) = \beta_1(x_1 - M_1) + \beta_2(x_2 - M_2) + \ldots + \beta_p(x_p - M_p)$; $x_1, x_2, \ldots, x_p$ are scores for the presence of risk factors in the individual; $M_1, M_2, \ldots, M_p$ are mean values of the risk factors in the group; and S(t) is 10-year survival rate at these mean values. The equation estimates P which is 10-year risk for CHD. This equation has the following features:

1. The risk factors considered are age, smoking (yes/no), diabetes, blood pressure category, total cholesterol category, low density lipoprotein (LDL)-C category and high density lipoprotein (HDL)-C category. Many risk factors have not been incorporated.
2. The area under the curve is in the range of 0.75. Thus, there is an inherent uncertainty to the extent of 25%. This kind of uncertainty is a necessary component of any model but here it is rather high. This implies that the predictivity of the model is low.
3. The equation is useful for predicting the risk of CHD in *individual* subjects whose status with regard to the seven risk factors is known. The presence of various risk factors in individual subjects is assessed against the mean presence in the group to which the individual belongs (*see* f(x,M)). Although the equation can be used for say, each age group, it looks stretching it too much.
4. The equation cannot be directly used in other countries. It requires recalibration for local set-up. For this, data on the risk factors and 10-year survival rate are required for a large group of local subjects. These are not available.

For the reasons enumerated above, the equation is unsuitable in the present exercise on CHD projection at the national level.

diseases are replacing infectious diseases. The age–gender-wise projections for each year till 2016 are available in a report of the Registrar General of the Government of India (1996). The gross picture of the trend in population is shown in Fig. 1. Those in the age group of 60+ years will face the major onslaught of CVDs.

We could not locate the rural–urban break-up of the projected age-wise population anywhere. Since the prevalence of CVD is very different in rural areas compared with urban areas, this break-up is important for forecasting because urbanization is occurring rapidly. We captured the linear trend

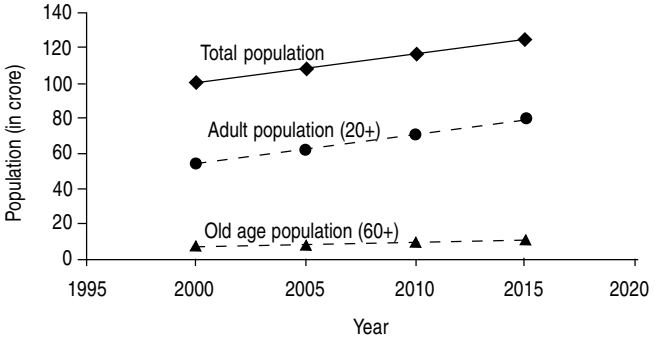

Fig. 1 Projected trends in population

Source: Report of the Registrar General of India 1996

in rural–urban ratio from the data of three censuses (1971, 1981 and 1991) and from the Sample Registration System (SRS) for the year 2000. We used this trend to forecast the ratio in the years 2005, 2010 and 2015. This was done separately for each age group and gender as shown in Fig. 2. The value of R^2 is more than 80% in 9 out of 10 age–gender groups, and the projections looked realistic. The projected rural–urban ratio so obtained was used on the projected population to get the rural–urban break-up of population in different age–gender groups. The population and the age–gender–area-wise break-up is given in Table 1.

Projection of cases

Trends in the prevalence of coronary heart disease

Coronary heart disease is the predominant CVD. CHD includes conditions such as cardiomyopathies, acute MI, angina pectoris, congestive heart failure and inflammatory heart disease (these are not necessarily mutually exclusive terms). Some useful data are available for CHD. Although longitudinal data from community-based studies from several places were desirable, they are not available. Hospital-based data were not useful in the present exercise because of their high selectivity. When the place is ignored, 4–5 points of data on prevalence rate were available for each of the age–gender groups in urban areas, and to a lesser degree in rural areas. The age groups we chose were 20–29, 30–39, …, 60–69 years. Wherever the reported age groups did not match exactly with these intervals, the data were put into the nearest group. When prevalence from some studies was available separately for 5-yearly intervals such as 30–34 and 35–39, the average was used for 30–39 years. These prevalences were used to fit a linear trend. Although a curvilinear trend is plausible, the data were inadequate to try this. Statistically, forecasting for 15 years on the basis of trend in the past 15 years is not a wise proposition. Yet, it is better to have something rather than nothing. Our experience suggests that forecasting on the basis of such scanty data may not be a worthless exercise although it has obvious limitations.

The CHD prevalence trends for various age–gender groups in urban areas are given in Fig. 3 and in rural areas in Fig. 4. For an exercise such as this, when the data are highly fluctuating and scanty, we did not consider it necessary to test the statistical significance of the trend (*Note*: The NCMH Expert Group was of the opinion that the exercise be redone after deleting one data point relating to the year 1974 that might be pushing the estimates upwards. This has been done and reported in Appendix 2, where we found that when that data point was deleted, the estimates of CHD caseload increased further).

Whereas the trend apparently looked fine for all age–gender groups in urban areas, there was only one data point for the 20–29 years age group (males as well as females) in rural areas (Fig. 4). We assume that the prevalence in

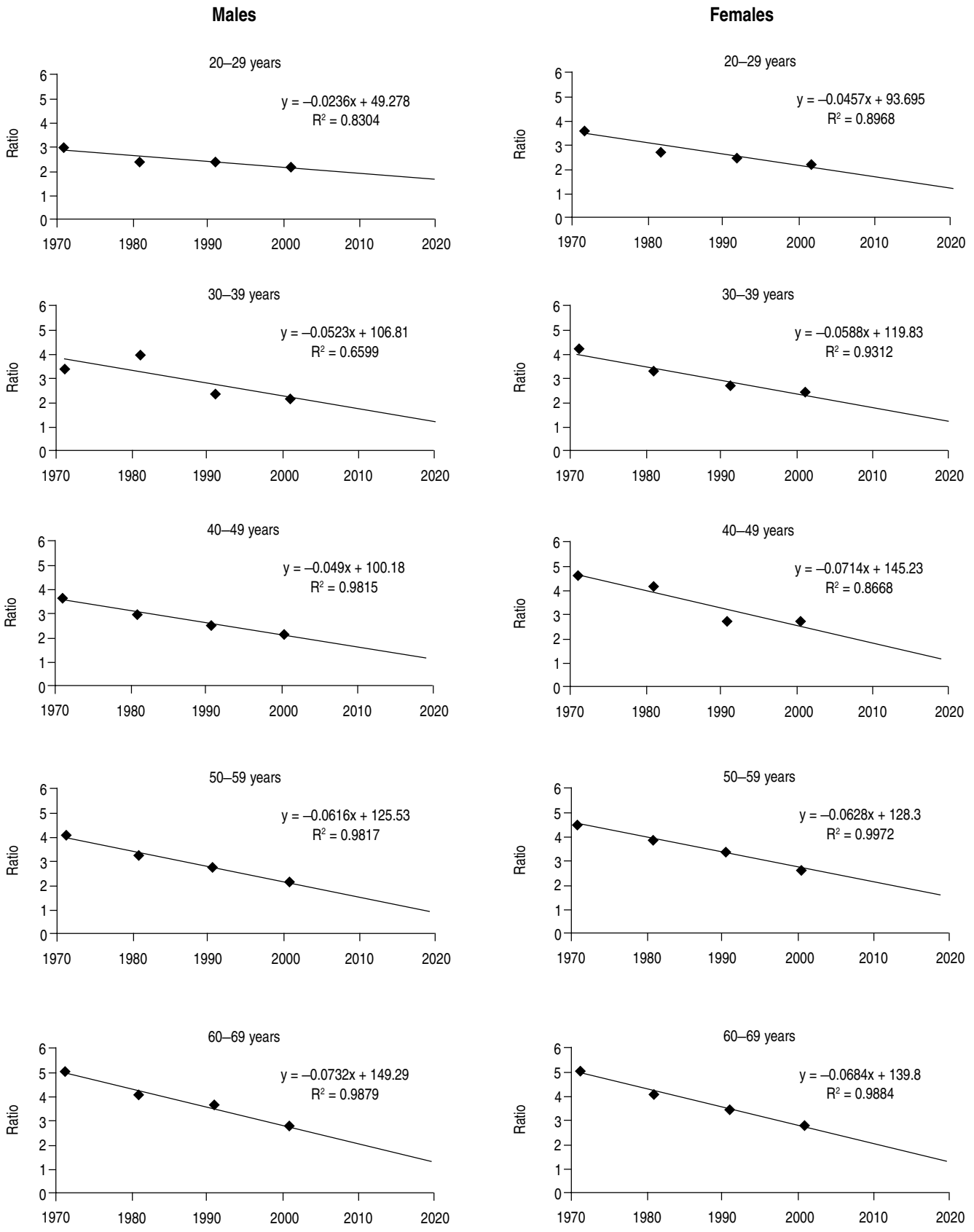

Fig. 2 Trends in rural–urban ratio in male and female populations

Sources of data: Indian Census 1971, 1981, 1991 and Sample Registration System (SRS) Survey 2000

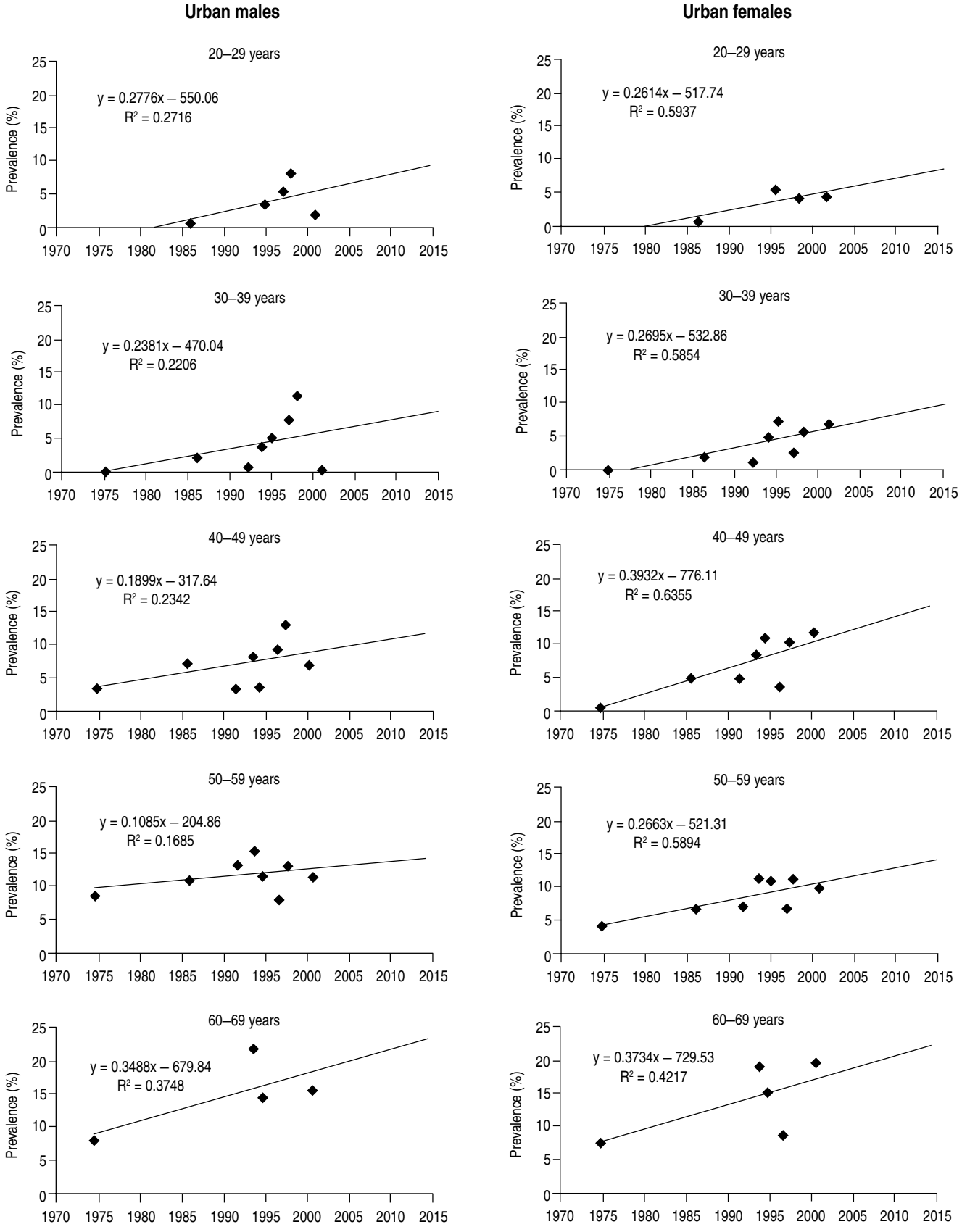

Fig. 3 Trends in CHD prevalence—Urban

Source of data: Centre for Chronic Disease Control

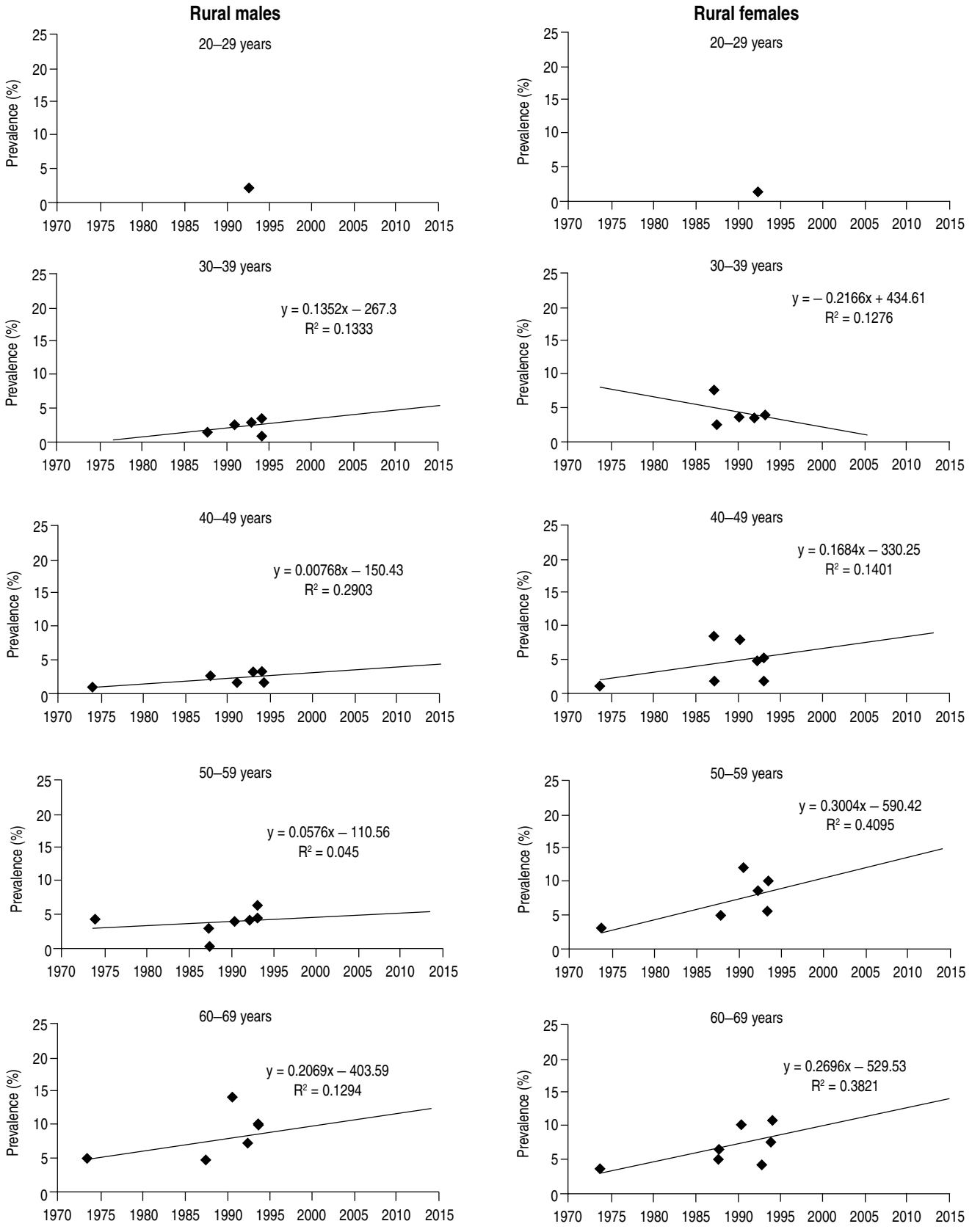

Fig. 4 Trends in CHD prevalence—Rural

Source of data: Centre for Chronic Disease Control

Table 1. Projected population of India by age, sex and area

Year/age	Population			Projected population			Rural–urban ratio (calculation from census)		By multiplication: Rural		By multiplication: Urban	
group	Both sexes	Male	Female	Age	Male	Female	Male	Female	Male	Female	Male	Female
2000												
0–4	110,298,000	56,527,000	53,771,000	20–29	87,138,000	81,813,000	2.078	2.295	58,828,058	56,983,561	28,309,942	24,829,439
5–9	119,711,000	61,155,000	58,557,000	30–39	69,778,000	68,176,000	2.210	2.230	48,040,305	47,068,879	21,737,695	21,107,121
10–14	122,401,000	63,574,000	58,827,000	40–49	53,059,000	47,182,000	2.180	2.430	36,373,780	33,426,315	16,685,220	13,755,685
15–19	105,816,000	56,107,000	49,709,000	50–59	36,011,000	31,972,000	2.330	2.700	25,196,886	23,330,919	10,814,114	8,641,081
20–24	88,178,000	46,387,000	41,792,000	60–69	21,785,000	20,697,000	2.890	3.000	16,184,743	15,522,750	5,600,257	5,174,250
25–29	80,772,000	40,751,000	40,021,000	70+	13,471,000	12,835,000						
30–34	73,997,000	36,944,000	37,053,000	Others	237,363,000	220,864,000						
35–39	63,957,000	32,834,000	31,123,000	Total	518,605,000	483,539,000						
40–44	54,712,000	28,865,000	25,847,000									
45–49	45,528,000	24,194,000	21,335,000									
50–54	37,839,000	20,069,000	17,769,000									
55–59	30,144,000	15,942,000	14,203,000									
60–64	23,594,000	12,179,000	11,415,000									
65–69	18,888,000	9,606,000	9,282,000									
70–74	12,576,000	6,411,000	6,165,000									
75–79	7,653,000	3,892,000	3,760,000									
80+	6,078,000	3,168,000	2,910,000									
Total	1,002,142,000	518,604,000	483,538,000									
2005												
0–4	112,341,000	57,821,000	54,519,000	20–29	101,923,000	91,038,000	1.960	2.067	67,489,554	61,350,082	34,433,446	29,687,918
5–9	107,964,000	55,275,000	52,688,000	30–39	76,996,000	76,480,000	1.949	1.936	50,882,383	50,430,954	26,113,617	26,049,046
10–14	118,498,000	60,492,000	58,007,000	40–49	60,491,000	56,148,000	1.935	2.073	39,880,779	37,876,604	20,610,221	18,271,396
15–19	121,992,000	63,340,000	58,652,000	50–59	42,067,000	37,716,000	2.022	2.386	28,146,749	26,577,193	13,920,251	11,138,807
20–24	105,300,000	55,822,000	49,478,000	60–69	24,745,000	23,191,000	2.524	2.658	17,723,150	16,851,197	7,021,850	6,339,803
25–29	87,661,000	46,101,000	41,560,000	70+	15,708,000	15,471,000						
30–34	80,187,000	40,437,000	39,750,000	Others	236,928,000	223,866,000						
35–39	73,289,000	36,559,000	36,730,000	Total	558,858,000	523,910,000						
40–44	63,074,000	32,325,000	30,750,000									
45–49	53,565,000	28,166,000	25,398,000									
50–54	43,996,000	23,247,000	20,749,000									
55–59	35,787,000	18,820,000	16,967,000									
60–64	27,532,000	14,387,000	13,145,000									
65–69	20,404,000	10,358,000	10,046,000									
70–74	15,212,000	7,580,000	7,632,000									
75–79	8,735,000	4,373,000	4,361,000									
80+	7,232,000	3,755,000	3,478,000									
Total	1,082,768,000	558,857,000	523,911,000									
2010												
0–4	120,292,000	61,741,000	58,551,000	20–29	118,496,000	107,566,000	1.842	1.838	76,801,419	72,488,224	41,694,581	35,077,776
5–9	109,763,000	56,381,000	53,383,000	30–39	85,786,000	80,684,000	1.687	1.642	53,859,688	53,203,074	31,926,312	27,480,926
10–14	106,819,000	54,715,000	52,104,000	40–49	67,596,000	66,530,000	1.690	1.716	42,467,375	44,880,146	25,128,625	21,649,854
15–19	117,994,000	60,217,000	57,776,000	50–59	48,972,000	44,577,000	1.714	2.072	30,927,785	31,411,908	18,044,215	13,165,092
20–24	121,384,000	63,016,000	58,368,000	60–69	29,364,000	27,395,000	2.158	2.316	20,065,710	19,133,540	9,298,290	8,261,460
25–29	104,678,000	55,480,000	49,198,000	70+	18,026,000	18,203,000						
30–34	87,032,000	45,755,000	41,278,000	Others	351,550,000	329,380,000						
35–39	79,437,000	40,031,000	39,406,000	Total	601,294,000	566,769,000						
40–44	72,318,000	36,019,000	36,299,000									
45–49	61,808,000	31,577,000	30,231,000									
50–54	51,844,000	27,116,000	24,729,000									
55–59	41,704,000	21,856,000	19,848,000									
60–64	32,800,000	17,045,000	15,755,000									
65–69	23,959,000	12,319,000	11,640,000									

(*Cont.*)

Table 1 (*cont.*) Projected population of India by age, sex and area

Year/age	Population			Projected population			Rural/urban ratio (calculation from census)		By multiplication: Rural		By multiplication: Urban	
group	Both sexes	Male	Female	Age	Male	Female	Male	Female	Male	Female	Male	Female
70–74	16,363,000	8,133,000	8,230,000									
75–79	11,046,000	5,414,000	5,632,000									
80+	8,820,000	4,479,000	4,341,000									
Total	1,168,062,000	601,293,000	566,769,000									
2015												
0–4	122,690,000	63,068,000	59,622,000	20–29	122,622,000	115,633,000	1.724	1.610	77,606,581	71,320,680	45,015,419	44,312,320
5–9	116,840,000	60,002,000	56,838,000	30–39	100,449,000	89,877,000	1.426	1.348	59,035,271	51,598,891	41,413,729	38,278,109
10–14	109,478,000	56,193,000	53,286,000	40–49	74,738,000	74,734,000	1.445	1.359	44,170,311	43,053,627	30,567,689	31,680,373
15–19	106,349,000	54,467,000	51,882,000	50–59	56,044,000	53,216,000	1.406	1.758	32,750,567	33,920,859	23,293,433	19,295,141
20–24	117,490,000	59,947,000	57,543,000	60–69	34,547,000	32,535,000	1.792	1.974	22,173,433	21,595,188	12,373,567	10,939,812
25–29	120,766,000	62,675,000	58,090,000	70+	20,909,000	21,181,000						
30–34	104,021,000	55,108,000	48,913,000	Others	233,730,000	221,628,000						
35–39	86,305,000	45,341,000	40,964,000	Total	643,039,000	608,804,000						
40–44	78,482,000	39,489,000	38,993,000									
45–49	70,990,000	35,249,000	35,741,000									
50–54	59,960,000	30,468,000	29,492,000									
55–59	49,299,000	25,576,000	23,724,000									
60–64	38,379,000	19,875,000	18,504,000									
65–69	28,703,000	14,672,000	14,031,000									
70–74	19,446,000	9,807,000	9,639,000									
75–79	11,948,000	5,801,000	6,147,000									
80+	10,696,000	5,301,000	5,395,000									
Total	1,251,841,000	643,037,000	608,804,000									

Note: Differences in some totals are due to rounding off
Source: Registrar General of India 1996

this age group will remain unchanged from this value till the year 2015 in rural areas.

In rural females, in the 30–39 years age group, the trend shows a decline and approaching zero prevalence. Since this does not seem plausible, we assume that the rate last seen would remain constant till the year 2015.

The projected prevalence rates of CHD in India in different age–gender groups in urban and rural areas are given in Table 2. The assumption is that they too follow the past trends. The prevalence in rural areas is much lower than that in urban areas, and is not much different among males and females.

The ICMR Task Force project reported for 1991–94 a prevalence of 23.2% in urban males in Delhi in the 60–64 years age group based on the history and ECG evidence, which is unusually high. This could be because CHD includes angina pectoris, which is quite common, and also includes CHD arising from conditions such as diabetes and hypertension. Another explanation of such a high projection could be the indiscriminate eating and exercise habits of the younger generation. A recent study in Delhi found that 1 in 4 adolescents and young adults suffers from insulin intolerance, which predisposes to diabetes and subsequent coronary conditions, and 1 in 8 has a high level of C-reactive

Table 2. Forecasting the prevalence rate (%) of coronary heart disease (CHD) in India

		20–29 years		30–39 years		40–49 years		50–59 years		60–69 years	
Year	Area	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
2000	Urban	5.14	5.06	6.16	6.14	8.16	10.29	12.14	11.29	17.76	17.27
	Rural	1.80	1.30	3.10	2.90	3.17	6.55	4.64	10.38	10.21	9.67
2005	Urban	6.53	6.37	7.35	7.49	9.11	12.26	12.68	12.62	19.50	19.14
	Rural	1.80	1.30	3.78	2.90	3.55	7.39	4.93	11.88	11.24	11.02
2010	Urban	7.92	7.67	8.54	8.84	10.06	14.22	13.23	13.95	21.25	21.00
	Rural	1.80	1.30	4.45	2.90	3.94	8.23	5.22	13.38	12.28	12.37
2015	Urban	9.30	8.98	9.73	10.18	11.01	16.19	13.77	15.28	22.99	22.87
	Rural	1.80	1.30	5.13	2.90	4.32	9.08	5.50	14.89	13.31	13.71

Table 3a. Forecasting the number of male and female cases of coronary heart disease (CHD) in India

	20–29 years		30–39 years		40–49 years		50–59 years		60–69 years		Total	
Year/area	Males	Females	Males	Females	Males	Females	Males	Females	Males	Females	Males	Females
2000												
Urban	1,455,131	1,256,370	1,339,042	1,295,977	1,361,514	1,415,460	1,312,833	975,578	994,606	893,593	6,463,126	5,836,978
Rural	1,058,905	740,786	1,489,249	1,364,997	1,153,049	2,189,424	1,169,136	2,421,749	1,652,462	1,501,050	6,522,801	8,218,007
Total	2,514,036	1,997,156	2,828,291	2,660,975	2,514,563	3,604,884	2,481,969	3,397,327	2,647,068	2,394,643	12,985,927	14,054,985
2005												
Urban	2,247,815	1,890,230	1,919,481	1,950,422	1,877,488	2,239,342	1,765,436	1,405,885	1,369,542	1,213,248	9,179,762	8,699,127
Rural	1,214,812	797,551	1,921,319	1,462,498	1,417,363	2,799,839	1,387,072	3,157,902	1,992,880	1,856,665	7,933,445	10,074,454
Total	3,462,627	2,687,781	3,840,800	3,412,920	3,294,851	5,039,181	3,152,508	4,563,787	3,362,421	3,069,913	17,113,207	18,773,581
2010												
Urban	3,300,543	2,691,869	2,726,826	2,427,940	2,527,688	3,079,042	2,386,347	1,836,925	1,975,701	1,735,237	12,917,106	11,771,013
Rural	1,382,426	942,347	2,397,833	1,542,889	1,672,365	3,695,431	1,613,193	4,204,170	2,463,869	2,366,054	9,529,686	12,750,891
Total	4,682,969	3,634,215	5,124,660	3,970,829	4,200,054	6,774,473	3,999,541	6,041,095	4,439,569	4,101,291	22,446,792	24,521,903
2015												
Urban	4,188,235	3,979,689	4,030,177	3,897,668	3,365,044	5,128,419	3,206,923	2,949,166	2,844,931	2,502,044	17,635,310	18,456,987
Rural	1,396,918	927,169	3,027,329	1,496,368	1,909,041	3,907,547	1,802,591	5,049,459	2,952,060	2,961,564	11,087,939	14,342,107
Total	5,585,153	4,906,858	7,057,506	5,394,036	5,274,085	9,035,966	5,009,515	7,998,625	5,796,991	5,463,608	28,723,249	32,799,094

protein that increases the risk of heart disease later in life (HT 2004). Our projections based on trends in prevalences have an inbuilt provision to take care of such changes.

Using these projected prevalence rates onto the projected population gives the number of cases as shown in Tables 3a and b. The estimate for the year 2000 is nearly 2.7 crore cases of CHD which more than doubles to nearly 6.1 crore cases in the year 2015. The pattern across age groups is nearly the same (Fig. 5). More than half of this rise can be ascribed to demographic changes but the contribution of increased prevalence of risk factors is also substantial.

Trends in the prevalence of other cardiovascular diseases

Appropriate area–gender-wise data are not available for stroke, rheumatic heart disease (RHD) and congenital heart disease. Age group-wise information is available only for stroke. Since RHD is primarily a disease of childhood and congenital heart disease is seen in infants, some workable projections could still be obtained. In the absence of any worthwhile information, it would be statistically incorrect to interpolate to males–females and rural–urban areas, and to younger age groups.

Trends in the prevalence of stroke revealed by various

Table 3b. Forecasting the number of cases (males and females) of coronary heart disease (CHD) in India

Year/area	20–29 years	30–39 years	40–49 years	50–59 years	60–69 years	Total
2000						
Urban	2,711,501	2,635,019	2,776,974	2,288,412	1,888,199	12,300,104
Rural	1,799,691	2,854,247	3,342,472	3,590,885	3,153,512	14,740,808
Total	4,511,192	5,489,266	6,119,446	5,879,296	5,041,711	27,040,912
2005						
Urban	4,138,045	3,869,904	4,116,830	3,171,320	2,582,790	17,878,889
Rural	2,012,363	3,383,816	4,217,201	4,544,974	3,849,544	18,007,899
Total	6,150,408	7,253,720	8,334,032	7,716,294	6,432,334	35,886,789
2010						
Urban	5,992,412	5,154,766	5,606,731	4,223,273	3,710,938	24,688,119
Rural	2,324,772	3,940,722	5,367,797	5,817,363	4,829,922	22,280,577
Total	8,317,184	9,095,489	10,974,527	10,040,636	8,540,860	46,968,695
2015						
Urban	8,167,924	7,927,846	8,493,463	6,156,089	5,346,975	36,092,297
Rural	2,324,087	4,523,697	5,816,588	6,852,050	5,913,624	25,430,046
Total	10,492,011	12,451,542	14,310,051	13,008,140	11,260,599	61,522,343

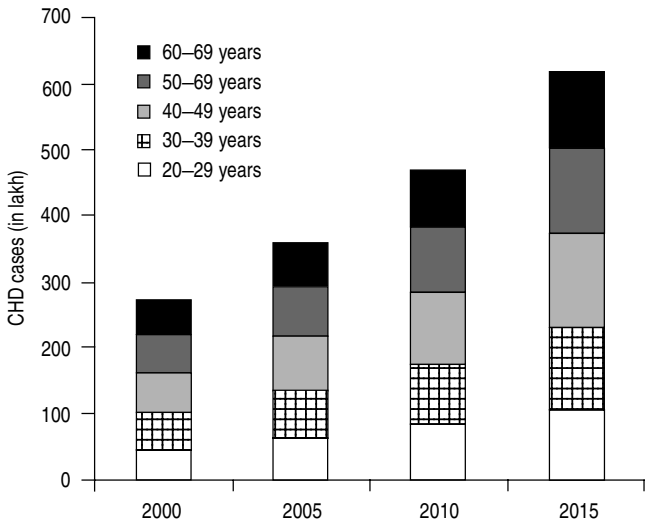

Fig. 5 Estimates and trends of coronary heart disease (CHD) cases in various age groups

studies in different areas in different years are shown in Fig. 6. The value of R^2 is too small and the trends are weird: decreasing and reaching to zero by the year 2015 in the 20–39 and 40–59 years age groups but increasing to 11.5 per 1000 in the 60–69 years age group. Because of these highly unstable features, we ignored the time trend and instead depended only on the age group-specific averages. Available data did not allow study of the 10-year age intervals. These average prevalences were used on the projected population of different ages to forecast the number of cases (Table 4). The total number of stroke cases estimated for the year 2000 are 1,081,000 and projected for the years 2005, 2010 and 2015 are 1,248,000, 1,451,000 and 1,667,000, respectively.

According to the data supplied to us, the average prevalence of RHD in the assumed age group of 6–16 years is 2.935 per 1000 children (Table 5). This age group does not conform to the standard age groups for which population data are readily available—thus the size was worked out by additional calculations for relevant proportions. The fewer cases in the year 2010 reflect a slight decrease in population of those 6–16 years of age by that year because of the ongoing demographic transition.

Congenital heart disease afflicts newborns and the number of cases can be easily projected on the basis of expected live-births in the next 10–15 years. For this we studied the trend in the birth rate over the past 30 years (1971–2000) for which we relied on data from SRS reports. Since 30 data points were available it was possible to examine the adequacy of fit of various curves. Figure 7 shows the results of linear, quadratic and cubic fit to the birth rate data. Cubic fit projected a birth rate of nearly 2 per 1000 population for the year 2015, and quadratic fit a rate of nearly 16. Both are absurd and were discarded. The linear fit forecast for birth rate is 24.99 for the year 2005, 23.29 for the year 2010, and 21.58 for the year 2015. These estimates seem

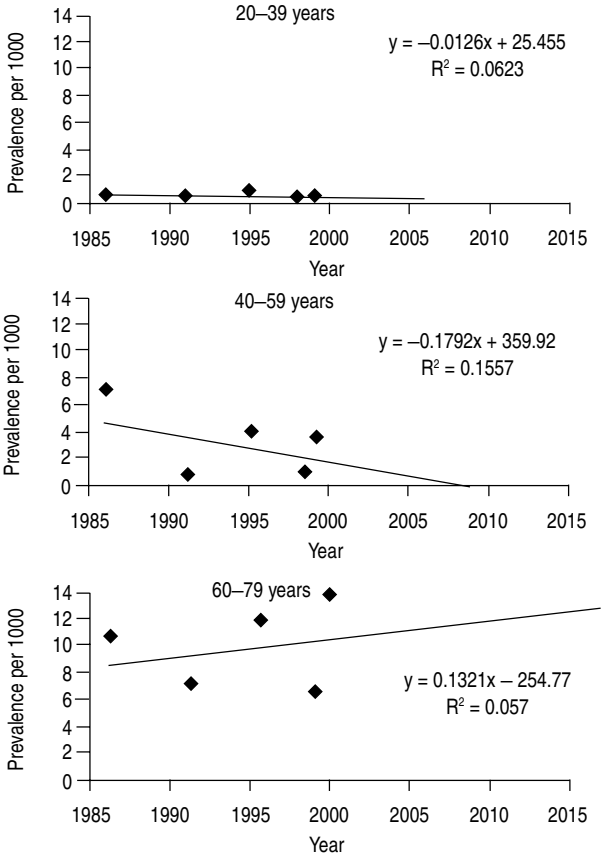

Fig. 6 Linear trend in the prevalence of stroke

Source of data: Centre for Chronic Disease Control

Table 4. Forecasting of cases of stroke

Year/ age	Estimated prevalence of stroke per 1000	Estimated population	Estimated cases
2000			
20–39	0.3022	306,904,000	92,746
40–59	2.7188	168,223,000	457,365
60–79	8.4733	62,711,000	531,369
Others		464,304,000	
Total		1,002,142,000	1,081,480
2005			
20–39	0.3022	346,437,000	104,693
40–59	2.7188	196,422,000	534,032
60–79	8.4733	71,883,000	609,086
Others		468,027,000	
Total		1,082,769,000	1,247,812
2010			
20–39	0.3022	392,531,000	118,623
40–59	2.7188	227,674,000	619,000
60–79	8.4733	84,168,000	713,181
Others		463,688,000	
Total		1,168,061,000	1,450,804
2015			
20–39	0.3022	428,582,000	129,517
40–59	2.7188	258,731,000	703,438
60–79	8.4733	98,476,000	834,417
Others		466,053,000	
Total		1,251,842,000	1,667,372

Table 5. Forecasting of cases of rheumatic heart disease

Year/age	Prevalence of RHD per 1000	Estimated population	Estimated cases
2000			
6–16	2.935	260,496,200	764,556
Others		741,645,800	
Total		1,002,142,000	
2005			
6–16	2.935	253,666,000	744,510
Others		829,103,000	
Total		1,082,769,000	
2010			
6–16	2.935	241,827,000	709,762
Others		926,234,000	
Total		1,168,061,000	
2015			
6–16	2.935	245,489,600	720,512
Others		1,006,352,400	
Total		1,251,842,000	

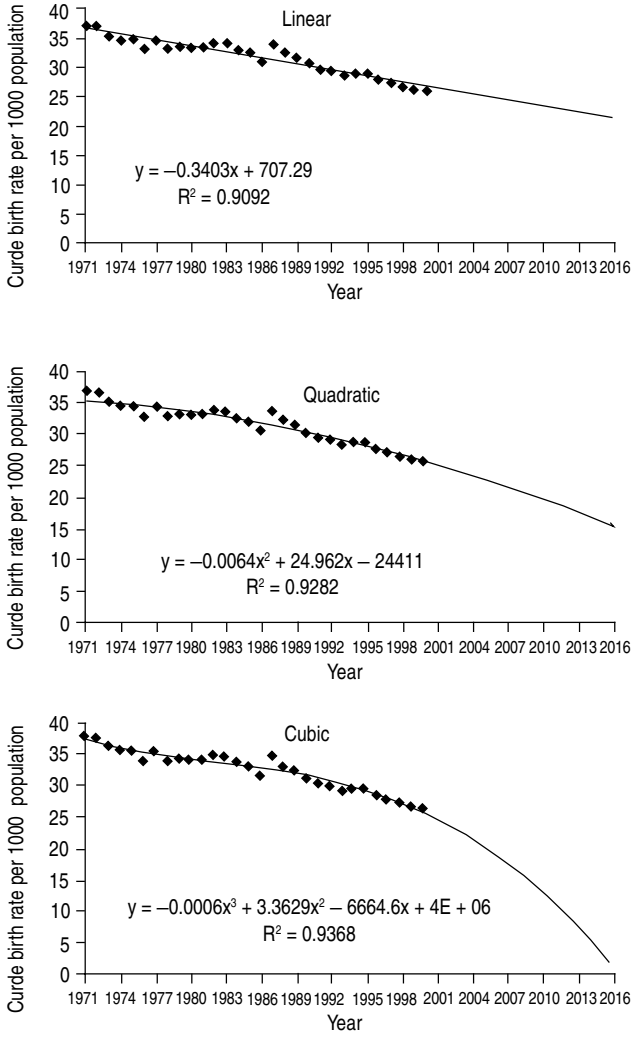

Fig. 7 Linear, quadratic and cubic fit to the birth rate data

Table 6. Forecasting of cases of congenital heart disease

Year	Estimated population	Birth rate per 1000	Number of live-births	Estimated prevalence per 1000 live-births	Estimated cases
2000	1,002,142,000	25.8*	25,855,264	5.98667	154,787
2005	1,082,768,000	24.9885	27,056,748	5.98667	161,980
2010	1,168,062,000	23.2870	27,200,660	5.98667	162,841
2015	1,251,841,000	21.5835	27,019,110	5.98667	161,754

*From Sample Registration System Survey 2000

plausible. R^2 for linear fit also exceeded 90%.

The estimated average prevalence of congenital heart disease as revealed by the data supplied to us is nearly 6 per 1000 live-births. No data were available to project the trend. Thus this rate was used on the projected births to get the projection of cases. The estimates for the years 2000, 2005, 2010, 2015 are given in Table 6.

A summary of the estimated prevalence rates of stroke, RHD and congenital heart disease is given Table 7.

Table 7. Prevalence rate per 1000 for stroke, rheumatic heart disease (RHD) and congenital heart disease

	Stroke (age in years)			RHD (age in years)	Congenital heart disease per
Year	20–39	40–59	60–79	6–16	1000 live-births
2000–2015	0.302	2.719	8.473	2.935	5.987

Note: No time trend could be detected from the available data. Thus their revalence rates have neither decreased nor increased.

Estimates of mortality

Death is the only certainty in life. It can only be postponed, not denied. If I do not die of tuberculosis, I may die of cancer. Various causes of death compete with one another, and one replaces the other. This has raised the question of what causes should be prevented and what should be promoted for death in old age (Indrayan 2001), while efforts are made to prevent all deaths in young age.

CVDs are extremely important in the context of epidemiological transition. Chronic diseases of old age are the major causes of death as the life expectancy increases and as communicable and undernutrition-based diseases respond to control efforts. Khor (2001) projected that non-communicable diseases including CVDs are expected to account for 7 out of 10 deaths in developing countries in the year 2020 compared to less than half in the year 2001. This is a likely scenario for India too.

Mortality due to coronary heart disease

Data on CVD mortality are even more scanty. A hospital-based study in Ahmedabad found a mortality rate of

Table 8. Estimated mortality from coronary heart disease (CHD)

	Deaths in age group (years) (4%)			Deaths in age group (years) (6%)		
Year	20–29	30–39	40–49	50–59	60–69	Total deaths
2000	180,448	219,571	244,778	352,758	302,503	1,300,057
2005	246,016	290,149	333,361	462,978	385,940	1,718,444
2010	332,687	363,820	438,981	602,438	512,452	2,250,378
2015	419,680	498,062	572,402	780,488	675,636	2,946,268

5.39% among cases of MI and 1.83% among cases of angina pectoris in the year 1996–97. One study in Karnataka reported a case-fatality rate of only 0.96% in a 3-year follow-up whereas it was 12.35% within 6 weeks of hospital stay in Chennai in the year 1970. Yearwise information is not available to evaluate the trend. However, it is exponentially declining in the UK (www.dh.gov.uk/PolicyAndGuidance/HealthAndSocialCareTopics CoronaryHeartDisease/fs/en), and also in the USA (www.khi.org/transfers/Marks.ppt). In the long run, the decline in India may be steeper because the technology is already available. The present evidence, through weak, suggests an average mortality of 4% in the age group of 20–49 years and 6% in those 50+ years due to CHD. This may remain so till the year 2015 if the current situation continues for the next 10–15 years. Based on this premise, the estimated mortality due to CHD is given in Table 8. This estimate is 1,300,000 for the year 2000, and the projection for the year 2015 is 2,946,000 deaths (Table 8).

Mortality due to other cardiovascular diseases

The case-fatality rate due to stroke varies from 11.7% to 32.4%. Stroke is a serious condition and the mortality is high. A case-fatality rate of 25% could be the average (Table 9). The case-fatality rate of RHD was supplied to us from two studies. In Haryana, this was 16% in 1999, and in Cuttack, 11.2% in 1981–90 and 8.3% in 1991–2000. This decline could be real because of the advanced technology now available in hospitals to save lives. Further reduction in case fatality seems highly unlikely. If a mortality of 8% is estimated for RHD cases, the number of deaths due to this cause are estimated as 61,000 for the year 2000; 60,000 for the year 2005; 57,000 for the year 2010; and 58,000 for the year 2015 (Table 10). No data were available on mortality from congenital heart disease.

The mortality estimates are derived from hospital-based studies and these may not be truly representative of community conditions. This is because health care services are not available to a large percentage of cases; as a result, the statistics provide a higher estimate compared to other cases. On the other hand, higher estimates will be obtained if hospitals get only severe cases. Although hard data are not available, we expect that the two would nearly balance and case-fatality rate seen in hospitals would be nearly the same as in the general population.

Table 9. Estimated mortality from stroke

Year	Age (years)	Case-fatality rate (%)	Estimated cases	Estimated deaths
2000	20–79	25	1,081,480	270,370
2005	20–79	25	1,247,812	311,953
2010	20–79	25	1,450,804	362,701
2015	20–79	25	1,667,372	416,843

Table 10. Estimated mortality from rheumatic heart disease (RHD)

Year	Age (years)	Case-fatality rate (%)	Estimated cases	Estimated deaths
2000	6–16	8	764,556	61,165
2005	6–16	8	744,510	59,561
2010	6–16	8	709,762	56,781
2015	6–16	8	720,512	57,641

Summary

A summary of our projections is given in Table 11. A total of nearly 6.4 crore cases of CVD are likely in the year

Table 11. Summary of projections of cardiovascular disease (CVD) cases and deaths

Year	CHD	Stroke	RHD	Congenital heart disease	Total cases
2000	27,040,912	1,081,480	764,556	154,787	29,041,735
2005	35,886,789	1,247,812	744,510	161,980	38,041,090
2010	46,968,695	1,450,804	709,762	162,841	49,292,102
2015	61,522,343	1,667,372	720,512	161,754	64,071,981
			Deaths		
Year	CHD	Stroke	RHD	Congenital heart disease	Total deaths (CHD+stroke +RHD)
2000	1,300,057	270,370	61,165	No data available	1,631,591
2005	1,718,444	311,953	59,561	No data available	2,089,958
2010	2,250,378	362,701	56,781	No data available	2,669,860
2015	2,946,268	416,843	57,641	No data available	3,420,752

CHD: coronary heart disease, RHD: rheumatic heart disease

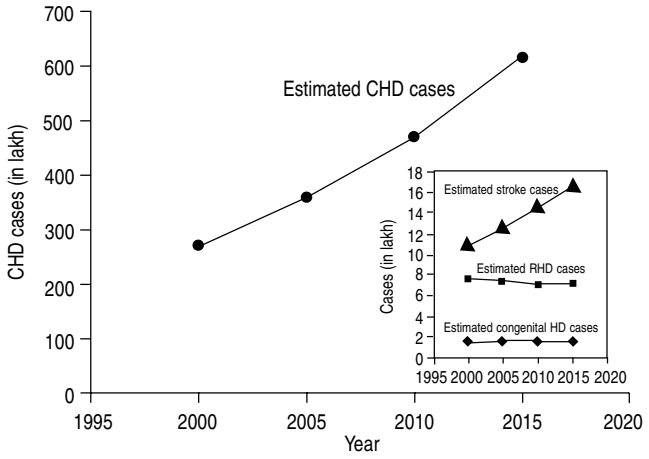

Fig. 8 Estimated trends of cases due to cardiovascular disease (CVD)

CHD: coronary heart disease; RHD: rheumatic heart disease

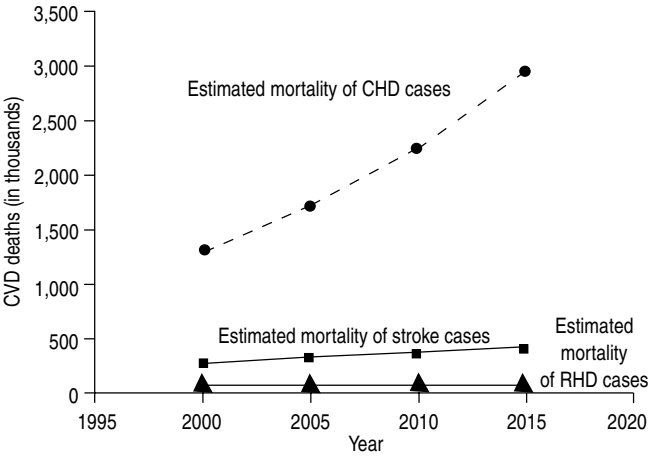

Fig. 9 Estimated trends in mortality due to cardiovascular disease (CVD)

2015, of which nearly 96% would be CHD cases (Fig. 8). Deaths from this group of diseases are likely to amount to be a staggering 34 lakh (Fig. 9).

Since the crude death rate is 8 per 1000 population even in many developed countries, it would not be wrong to assume that India has also reached its limit, and this rate would continue to be static in the near future. If so, nearly 1 crore deaths would occur in the year 2015. Thus, CVD is expected to contribute to nearly one-third of the mortality pie if the previous trend continues (Table 12).

The economic cost of CVD per case may not be staggering at present but future technology would be expensive. Given the finite resources, the management of CVD at the macro level will pose a tougher challenge.

Table 12. Deaths due to cardiovascular disease (CVD) as a percentage of total deaths

Year	Population	Crude death rate per 1000	Total deaths by all causes	Total CVD deaths	Percentage of CVD to total deaths
2000	1,002,142,000	8	8,017,136	1,631,591	20.35
2005	1,082,768,000	8	8,662,144	2,089,958	24.13
2010	1,168,062,000	8	9,344,496	2,669,860	28.57
2015	1,251,841,000	8	10,014,728	3,420,752	34.16

References

Boyle JP, Honeycutt AA, Venkat Narayan KM, *et al.* Projection of diabetes burden through 2050: Impact of changing demography and disease prevalence in the US. *Diabetes Care* 2001;**24:**1936–40.

GenX eating dangerously. *Hindustan Times*, 25 September 2004, p.1.

Indrayan A. Can I choose the cause of my death? *BMJ* 2001;**322:**1003.

Khor GL. Cardiovascular epidemiology in Asia-Pacific region. *Asia Pac J Clin Nutr* 2001;**10:**76–80.

Registrar General of India. *Population projection for India and States, 1996–2016.* New Delhi: Registrar General; 1996:91–4.

Singh SP, Sen P. Coronary heart disease: The changing scenario. *Indian J Prev Soc Med* 2003;**34:**74–80.

Wilson PWF, D'Agostino RB, Levy D, Belanger BS, Silbershatz H, Kannel WB. Prediction of coronary heart disease using risk factor categories. *Circulation* 1998; **97:**1837–47.

Limitations of this exercise in forecasting mortality due to CVD

- Since all diseases are not considered in this exercise, there is a possibility of over-counting. The total mortality from all causes put together should not exceed the deaths calculated from the crude death rate. In the absence of other diseases from this exercise, such epidemiological consistency could not be ensured. It is possible that a case of diabetes dying of MI is counted twice—once in diabetes and again in CHD.
- Different studies use different criteria for identifying or labelling a patient of CVD. For example, one study has used only ECG whereas another has used clinical history. We have ignored this distinction because of lack of data with uniform criteria.
- This exercise does not include the population in the age group of 70+ years for CHD because of lack of data for this age group in whom the prevalence and death rates may be high. However, only 3% of the population in India is in this age group, and the total cases or deaths in terms of absolute numbers may not be much affected.
- Our projections are based on very few data points, which could affect their reliability.
- Only linear trends could be explored because of lack of data.
- Some CVDs such as arrhythmias and inflammatory heart disease (carditis and cardiomyopathy) may be excluded from this exercise. No data were supplied to us on these conditions.

Appendix 1

Projections for the prevalence of diabetes

Since diabetes is a risk factor for CVD, the information supplied to us contained data points on the prevalence of diabetes in various age–gender groups for various years since 1990. These are plotted in Fig. A1.1 for males and females of various age groups. No time trend could be detected. The value of R^2 is very small except for males 50–59 years of age.

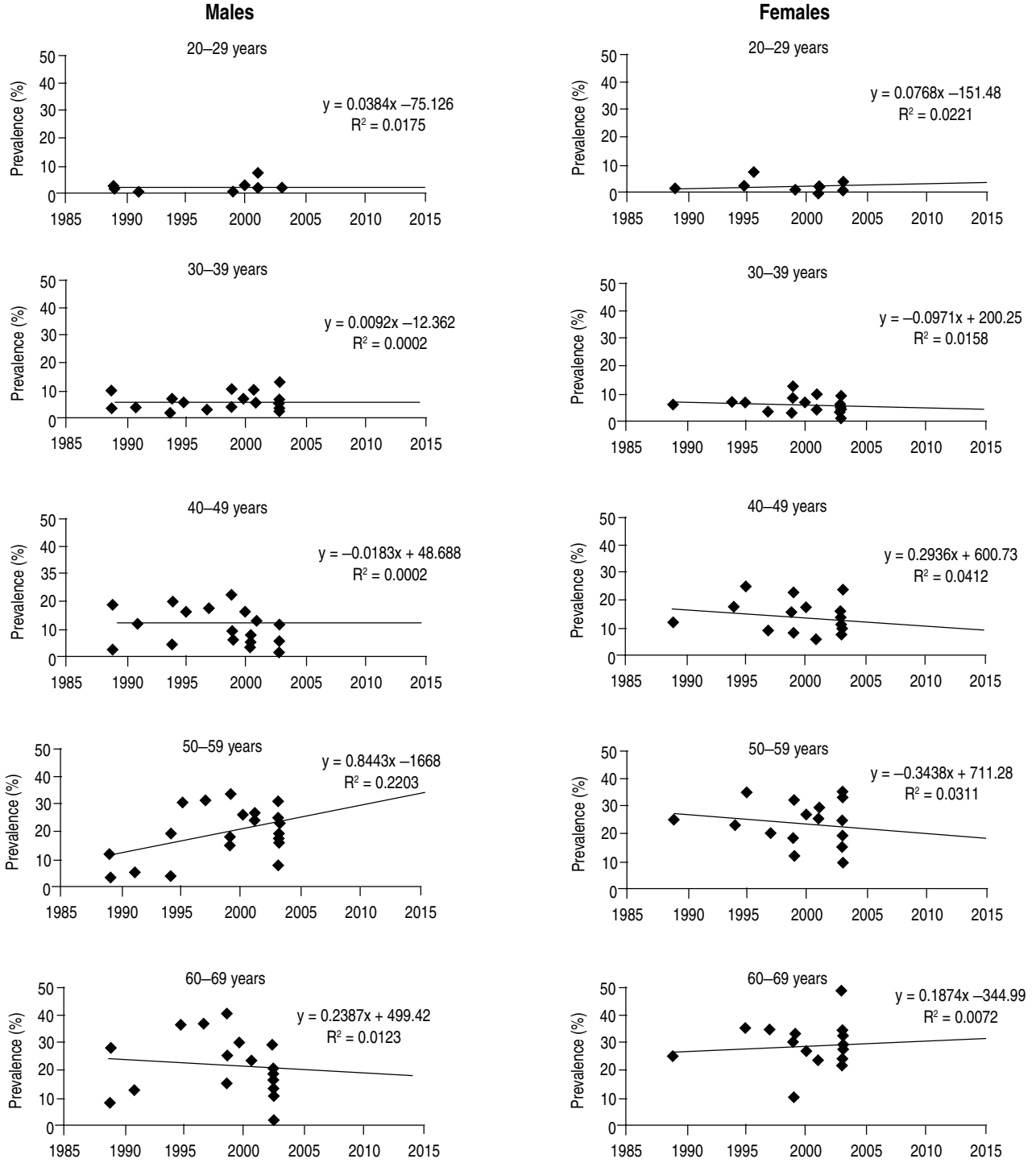

Fig. A1.1 Absence of a trend in the prevalence of diabetes

Source of data: Centre for Chronic Disease Control

Table A1.1 Prevalence of diabetes in rural and urban areas of India

State	Year	Age group	Prevalence (%)				References
			Male		Female		
			Urban	Rural	Urban	Rural	
Delhi	1991–94	35–44	8.1	1.7	7.0	2.5	ICMR Task Force Project on
		45–54	19.6	3.7	17.5	1.6	Collaborative Study of Coronary
		55–64	18.8	3.9	23.3	4.9	Heart Study
Tamil Nadu	1989	20–24	—	—	2.0	—	
		25–34	1.1	1.9	0.6	—	Ramachandran A, Snehalatha C,
		35–44	10.5	3.8	5.7	0.9	Dharmaraj D, Viswanathan M.
		45–54	18.5	1.6	12.2	3.7	*Diabetes Care* 1992;**15:**1348–55
		55–64	11.8	3.6	25.0	1.7	

Under these circumstances the only plausible hypothesis is that the age–gender rates of diabetes have neither increased nor decreased in our population over the past 15 years or so, although diabetes might have increased due to ageing and urbanization of the population. This may remain so till the year 2015.

Table A1.2 Estimated prevalence rate of diabetes per 1000 in India

Area	20–29		30–39		40–49		50–59		60–69		70+	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
Urban	17.29	20.70	60.13	59.74	121.04	135.39	198.50	236.26	222.26	297.31	215.20	221.33
Rural	4.32	5.18	15.03	14.93	30.26	33.85	49.63	59.07	55.57	74.33	53.80	55.33

Since not many studies have been reported from rural areas, the best strategy in such data-deficient situations is to obtain the rural prevalence as a percentage of the urban prevalence. Comparable data from a few studies are given in Table A1.1. The data in Table A1.1 suggest that the rural prevalence could be nearly one-fourth of the urban prevalence. With this assumption, the age–gender-wise prevalence rates are as shown in Table A1.2. Females are probably affected more than males. Using these rates on the projected population gives the projected caseload of diabetes (Table A1.3). These projections take into account factors such as population growth, ageing and urbanization.

Diabetes mortality

No data on diabetes mortality were supplied to us.

Table A1.3a Forecasting the total number of male and female cases of diabetes in India

Year and area	Age groups						
	20–29	30–39	40–49	50–59	60–69	70+	Total
2000							
Urban	1,003,310	2,567,970	3,882,005	4,188,171	2,783,100	1,401,300	15,825,855
Rural	549,102	1,425,108	2,232,090	2,628,455	2,053,095	1,100,412	9,988,262
Total	1,552,412	3,993,077	6,114,095	6,816,626	4,836,195	2,501,712	25,814,117
2005							
Urban	1,209,725	3,126,311	4,968,478	5,394,860	3,445,602	1,807,951	19,952,927
Rural	609,128	1,518,041	2,488,845	2,966,586	2,237,319	1,267,086	11,087,005
Total	1,818,853	4,644,352	7,457,323	8,361,446	5,682,920	3,075,038	31,039,932
2010							
Urban	1,505,267	3,744,045	6,358,087	7,010,131	4,522,903	2,304,049	25,444,482
Rural	692,391	1,558,528	2,707,848	3,310,679	2,537,127	1,420,910	12,227,483
Total	2,197,658	5,302,573	9,065,935	10,320,810	7,060,030	3,724,959	37,671,965
2015							
Urban	1,695,361	4,776,839	7,989,191	9,182,478	6,002,731	2,961,490	32,608,091
Rural	704,444	1,658,043	2,793,878	3,628,810	2,837,214	1,578,669	13,201,058
Total	2,399,805	6,434,881	10,783,069	12,811,288	8,839,946	4,540,160	45,809,149

Table A1.3b Forecasting the total number of cases of diabetes in India

	Age groups													
	20–29		30–39		40–49		50–59		60–69		70+		Total	
Year/area	Males	Females	Males	Females	Males	Females	Males	Females	Males	Females	Males	Females	Males	Females
2000														
Urban	489,340	513,969	1,307,097	1,260,873	2,019,637	1,862,367	2,146,602	2,041,569	1,244,731	1,538,369	682,108	719,192	7,889,515	7,936,340
Rural	254,212	294,890	722,171	702,937	1,100,702	1,131,388	1,250,395	1,378,059	899,318	1,153,777	570,007	530,404	4,796,807	5,191,455
Total	743,552	808,859	2,029,268	1,963,809	3,120,340	2,993,755	3,396,997	3,419,629	2,144,049	2,692,146	1,252,115	1,249,596	12,686,322	13,127,795
2005														
Urban	595,186	614,540	1,570,223	1,556,088	2,494,733	2,473,744	2,763,170	2,631,690	1,560,699	1,884,903	864,764	943,188	9,848,775	10,104,152
Rural	291,641	317,487	764,895	753,146	1,206,827	1,282,018	1,396,782	1,569,803	984,801	1,252,518	646,823	620,264	5,291,769	5,795,236
Total	886,826	932,027	2,335,118	2,309,234	3,701,561	3,755,762	4,159,952	4,201,493	2,545,500	3,137,421	1,511,586	1,563,451	15,140,543	15,899,388
2010														
Urban	720,695	784,572	1,919,743	1,824,302	3,041,657	3,316,430	3,581,777	3,428,354	2,066,668	2,456,235	1,087,218	1,216,830	12,417,757	13,026,724
Rural	331,880	360,511	809,652	748,876	1,285,100	1,422,748	1,534,791	1,775,888	1,114,967	1,422,160	717,886	703,024	5,794,276	6,433,207
Total	1,052,575	1,145,083	2,729,394	2,573,179	4,326,757	4,739,178	5,116,568	5,204,242	3,181,635	3,878,395	1,805,105	1,919,854	18,212,034	19,459,931
2015														
Urban	778,096	917,265	2,490,225	2,286,613	3,700,020	4,289,171	4,623,746	4,558,732	2,750,189	3,252,543	1,394,365	1,567,125	15,736,642	16,871,449
Rural	335,359	369,085	887,454	770,589	1,336,632	1,457,246	1,625,247	2,003,563	1,232,085	1,605,130	798,437	780,232	6,215,214	6,985,844
Total	1,113,455	1,286,350	3,377,679	3,057,202	5,036,652	5,746,417	6,248,993	6,562,295	3,982,273	4,857,673	2,192,802	2,347,357	21,951,856	23,857,293

Appendix 2

Revision following the suggestion of the experts

The NCMH called a meeting of experts to review the draft report we had submitted earlier. Primarily, two revisions were suggested. We were asked to

—use population projection of the Registrar-General (R-G) instead of the US Census Bureau that we had used earlier. This revision has been done and is reflected in this final report. Due to the higher population estimates of the R-G, the corresponding estimates of CVD caseload and deaths have increased.

—remove the 1974 data point that is suspected to cause a steep rise in the regression line used for projecting the prevalence of CHD. It was expected that the estimates would come down when this point is deleted. The experts were of the view that the estimates seemed to be on the higher side.

The new graphs obtained after deleting the 1974 data point are shown in Figs A2.1 and A2.2 for urban and rural areas,

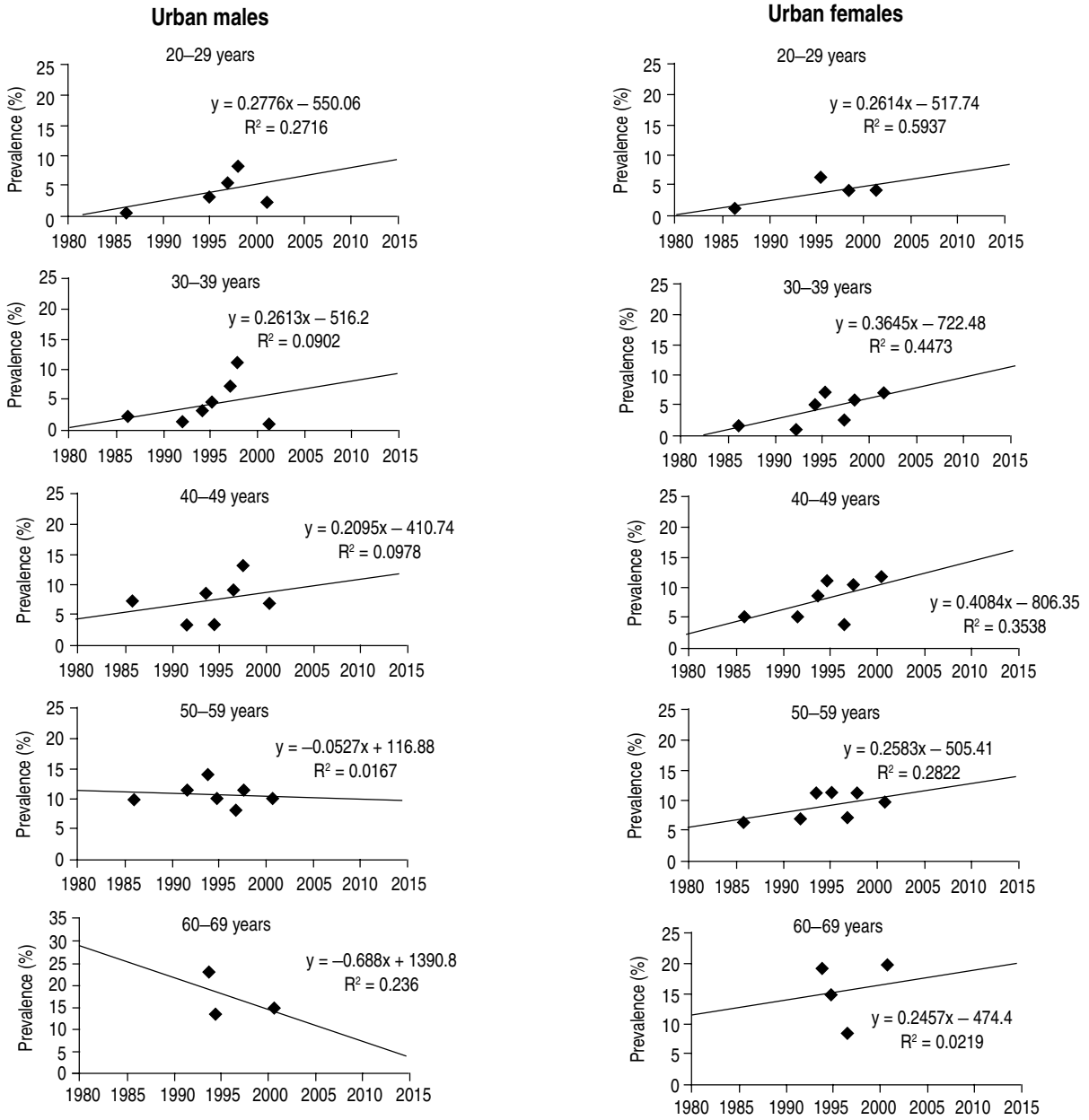

Fig. A2.1 Trends in the prevalence of coronary heart disease (CHD)—Urban (after excluding 1974 data point)

Source of data: Centre for Chronic Disease Control

respectively. Comparison of these with the previous graphs (Figs 3 and 4 in the main paper) show that this deletion had the reverse effect, particularly for rural areas. Table A2.1 has these estimates for each age group. When such 'revised' prevalence estimates were used on the estimated population, the projected CHD caseload for the year 2015 rose from nearly 6.15 crore estimated earlier to more than 6.60 crore (Table A2.2). Thus, deletion of the 1974 data point had a reverse effect of what was anticipated. We would like to stick to the estimates of 6.15 crore for the year 2015 provided in the main paper.

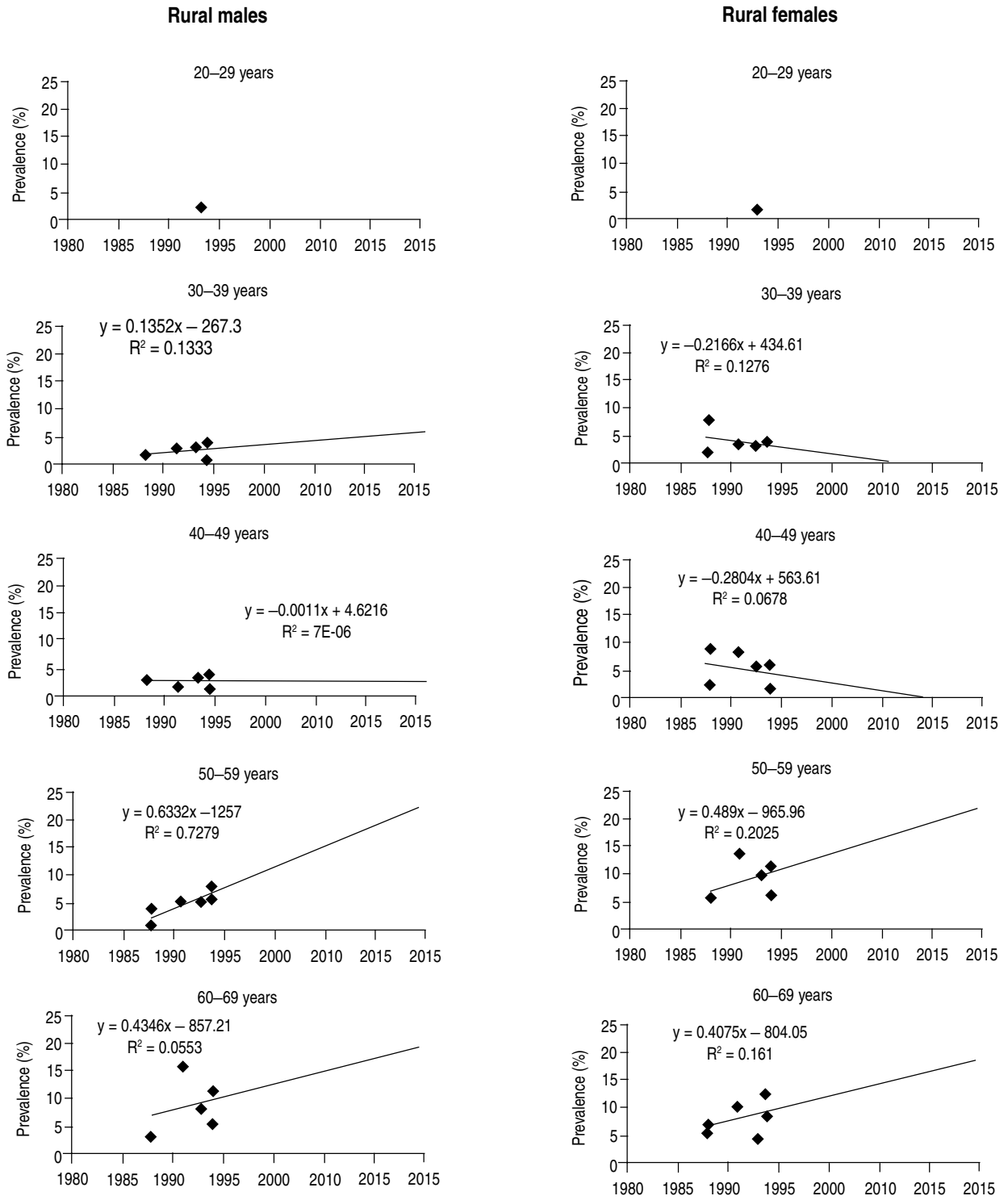

Fig. A2.2 Trends in the prevalence of coronary heart disease (CHD)—Rural (after excluding 1974 data point)

Source of data: Centre for Chronic Disease Control

Table A2.1 Forecasting the prevalence rate (%) of coronary heart disease (CHD) in India (after excluding 1974 data point)

Year and area	Age groups									
	20–29		30–39		40–49		50–59		60–69	
	Males	Females	Males	Females	Males	Females	Males	Females	Males	Females
2000										
Urban	5.14	5.06	6.39	6.52	8.26	10.45	11.00	11.19	14.81	17.00
Rural	1.80	1.30	3.10	2.90	3.45	5.50	9.40	12.04	11.99	10.95
2005										
Urban	6.53	6.37	7.70	8.34	9.31	12.49	11.00	12.48	14.81	18.23
Rural	1.80	1.30	3.78	2.90	3.45	5.50	12.57	14.48	14.16	12.99
2010										
Urban	7.92	7.67	9.00	10.17	10.36	14.53	11.00	13.77	14.81	19.46
Rural	1.80	1.30	4.45	2.90	3.45	5.50	15.73	16.93	16.34	15.03
2015										
Urban	9.30	8.98	10.31	11.99	11.40	16.58	11.00	15.06	14.81	20.69
Rural	1.80	1.30	5.13	2.90	3.45	5.50	18.90	19.38	18.51	17.06

Table A2.2a Forecasting the number of male and female cases of coronary heart disease (CHD) in India (after excluding 1974 data point)

Year/area	Age groups										Total	
	20–29		30–39		40–49		50–59		60–69			
	Males	Females	Males	Females	Males	Females	Males	Females	Males	Females	Males	Females
2000												
Urban	1,455,131	1,256,370	1,389,039	1,376,184	1,378,199	1,437,469	1,189,553	966,937	829,398	879,623	6,241,319	5,916,582
Rural	1,058,905	740,786	1,489,249	1,364,997	1,254,895	1,838,447	2,368,507	2,809,043	1,940,551	1,699,741	8,112,108	8,453,015
Total	2,514,036	1,997,156	2,878,288	2,741,182	2,633,095	3,275,916	3,558,060	3,775,980	2,769,949	2,579,364	14,353,427	14,369,597
2005												
Urban	2,247,815	1,890,230	2,009,835	2,173,142	1,918,296	2,282,463	1,531,228	1,390,290	1,039,936	1,155,651	8,747,110	8,891,775
Rural	1,214,812	797,551	1,921,319	1,462,498	1,375,887	2,083,213	3,536,920	3,849,706	2,510,130	2,188,549	10,559,068	10,381,518
Total	3,462,627	2,687,781	3,931,153	3,635,639	3,294,183	4,365,676	5,068,148	5,239,997	3,550,066	3,344,200	19,306,178	19,273,293
2010												
Urban	3,300,543	2,691,869	2,874,326	2,793,436	2,602,069	3,146,590	1,984,864	1,813,228	1,377,077	1,607,432	12,138,878	12,052,555
Rural	1,382,426	942,347	2,397,833	1,542,889	1,465,124	2,468,408	4,865,559	5,318,036	3,277,934	2,874,814	13,388,877	13,146,495
Total	4,682,969	3,634,215	5,272,159	4,336,325	4,067,194	5,614,998	6,850,423	7,131,264	4,655,011	4,482,247	25,527,755	25,199,049
2015												
Urban	4,188,235	3,979,689	4,269,548	4,588,588	3,485,481	5,251,339	2,562,278	2,906,717	1,832,525	2,262,955	16,338,067	18,989,288
Rural	1,396,918	927,169	3,027,329	1,248,555	1,523,876	2,367,949	6,189,202	6,572,166	4,104,081	3,684,679	16,241,406	14,800,519
Total	5,585,153	4,906,858	7,296,877	5,837,144	5,009,356	7,619,288	8,751,480	9,478,883	5,936,606	5,947,634	32,579,472	33,789,807

Table A2.2b Forecasting the total number of coronary heart disease (CHD) cases in India (after excluding 1974 data point)

Year and area	Age groups					Total
	20–29	30–39	40–49	50–59	60–69	
2000						
Urban	2,711,501	2,765,223	2,815,668	2,156,490	1,709,021	12,157,902
Rural	1,799,691	2,854,247	3,093,343	5,177,550	3,640,292	16,565,123
Total	4,511,192	5,619,470	5,909,011	7,334,039	5,349,312	28,723,025
2005						
Urban	4,138,045	4,182,976	4,200,759	2,921,518	2,195,587	17,638,885
Rural	2,012,363	3,383,816	3,459,100	7,386,627	4,698,679	20,940,585
Total	6,150,408	7,566,793	7,659,859	10,308,145	6,894,266	38,579,471
2010						
Urban	5,992,412	5,667,762	5,748,659	3,798,092	2,984,509	24,191,433
Rural	2,324,772	3,940,722	3,933,533	10,183,595	6,152,749	26,535,371
Total	8,317,184	9,608,484	9,682,191	13,981,687	9,137,258	50,726,805
2015						
Urban	8,167,924	8,858,137	8,736,819	5,468,994	4,095,480	35,327,354
Rural	2,324,087	4,275,884	3,891,825	12,761,368	7,788,760	31,041,925
Total	10,492,011	13,134,021	12,628,645	18,230,363	11,884,240	66,369,279

Suggested treatment for selected cardiac disorders at secondary-level health care facilities in low-resource countries

CENTRE FOR CHRONIC DISEASE CONTROL, NEW DELHI

Table 1. Suggested treatment for chronic cardiovascular disorders

Condition	Drug	Dose*	Remarks
Post-MI CHD (uncomplicated)	• Aspirin	• 75–150 mg	
	• ACE inhibitors	• Enalapril 10 mg or equivalent	• Needs individual titration
	• Beta-blockers	• Atenolol 50 mg or equivalent	• Needs individual titration
	• Statins	• Atorvastatin 10–20 mg or equivalent	• Needs individual titration
	• Nitrates	• Nitrates (may be required for post-MI angina)	• For symptomatic relief in case of chest pain
Chronic stable angina (uncomplicated)	• Aspirin	• 75–150 mg	
	• Beta-blockers	• Atenolol 50 mg or equivalent	• Needs individual titration
	• Nitrates	• As required	• For symptomatic relief in case of chest pain
	• Additional antianginal drugs, if required	• Calcium-channel blockers	• Needs individual titration
	• Statins	• Atorvastatin 10–20 mg or equivalent	• Needs individual titration
	• ACE inhibitors	• Enalapril 10 mg or equivalent	• Needs individual titration
Congestive heart failure (uncomplicated)	• ACE inhibitors or angiotensin II receptor blockers	• Maximally tolerated dose	
	• Beta-blockers	• Carvedilol/bisoprolol/metoprolol (maximally tolerated dose)	
	• Diuretics	• Frusemide (as required)	
	• Nitrates	• As required	
	• Digoxin		• Needs individual titration
	• Spironolactone	• 25–50 mg	• In advanced cases of heart failure
Hypertension (uncomplicated)	*(When pharmacological treatment is indicated)*		
	• Beta-blockers	• Atenolol 50 mg or equivalent	• Chosen depending on the underlying problem, combinations/multiple treatment may be required
	• Calcium-channel blockers	• Amlodipine 5 mg	
	• Thiazides	• Hydrochlorothiazide 25 mg or Indapamide or equivalent	• Dose titration required
	• ACE inhibitors	• Enalapril 10 mg or equivalent	
	• Angiotensin II receptor blockers	• Losartan 50–100 mg or equivalent	
RHD—mitral stenosis (uncomplicated)	• Antiarrhythmic drugs	• Digoxin 0.25 mg OD	• In case of atrial fibrillation, heart failure
	• Diuretics	• Frusemide 40 mg OD	
	• Secondary penicillin prophylaxis penicillin V/erythro-mycin orally if penidure NA	• Penidure 1.2 mU once in 3 weeks or	
	• Anticoagulants	• Warfarin/dicoumarol (dose titrated)	• In cases with atrial fibrillation or history of embolism
	• Balloon mitral valvotomy/ surgery	• Required in most as a therapeutic measure	

MI: myocardial infarction; CHD: coronary heart disease; ACE: angiotensin-converting enzyme; RHD: rheumatic heart disease

*Only suggested, can be modified as per the requirement/infrastructure and discretion of the physician

Table 2. Suggested treatment for acute cardiovascular disorders

Condition	Drug	Dose*	Remarks
Acute ST elevation MI (uncomplicated)	• Inj. streptokinase	• 1.5 mU once	
	• Nitroglycerine/nitrates	• As required for 24–48 hours	
	• Morphine	• 2–5 mg (3–4 times)	
	• Metoprolol	• 25–50 mg 2 times a day	• Needs individual titration
	• Aspirin	• 325 mg initial dose and later 150 mg OD	• Indefinite duration
	• Heparin	• 5000 units initial dose and 1000 units/hour (maximum) for 48–72 hours	• If required
	• Atorvastatin	• 10 mg OD	• Indefinite duration
	• Enalapril or any other equivalent ACE inhibitor	• 1.25–5 mg initially	• Titrated dose
Non-ST elevation acute coronary syndrome (uncomplicated)	• Heparin	• 24,000 IU/day for 24–72 hours	• Low molecular-weight heparin expensive but does not need monitoring
	• Inj. morphine	• 15–20 mg	
	• Inj. nitroglycerine	• 50 mg (total dose)	• Dosage depends on individual requirement
	• Metoprolol	• 50–100 mg BD	• Needs individual titration
	• Atorvastatin	• 10–20 mg OD	
	• Aspirin	• 150 mg OD	• Needs individual titration
	• Clopidogrel	• 75 mg OD	• Needs individual titration
Congestive heart failure—acute (uncomplicated)	• Oxygen inhalation	• As required	
	• Inj. frusemide	• As required (40 mg upwards)	
	• Inj. nitroglycerine	• Titrated dose	
	• Inj. dopamine/dobutamine	• As required for symptomatic relief	
	• Ramipril	• 5–10 mg or as tolerated	

MI: myocardial infarction; ACE: angiotensin-converting enzyme
*Only suggested, can be modified as per the requirement/infrastructure and discretion of the physician

Table 3. Requirements for the management of acute cardiovascular disorders

Condition	Staff requirement	Duration of stay	Investigations (number)	Other investigations	Remarks
Acute ST elevation MI, non-ST elevation MI (uncomplicated)	In an ICU with 10–15 beds, for each shift (2–3/day) • One resident physician—continuous • One nurse for 2 beds • Two orderlies • One ECG technician on call • One senior doctor on call	• In CCU 3–5 days • In hospital 5–7 days	• Cardiac enzymes (3) • Lipid profile (1) • Haemogram (1) • LFT (1) • RFT (1) • Coagulation parameters—as required • ECG (5 for whole duration) • ECHO (1 or as required) • Blood sugar (1)	At discharge TMT—1 (variable) ECHO—1 Angiogram—variable	50%–70% may require further referral for revascularization
Congestive heart failure due to valvular, hypertensive or ischaemic disease (uncomplicated)	In Intensive Care Unit with 10–15 beds, for each shift (2–3/day) • One resident physician—continuous • One nurse for 2 beds • Two orderlies • One ECG technician on call • One X-Ray technician on call • One senior doctor on call	5–7 days	• ECHO 1–2 • X-ray (1–3) • ECG (3) • Lipid profile (1) • LFT (1) • RFT (3) • Blood sugar (1) • Haemogram (1) • Cardiac enzymes (1)		

MI: myocardial infarction; ICU: intensive care unit; LFT: liver function tests; RFT: renal function tests; ECG: electrocardiogram

Table 4. Requirements for the management of chronic cardiovascular disorders

Condition	Staff requirement	Frequency of visits to OPD	Time taken in the OPD	Investigations	Frequency/year	Remarks
RHD—mitral stenosis (uncomplicated)	• One doctor • 1–2 nurses • 1 counsellor/dietician • 1 ECG technician • 2–3 orderlies	Once in 3 months	10 minutes	• ECHO • ECG • X-ray • Serum electrolytes • Prothrombin time	1 2 1 2 3–4 times or more in a year (if on anticoagulants)	Many will require referral to a tertiary-level health facility for therapeutic valvuloplasty
Chronic stable angina (uncomplicated)	• One doctor • 1–2 nurses • 1 counsellor/dietician • 1 ECG technician • 2–3 orderlies	2–4 visits a year (variable)	10 minutes	• ECG • X-ray • ECHO • Lipid profile • RFT • Serum electrolytes	2–3 times a year 1 variable 2/year (variable) 2–4 times/year (variable) 2–4 times/year (variable)	
Post-MI CHD (uncomplicated)	• One doctor • 1–2 nurses • 1 counsellor/dietician • 1 ECG technician • 2–3 orderlies	3–4 visits/year (variable)	10 minutes	• ECG • X-ray • ECHO • Lipid profile • RFT • Serum electrolytes	2–3/year (variable) 1 (variable) 2 2–3/year (variable) 2–3/year (variable) 2–3/year (variable)	Variable proportion (30%–40%) may referral for revascularization

RHD: rheumatic heart disease; ECG: electrocardiogram; RFT: renal function tests; MI: myocardial infarction; CHD: coronary heart disease

Cancer: Current scenario, intervention strategies and projections for 2015

M. KRISHNAN NAIR*, CHERIAN VARGHESE†, R. SWAMINATHAN‡

The term cancer refers to a group of diseases which share similar characteristics. Cancer can affect all living cells in the body, at all ages and in both genders. The causation is multifactorial and the disease process differs at different sites. Tobacco is the single most important identified risk factor for cancer. A host of other environmental exposures, certain infections as well as genetic predisposition play an important role in carcinogenesis. Diagnostic work-up, treatment methods and outcome of treatment are not uniform for all cancers. Advanced technology is required in many situations and ongoing research initiatives might lead to better understanding of the disease and its control.

The control of cancer requires the effective implementation of knowledge derived from more than two decades of successful research. It is now known that over one-third of cancers are preventable, and one-third potentially curable provided they are diagnosed early in their course. The quality of life of patients with incurable disease can be improved with palliative care.

A rational concept to put science into practice has to be formulated to counter this disease. In cancer, even with limited resources, an impact can be achieved if the right priorities and strategies are established and implemented.

The carcinogenic agents that people breathe, eat, drink and are otherwise exposed to, largely determine the occurrence of the disease. Personal habits such as the use of tobacco play a key role; people develop such habits in response to the social circumstances of life. Thus, the social origin of lifestyle must be considered in cancer prevention.

The high rates of cervical and breast cancers have created a higher cancer burden in women than men and hence these diseases are of major societal and familial consequence.

*Regional Cancer Centre, Thiruvananthapuram, Kerala
e-mail: mknairtvm@yahoo.com

†WHO India Office, New Delhi

‡Division of Epidemiology and Cancer Registry, Cancer Institute (WIA), Chennai 600020

Current scenario

India has a National Cancer Control Programme which was established in 1975–76. This has contributed to the development of Regional Cancer Centres (RCCs), oncology wings in medical colleges and support for purchase of teletherapy machines. The District Cancer Control Programme was initiated but did not result in sustainable and productive activity. Leading cancer sites in various cancer registry areas are shown in Fig. 1.

The present scenario is summarized as follows:

Cancer prevention

There is no uniform cancer prevention strategy for the entire country. Awareness programmes have been undertaken in a few places, but there is no uniform standardized information, education and communication (IEC) strategy for cancer prevention. There is no education on risk factors, early warning signals and their management. Cancer screening is not practised in an organized fashion in any part of India. There are sporadic attempts at opportunistic interventions and small-scale research studies for field interventions.

Infrastructure for diagnosis

Diagnostic infrastructure in the country is limited. There are many districts in the country which do not have a pathologist and pathology/cytology services, which are crucial for diagnosing cancer.

Financial and geographic constraints, and lack of manpower have contributed to the urban concentration of facilities. An unestimated number of cancers diagnosed in the population are not treated. Untreated patients are likely to demand more resources from society.

Cancer treatment

Treatment facilities are also mostly limited to urban areas of the country. There are no uniform protocols for manage-

The material presented in this publication are the views of the authors and do not imply the expression of any opinion whatsoever on the part of the organization they belong to.

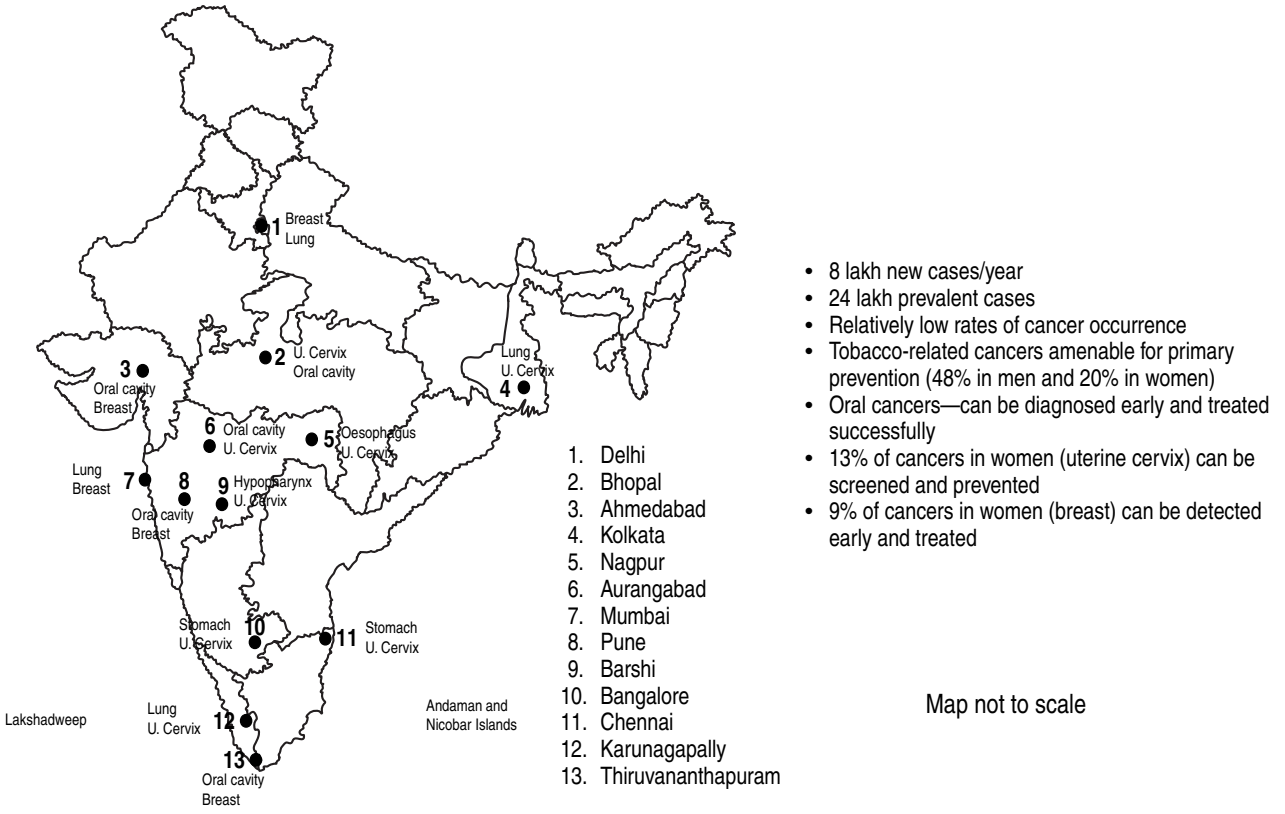

Fig. 1 India: Cancer pattern

ment, and the availability and affordability of cancer treatment shows wide disparities.

The majority of patients with cancer present to a cancer treatment centre in late stages of the disease (80% are advanced) and this adds to the already high morbidity, mortality and expenditure.

Treatment results are about 20% less than what is observed for similar conditions in more developed countries, mostly due to late diagnosis and inappropriate treatment.

Paediatric cancers are highly curable but this has not been achieved in India due to lack of access to quality care and lack of support systems.

Pain relief and palliative care

Oral morphine is the mainstay for cancer pain relief and is still not widely available in the country. There is a serious limitation of manpower for providing palliative care.

Finances

The funds for the cancer programme are mainly from the Government and needs to be augmented. Private initiatives are few and are unlikely to cater to a large population across different socioeconomic strata, as it is often not a financially viable venture.

Coordination

All elements of cancer control, from surveillance to palliative care, are not linked and coordinated.

Strategies for cancer control

A number of strategies can be considered for the control of cancer. An activity should only be introduced if data that strongly support its effectiveness are available, either from research programmes or cancer control programmes elsewhere.

Prevention

At least 30% of the future cancer burden is potentially preventable by tobacco control. Spread of tobacco addiction, promoted by commercial interests in the world, is responsible for the lung cancer epidemic that is already taking hundreds of thousands of lives annually; unless checked, cigarettes will in the next decade cause more than 1 crore deaths from cancer.

Action is also possible on dietary modification. Evidence that excessive fat in the diet may induce some cancers and that whole grains, vegetables and fruits are protective has accumulated in recent years. The same diet that lowers the risk of cardiovascular disease may inhibit the development of diet-associated cancers.

Table 1. Risk factors for cancer

Factor or factor class	Percentage of cancer deaths in the age group of 35–64 years caused by various factors Best estimate
Tobacco	30–40
Alcohol	3–10
Diet	Not known
Reproductive and sexual behaviour	10
Occupation	6–8
Pollution	2
Industrial products	1
Medicines and medical procedures	1
Geophysical factors	3

Excessive alcohol consumption increases the risk of cancers of the oral cavity, pharynx and oesophagus; it is also strongly associated with cancer of the liver.

Infections with certain viruses are associated with cancer; for example, liver cancer and the hepatitis B virus, and cancer of the cervix and the human papillomavirus. Hepatitis B vaccination can be undertaken in regions where the prevalence of chronic carriers exceeds 10%. Table 1 gives the risk factors for deaths due to cancer.

Early detection

If cancer can be detected early, treatment may be curative. One means to that end is educating people regarding early signs of the disease: lumps, sores that do not heal promptly, abnormal bleeding, and persistent indigestion or hoarseness. Medical attention should be sought when these occur. Early diagnosis of cancers that are curable if detected early (cervix, breast, mouth) can be promoted in India using public education and training of primary health care workers.

A second approach to early cancer detection is through population screening; namely, the identification of people with asymptomatic disease by applying simple tests. Cancer screening should be applied only when its effectiveness has been demonstrated; programmes should be introduced only when there is adequate manpower to perform the tests, with mechanisms to achieve adequate population coverage, facilities for diagnosis, treatment and follow-up of individuals with abnormal test results, and when the extent of disease in the population justifies the effort and cost. Currently, screening can only be advocated for cancers of the cervix and breast. It is important that such programmes concentrate on those at greatest risk of invasive cancer, for cervix cancer women aged 35–60 years, for breast cancer women aged 40 years or more (but for mammography programmes those aged 50–69 years).

Table 2. Curable cancers for which treatment is justifiable

Cancer	Load (%)	Primary modality
Childhood cancer	5	CT/S/RT
Breast	20	S/RT/CT/HT
Cervix	18	RT/S
Oral	11	RT/S
Gestational trophoblastic disease	1	CT
Germ cell tumours	3	CT/S
Colon	7	S/CT
Osteosarcoma	2	CT/S
Soft tissue sarcomas	2	S/RT
Central nervous system	2	S/RT
Total	71	

CT: chemotherapy; S: surgery; RT: radiotherapy; HT: hormone therapy

Treatment

The primary objectives of cancer treatment are cure, prolongation of useful life and improvement in the quality of survival. Mechanisms should be set up to decide on guidelines for integrating treatment resources with early diagnosis and screening programmes, and for providing therapeutic standards for the most important cancers in India.

Care for cancer patients typically starts with recognition or suspicion of the disease by the patient and primary health care worker. Specialized services for diagnosis and treatment, and referral, if appropriate, to a centre for cancer treatment comprise the next element of the system. Curative treatment involves surgery, radiation, chemotherapy, hormone therapy or some combination of these modalities. For some kinds of cancer, including those affecting the uterine corpus, testis, melanoma and female breast, state-of-the-art therapy yields a 75% or greater 5-year survival rate. On the other hand, survival for patients with cancers of pancreas, liver, stomach and lung is less than 15%. Though simple forms of cancer treatment have to be provided at a conservative level in medical colleges and district level hospitals, the high technology required for some forms of cancer therapy heighten the desirability of concentrating such treatment in a few places in the country. Table 2 lists the curable cancers in India.

Major reliance on treatment as a cancer control strategy, however, favours an expensive and narrow approach to the problem. High technology for cancer treatment imposes a heavy financial investment, tends to select patients inequitably, and detracts from appropriate emphasis on prevention. In the developing as well as developed world, focus on treatment as the main thrust against cancer is a poor strategy.

Palliative care

Having a good quality of life is a highly significant aim for patients with cancer, whether or not cure is possible. Cancer pain relief and palliative care are important and integral parts of cancer care. Relatively simple and inexpensive treatment to control pain should be available throughout the country as a priority. Palliative therapy and care, including symptom control and pain relief, will be important for years to come, especially in developing countries, because of the large number of patients for whom curative therapy is not possible. Guidelines for cancer pain relief have been produced and are available from the World Health Organization (WHO). Actions to ensure the availability of oral morphine through amendment of regulations that might inhibit the use of oral morphine for cancer pain relief, and training of health professionals in palliative care, are critical.

Interventions

Primary prevention

- The most useful prevention strategy is reduction in tobacco consumption (all forms). Currently about 50% of cancers in men and 20% of cancers in women are related to tobacco use. These cancers can be prevented to a large extent through a comprehensive tobacco control programme which will include awareness, education, legislation, community participation and tobacco cessation services.
- A healthy lifestyle, which includes eating plenty of fruits and vegetables, avoidance of alcohol and adequate physical activity, is protective for many of the non-communicable diseases including cardiovascular disease and diabetes, and can be considered as part of the overall health promotion programmes.
- Cancers related to infectious agents such as human papillomavirus and hepatitis B virus can be prevented through vaccination strategies.

Screening

- Screening is the application of a relatively simple and inexpensive test to asymptomatic subjects to classify them as being likely or unlikely to have cancer. A screening test in itself will not prevent cancer; it needs to be followed up through a systematic approach.
- Opportunistic screening or case finding can be attempted, but may not result in significant reduction in the incidence of cancer in a population as the coverage will be poor. However, it might help to increase the awareness and produce the human resources needed for future programmes, which include population-based screening in an organized manner with proper mechanisms for call–recall and quality control.
- At present, cytology-based Papanicolaou smear screening is the only proven strategy for cervical cancer and this can be undertaken in areas which are covered by cancer registries. Alternative strategies are being researched and might prove beneficial in certain settings.
- At present, mammography as a screening tool is not applicable to India. Breast cancer awareness can be propagated along with provision for fine-needle aspiration cytology, pathology services and surgical interventions. Once-a-year clinical breast examination can be made feasible for women above the age of 40 years, which can be carried out by general practitioners or trained health workers.
- Cancers in accessible parts of the body like the oral cavity may be detected at an early stage or even in a precancerous stage through simple inspection and examination, which can be practised by a trained health care worker.
- Self-examination of the oral cavity (MSE) and breast (BSE) can be useful methods and each can be propagated widely as a strategy.

Early detection, diagnosis and treatment/referral chain

- Cancer detection and diagnostic facilities have to be made available at medical colleges and district-level hospitals if they are to be accessible, and a clear referral chain should be established to ensure that those who require further treatment are referred to higher-care centres such as RCCs.
- Medical colleges should have provision for the management of all early common cancers.
- Dedicated centres need to be established for the management of paediatric cancers.
- RCCs should be equipped for comprehensive cancer control, treatment and research.

Palliative care

- Oral morphine has to be made available at the district level throughout the country. Various categories of health professionals need to be trained in the WHO step-ladder approach to pain management.
- Palliative care should be treated as an integral part of cancer management.

Figure 2 depicts the appropriate course and mix of curative and palliative care services and the relative positions of the two approaches.

Surveillance and monitoring

- The cancer registry programme has to be expanded and be made the monitoring component of the cancer control programme.
- At least 50% of the population has to be made aware

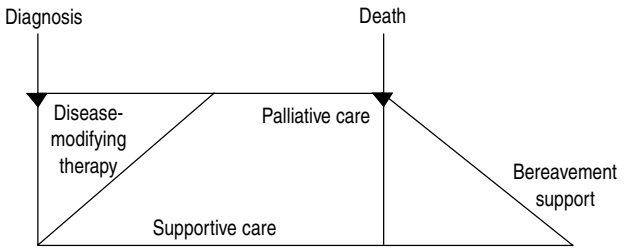

Fig. 2 The relationship between disease-modifying therapy, supportive care and palliative care

of the disease, its risk factors, prevention potential and curability.
- Paramedical personnel and field-level health workers have to be trained for providing awareness, documentation and ensuring compliance to referral and treatment.
- A comprehensive tobacco control programme must be implemented to reduce the prevalence of tobacco use by 10% from the current levels.

Vision 2015

- Affordable and accessible diagnostic, therapeutic and palliative care services should be made available in India.
- Tobacco control has to be strengthened and the present status of women and children as non-users of tobacco should be sustained at any cost.

Key components

Infrastructure and human resources

- Early detection of oral, breast and cervical cancers will have to be made possible through oncology wings in medical colleges and District Cancer Centres through augmentation of infrastructure and capacity enhancement.
- 300 more teletherapy machines will have to be made available in the country, taking into consideration the geographic gaps in the present distribution. Machines should be chosen in such a way that they are environmentally acceptable and recurring costs are minimal (a combination of cobalt and low-energy accelerators and simple brachy therapy [BT] equipment can be selected for this).
- Dedicated paediatric cancer treatment facilities will have to be established in all States.
- 1200 radiographers, 600 radiation oncologists and 300 radiation physicists will have to be made available; 300 surgeons and 300 physicians will need to be given re-orientation training in surgical and medical oncology.
- 2400 nurses, 600 doctors and pharmacists trained in pain relief and palliative care will have to be made available.
- Medical and surgical oncology training to be augmented and drug therapy for curable cancers including paediatric cases made available in all RCCs.

Drugs

- An essential drugs list of cancer chemotherapeutic drugs will have to be prepared and availability of all drugs in the essential list ensured. Protocol-based chemotherapy should be made available.
- Oral morphine will have to be made available in all districts of the country.

Surveillance and monitoring

- All RCCs should have two population-based registries, one covering the urban area and one for the rural area.

Community participation

- Community participation in the cancer programme can be ensured by having development committees for District Cancer Centres and oncology wings in medical colleges with people's representatives, religious leaders, teachers, etc.

Non-governmental organizations (NGOs) and civil society

- NGOs and civil society initiatives will have to be promoted and linked to cancer diagnostic and treatment centres.

Linkages

- Linkages with the Reproductive and Child Health Programme, National AIDS Control Programme, Nutrition Programme and Hepatitis B Control Programme will have to be established.

Finances

- Alternate sources of financing will have to be explored.

Channels for delivery of services

- RCCs/oncology wing of medical colleges/District Cancer Centres will have to be responsible for the delivery of all cancer-related services. Medical colleges will need to be restructured to enable oncology wings to function with more objectivity.
- All services at the district level will have to be provided by the District Cancer Centre—a charitable society spearheaded by the district administration. The district administration will thus become responsible for providing all services related to cancer in the community.

Estimate of cancer burden for 2004 and projection for 2015

Estimation of the incidence rate of cancer for the entire country in the year 2004 was done by selecting registries on the basis of data quality and location; selecting the

same time window, say 1997–98 as the mid-period; representation of urban and rural registries as available and taking their average (Tables 3 and 4).

Estimation of the population at risk of developing cancer was done by the exponential growth rate method, and using data from the Census of 1991 and 2001.

Estimation of the death rate for cancer was done by using mortality data of the Chennai and Mumbai registries only, based on data quality.

Estimation of prevalence was done by assuming the average duration of disease as 2.5 years.

Estimation of the cancer incidence for the entire country in the year 2015 is done by assuming a constant rate without any change as done for the year 2004 (Tables 5 and 6). The only difference will be in the estimated number of incident cases. Trend data in incidence rate have not been used for reasons of instability.

Estimation of the population at risk of developing cancer was done by the exponential growth rate method and using data from the Census of 1991 and 2001.

Estimation of the death rate of cancer cases by the same method as in 2004.

Estimation of the prevalence was done by the same method as in 2004.

Bibliography

1. National Cancer Registry Programme. *Two year report of the Population-based Cancer Registries 1997–1998. Incidence and distribution of cancer*. New Delhi: Indian Council of Medical Research; 2002.
2. *National cancer control programmes; policies and managerial guidelines*. 2nd ed. World Health Organization; 2002.

Table 3. Cancer incidence, prevalence and mortality: Estimate for India, 2004

	Male	Female	M+F
Incidence, all ages			
CIR/10^5	66.2	81.6	73.6
ASR/10^5	95.1	112.1	104.2
Cumulative risk (0–74 years)	One in 9	One in 8	One in 9
Incident cases	374,506	432,174	806,680
Prevalent cases	936,265	1,080,435	2,016,700
Incidence, 35–64 years			
CIR/10^5	119.5	176.5	153.7
ASR/10^5	155.1	234.3	202.6
Mortality			
CMR/10^5	51.8	46.4	49.1
Deaths	293,219	245,638	538,858

CIR: crude incidence rate; ASR: age standardized rate; CMR: crude mortality rate

Table 4. Common cancers in India, 2004

		CIR/10^5		ASR/10^5		Ratio at risk
Site of cancer	Incident cases (%)	All ages	35–64 years	All ages	35–64 years	0–74 years
Males						
Oral cavity*	40,700 (10.9)	7.2	17.6	10.3	21.5	1:81
Lung	34,983 (09.3)	6.2	9.8	9.4	15.2	1:85
Pharynx†	31,716 (08.5)	5.6	11.6	8.3	14.4	1:99
Oesophagus	24,729 (06.6)	4.4	9.7	6.6	11.9	1:123
Stomach	22,222 (05.9)	3.9	7.9	5.8	9.9	1:137
Females						
Cervix	112,609 (26.1)	21.3	57.4	28.5	70.7	1:32
Breast	90,723 (21.0)	17.1	39.7	22.8	53.1	1:40
Ovary	24,246 (05.6)	4.6	9.8	6.2	13.2	1:144
Oral cavity*	22,741 (05.3)	4.3	9.7	6.5	12.8	1:20
Oesophagus	17,220 (04.0)	3.3	6.8	5.0	8.7	1:163

CIR: crude incidence rate; ASR: age standardized rate

*Oral cavity includes lip, tongue, gum, floor of the mouth, cheek and palate

†Pharynx includes oro-, naso- and hypopharynx

Table 5. Cancer incidence, prevalence and mortality: Estimate for India, 2015

	Male	Female	M+F
Incidence, all ages			
CIR/10^5	66.2	81.6	73.6
ASR/10^5	95.1	112.1	104.2
Cumulative risk (0–74 years)	One in 9	One in 8	One in 9
Incident cases	461,681	536,772	998,453
Prevalent cases	1,154,203	1,341,930	2,496,133
Incidence, 35–64 years			
CIR/10^5	119.5	176.5	153.7
ASR/10^5	155.1	334.3	202.6
Mortality			
CMR/10^5	51.8	46.4	49.1
Deaths	361,474	305,000	666,563

CIR: crude incidence rate; ASR: age standardized rate; CMR: crude mortality rate

Table 6. Common cancers in India, 2015

		CIR/10^5		ASR/10^5		Ratio at risk
Site of cancer	Incident cases (%)	All ages	35–64 years	All ages	35–64 years	0–74 years
Males						
Oral cavity*	50,174 (10.9)	7.2	17.6	10.3	21.5	1:81
Lung	43,126 (09.3)	6.2	9.8	9.4	15.2	1:85
Pharynx†	39,098 (08.5)	5.6	11.6	8.3	14.4	1:99
Oesophagus	30,485 (06.6)	4.4	9.7	6.6	11.9	1:123
Stomach	27,395 (05.9)	3.9	7.9	5.8	9.9	1:137
Females						
Cervix	139,864 (26.1)	21.3	57.4	28.5	70.7	1:32
Breast	112,680 (21.0)	17.1	39.7	22.8	53.1	1:40
Ovary	30,114 (05.6)	4.6	9.8	6.2	13.2	1:144
Oral cavity*	28,245 (05.3)	4.3	9.7	6.5	12.8	1:120
Oesophagus	21,388 (04.0)	3.3	6.8	5.0	8.7	1:163

CIR: crude incidence rate; ASR: age standardized rate

*Oral cavity includes lip, tongue, gum, floor of the mouth, cheek and palate

†Pharynx includes oro-, naso- and hypopharynx

Mental, neurological and substance abuse disorders: Strategies towards a systems approach

GURURAJ G.*, GIRISH N.*, ISAAC M.K.†

Mental health has gained recognition as a major public health problem only in recent years. The National Mental Health Programme (NMHP) is being expanded to 100 districts from the existing 27 districts. This study estimates the burden of mental disorders, identifies causal mechanisms and lists the possible intervention strategies.

Mental disorders constitute a wide spectrum ranging from mild anxiety states to very severe forms of behavioural and thought abnormalities. The causes of mental disorders are varied and complex in nature, and vary from condition to condition. Despite newer technological understanding, the enigma of the causation of mental disorders continues as a complex interaction of the brain, mind and milieu.

Although India has a vast network of community health centres and primary health centres along with a rapidly growing private sector, there is a severe shortage of trained mental health professionals. Delivering cost-effective and meaningful mental health services in India requires reorientation and reorganization of existing health systems.

This report has been developed based on secondary data from available sources along with an in-depth review of existing databases in the Indian region. Only core disorders have been addressed in this report. The ICD-10 system of classification has been used. Suicide has not been included; it has been covered under the Injury Report of the Commission. Similarly, tobacco use is covered in a separate section of the report. Only epilepsy and dementia have been included under neurological disorders.

PART I

The burden and impact of mental disorders—the global scenario

Mental and behavioural disorders account for 12% of the global burden of disease. The WHO, in its *World Health Report 2001* has drawn attention to the fact that of the nearly 45 crore estimated to be suffering from mental and behavioural disorders globally, 'only a small minority' are adequately cared for. The spending in terms of the country's mental health budget does not exceed 1% of the total health expenditure. The global prevalence of mental and behavioural disorders among the adult population is estimated to be 10% and contributed to four of the ten leading causes of disability, with one in four families suffering the burden. Depression, anxiety and alcohol use were the commonest disorders in a primary care setting, contributing to nearly 20% of the caseload. It is estimated that by 2020, 15% of the disability-adjusted life-years (DALYs) lost would be due to mental and behavioural disorders, up from 10% in 1990 and 12% in 2000 (for details about disability-adjusted life-years [DALYs], please visit http://www.worldbank.org/html/extdr/hnp/hddflash/workp/wp_00068.html). The lifetime prevalence of developing one or more mental and behavioural disorders is estimated to be 25%. At the global level, considering the years lived with disability, unipolar depression stands out prominently. Putting together all neuropsychiatric conditions, the proportional contribution to the total years lived with disability was 23.7% (males: 24.2%, females: 24.9%). The burden is 40% if the most productive age group (15–44 years) is considered (WHO 2001). The poor, the homeless, the unemployed and persons with low education are at higher risk (Francisco 2004). Three areas of concern have been identified: (i) the real burden of mental and behavioural disorders, (ii) the human, social and economic costs of these, and (iii) the need for dismantling barriers in the provision of adequate services (WHO 2001).

Departments of Epidemiology* and Psychiatry†
National Institute of Mental Health and Neuro Sciences
Bangalore 560029
e-mail: guru@nimhans.kar.nic.in

Measuring the burden of mental disorders

Psychiatric epidemiology in India

Psychiatric epidemiology as a branch of psychiatry and public health investigates how mental disorders are distributed in the population and extends to identifying possible causes and measuring the impact of interventions. Dr Dube undertook the first major survey of psychiatric problems in 1961 at Agra, India (Dube 1970). This is considered a watershed event in the development of psychiatric epidemiology in India. The key feature of studies undertaken during 1960–80 was that they were descriptive, population-based studies of psychiatric morbidity in several parts of India. These studies dispelled several myths (Murthy 1987a). The studies undertaken in the late 1980s and during the 1990s focused on specific disorders in specific populations and in specific settings. There have been large-scale epidemiological studies in large populations on specific problems with methodological advancements focusing on issues of case definition, screening, diagnosis and classification (ICMR 1987, WHO and MOHFW 2003). Since the 1990s, the studies with their improved methodologies focused on new and emerging problems. This period saw a greater need for organizing services across the country and also established the incidence, course and outcome of schizophrenia as well as evaluation of interventions in mental health care. '… the period between 1950 and 2000 has witnessed tremendous growth of the discipline (psychiatric epidemiology) across the country with studies getting more and more refined and advancing clearly' (Gururaj *et al.* 2004).The recent publication *Mental Health—an Indian perspective 1946 to 2003* provides an overview of the major developments in post-Independent India (Agarwal 2004). Indian psychiatric epidemiological studies (focusing on all mental disorders) can be broadly classified as cross-sectional studies, interval studies, longitudinal studies, studies adopting the case–control approach, systematic reviews and meta-analysis (only one).

Total prevalence of major mental disorders

Several policy-making bodies in India have arrived at an estimate of their own, the precise basis of which is not clearly known. As early as 1911, Overbeck and Wright estimated the prevalence to be 26–28/1000 for the Indian population. Subsequently, the mental health advisory committee of India estimated the prevalence to be 2% of the total population (Chandrashekar and Isaac 1999).

Following the path-breaking effort by Dube in Agra, the majority of the classical Indian psychiatric epidemiology studies in the past four decades focused on general psychiatric morbidity in small-to-medium populations. The population samples were more often convenient samples. Chandrashekar and Isaac (1999) reviewing these studies reveal the wide variation in prevalence rates; ranging from 10–370/1000 population in different parts of the country. The reasons for this wide variation are several: factors such as selection of the study population (urban/rural/tribal); choice of method (door-to-door or hospital-based, two- or three-stage survey); case identification methods (layperson, trained health personnel, clinical psychologist, psychiatrist); different screening instruments (symptom checklists with different numbers; use of different standardized survey instruments; questionnaires; interview schedules [both structured and unstructured]); case ascertainment methods (use of different gold standards); different and unspecified case definitions and use of inappropriate statistical procedures. The individual studies along with select details are listed in Appendix 1.

Two recent studies have attempted to generate all-India prevalence rates. Reddy and Chandrashekar (1998) in a meta-analysis reported the total prevalence to be 58/1000 (confidence interval [CI] 55.7–60.7) with 48.9/1000 for the rural population and 80.6/1000 for the urban population. Ganguli (2000), reviewing major Indian studies, computed the total rate to be 73/1000 (range: 18–207). These studies utilized different inclusion and exclusion criteria and hence the number and type of studies included are not the same. Reddy and Chandrashekar (1998) reanalysed the original numbers from community-based studies undertaken as a door-to-door survey. A major limitation with this study is that different study designs with differing instruments were included. Ganguli (2000), computing the prevalence rates from region-representative major Indian studies, however, does not provide the basis for the computation. The two studies thus provide two different all-India prevalence rates (58/1000 population and 73/1000 population). This difference persists with the rural and urban rates. The difference in the rural–urban rates stands out prominently in the meta-analysis.

The prevalence rates (Appendix 1) across the regions of the north and east are similar, as are the rates in the south and west of India. Though the combined rates for the south are not available, they could be inferred from the individual urban and rural rates.

The only available longitudinal study (for all disorders) is over a one-year period (Nandi *et al.* 1976). This is thoroughly inadequate to document a change in mental morbidity. In such a situation, interval studies provide us with some insights with respect to the changing trend of the burden. Nandi *et al.* observed that there was no significant increase in the prevalence of total mental morbidity during 1972–1982 (Nandi *et al.* 1986) or 1972–1992 (Nandi *et al.* 2000). It was either decreasing or was 'stable'. Raghurami Reddy observed that there was no change in the total prevalence rate (Reddy *et al.* 1994). The worst-case scenario in this case would be assuming stable rates or a near-constant prevalence rate over the period of time. This finding is further corroborated when the trends of individual disorders such as schizophrenia also report static prevalence rates (Murthy 1999). Thus, it could be summarized that: despite a near-static prevalence rate over a period of time, the

number of people requiring mental health care would increase with a corresponding increase in the population.

Interestingly, Ganguli (2000) found a minor difference between overall rural and urban rates. When comparing the three sets of paired studies, it was observed that urban rates increased by a factor of 1.6 (range: 1.4–1.9), which implies that for every 100 rural persons with any mental disorder, there are 157 urban persons with a mental disorder. However, after computation, the difference in the final rates for rural and urban areas was 3.5 per 1000 population. Contrary to the above line of thinking by Ganguli, in the meta-analysis, the rural–urban differences noted by Reddy and Chandrashekar (1998) are striking, to the extent that urban rates (79.1/1000) are twice that of rural rates (37.1/1000 population).

In summary, the range of prevalence rates for major mental disorders from the available epidemiological literature is very wide. While extrapolating from one good study is inappropriate for deriving national estimates, pooling from a few incomparable studies is also incorrect. Hence, for purposes of estimating the number of persons with any mental and behavioural disorder in India, a median conservative estimate of 65/1000 population has been utilized. This estimate is the median value derived from the two studies of Reddy *et al.* (1998) and Ganguli (2000). Rates are higher in females by approximately 20%–25%. The overall individual burden for urban and rural areas cannot be estimated with the available studies. However, it may be surmised that the number of people affected with any mental and behavioural disorder from rural areas would be higher, corresponding to the proportion of the population living in these areas. Better estimates for age, sex and place of residence may be made after completion of the ongoing Indian component of the World Mental Health Survey (WHO and MOHFW 2003).

Schizophrenia

Schizophrenia has been recorded in Indian history nearly 3300 years back by Charaka (Rajkumar 1987). The major studies pertaining to schizophrenia from India are the International Pilot Study of Schizophrenia and Determinants on the outcome of severe mental disorders in India (WHO 1975; Jablensky 1995). The first large-scale study on prevalence and service evaluation was undertaken by ICMR–DST in four centres of India—Bangalore, Baroda, Calcutta and Patiala (ICMR 1987). More recently, longitudinal studies and those investigating the course and outcome have been undertaken in the country (ICMR–SOFACOS 1988; ICMR–SOFPUC 1990).

Prevalence

Beginning with the first epidemiological study by Govindaswamy during 1958–59 (Govindaswamy *et al.* 1959), several researchers have examined the prevalence and sociodemographic correlates of schizophrenia. The prevalence (Table 1) varied from 1.1 to 4.3 for schizophrenia in particular. The Chennai study, covering a large population of 101,229, observed the prevalence to be 2.5/1000 (Padmavathi *et al.* 1987). From an all-India perspective, the ICMR multicentre study (ICMR 1987) undertaken across four different regions of the India provides better methodological and research vigour (combined methods of case identification). The prevalence rate for schizophrenia varied from 1.8/1000 in Bangalore to 3.1/1000 in Patiala. The meta-analysis by Reddy and Chandrashekar (1998) estimated the prevalence of schizophrenia to be 2.7 (2.2–3.3)/1000 population, while Ganguli computed the prevalence to be 2.5 (1.1–14.2)/1000 population (Ganguli 2000). Against this backdrop, a rate of 3/1000 has been taken as the all-India prevalence of schizophrenia, the range being 2–3/1000 population.

The evidence for the prevalence in urban and rural areas is contrasting. The meta-analysis reports an urban preponderance (U: 2.9, R: 2.6 per 1000 population) while the systematic analysis reports a rural dominance (R: 3.6, U: 2.5 per 1000 population). The age–sex distribution reveals that men are affected more often compared with women. Rates ranging from 2.3 to 21.3 have been reported in males. Elnagar *et al.* (1971) observed higher rates among women (5.8). The pooled study indicates higher rates among women (3.2 as compared to 2.3 among men).

Incidence

The incidence of schizophrenia was estimated to be 4.6/10,000 population in urban and rural parts of Chandigarh (Wig *et al.* 1993). Nandi *et al.* (1976) noticed an incidence rate of 9.3/10,000 and Rajkumar *et al.* (1993) a rate of 3.5/10,000. Wig *et al.* (1993) found the highest incidence among rural males and females in the age group of 50–54 years. A crude incidence rate of 4/10,000 can be adopted for estimating the burden of schizophrenia in India.

Course and outcome

Under the aegis of the International Pilot Study of Schizophrenia, Dube *et al.* (1984) reported that 56% of the cases initially included were normal at the end of two years and the proportion rose to 67% over the 5 years of follow-up. At the end of the 13-year follow-up, 13% continued to suffer from the disease without remission. They conclude that the 'prospect of recovery is not as bleak as is generally believed'. In addition, they reported a higher mortality rate and also found that it was due to suicide alone. In the full version of the study, the International Study of Schizophrenia, Harrison *et al.* (2001) reported that a more favourable long-term outcome was achieved across the countries in a significant proportion and conclude that 'early intervention' programmes should be initiated to achieve the desirable objectives. Sharma *et al.* (1998) and Thara (2004b) reported

Table 1. Prevalence rates of schizophrenia in Indian studies

Author and year	Place	Sample size	Crude rate per 1000 population
All India			
Reddy and Chandrashekar 1998	Combined	33,572	2.7 (R-2.6, U-2.9)
Ganguli 2000	Combined	NA	2.5 (R-3.6, U-2.5)
East			
Nandi *et al.* 1992	Combined	1,424	1.5 (M-0; F-3.1)
Elnagar *et al.* 1971	Rural	1,383	4.33 (M-2.7 ; F-5.8)
Nandi *et al.* 1975	Rural	1,060	2.8
ICMR (Calcutta) 1987	Rural	34,582	2.05
Nandi *et al.* 2000	Rural	3,488	3.0
Nandi *et al.* 2000	Rural	2,183	4.0
North			
Dube 1970	Combined	29,468	2.17 (M-2.4; F-1.9)
Thacore *et al.* 1975	Urban	2,696	1.9
Sethi *et al.* 1972	Rural	2,691	1.1
Murthy *et al.* 1978	Rural	2,500	2.0
Sachdeva *et al.* 1986	Rural	1,989	2.0 (M-2; F-2)
ICMR (Patiala) 1987	Rural	36,595	3.09
South			
Verghese *et al.* 1973	Rural	26,039	2.6
Mehta *et al.* 1985	Rural	5,941	1.9 (M-2; F-1.7)
ICMR (Bangalore) 1987	Rural	35,548	1.83
Gopinath 1968	Rural	423	7.1
Shaji *et al.* 1995	Rural	1,094	3.6 (M-3, F-4)
Surya *et al.* 1962	Urban	2,731	1.5
Premarajan *et al.* 1993	Urban	1,115	2.5 (M-0; F-5.2)
Padmavathi *et al.* 1987	Urban	101,229	2.5 (M-2.9; F-2.1)
Vimala *et al.* 1998	Rural	32,000	1.9 (M-2; F-3)
West			
Shah *et al.* 1980	Urban	2,712	1.5
ICMR (Baroda rate) 1987	Rural	39,655	1.77
Sharma *et al.* 2001	Combined	4,022	14.2 (M-21.3; F-7.3)

Note: The male- and female-specific rates are given in brackets

that schizophrenics from India stand a better chance of recovery as they are 'more socially integrated'. Though studies from India have demonstrated that the course of the illness was more favourable, with a short duration of illness indicating better prognosis, the major problem is the huge treatment gap. In the *Country Report on Schizophrenia*, Murthy (1999) reviewed the burden of schizophrenia and observed that males were more disabled than females, drug compliance was significantly associated with all aspects of disability, home care was better than hospital care in reducing the burden and the quality of life was better among those who were employed and literate. Further, the urban population perceived a higher burden, and the major needs of patients and families were employment and vocational rehabilitation, while psychosocial rehabilitation and accommodation were a lesser priority.

Thara and Srinivasan (1998) examined the impact of medication alone and a combination of medication and psychosocial intervention on schizophrenics with moderate disability. They concluded in their one-year study that along with medication, psychosocial intervention brought about a significant improvement in work-related areas, under-activity, social withdrawal and participation in family life. Gururaj and Isaac (2004) observe that the better course and outcome of schizophrenia in the Indian region can be attributed to several factors such as low expressed emotions among relatives, greater tolerance, better quality of social support and lower expectations from patients. High attrition rates, related partly to greater mortality among schizophrenics, pose difficulty in inferring favourable outcomes. In addition, unlike in developed countries, multiple sources of care (psychiatric pluralism) in developing countries such as India is said to contribute to better outcomes (Halliburton 2004).

Mood disorders

ICD-10 classifies mood disorders into: manic episode, depressive episode, bipolar disorder, recurrent depressive disorder, persistent mood disorder and other mood disorder. In earlier classificatory systems and in different epidemiological studies, bipolar affective disorder and manic episode were distinctly clubbed together as against depressive disorder.

This differential classification is the basis for many

uncertainties in rates in reviews of past epidemiological studies. For example, unipolar depression or depressive episode is known to be the fourth-leading cause of the global DALYs lost in all ages and both sexes for the year 1998 (WHO 2001). Earlier studies report lower prevalence rates. The key reasons were underreporting of the episodes of depression, focus on priority mental disorders (which did not include depression), the different 'screening' instruments used, selective reporting of manic psychoses, sociocultural context of reporting mild to even moderate depressive episodes. A review of the available epidemiological studies related to mood disorders has to contend with these differing observations. To maintain uniformity, the prevalence of all types of mood disorders have been combined and include mania, manic depression and depression.

The different community-based studies on mood disorders are given in Table 2. The prevalence rate of mood disorders varies from study to study, region to region and from author to author. The crude prevalence rates per 1000 population vary from as low as 0.5 to as high as 78 in different studies across regions, with different rates being reported by various authors. Studies from northern India reveal that the combined rates vary from 1.3 to 4.7, while rural and urban rates vary from 1.5 to 13, and 1.9 to 6.1/1000, respectively. The southern region reports rural rates of 0.5–3 and an urban rate of 20. The eastern parts of India report the highest rate; the rural rates being in the range of 2.9–78 and the urban rate being 43. The reasons for these variations are not clear. On a national basis, Ganguli (2000) computes the rural and urban rates to be 34/1000 and 37/1000, respectively, while Reddy and Chandrashekar (1998) estimate it to be 11/1000 and 18/1000, respectively.

The incidence study of depression by Nandi *et al.* (1976) undertaken in a rural area between 1972 and 1973 estimated the rates to be 4.63/1000 population. The same authors reporting in 1986 reveal that nearly 5% of the healthy cohort of 1972 had developed depression in the 10-year interval and the difference between the overall prevalence rates in the 10-year interval was greater in 1982 (37.7 in 1972 and 53.3 in 1982), greater by a factor of 1.5 in 1972

Table 2. Crude prevalence rates and gender-specific rates of mood disorders

Author and year	Place	Sample	Crude rate/1000	Rates in males	Rates in females
Reddy and Chandrashekar 1998	All India	NA	12.3 (Urban: 17.9; Rural: 11)	9.1	15.6
Ganguli 2000	All India	NA	34 (Urban: 37, Rural: 34)	NM	NM
East					
Nandi *et al.* 1992	West Bengal (combined)	1,424	21	26	43
Elnagar *et al.* 1971	West Bengal (rural)	1,383	2.9	1.4	4.4
Nandi *et al.* 1975	West Bengal (rural)	1,060	37.74	18	22
Nandi *et al.* 1978	West Bengal (rural)	1,259	25.4	16.8	34.8
Nandi *et al.* 1979	West Bengal (rural)	3,718	37.4	24.8	50.5
ICMR 1987	Calcutta (rural)	34,582	3.9	NM	NM
Nandi *et al.* 1992	West Bengal (rural)	653	25	12	38
Nandi *et al.* 2000	West Bengal (rural)	3,488	78	NM	NM
Nandi *et al.* 2000	West Bengal (rural)	2,183	51	NM	NM
Banerjee *et al.* 1986	West Bengal (urban)	771	42.8	38.55	47.12
North					
Sethi and Gupta 1970	Lucknow (combined)	8,583	4.7 (Urban: 6.1, Rural: 1.5)	NM	NM
Dube 1970	Agra (combined)	29,468	1.26	1	1.56
Sachdeva *et al.* 1986	Faridkot, Haryana (rural)	1,989	13	15	12
ICMR 1987	Patiala (rural)	36,595	5.5	NM	NM
Thacore *et al.* 1975	Lucknow (urban)	2,696	1.9	NM	NM
South					
Mehta *et al.* 1985	Vellore (rural)	5,941	1.5	0.3	2.7
ICMR 1987	Bangalore (rural)	35,548	1.35	NM	NM
Shaji *et al.* 1995	Kerala (rural)	1,094	3	2	4
Verghese *et al.* 1973	Vellore (urban)	26,039	0.5	NM	NM
Premarajan *et al.* 1993	Pondicherry (urban)	1,115	20.2	4.9	36.4
West					
Sharma *et al.* 2001	Goa (combined)	4,022	12.4	15.9	9.3
ICMR 1987	Baroda (rural)	39,655	0.9	NM	NM
Shah *et al.* 1980	Ahmedabad (urban)	2,712	14.8	NM	NM

NM: not mentioned

and 53.3 in 1982 (Nandi *et al.* 1986). The rate in 1992 in the same population was estimated to be 74/1000 (Nandi *et al.* 2000). This definite increase in the occurrence of mood disorders is evident and should be considered for the larger implications of planning service delivery.

The rates of mood disorders were higher in urban areas compared with rural areas (18 *v.* 11) as reported by Reddy and Chandrashekar (1998). Higher rates were also noticed for individual disorders of mania, manic depression and depression. The highest differences were noticed for depression. The sex differences indicate higher rates among women (16 *v.* 9) with a ratio of 2:1. Similar observations are noticed for manic depression and depression, with opposite results for mania (1.2 *v.* 0.1). Studying postpartum depression in rural Vellore, Chandran *et al.* (2002) report the incidence to be 11%.

Health facility-based studies have been undertaken in different settings which include primary health centre, tertiary teaching hospital, private psychiatric clinic or psychiatric outpatient department. The proportion of cases range from 20% to 43% of all cases seen. Thus, it could be said nearly one-third of the patients seeking help from a health care facility have depressive symptoms (Gururaj *et al.* 2005). However, it may be noted that depressive symptoms are routinely not enquired about by the treating/managing professional. These missed opportunities are of greater significance with respect to depression as a whole. Patel *et al.* (2002); emphasizing the need for integrating the mental health components into the ongoing maternal and child health services, in their prospective study examined 270 mothers, 6–8 weeks after delivery and found that 23% fulfilled the criteria for postnatal depression. Further, 22% of them continued to have depressive symptoms at the end of 6 months; 8% had developed depression during the puerperium. Overall, 14% of the study group continued to have symptoms of chronic depression till 6 months after delivery.

Common mental disorders

Common metal disorders (CMDs) are a functional clinical classification of the group of disorders that describe the 'deeper psychological distress states' of an individual. They include anxiety disorders, somatoform disorders, dissociative disorders, phobia and depression. The classification of CMDs for primary health care according to ICD-10 includes depression, phobic disorder, panic disorder, generalized anxiety, mixed anxiety and depression, adjustment disorder, dissociative disorder, unexplained somatic symptoms, neurasthenia and sleep problems. The patients usually present with clearly defined symptoms or somatic complaints: 'some patients may admit to having emotional symptoms' (Patel *et al.* 1998).

The occurrence of CMD has ranged between 13% and 50% in primary care settings to as high as 49% to 57% in hospital settings (Table 3). Patel *et al.* (1998) report that among 97% of the subjects presenting with a somatic complaint, 46.5% of them had a biomedically defined CMD; of these, 51% attributed it to a psychological illness. WHO estimated a global point prevalence of unipolar depression to be 1.9% among men and 3.2% among women (WHO 2001). Reddy and Chandrashekar (1998) estimated the overall prevalence of neuroses to be 0.69%. As a group, CMDs range between 12.7% and 57% of outpatient attendance. Considering these data, a conservative estimate of 2% is considered for purposes of calculating the burden.

Table 3. Common mental disorders in Indian studies

Author and year	Remarks
Harding *et al.* 1980	12.7% of patients in a primary care setting in India
Bagadia *et al.* 1985	57% scored high on GHQ in hospital OPD
Shyamsundar *et al.* 1986	36% among general practitioners
Sen 1987	50% in 3 PHC outpatient departments
Bhatia *et al.* 1989	49% neurotic in surgical OPD
Puri 1995	22% complained of depressed mood from 100 diagnosed depressives
Kishore *et al.* 1996	42% in a PHC in Haryana
Patel 1998	47% of the 97% with somatic complaints had biomedically defined CMD
Amin *et al.* 1998	21% depressives in a health clinic
Nambi *et al.* 2002	44% among those who had unexplained somatic symptoms
Pothen *et al.* 2003	34% in the primary care system
Gururaj *et al.* 2004	Overall prevalence 13% in the community study of 10,168 individuals

GHQ: General Health Questionnaire; OPD: outpatient department; PHC: primary health centre

Substance abuse

Alcohol

The use of psychotropic substances, especially alcohol, has been the focus of sanction or otherwise in different cultures and has also varied over a period of time. The Vedic scriptures of Indian origin have documented the use of *soma-sura* (intoxicating beverages) as early as 2000–800 BC in India. Even the ancient Indian texts of Charaka and Sushruta (around AD 300) make distinctions between normal and excessive drinking. These texts and scriptures also identified the harmful effects of drinking (Isaac 1998). Alcohol was referred to as an evil, yet 'glamorized and accepted' by certain classes. The market-oriented policies of successive governments pertaining to production–distribution–availability, including permitting advertisements of alcohol have been implicated in greater use of alcohol in Indian communities (Lal and Singh 1978, Benegal *et al.* 2000).

During the past two decades, alcohol-related psychiatric problems have been studied in psychiatric morbidity surveys in general and specific populations. The findings from various

Table 4. Community-based studies of alcohol use

Author and year	Place	Sample size	Screening instrument	Crude rate per 1000	Remarks: Focus of enquiry on
Gopinath 1968	Rural community	423	Survey questionnaire	2.36	Alcoholism
Elnagar *et al.* 1971	Rural community	1,383	3-stage survey	10.84	Alcohol and drug addiction
Verghese *et al.* 1973	Urban community	2,904	Mental health item sheet	4.8	Chronic alcoholism
Thacore 1975	Urban community	2,696	Prepared schedule	19	Habitual excessive drinking
Lal *et al.* 1978	Urban community	6,699	QFI		Alcohol users
Mohan *et al.* 2001	Urban community	6,004	Structured questionnaire	30	
Ponnudrai *et al.* 1991	Urban community	2,334	MAST		Suffering from alcoholism
Premarajan 1993	Urban community	1,115	IPSS	34.5 (M-66%)	Alcohol dependence
Hazarika *et al.* 2000	Urban community	312	Not mentioned	365 (M-40%, F-33%)	Alcohol users
Sharma *et al.* 2001	Urban community	4,022	RPES	1	Alcohol dependence
Meena *et al.* 2002	Urban community	142,000	WHO questionnaire	198	Alcohol users
Mohan *et al.* 2002*	Urban community	10,312	Structured questionnaire	59	Alcohol users
Chaturvedi *et al.* 2004	Urban community	5,135	Pretested questionnaire	300	Substance abuse
Varma *et al.* 1980	Urban + rural community	1,031	Structured questionnaire	237 (M-41%)	Alcohol users
Gururaj *et al.* 2004a	Rural, semi-rural, slum and urban community	10,168	Structured questionnaire	90	Alcohol users
Adityanjee *et al.* 1989	Urban hospital	352	MAST, CAGE	3	Alcohol-related problems
Satija *et al.* 1997	Urban hospital	349	ASI, MAST and AUDIT	146	Alcohol users
Vohra *et al.* 2003	Urban hospital	30	SCID	117	Alcohol dependence

*This is an incidence study and the rates are incidence rates, all other rates are prevalence rates

studies (Table 4) reveal that the reported prevalence of alcoholism varies widely, ranging between 1 and 550/1000, and is dependent on the definition adopted and/or screening instrument used. Bang and Bang (1991) in 104 villages of the Gadchiroli district of Maharashtra, observed that nearly 100,000 men consumed alcohol, of whom one-fifth were addicts.

The head of the household survey undertaken by Mohan *et al.* (1992) in Delhi is more robust when compared with other epidemiological studies. The study reported that 26% of residents in urban slums were substance abusers, the majority involving alcohol. In a recent survey of 32,400 people in and around Bangalore, 1.2% of men were found to suffer from alcohol dependence syndrome (ICMR–CAR–CMH 1990). The meta-analysis by Reddy and Chandrashekar (1998) revealed an overall prevalence of 6.9/1000 for India with urban and rural rates of 5.8 and 7.3/1000 population. The rates among men and women were 11.9 and 1.7, respectively. In a community-based study (Gururaj *et al.* 2004) in 4 areas—rural, semi-rural, slum and urban—habitual alcohol users accounted for 9%. Specific population surveys of alcohol use that have been carried out in India include populations such as school students, industrial workers and medical personnel, and ranged between 10% and 60% (Gururaj *et al.* 2005).

Gururaj and Isaac (2004) observe that 'in accordance with the growth of consumption of alcohol all over the country, the hospital admission rates due to the adverse effects of alcohol consumption are also increasing. Several studies indicate that nearly 20%–30% of hospital admissions are due to alcohol-related problems (direct or indirect) in health care settings'. An estimate by Anand (2000) placed the burden due to alcohol as the 'numero uno' among all non-communicable disorders. The estimate of the numbers who consume alcohol in the country was 8.9 crore with an assumption that 17% of males use alcohol and 0.85% of them are dependent on it. The prevalence of alcohol use among women was estimated to be 0.5%.

The masking of the societal and family impact of alcohol use is obvious in earlier studies. Long-term alcohol consumption is linked to a wide variety of social (family disruption, marital disharmony, impact on children, deprivation of the family, work absenteeism, growing rates of crime and violence, etc.) and health (cirrhosis of the liver, road traffic injuries, suicides, etc.) problems. The immediate effects of alcohol consumption include greater incidence of injuries to self and also to others (WHO 2003). In a comparison study of alcohol users versus non-users (Gururaj *et al.* 2004), it was observed that for an alcohol user, the health status of the family and self is worse, they sustain more injuries and inflict more harm to themselves, beat and abuse the wife–children–parents, have a disastrous family life, they are found deficient in managing financial resources (both personal and family), have greater problems in the workplace and face increasing psychological problems. It is suggested that the prevalence rates of alcohol use be utilized for purposes of arriving at the burden.

Drug abuse

Channabasavanna (1989), reviewing the use of drugs in India, lists nine groups of drugs being used in India (*Cannabis* and its products; tranquillizers [hypnotics and sedatives]; barbiturates; amphetamines; hallucinogens; other narcotic drugs like opium, pethidine, morphine, heroin and cocaine; tobacco; alcohol and pain killers). In the earliest available epidemiological study, Elnager *et al.* (1971) reported the prevalence of drug abuse to be 2/1000. Several studies, while evaluating drug abuse, revealed the rates to vary from 1% to 52% based on different methodologies. Ahmed and Sen (1998), using the WHO questionnaire, arrived at a prevalence rate of 52% in Delhi, using DSM-IIIR as the diagnostic instrument, Mohan *et al.* (2002), Satija *et al.* (1997) reported prevalence rates of 3% and 27% among the Delhi urban community and Jaipur industrial workers, respectively.

The United Nations office on drugs and crime and the Ministry of Social Justice and Empowerment, Government of India, 2004 has recently completed a report of the extent, pattern and trends of drug abuse in India. Triangulating the different methodologies, the study has attempted to provide a realistic picture of the extent, pattern and trends of drug abuse in the country. Four different approaches have been utilized: National Household Survey (two-stage stratified random sample through probability proportional to size), Rapid Assessment Survey of Drug abuse (the non-random sample, key informant interviews and focus group discussions), Drug Abuse Monitoring Systems (DAMS) and focused thematic discussions. According to the national household survey, the current one-month period use for alcohol, cannabis and opiates were 21.4%, 3%, and 0.7%, respectively. Other drug abuse was reported by 3.7%. Applying the prevalence estimates to the population figures of 2001, the report estimates and projects that there are 6.25 crore alcohol users, 87 lakh cannabis users and 20 lakh opiate users in the country. These numbers, when applied to the total Indian population of 102.7 crore of 2001, provide prevalence rates of 60/1000, 8/1000 and 2/1000 population, respectively. Dependent users were 17% for alcohol, 26% for cannabis and 22% for opiates. The rate from this methodologically better study has been used to arrive at the national burden.

Child and adolescent mental health problems

Children and adolescents form nearly 37% of the total population of India (Census 2001: http://censusindia.net). The entire gamut of childhood and adolescent disorders can be broadly grouped into two: (i) Mental retardation (disorders of psychological development), and (ii) behavioural and emotional disorders, with the onset in childhood and adolescence. The available general community surveys have not utilized the necessary specific tools for addressing the disorders of children and adolescents. The specific surveys for childhood have used different instruments, which makes it difficult to compare them. Adding to the problems in these surveys are the different age groups covered, differing sizes of the population and the setting of the study—urban, rural, combined. School-based studies do not provide true prevalence rates as the denominator is difficult to define. In addition, school-based studies obviously do not cover 'out-of-school' children. The result is that the morbidity among this group is left untouched. Because of the huge school drop-out rates in primary school (Government of India 2004), the estimates from school-based surveys reflect only a portion of the total burden of mental and behavioural disorders in all children. Despite this, these estimates would provide 'guidelines for planning both community- and school-based services'.

Mental retardation

Mental retardation (MR) has been defined as the 'sub-average general intellectual functioning which originates during the developmental period and is associated with impairment in adaptive behaviour'. It can be defined in combination or in isolation with measurement of intelligence, neurological functioning, social adaptation and behavioural competence (Kiely 1987). From a public health point of view, the continued occurrence of mental retardation due to a potentially preventable cause such as iodine deficiency disorder reflects the extent of impact of the ongoing programme. Early and appropriate planned comprehensive interventions in this group is the need of the hour.

Most of the Indian epidemiological studies have included MR in their ambit (Table 5). A review of Indian studies on mental retardation by Prabhu (1987) revealed that the prevalence rates vary from 0.22 to 32.7/1000 population. Madhavan (1987), in a collective review, found the rates to vary from 3.4/1000 to 30/1000 in India. Gupta and Sethi (1970) in a population-based survey in Uttar Pradesh reported a prevalence of 2.1% among 500 rural and 1000 urban households surveyed. Severe MR was present in 1.5%. Specific surveys in schoolgoing children have shown a prevalence of 2.91–20.6/1000 (Bhola and Kapur 2003). Reddy and Chandrashekar (1998) established a weighted prevalence rate of 6.9/1000 based on a meta-analytical approach, with a higher occurrence among males. The urban and rural rates were found to be 8.9 and 6.4/1000, respectively. Ganguli (2000) in a review of 10 studies observed a prevalence rate of 3.7 and 9/1000, respectively. Srinath and Girimaji (1999) in a review of child and adolescent mental health problems in India conclude that 2% and 0.5% of children in India suffer from mild and severe forms of MR, respectively. The National Sample Survey Organization in its 58th round estimated that there are nearly 10 lakh children who are disabled due to MR (NSSO 2003). Despite the fact that the Survey may have picked up only the moderate–severe forms of the disorder, these pan-country authoritative

Table 5. Community-based studies reporting mental retardation (combined, rural and urban)

Author and year	Place	Sample size	Crude rate/1000
Prabhu *et al.* 1985	Review	Not applicable	(2 crore)
Ganguli 2000	Combined	Not applicable	5.3
Reddy and Chandra-shekar 1998	Meta-analysis	33,572	6.9
Gupta *et al.* 1970	Combined	1,500	23.3 (M-131; F-69)
Gupta and Sethi 1970	Combined	8,583	23.3
Dube 1970	Combined	29,468	3.7 (M-3; F-4)
Verghese *et al.* 1973	Combined	26,039	3.2
Surya *et al.* 1962	Urban	2,731	0.07
Thacore *et al.* 1975	Urban	2,696	14
Shah *et al.* 1980	Urban	2,712	1.8
Banerjee 1986	Urban	771	2.59
Gopinath 1968	Urban	423	4.72
Premarajan *et al.* 1993	Urban	1,115	18.3
Elnagar *et al.* 1971	Rural	1,383	1.4
Nandi *et al.* 1973	Rural	2,250	8.1
Nandi *et al.* 1975	Rural	1,060	2.83
Murthy *et al.* 1978	Rural	2,500	0.28
Nandi *et al.* 1979	Rural	3,718	7 (M-7.4; F-6.6)
Naryanan 1981	Rural	6,708	78.4 (M-50; F-28)
Subrahmanya 1983	Rural	1,498	27.4 M-2; F-1
Mehta *et al.* 1985	Rural	5,941	3.2 (M-4.7; F-1.7)
Sachdev *et al.* 1986	Rural	1,989	2.51 (M-1; F-15)
Shaji 1995	Rural	1,094	2.84 (M-3; F-3)
Nandi *et al.* 2000	Rural (1992)	3,488	12.33
Nandi *et al.* 2000	Rural (1972)	2,183	8.7

estimates spell out greater numbers than the guesstimates relied on earlier.

Child and adolescent mental health problems

Several population-based/hospital-based/specific children-based epidemiological studies have been completed in India (excluding MR). Bhola and Kapur (2003) in their review identified and listed 55 epidemiological studies (both community-based and school-based) during the period 1964–2002. Compared to adult epidemiological surveys, studies on children are much more difficult due to problems in definition of deviance, emotion and perception, understanding disability by parents, teachers and interviewers, and measurement issues.

Reviewing the studies on mental health problems in children, Kapur (1993) observed that community surveys identified only severe problems such as enuresis, stuttering, sleep disorders, MR and epilepsy. The prevalence of child mental health problems from earlier studies in different populations varied from 7 to 172/1000 children (Seshadri 1993). Srinath and Girimaji (1999) in their review on childhood psychiatric and emotional problems report that the prevalence ranges from 25 to 356/1000 in field studies. Bhola and Kapur (2003), however, note the range to be 5/1000 to 294/1000. Mental retardation, epilepsy and enuresis are reported as highly prevalent disorders in community-based studies. The 23 school-based studies listed during the period 1978–2002 identified enuresis, MR, conduct disorders and attention deficit hyperactivity disorder (ADHD) as being the most prevalent. In general, it has been realized that schoolgoing children report higher psychological distur-bances; urban children report more problems compared to rural children; boys report more problems than girls. Scholastic backwardness has been a major problem in the Indian region. It was thus concluded that scholastic and learning-related problems of children need to be examined simultaneously with mental health problems.

To overcome the lacunae arising from methodological issues, the Indian Council of Medical Research (ICMR) undertook a study in Bangalore and Lucknow during 1997 (ICMR 2001). Adopting a two-stage survey and using standardized instruments for different age groups (Child behavior check list; Rutter teacher's questionnaire; Diagnostic interview schedule for parents and teachers (DISC); Parents interview schedule; Children's global assessment scale; Intelligence assessment; Assessment of felt treatment needs; Physical examination), the prevalence of child and adolescent disorders in this study was observed to be 12.8% in 1–16-year-old children. These recent figures from ICMR of 128/1000 have been taken to be the prevalence estimates for child mental health care services.

Geriatric mental health problems

With the changing demographic scenario declining mortality has led to an increase in the elderly population. From being 6.5% of the total population in 1981, the elderly constituted 8% of the total population in 2001. In absolute numbers, it means a jump from 5.2 crore in 1981 to 8.3 crore in 2001 (http://censusindia.net). The problems of the 60+ years age group are manifold. Rao (1997) found that visual and locomotor problems were the two prominent symptoms and 8.4% had symptoms related to mental distress. The results from general community epidemiological surveys can be misleading as they do not specifically target the elderly to arrive at estimates of prevalence. A wide range of estimates (per 1000 age-specific population) ranging from 22 to 333 has been observed (Gururaj *et al.* 2005). Shaji *et al.* (1995) reporting from Kerala on the prevalence of priority mental disorders reported it to be 95/1000 population. Nandi *et al.* (1997) reported an astonishing 61% of their study subjects to have been 'mentally ill'. The higher rate from recent surveys is probably due to studying

priority disorders in a rapidly greying population. Estimates from community-based studies are further hampered by the non-recognition of mental illness of the elderly while reporting diseases. In addition, the diagnosis of the presence of a mental illness may be at the institution where care is being sought, thus resulting in much of the underreporting from community-based surveys. The meta-analysis of Reddy and Chandrashekhar (1998) estimated the prevalence to be 31/1000 among the 60+ years age group. As noted earlier, the common problems of the elderly are affective disorders and organic brain syndromes. Affective disorders as a whole have been covered under mood disorders and also under common mental disorders, hence the following paragraphs cover the major cause of organic brain syndrome—dementia.

Dementia

Dementia is defined as the 'global deterioration of the individual's intellectual, emotional and cognitive faculties in a state of impaired consciousness' (Roth 1980). This has to be chiefly differentiated from the impairment of memory and intellect, which happens as part of the normal ageing process. The diagnosis of dementia is made with demonstrable evidence of impairment of memory and/or intellectual functioning, the severity of which interferes with normal social or occupational functioning. Dementia of Alzheimer type (DAT) contributes to 60% of all dementias affecting people over the age of 60 years, while a number of other conditions are responsible for non-Alzheimer dementia (Rao 1997), chief among them being multi-infarct dementia (Jha and Patel 2004).

Geriatric psychiatric epidemiological studies focusing on dementia have been few and limited in India. The prevalence of dementia was found to be 3.5% in rural Thirupur (Rajkumar *et al.* 1997), 3.4% in Thiruvaniyoor in Kerala (Shaji *et al.* 1996), 1.07% among those 65+ years of age (Chandra *et al.* 1998). Vas *et al.* (2001) in their multistage survey in Bombay found the prevalence of dementia to be less than reported elsewhere: 0.43% for those over 40 years and 2.44% for those over 65 years of age. Alzheimer disease was found in 1.5% of those over the age of 65 years. The ratio of DAT to vascular dementia was found to be 2:1. This finding is of interest as the latter is preventable. Shaji *et al.* (1996) reported similar findings from their study in Kerala. Geriatric mental health problems are assumed to be present among 31/1000 population who are above 60 years based on a meta-analysis study (Reddy and Chandrashekar 1998) or 2.48/1000 population of all ages. For the present report, specific rates of dementia are assumed to be 1.9% in the 65+ years age group (or 1.52/1000 population of all ages), derived from the median value of the rates from the two community-based studies on dementia.

Epilepsy

Epilepsy is considered to be the most common disorder of the brain and one of the oldest recorded medical conditions. During the 1960s and 1970s, epilepsy was regarded as a mental and behavioural problem and included as part of mental morbidity surveys. The sample sizes for different studies varied between 423 and 102,557. In addition, the difficulties in comparing different studies are primarily due to differing case definitions. The overall rates range between 2.2 and 11.9/1000 population (Gururaj *et al.* 2005). Urban and rural differences have been clearly documented in the largest ever Indian community-based neurological study in Bangalore. The rural rates were nearly twice that of the urban rates; females were more affected than males (Gourie Devi *et al.* 2004). Those less than 20 years of age constituted 38% of the total cases. Preventable causes such as birth trauma, infections and head injuries contributed to the majority of epilepsy among children. WHO (2001b) estimated that 'India alone has approximately 80–100 lakh people suffering from epilepsy'. For the purposes of estimation in this report, an estimate of 9/1000 has been utilized, which is similar to the rate from the large sample Bangalore Urban Rural Neurological problem study.

Psychiatric morbidity among the disaster afflicted

In the Indian context, the mental health needs of the disaster afflicted is an emerging area. Unpublished reports estimate the prevalence of post-traumatic stress to be as high as 56% among survivors of a disaster (Orissa cyclone) 6 months after the event (Sekar K, personal communication). It is estimated that the rates for psychopathology increase by about 20%–40% in the period after the disaster (Murthy 2000). These rates are twice those for common mental disorders. Systematic efforts undertaken after the Bhopal gas tragedy, earthquake in Maharashtra and Gujarat, the Orissa supercyclone and the recent tsunami tragedy have led to the conclusion that disaster mental health is an area that needs immediate attention.

Burden of mental, neurological and substance use disorders in India, 2000–2015

To make calculations less cumbersome, the different estimates which had specific subgroups have been computed to make them applicable to the population of all ages and both sexes. For example, the prevalence rates for dementia are 19 per 1000 in the 65+ years age group and 1.52 per 1000 population of all age groups.

- The prevalence of major mental and behavioural disorders at any given point of time was estimated as 65/1000 population in all ages and both sexes based on the average value of two pooled studies (Reddy and Chandrashekhar 1998; and Ganguli 2000) (57/1000 and 73/1000, respectively).
- The prevalence of schizophrenia has been considered as 3/1000 for all ages and both sexes.
- Considering the wide range in the prevalence rate of mood disorders, a range of 12/1000 (lower) and 21/1000 (upper), a median value of 16/1000 population (all ages and both sexes) has been used for calculating the burden.
- Most of the studies on CMDs were done in a health facility setting, which makes it difficult to make realistic estimates. A conservative estimate of 2% has been used based on the meta-analysis study of Reddy and Chandrashekhar (1998) and WHO estimates.
- The prevalence rate for alcohol, cannabis and opiate use in the community-based representative National Household Survey was 214/1000, 30/1000 and 7/1000, respectively. Seventeen per cent of alcohol users, 26% of cannabis users and 22% of opiate users were classified as dependent users based on ICD-10, the median value being 22%. The rates when applied to the total population of all ages and both sexes would be current alcohol users—60/1000, cannabis—8/1000 and opiates—2/1000 population. Based on a 17% dependency rate among current alcohol users, an alcohol dependency rate of 1% of all ages and both sexes is obtained.
- Estimates for MR are based on the 58th Round of the National Sample Survey. During the survey year of 2002, 994,600 persons in India suffered from MR. Based on this, the rate of MR in India would be 9.5/10,000 population of all ages, or 1/1000 population (all ages, both sexes).
- Child and adolescent mental health problems are estimated at 128/1000 child population (1–16 years) based on the methodologically better WHO study, or 43/1000 population of all ages.
- Geriatric mental health problems are assumed to be present among 31/1000 population above 60 years based on a meta-analysis study or 2.48/1000 population of all ages. Specific rates of dementia are 19/1000 in the 65+ years age group or 1.52/1000 population of all ages.
- The prevalence rate for epilepsy varies from 2 to 11/1000 population. A conservative estimate of 9/1000, similar to the rate from the large sample Bangalore Urban Rural Neurological problem study has been utilized for estimation.

For the year 2001, an estimated 6.7 crore people with major mental disorders, 2.05 crore with CMDs and 1.02 crore with alcohol dependency problems required services in India. Table 6 provides the all-India projections for 2005, 2010 and 2015. In the absence of trend studies and available interval studies indicating no change in the prevalence of overall mental and neurological morbidity, the same prevalence rates were used for different reference years to the population of respective States. Although the rates are steady, the numbers would swell and definitely have an impact on the provision of health services. The States with a high burden of mental problems (more than 50 lakh cases) are: UP, Maharashtra, Bihar, West Bengal and AP.

Table 6. All-India estimates of mental, select neurological and substance use disorders

Type of mental and behavioural disorder	Prevalence*	2001	2005	2010	2015
All-India population (in lakh)[1]		10,280	10,760	11,620	12,450
Major mental/behavioural disorders	65	66,859,671	70,000,710	75,548,395	80,978,755
Schizophrenia	3	3,085,831	3,230,802	3,486,849	3,737,481
Mood disorders	16	16,457,765	17,230,944	18,596,528	19,933,232
Cannabis users	8	8,228,883	8,615,472	9,298,264	9,966,616
Opiate users	2	2,057,221	2,153,868	2,324,566	2,491,654
Mental retardation	1	1,028,610	1,076,934	1,162,283	1,245,827
Child and adolescent disorders	43	25,509,536	26,707,963	28,824,618	30,896,510
Geriatric disorders	3	2,550,954	2,670,796	2,882,462	3,089,651
Dementia	2	1,563,488	1,636,940	1,766,670	1,893,657
Epilepsy	9	9,257,493	9,692,406	10,460,547	11,212,443
Common mental disorders	20	20,572,207	21,538,680	23,245,660	24,916,540
Alcohol users	60	61,716,620	64,616,040	69,736,980	74,749,620
Alcohol dependency†	10	10,286,103	10,769,340	11,622,830	12,458,270

*Rate per 1000 population all ages and both sexes. Rates after adjusting to the age distribution. The numbers do not add up as the estimates have been arrived at from individual or pooled or representative studies. Please see the text for basis of these estimates.

†This group does not include hazardous alcohol users, whose number would be approximately 24 crore.

[1]*Source:* Population of India, 2001 (www.Censusindia.net)

PART II

Causation of mental disorders: The enigma continues…

'… social constructs regarding mechanism of symptoms have special meanings in relation to the shared belief of a community about the nature of an illness'
—MURPHY 1964

The premise and the paradigm

The causes of mental illness are complex, varied, differing from condition to condition and influenced by several sociodemographic and biological attributes. The previous century witnessed major advances in treatment despite an elusive aetiological–pathological–physiological process (Mitchell and Pavlavic 2000). Reviewing the recent advances, Kessler (2000) found that the problems encountered in psychiatric epidemiology were in 'conceptualizing and measuring mental disorders' and pointed out the need to focus on modifiable risk factors rather than on broad, non-specific risk markers.

Developments in the past few decades laid a firm foundation for a biological basis of mental disorders along with the then prevalent behavioural theories of causation of mental disorders. The neurotransmitter basis for many of the mental disorders has been extensively documented and the putative pathways have also been identified. Neuroimmunology, dynamic neuroimaging and genetic studies (twin studies, sibling studies, family studies and association studies) have added newer possibilities for the causation of mental disorders (Vyas and Ahuja 1999). Many of these different hypotheses of the causation of mental disorders are still in the investigative/trial stage.

The different hypotheses for the causation of mental disorders were broadly identified as belonging either to Nature (biological) or Nurture (environmental including sociocultural). With increasing research, it has become evident that mental illnesses are due to a complex interaction of social (including economic, environmental), psychological and biological factors (Fig. 1) and have a differential impact on the prevalence, onset and course of mental and behavioural disorders. It may be noted that a strict classification into biological, psychological and social factors poses a 'formidable barrier' to the 'true understanding' of mental and behavioural disorders (WHO 2001). Hence, Rose (2001) called for a non-dichotomizing developmental approach to identify causation: 'both the genome and envirome are abstraction of the continuous dialectic'. This point is further illustrated when one considers the directions taken by research in the causation, onset, course and outcome of schizophrenia (Walker *et al.* 2004). Eisenberg (2004), critiquing the euphoria of the genetic model, celebrates the success of social psychiatry: 'genes set the boundaries of the possible; environment parses out the actual.' Hyman (2000) observes

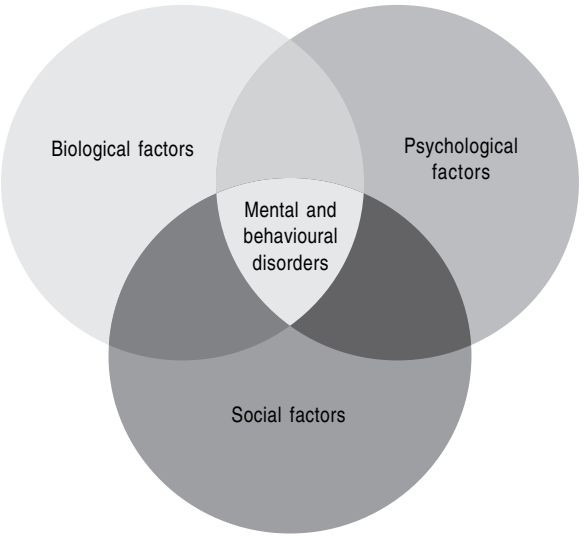

Fig. 1 Factors for the causation of mental disorders
Source: WHO 2001

that the proposal of 'symptom clusters, course of illness, family history and treatment history' turning ('coalescing') into a relatively simple diagnostic category is 'frayed'. In the backdrop of new research to link molecules, milieu and the mind, Murthy (2005) observes that the emerging evidence has an 'important implication for the role of mental health professionals' who need to recognize the bio–psycho–social approach in the practice of psychiatry. While the public health approach identifies causes amenable to cost-effective interventions on the larger population base (WHO 2001), an alternate approach, the risk concept, has also been utilized in recent years (WHO 2005). The *World Health Report* lists the key determinants of mental health to be poverty (and its associated conditions of unemployment, low educational attainment, deprivation, homelessness), gender, age, conflicts and disasters, major physical diseases, family and environmental factors (WHO 2001).

Causation in the Indian region

Well-designed, population-based analytical studies on the causation of mental disorders are lacking in India. Complex methodological issues have further added to the existing problems in undertaking research. A review of the current research efforts in the country reveals that there has been a gradual shift and progress in identifying the social, demographic and cultural correlates of disorders (Gururaj and Isaac 2004). The epidemiological enquiries undertaken in India have primarily focused on arriving at the extent of morbidity; recent studies which have demonstrated more methodological rigour (identifying the sociodemographic

correlates) permit the identification of 'at-risk' groups or subpopulations. Apart from this, the different variables studied include: age, gender, sex, place of dwelling, education, occupation, income, religion, migration, marital status, caste, type of family (nuclear, non-nuclear). While no clear conclusions can be drawn based on these data, certain pointers emerge related to 'at risk' groups and have led to formation of some hypotheses.

- **Age and sex distribution:** In the study by Dube (1970), the age of onset of any mental illness was 15–24 years, with two-thirds of the cases being in the 15–55 years age group, except for mental deficiency which was among children. There was a female preponderance in the ratio of males to females (1:3); in addition, the age at onset was significantly higher for females for all mental disorders. The ICMR study on severe mental morbidity revealed divergent results, with the 60+ years age group having a greater proportion of morbidity, except in Bangalore where two peaks at the extremes of the adult age group (15–19 years and 40–59 years) were noticed. On the whole, the age group of 60+ years experienced greater morbidity; the age groups of 40–59 and 30–39 years come next. There was a male preponderance in two of the centres (Bangalore and Baroda) and a female preponderance in the Patiala centre. The four period-representative studies (1960s, 1970s, 1980s and 1990s) point towards an age range of 30–45 years with three of the four studies showing a female preponderance (Gururaj *et al.* 2005). Reddy *et al.* (1994) observed in their study that the extremes of age had a higher morbidity with more neurotics in the 21–30 years age group (18% *v.* 7%). The meta-analysis of Reddy and Chadrashekar (1998) showed a greater proportion of mental disorders among the 35–44 years age group and among females. In essence, regarding age–sex distribution, it could be inconclusively said that adult females in the age group of 30–45 years are more at risk for mental illness. Astbury (2000), realizing the information gaps in studying gender disparities, lists out the systemic 'fault lines' (status, roles, options and treatment in society) which predispose women and push them into the more vulnerable group.
- **Urban/rural differences:** Dube (1970) found a higher rate in the non-rural population (rural 18/1000 *v.* 25/1000 non-rural), as did the pooled meta-analysis of Reddy and Chandrashekhar (1998) (rural 49/1000, urban 81/1000 population). Ganguli (2000) found a small difference (rural 71/1000 and urban 73/1000 population). The only other recent combined study in both urban and rural areas (Sharma *et al.* 2001) did not show any difference in the rates. However, considering the fact that a large part of the population of India lives in rural areas, the burden of mental morbidity and need for services will be higher in these areas.
- **Income:** Income has been strongly implicated in many individual studies: those with low-income levels show a higher prevalence of mental illness. However, it continues to be debated whether income levels are a result of the mental illness or a cause. Contrary to expectation, the ICMR study (ICMR 1987) showed that the high-income category had a greater proportion of mental illness. The Calcutta centre found a higher prevalence among the extremes of income levels. Income levels only need to be considered as a proxy for lack of resources among the mentally ill, the result of which would drive them further down the income categories (the social causation versus the downward drift hypotheses—Mueser and McGurk 2004). Thus, despite there being no conclusive evidence from available studies for severe mental disorders, those with low-income levels need to considered as a more vulnerable group. Patel *et al.* (2003), reviewing the literature pertaining to CMDs, found the evidence supporting a specific association with income levels to be weak. However, poverty as a 'marker' of deprivation is found to result in a vicious cycle of poverty–mental and behavioural disorders–economic impact. Further, it was observed that just overcoming poverty is insufficient to comprehensively address deprivation, but would need 'equitable distribution of resources'.
- **Education:** Education as variable of study is important both from identifying the at-risk group and in guiding the planning of interventions, including continuity of care. The different definitions adopted in defining the education levels among various studies pose problems in comparison. Verghese *et al.* (1973) and ICMR (1987) found a greater proportion of low literacy levels among the mentally ill.
- **Occupation:** Dube (1970) reported that 'non-earning employment, unremunerative work with all monotony and lack of motivation and lack of security may be more stressful contributing to mental illnes'; in contrast, 'earners had better self-esteem, better interpersonal relationships (thus) preventing precipitation of a breakdown'. In addition, 'indebtedness without undue anxiety', 'multiple drug users', 'short temperament', 'vocational maladjustment', 'special strains (anxiety to financial overstrain, heavy indebtedness, severe anxiety on account of disease, marriage, domestic problems)' had higher morbidity. This feature has been consistent even with the ICMR study (1987) and that of Verghese *et al.* (1973).
- **Marital status:** All reports indicate that those who are widowed, separated or divorced have greater mental morbidity. Contrary to western data, the Indian married population have a greater proportion of the mentally ill. Various reasons have been ascribed for this finding, a major one being the possibility of not differentiating but accommodating a mentally ill person in day-to-day activities.
- **Migration:** Migration as a specific variable of study has been reported by Dube (1970). It has been observed that Punjabi refugees were more afflicted than Sindhi refugees. Nandi *et al.* (1992), using a case–control approach,

studied the migration of a tribal and a resettled population and found that neurotic illness was more prevalent among those who had migrated to an 'urban' area but had little effect on the total mental morbidity. Reviewing the literature on migration and mental health, Bhugra (2004) implores that the role of social and cultural factors is paramount in both aetiology and management of psychiatric illnesses.

- **Type of family:** Living alone was a specific risk factor for severe mental morbidity as revealed by the ICMR study (1987). The contradictory findings of Dube (1970) and Verghese *et al.* (1973) regarding the type of family that is at greater risk needs to be seen in the larger sociocultural context. The former reports a greater risk among the joint family while the latter report a greater risk among nuclear families.

In the absence of longitudinal studies that would help to identify the time-trends of the disease and evaluate causal mechanisms, interval studies are invaluable. Nandi *et al.* reported their findings on the prevalence and sociodemographic correlates of mental illness in the villages of Gambhirgarchi and Paharpur in the 24 Parganas district in West Bengal over two time intervals: 1972–82 and 1972–92 (Nandi *et al.* 1986, Nandi *et al.* 2000). The small sample sizes do not permit generalizations. However, the change, which has been documented over a period of two decades, is a definite pointer. The enhanced 'affluence' of the communities does not reflect on the overall morbidity pattern. The 'cohort effect' could be a plausible reason. The finding of a lesser proportion of morbidity among those healthy in the earlier surveys further bolsters this point.

In a community-based analytical study, Chandran *et al.* (2002) indicated that incident cases of postpartum depression are predicted by low income (RR=3), an adverse life event in the year preceding delivery (RR=4), problems with the in-laws (RR=3), poor relationship with parents (RR=3), birth of a daughter when a son was desired (RR=3) and lack of physical help at home during the postpartum period (RR=3). In epilepsy, studies point to the relative risk of genetic factors (RR: 0.7–5.6) with the other major contributory risk factors being brain injuries (RR: 1.4–12.7), febrile convulsions (RR: 3.0–14.2) and pyogenic meningitis (RR: 7–40) (Gourie Devi *et al.* 1999). The findings from such studies are particularly important as they point out the urgent need for identifying, incorporating or strengthening the mental health component of existing health and other developmental programmes for disease prevention: Maternal and Child Health, Integrated Child Services Development Scheme, Poverty Alleviation Schemes, etc.

In recent years, further research into causation has identified specific areas of association. For schizophrenia and other psychoses, the genetic basis of these illnesses is gathering momentum. Reduced brain volumes with deficient brain neurocircuits in specific areas from twin studies suggest a biological basis for the disorder. These studies also suggest a key role for prenatal and postnatal stressors being responsible for determining the onset, course and outcome of the illness (Walker *et al.* 2004). Dementia is recognized primarily as a neurodegenerative disorder with a vascular basis (Peng 2003). Alcohol and substance use disorders are predominantly sociocultural phenomena in the early stages of experimentation which, with later abuse, acquire a distinct biological basis rooted in altered neurochemistry and functioning. Depressive disorders as noted above have a strong mooring within the sociocultural and demographic correlates, in turn linked with endocrinal imbalance. The dominant theme of present-day research is to explore the bio–psycho–social models rather than individual components.

A major objective of enquiry into the causal mechanisms or determinants of an illness is to arrive at possible interventions. In India, currently, the major issue is with regard to problems pertaining to the three A's of service delivery: availability, accessibility and affordability. The public health determinants and causes of morbidity need to be attended to and addressed as an utmost priority. For better planning and bundling of interventions and thereby services, the classification of causes also need to be modified. WHO has classified interventions for mental and behavioural disorders into prevention, treatment and rehabilitation. Correspondingly, there is a need to examine the causation of mental disorders from a different perspective from those that can be prevented (preventable mental illness/disorder: e.g. MR due to preventable causes), easily managed (treatable mental illness/disorder: e.g. CMDs, especially depression, anxiety disorders, deviant behavioural problems of children, alcohol and other substance use disorders, etc.) and those disorders requiring long-term care along with appropriate rehabilitation (all types of moderate to severe functional psychoses—schizophrenia, mania, manic–depressive episodes, all types of established MR, dementia and others). Some examples of risk factors directly and indirectly related to health and non-health parameters for selected conditions is provided in Table 7. Detailed discussion of causation is beyond the scope of this report.

Table 7. Causal (risk) analysis of mental and behavioural disorders

Condition	Direct (medical) causes	Indirect/distant (non-medical) causes
Schizophrenia	• Immunological factors • Genetic predisposition • History of alcohol and drug abuse • Course and outcome of conditions • Personality/nature of individual • Past viral infections • Presence of violence	• Lack of income and employment • Poverty • Stigma • Marital status (not clear) (single/ widowed/divorced/separated) • Available drugs beyond the reach of the poor • Living alone • Lack of family support systems • Gender and age (equivocal) • Social adversities • Family status
Alcohol and durg abuse	• Predisposition of the individual • Personality profile • Family history of usage	• Easy availability of alcohol and drugs • Extensive promotion in the media • Liberalized values among the people • Lack of clear policies on production, availability, distribution and promotion • Peer group influences
Mental retardation	• Lack of obstetrics services • Neonatal sepsis • Infections of the nervous system • Inborn errors of metabolism • Absence of premarital and genetic counselling • Lack of investigative facilities for metabolic errors • Previous medical conditions (hypertension)	• Poor life-skills • Lack of rehabilitative facilities • Social factors • Difficulties in health care due to lack of resources • Absence of policies on rehabilitation • Lack of iodine in the nutrition
Dementia	History of mental disorders	• Emerging social issues such as isolation • Deprivation of basic care • Absence of supportive care

Note: General service-related issues such as help-seeking behaviour, lack of physical and human resources, low levels of mental health, literacy and failure to implement existing programmes are intermediate spokes in the complex chain of causation for all mental disorders.

PART III

Interventions for mental and behavioural disorders

Knowing is not enough; we must apply
Willing is not enough; we must do

—Goethe

Learning from the past

The twentieth century bears testimony to the improvement in the way mental illness has been managed. Moving away from the mental asylum approach, the concept of mental hospitals is undergoing a major change in their role and functioning. Reflecting the movement away from custodial care of the mentally ill, the first 'General Hospital Psychiatry Unit' was established in Calcutta in 1933 (Parkar *et al.* 2001). On the eve of Independence, the Health Survey and Development Committee headed by Sir Joseph Bhore (1942–46), was a notable effort to comprehensively plan for the health of the people of India. Noting the non-availability of data on the burden of mental disorders, the Bhore Committee in its report, assumed 'the population of mental patients be taken as two per thousand population'. Accordingly, the infrastructure was recommended to make available facilities for 800,000 patients 'as against the existing 10,000 beds' (Chandrashekar and Isaac 1999). In 1954, on the recommendations of the Bhore Committee, the All India Institute of Mental Health, Bangalore was set up to increase the availability of trained manpower in the country. Apart from making available trained human resources, emphasis was on research endeavours which would be suited for planning better services. The Mudaliar Committee (1959), commenting on the lack of 'reliable statistics regarding the incidence of mental morbidity in India', (Chandrashekar and Isaac 1999) recommended that 'each district should have a psychiatric clinic and five to ten beds may be earmarked for psychiatric cases' (Agarwal *et al.* 2004).

Amid the debates in causation and intervention, despite documented social vulnerabilities, proven pharmacological remedies and psychotherapies, the overall burden of existing mental disorders continues all over the world. Andrews *et al.* (2000) reviewed the data from the Australian National Survey of mental health and well-being on the two commonest mental disorders (generalized anxiety disorder and depression)

and observed that 'too many people do not seek treatment and when they do, efficacious treatments are not always used effectively'. Indian studies too have found a huge treatment gap. For example, epilepsy is an eminently preventable, easily identifiable and diagnosable major public health problem, and 70%–80% of seizures are effectively controlled with one or two drugs. However, the treatment gap is said to be in the range of 50%–70% (Gourie Devi *et al.* 1999). In the recently concluded consensus study in developing countries for resource utilization in select neuropsychiatric conditions, Ferri *et al.* (2004) found that current treatment coverage was below 20% for Alzheimer dementia to about 60% for epilepsy across 7 developing nations. For India, the current coverage rate was schizophrenia—40%, bipolar disorder—38%, depression—28%, panic disorder—16%, alcohol misuse—20%, alcohol dependence—23%, Alzheimer disease—9% and epilepsy—45%. Only a fraction of those currently covered adhered to the management protocol; this ranged between 15% and 45%. There is therefore gross underutilization of even the minimal available services. The reasons for underutilization of services and thereby the causes for mental disorders can be varied: they could either lie with the patient or the family (stigma, distance, long duration of therapy, no permanent cure, etc.) or the service provider (non-recognition of the illness/disorder either as stand-alone or with co-morbidities such as substance abuse, inadequate clinical skills to manage the illness after 'case detection'), or with the health system (inadequate drugs and infrastructure, inappropriate development of human resources, adopting culturally inappropriate, western treatment/management protocols in their entirety) (Thara 2004a).

Several experiments in the Indian region have successfully demonstrated the possibility, feasibility and integration of mental health services with primary care services, in line with international thought to deprofessionalize health care, decentralize health services and further the 'community approach'. Several mental health professionals have documented their experience across India. Reddy (1983) lists the major developments in mental health care delivery as organization of outpatient services, family psychiatric services, rehabilitation services, community mental health services for both rural and urban populations, training of school teachers and lay volunteers, domiciliary care programme, extension services (satellite clinics and self-help groups of parents). The fact that basic and essential mental health care can be delivered resulted in the birth of the National Mental Health Programme (NMHP) for India in 1982. The Central Council of Health and Family Welfare in its meeting held in 1982 recommended that 'mental health must form an integral part of health programmes and as such should be included in all national policies and programmes in the field of health; and education and social welfare'. The key messages from different studies/reviews which have investigated outreach activities and related aspects are as follows:

- Epilepsy, neurotic disorders, mental retardation, psychoses and other disorders of the CNS form the major neuro-psychiatric problems at the community level (Kapur *et al.* 1982, Gururaj *et al.* 1988).
- The possibility of extending mental health services through neuropsychiatric camps, satellite clinics and satellite units is a cost-effective method to deliver mental health services to a large majority of the needy population. (Wig *et al.* 1980, Kapur *et al.* 1982, Mathai 1984, Reddy *et al.* 1986).
- Follow-up activities and establishing proper referral patterns are vital to the success of the programme (Gururaj *et al.* 1988).
- Lack of resources, the population explosion, misplaced priorities and adopting unrealistsic approaches have continued to hamper the reaching of the desired objectives (Verma 1986).
- Rehabilitation through work therapy needs to move from the passive 'make work' approach to the active 'participatory' work restoration (Menon 1996).
- Augmenting family self-help groups is possible and feasible (Reddy *et al.* 1986).
- Psychotherapy models based on indigenous philosophical systems can be tried (Balodhi 1990).
- The use of affordable, simple drugs in general health care settings is associated with improved clinical and economic outcomes, especially in the short term (Patel and Kleinman 2003).
- Training primary care medical officers in mental health care is possible, feasible, effective and beneficial (Reddy *et al.* 1986, Sriram *et al.* 1990).
- There is a need for collaborative programmes both in research and in training of human resources (Wig *et al.* 1977).
- The low proportion of the mentally ill utilizing the Government health infrastructure at the primary care level prevents a realistic economic analysis of the services provided (Chisholm *et al.* 2000).
- A quick 5-minute questionnaire to the adult respondent in the family was found to be most suitable for a primary mental health care set-up for screening, as it can identify adults with epilepsy, psychoses and those with other psychiatric problems (Isaac 1980).
- Mental health planning and policy development at the national level needs to address the varying aspirations of the different stakeholders (community, professionals, State Government, etc.) (Reddy *et al.* 1986, Murthy 2004).
- Integrating mental health care with the general health services has the twin benefits of avoiding problems associated with a highly institutionalized and professional mode of treatment and is a key means of providing basic mental care by a simple approach (Gururaj *et al.* 1988, Murthy 2004, Reddy *et al.* 1986).
- There is a great deal of scope for mental health professionals to liaise with the education, welfare and health sectors (Murthy 1987b, Gururaj *et al.* 1988).

- Communities (including educated urban groups) are largely uninformed about the various aspects of mental health and the information possessed by them remains uncrystallized (Prabhu *et al.* 1984).
- The various pathways for care of the mentally ill include formal, informal and non-formal health professionals. Longer delays in referral are found with native healers (Gater *et al.* 1991).
- Ignorance and stigma, duration of treatment, initial side-effects due to drugs, relative distance of the health facility, etc. pose problems for the mentally ill in rural areas (Wig *et al.* 1980).
- Other unmet needs, differing concepts of mental illness, professional non-commitment, differing demand and governmental priorities, absence of a social welfare net, vertical nature of national health programmes and such other factors have limited the care that could be provided for people with mental disorders in developing countries such as India (Jacob 2001).

It is to be noted that the above key messages do not constitute true scientific evaluations, despite many of them following scientific methodologies of enquiry. In this context, there is a dire need for in-depth systematic evaluation of the many innovations that have been attempted. For example, international literature has evaluated Assertive Community Treatments and case management as models of community-based care (Marshall *et al.* 1997, Marshall *et al.* 1998). However, the precise concept, components and configurations that can mirror the existing Indian situations need to be delineated first and then evaluated.

Amid improvements in service delivery, unfortunately, even as late as 2002, there was a substantial deficit of dedicated human resources for mental health services at the national level: 52% of the districts in India did not have psychiatric facilities; there was an acute shortage of psychiatrists (77%), psychologists (97%) and psychiatric social workers (90%) (Goel *et al.* 2004). This is notwithstanding the huge variations at the individual State level. In the light of health being a fundamental right, the National Human Rights Commission and the Supreme Court have played a very proactive role in bringing to the fore the quality aspect in the delivery of health care services.

The National Mental Health Programme (NMHP)

The NMHP recognized that services need to be planned for a minimum population of 1 crore who suffer from a serious mental disorder. It classified the burden as acute mental disorders, chronic or frequently recurring mental illnesses, emotional illness, and alcohol abuse and drug dependence. It specified service components with three sub-programmes—treatment, rehabilitation and prevention—to be implemented through primary health care. The different levels identified were the village/subcentre, primary health centre, district hospital, mental hospitals and teaching psychiatric units. Emphasizing equally on mental health training, it set out an 'outline plan of action' with a 'set of targets and of detailed activities'.

Ideally, the mental health services for the country need to be undertaken in the backdrop of the estimated disease burden, expanding on health care delivery based on previous experience and in the light of the policy directions. The Programme objectives and key approaches as enunciated in 1982 have been considered as a critical framework for the current report. The District Mental Health Programme (DMHP) came to be recognized as a strategy to implement the NMHP. In a major review in 2002 undertaken prior to the expansion under the Tenth Plan, it was found that there was considerable scope for improvement in many areas of the NMHP (Goel *et al.* 2004). The specific areas that needed strengthening included examination of issues related to duration of actual implementation of the Programme, inappropriate pilot districts being chosen for implementation, problems in recruiting appropriate personnel, lack of on-the-job or periodical refresher training programmes, inadequate monitoring (no standard reporting formats, need for simple recording and reporting systems), differential (and thus ineffective) IEC materials. 'No centre had undertaken community surveys of mental disorders as they were very much preoccupied in setting up service components of the scheme.' Where functioning was better, it was observed that the District Mental Health Clinic, inpatient facility at the district hospital and community outreach and liaison with primary health centres were relatively better organized (NIMHANS 2004). However, a major bottleneck was timely release of funds by both the Central and State Governments. In many instances, these factors seriously impacted the service delivery component. A specific instance has been the decrease in the number of patients seeking care (DMHP, Thiruvananthapuram, Kerala 2004).

In this context, another ambitious attempt is being made to restructure and restrategize the NMHP. Proposing an outlay of Rs 190 crore during the Tenth Plan period, the five major domains being addressed are modernization of mental hospitals, strengthening of medical college departments of psychiatry, IEC and training, research and an 'ambulating' DMHP (Agarwal *et al.* 2004). An all-India action plan with a Vision for 2020 has been proposed and the focus is on the efforts that need to be undertaken under the umbrella of the district health care system (Goel *et al.* 2004).

Resources for mental health care delivery in India

Since the time of Independence, India has steadily built up the health care infrastructure. This formidable human and infrastructure resource could be easily utilized to deliver better mental health care services. This has been proved beyond doubt in the various pilot projects across the country and also forms the backbone of the NMHP. The suggested resource management for mental and behavioural disorders is shown below.

At the district headquarters/district hospital:

- For curative activities:
  a. The General Hospital Psychiatry Unit (GHPU) headed by a psychiatrist/trained mental health person, supported by other staff with equivalent qualifications along with ancillary staff
  b. The existing general health care services at the *taluka* hospitals/community health centres/primary health centres to cater to the management of mental disorders
- For rehabilitative activities: The integrated district disability limitation/rehabilitation facility
- For preventive and promotive activities: The DMHP officer to liaise and coordinate with other staff headed by the District Health and Family Welfare Officer (monitoring referral services, periodic review for training and supervision of staff at the primary health centre and subdistrict hospitals (first referral unit/*Taluka* Hospital/ Community Health Centre). The responsible officer should also build liaison services with the related sectors of education, women and child welfare, etc.

Interventions in the mental health care delivery system

The burden

Based on the understanding and burden of mental health problems in India as outlined in Part I of this paper, it can be estimated that a minimum of 6.7 crore persons would need immediate care for severe and moderate (easily recognizable) problems, while at the district level there would be nearly 100,000 persons requiring mental health services. The break-up as per various diagnostic categories for the average district is provided in Table 8. The break-up of the estimate for the minimum district population and maximum district population is also given in Table 8.

Framework for intervention and goals of treatment

Early diagnosis and prompt treatment forms the key approach for all conditions. For disorders such as substance abuse (alcohol, tobacco, drugs), and CMDs (depression, anxiety, emotional problems), health promotion and specific protection can have a major impact that is equivalent in terms of early diagnosis and treatment. While no single approach will yield 100% positive results, combined approaches will be of great benefit to communities.

The major goal in management is to decrease the morbidity and disability (and to a certain extent mortality) associated with the disorders. The specific goal of treatment for psychotic disorders is to induce remission so as to decrease the frequency, severity and consequences of the episodes (exacerbations) and maximize social and psychological functioning of the individual. The principal tasks of treatment during the acute phase of the illness are reduction of symptoms and risk of harm, and improvement of functioning. In the post-acute phase, the aim is to consolidate the remission, maintain continued reduction in symptoms and prevent early relapse. Maintaining or improving the level of functioning and preventing recurrences are the specific tasks during the stable phase of the illness. Undertaking a comprehensive assessment before evolving a therapeutic plan in collaboration with the family members and ensuring adherence to treatment is critical (Indian Psychiatric Society 2004).

Specific activity/intervention

The various staff-specific activities that can be effectively undertaken by various health care functionaries are:

Table 8. Burden of mental and select neurological and behavioural illnesses in a district

No. of districts in India = 593	All India	Average district	Minimum district population	Maximum district population
Population	1,028,610,328	1,734,587	1,500,000	2,000,000
Major mental, select neurological and behavioural disorders	66,859,671	112,748	97,500	130,000
Schizophrenia	3,085,831	5,204	4,500	6,000
Mood disorders	16,457,765	27,753	24,000	32,000
Cannabis users	8,228,883	13,877	12,000	16,000
Opiate users	2,057,221	3,469	3,000	4,000
Mental retardation	1,028,610	1,735	1,500	2,000
Child and adolescent disorders	25,509,536	43,018	37,200	49,600
Geriatric disorders	2,550,954	4,302	3,720	4,960
Dementia	1,563,488	2,637	2,280	3,040
Epilepsy	9,257,493	15,611	13,500	18,000
Common mental disorders	56,573,568	95,402	82,500	110,000
Alcohol users	257,152,582	433,647	375,000	500,000
Alcohol dependency	82,288,826	138,767	120,000	160,000

Note: The numbers do not total up, please see box on page 236

- Community health volunteer: Case identification, targeted referral, liaison with patients, ensuring continuity of care
- Multipurpose worker: Screening, appropriate referral, liaison with other sectors, ensuring compliance
- Health supervisors: Follow-up of cases and further referral if needed, liaison with local school authorities for delivery of life-skills education and other health promotion activities
- Medical officer: Early diagnosis of cases, management of psychiatric emergencies, pharmacological management of cases, counselling as part of preventive and promotive mental health, follow-up and referral, maintaining records, staff training, community outreach activities, ensuring continuous availability of sufficient/minimal medicines, family education activities
- Medical staff at the CHC: Early diagnosis of cases, management of psychiatric emergencies, pharmacological management of cases, counselling as part of preventive and promotive mental health, management of organic mental disorders, follow-up and referral, organizing community outreach activities, ensuring continuous availability of sufficient/minimal medicines, family education activities
- General Hospital Psychiatry Unit: Outpatient and inpatient services, pharmacological therapy, ECT, psychosocial interventions, family education, training of health personnel, rehabilitation services
- DMHP officer: Monitoring, supervision/coordination of ongoing activities in the district, surveillance and planning for services, documentation—both administrative and technical, networking, advocacy and establishing linkages, planning for training

Support systems need to be strengthened with physical and technical resources. For mental health care, advanced equipment is not required; however, an uninterrupted supply of medicines is essential and crucial.

PART IV

Emerging issues and concerns

The *World Health Report* 2001 has listed 10 minimum actions required for delivery of mental health care. These include providing treatment in primary care; making psychotropic drugs available; giving care in the community; educating the public; involving communities, families and consumers; establishing national policies, programmes and legislations; developing human resources; linking with other sectors; monitoring community health; and supporting more research. In the Indian context, some priority issues are placed herewith for planning, implementation and evaluation of the mental health care delivery system, and listed under the broad headings of Research, Service and Policy.

Research

The need of the hour is

- To develop epidemiological databases (registries) of adequate sample sizes with better funding and coordination, utilizing culture-specific study instruments, which would aid in delineating the aetiology and management of mental disorders
- To undertake research into the emerging problems of alcoholism, child mental health, geriatric mental health, adolescent health, urban health and behavioural risk factor studies
- To initiate surveillance systems to study the time-trends of existing and emerging mental and behavioural disorders at possibly district and State levels
- To conduct operational research in mental health services to examine utilization, drug availability, manpower development, removal of stigma, barriers to care, and others
- To take a closer look at the association of issues such as poverty, urbanization and changing life patterns with mental health
- To designate national co-coordinating centres to examine specific issues. A group of centres can be given collective responsibility in specific areas with adequate financial and logistical support.
- To systematically evaluate the different components of the DMHPs. The existing mechanism of reviews based on programme statistics alone would be inadequate to address the issues arising in the expansion phase of the Programme.

Service

- Ensure that commonly required minimum medicines are available in all PHCs, *taluka* and district hospitals.
- Increase the participation of medical officers and other health workers in the mental health care delivery process.
- Incorporate other support systems such as availability of basic laboratory facilities, teaching materials and others in district hospitals and PHCs.
- Ensure better coordination of rehabilitation activities, especially at the district level.
- Reduce stigma at the community level so that more needy people access available resources.
- Promote more outreach programmes through extension services, mental health camps, school mental health activities and greater interaction with NGOs.
- Improve administrative mechanisms for availability of

funds, personnel and other support mechanisms from the Centre to the State.
- Evaluation should be an inbuilt component of a service delivery programme for mid-course corrections and refinement. Undertaking a systematic, regular, time-bound evaluation of activities pertaining to mental health at the district level should be done at periodic intervals.
- Expand the existing models being tested to larger populations to widen the scope of work.
- Strengthen health promotion efforts to move towards prevention of specific mental health problems such as suicide, substance abuse, common mental disorders, etc.
- Through targeted interventions focus on vulnerable populations such as women, children, the homeless, rural population, etc. for making minimum mental health care available in India.
- Develop capacity-building measures to enlist the cooperation of society at all levels, along with support for need-based human resources.
- Integrate the mental health programme with other ongoing national health programmes such as RCH and others.

Apart from the above-mentioned issues, five major areas require the attention of policy-makers and professionals.

- **Reorientation** of the system of cure (of merely dispensing pills) to a system of care (recognizing the overall individual's needs and not just the person as a disease entity).
- **Lab to land:** Critical reappraisal of pilot projects and translating their successes into the routine health care delivery system.
- **Empowerment:** The families in the ambit of community-based services need more support than is generally available currently. There is a need to ensure that the economic productivity of the family or its members are not compromised and they are able to participate better in the programmes.
- **Better integration:** No formal mechanisms exist at present for systematic integration of both the public and private sectors, including the formal and informal sectors. Integration mechanisms spelt out in the Mental Health Policy should also be reflected in Mental Health Act and the rules thereof.
- **Psychiatric rehabilitation:** In the absence of a planned or organized programme or policy on rehabilitation of the mentally ill there is a need to ensure quality and appropriate care uniformly across the country.

Policy

- Systemic reforms and changes in mental health policies and programmes are urgently required. Outdated laws need to be replaced with more humane approaches to mental health care.
- Focus on periodic, regular, systematic evaluation of ongoing community-based interventions to identify areas requiring further improvement to enhance the systems approach.
- Support broader policy frameworks under the NMHP by working with governments, NGOs and professionals to develop integrated models of mental health care.
- Policy reforms are required to reorient and re-energize existing mental hospitals in the country to redefine their role as centres of integrated activities and to function as referral centres.
- Augment manpower development and capacity-building programmes at all professional and non-professional levels to bridge the resource gap.
- Prioritize mental health problems for research and service delivery for effective utilization of meagre resources.
- Bridge the gap between research and service through promotion of operational research in a decentralized setting.
- Integrate mental health with education, child health, women's health, empowerment, legal systems, etc.
- Change the role of mental hospitals from being mere custodial care providers of patients to centres with integrated services, human resource development, capacity building and operational research.

As India moves towards a greater market-oriented economy and globalization, mental health problems will place a greater burden on the health care systems. Neglected for too long, mental health undoubtedly deserves a better place on the public health agenda in India. There is a need to move from a piecemeal and fragmented approach to an integrated and systems approach to provide better care for those with mental illness and improve their quality of life.

Acknowledgements

The authors acknowledge with gratitude the support and cooperation of the National Commission on Macroeconomics and Health, Ministry of Health and Family Welfare, Government of India; Dr D. Nagaraja, Director/Vice Chancellor, NIMHANS, Bangalore; Dr Mathew Verghese, Associate Professor and Dr Vivek Benegal, Associate Professor from the Department of Psychiatry, NIMHANS, Dr Murali T., Professor and Head of Psychiatric and Neurological Rehabilitation, NIMHANS, and Research Officers Ms Shailaja B.C., Dr Srivatsa D.V. The authors would like to express their sincere thanks for the critical comments on the earlier draft by Dr Vijay Chandra, Regional Advisor, Health and Behaviour, WHO-SEARO, New Delhi, Dr Vikram Patel, Reader in International Mental Health, London School of Hygiene and Tropical Medicine, Dr Prathap Tharyan, Professor and Head of Psychiatry, CMC, Vellore and Dr D.S. Goel, National Advisor for Mental Health, Ministry of Health and Family Welfare, Government of India.

References

Adityanjee MD, Wig NN. Alcohol related problems in the emergency room of an Indian general hospital. *Australian and New Zealand Journal of Psychiatry* 1989;**23:**274–8.

Agarwal SP (ed). *Mental health—an Indian perspective 1946–2003*. New Delhi: Directorate General of Health Services, Ministry of Health and Family Welfare, Government of India; 2004.

Agarwal SP, Icchpujani RL, Shrivastava, Goel DS. Restructuring the national Mental Health Programme. In: Agarwal SP (ed). *Mental health—an Indian perspective 1946–2003*. New Delhi: Directorate General of Health Services, Ministry of Health and Family Welfare, Government of India; 2004:119–31.

Ahmad A, Sen AK. Prevalence of drug abuse among students of Jamia Millia Islamia: A survey report. *Disabilities and Impairments* 1998;**12:**31–9.

Amin G, Shah S, Vankar GK. The prevalence and recognition of depression in primary care. *Indian Journal of Psychiatry* 1998;**40:**364–9.

Anand K. Assessment of burden and surveillance of major non-communicable diseases in India. New Delhi: World Health Organization (WHO), South East Asia Regional Office, Workshop document; 2000.

Andrews G, Sanderson K, Slade T, Issakidis C. Why does the burden of disease persist? Relating the burden of anxiety and depression to effectiveness of treatment. *Bulletin of the World Health Organization* 2000;**78:**446–54.

Astbury J. *The state of evidence: Gender disparities in mental health*. Geneva: WHO; 2000.

Bagadia VN, Ayyar KS, Lakdawala PD, Susainathan U, Pradhan PV. Value of the General Health Questionnaire in detecting psychiatric morbidity in general hospital out-patient population. *Indian Journal of Psychiatry* 1985;**27:**293–6.

Balodhi JP. Psychotherapy based on Hindu philosophy. *Journal of Personality and Clinical Studies* 1990;**6:**51–6.

Banerjee G. Mental hospitals and healing practices in colonial India. 2002. Available from URL: *www.psyplexus.com* (accessed on 15 Sept 2004).

Banerjee T, Mukherjee SP, Nandi DN, Banerjee G, Mukherjee A, Sen B, *et al.* Psychiatric morbidity in an urbanised tribal (Santhal) community: A field survey. *Indian Journal of Psychiatry* 1986;**28:**243–8.

Bang AT, Bang RA. Community participation in research and action against alcoholism. *World Health Forum* 1991;**12:**104–9.

Benegal V, Velayudhan, Jain S. The social cost of alcoholism (Karnataka). *NIMHANS Journal* 2000;**18:**67–76.

Bhatia MS, Agarwal P, Rastogi V, Aseri AK, Malik SC. Psychiatric morbidity in patients attending surgery OPD. *Annals of National Academy of Medical Sciences (India)* 1989;**25:**331–6.

Bhola P, Kapur M. Child and adolescent psychiatric epidemiology in India. *Indian Journal of Psychiatry* 2003;**45:**208–17.

Bhugra D. Migration and mental health. *Acta Psychiatrica Scandinavica* 2004;**109:**243–8.

Chandra V, Ganguli M, Pandav R, Johnston J, Belle S, Dekosky ST. Prevalence of Alzheimer'disease and other dementias in rural India: The Indo–US Study. *Neurology* 1998;**51:**1000–8.

Chandran M, Tharyan P, Muliyil JP, Abraham S. Postpartum depression in a cohort of women from a rural area of Tamil Nadu, India—Incidence and risk factors. *British Journal of Psychiatry* 2002; **181:**499–504.

Chandrashekar CR, Isaac MK. Development of psychiatric epidemiology in India. *NIMHANS Journal* 1999;**17:**297–306.

Channabasavanna SM. Overview of epidemiology of drug abuse. In: Ray R, Pickens RW (eds). *Proceedings of the Indo–US Symposium on Alcohol and Drug Abuse*. Bangalore: National Institute of Mental Health and Neurosciences; 1989.

Chaturvedi HK, Mahanta J. Socio-cultural diversity and substance use pattern in Arunachal Pradesh. *India Drug and Alcohol Dependence* 2004;**74:**97–104.

Chisholm D, James S, Waddington C, Mubbashar M, Saeed K, Murthy SR, *et al. Community mental health care in low-income countries: Developing and demonstrating methods for economic analysis.* London: Institute of Psychiatry, King's College London (IOP-UK) Institute for Health Sector Development; 2000.

District Mental Health Programme (DMHP), Thiruvanathpuram, Kerala (2004). Status report of DMHP Thiruvanathapuram, Kerala 2003–2004. Available from URL: *http:dmhp.org* (accessed on 24 Jan 2005).

Dube KC, Kumar N, Dube S. Long term course and outcome of the Agra cases in the International Pilot Study of Schizophrenia. *Acta Psychiatrica Scandivica* 1984;**44:**220–7.

Dube KC. A study of prevalence and biosocial variables in mental illness in a rural and an urban community in Uttar Pradesh. *Acta Psychiatrica Scandinavica* 1970;**46:**327–59.

Eisenberg L. Social psychiatry and the human genome: Contextualizing heritability. *British Journal of Psychiatry* 2004;**184:**101–3.

Elnagar MN, Maitra P, Rao MN. Mental health in an Indian rural community. *British Journal of Psychiatry* 1971;**118:**499–503.

Ferri C, Chisholm D, Ommeren MV, Prince M. Resource utilization for neuropsychiatric disorders in developing countries: A multinational Delphi consensus study. *Social Psychiatry and Psychiatric Epidemiology* 2004;**39:**218–27.

Franscisco A. *Mental and neurological health in the 10/90 report on health research 2003–2004*. Geneva: Global Forum for Health Research; 2004.

Ganguli HC. Epidemiological finding on prevalence of mental disorders in India. *Indian Journal of Psychiatry* 2000;**42:**14–20.

Gater R, de Almeida E, Sousa B, Barrientos G, Caraveo J, Chandrashekar CR, *et al.* The pathways to psychiatric care: A cross-cultural study. *Psychological Medicine* 1991;**21:**761–74.

Goel DS, Agarwal SP, Icchpujani RL, Srivastava. Mental health 2003: The Indian scene. In: Agarwal SP (ed). *Mental health: An Indian perspective 1946–2003*. New Delhi: Directorate General of Health Services, Ministry of Health and Family Welfare, Government of India; 2004.

Gopinath PS. Epidemiology of mental illness in Indian village. Prevalence survey for mental illness and mental deficiency in Sakalawara (MD thesis), 1968.

Gourie Devi M, Gururaj G, Satishchandra P, Subbukrishna DK. Prevalence of neurological disorders in Bangalore: A community-based study with a comparison between urban and rural areas. *Neuroepidemiology* 2004;**23:**261–8.

Gourie Devi M, Gururaj G, Satishchandra P. Report of the National Workshop on Public Health Aspects of Epilepsy for Senior Personnel of State Health Departments in India. *Annals of the Indian Academy of Neurology* 1999;**2:**43–8.

Government of India. *National human development report, 2001*. New Delhi: Government of India; 2002.

Government of India. *Selected socio-economic statistics, India, 2002*. New Delhi: Central Statistical Organization, Ministry of Statistics and Programme Implementation, Government of India; 2004.

Govindaswamy MV, Ramachandra Rao SK. Report of the work done on the 'Pilot studies on mental morbidity in selected parts of Mysore state' inquiry under Dr Govindaswamy up to the end of Sept 1958. *PRATIBHA–Journal of the All India Institute of Mental Health* 1959;**2:**32–42.

Gupta SG, Sethi BB. Prevalence of mental retardation in Uttar Pradesh. *Indian Journal of Psychiatry* 1970;**12:**264–72.

Gururaj G, Girish N, Benegal V. Psycho-social impact of alcohol—the hidden public health burden. Paper presented at the 31st Annual Conference of Indian Association of Preventive and Social Medicine, Chandigarh, 2004.

Gururaj G, Girish N, Isaac MK. Mental, neurological and substance abuse disorders: Strategies towards a systems approach. Report submitted to the National Commission of Macroeconomics and Health. Ministry of Health and Family Welfare, Government of India, New Delhi, 2005.

Gururaj G, Isaac MK, Girish N, Subbakrishna DK. Final report of the pilot study establishing health behaviour surveillance in respect of mental health. Report submitted to Ministry of Health and Family Welfare, Government of India and WHO India Country Office, New Delhi, 2004.

Gururaj G, Isaac MK. Psychiatric epidemiology in India—moving beyond numbers. In: Agarwal SP (ed). *Mental health—an Indian perspective 1946–2003*. New Delhi: Directorate General of Health Services, Ministry of Health and Family Welfare, Government of India; 2004.

Gururaj G, Reddy GNN, Subbakrishna DK. Service utilization pattern in extension services of NIMHANS. *NIMHANS Journal* 1988;**6:**91.

Halliburton M. Finding a fit: Psychiatry pluralism in south India and its implications for WHO studies of mental disorder. *Transcultural Psychiatry* 2004;**41:**80–98.

Harding TW, Arango De MV, Baltazar J, Climent CE, Ibrahim HHA, Ladrido-Ignacio L, *et al*. Mental disorders in primary health care—a study of their frequency and diagnosis in four developing countries. *Psychological Medicine* 1980;**10:**231–41.

Harrison G, Hopper K, Craig T, Laska E, Siegel C, Wanderling J. Recovery from psychotic illness: A 15- and 25-year international follow-up study. *British Journal of Psychiatry* 2001;**178:** 506–17.

Hazarika NC, Biswas D, Phukan RK, Hazarika D**,** Mahanta J. Prevalence and pattern of substance abuse at Bandardewa, a border area of Assam and Arunachal. *Indian Journal of Psychiatry* 2000;**42:**262–66.

Hyman SE. The genetics of mental illness: Implications for practice. *Bulletin of the World Health Organization* 2000;**78:**455–63.

ICMR. Collaborative study on severe mental morbidity report. Indian Council of Medical Research and Department of Science and Technology, New Delhi, 1987.

ICMR. Epidemiological study of child and adolescent psychiatric disorders in urban and rural areas, 2001. (Unpublished data)

ICMR–CAR–CMH. Report of the longitudinal study of mental health problems in a primary health centre area. Bangalore, 1990 (mimeograph).

ICMR–SOFACOS. Final report of the multi-centred collaborative study on factors associated with the course and outcome of schizophrenia. New Delhi: ICMR; 1988.

ICMR–SOFPUC. Longitudinal study of functional psychosis in an urban community. New Delhi: ICMR; 1990.

Indian Psychiatric Society. Task Force on clinical practice guidelines for psychiatrists in India. Draft proposals on clinical practice guidelines for psychiatrists in India presented at the National workshop, Jaipur 2004.

Isaac MK. Contemporary trends of alcoholism in India. In: Grant M (ed). *Alcohol and emerging markets: Patterns, problems and responses*. Ann Arbor, USA: Taylor & Francis International Centre for Alcohol Policies; 1998:145–76.

Isaac MK. A cost-effectiveness analysis of three different methods of psychiatric case finding in the general population. *British Journal of Psychiatry* 1980;**137:**540–6.

Jablensky A. Schizophrenia. Recent epidemiologic issues. *Epidemiologic Reviews* 1995;**17:**10–20.

Jacob KS. Community care for people with mental disorders in developing countries. *British Journal of Psychiatry* 2001;**178:** 296–8.

Jha S, Patel R. Some observations on the spectrum of dementia. *Neurology India* 2004;**52:**213–14.

Kapur M. Promotive and intervention strategies in the community. In: Kapur M *et al.* (eds). *Child mental health*. Proceedings of the Indo–US symposium. National Institute of Mental Health and Neuro Sciences, Bangalore, 1993.

Kapur RL, Chandrashekar CR, Shamsundar C, Isaac MK, Parthasastry R, Shalini S. Extension of mental health services through psychiatric camps: A new approach. *Indian Journal of Psychiatry* 1982;**24:**237–41.

Kessler RC. Psychiatric epidemiology: Selected recent advances and future directions. *Bulletin of the World Health Organization* 2000;**78:**464–74.

Kiely M. The prevalence of mental retardation. *Epidemiologic Reviews* 1987;**9:**194–218.

Kishore J, Reddaiah VP, Kapoor V, Gill JS. Characteristics of mental morbidity in a rural primary health centre of Haryana. *Indian Journal of Psychiatry* 1996;**38:**137–42.

Lal B, Singh G. Alcohol consumption in Punjab. *Indian Journal of Psychiatry* 1978;**20:**217–25.

Madhavan T. Childhood psychiatric epidemiology—community surveys. Workshop on research issues in psychiatric epidemiology in India, 1987 (unpublished).

Marshall M, Gray A, Lockwood A, Green R. Case management for people with severe mental disorders. Cochrane review; 1997.

Marshall M, Lockwood A. Assertive community treatment for people with severe mental disorders. Cochrane review; 1998.

Mathai JP *et al.* Psychiatric practice through satellite units—Report of an experience. *Indian Journal of Psychological Medicine* 1984;**7:**66–72.

Meena PK, Vohra AK. Prevalence and pattern of alcohol and substance abuse in urban areas of Rohtak city. *Indian Journal of Psychiatry* 2002;**44:**348–52.

Mehta P, Joseph A, Verghese A. An epidemiologic study of psychiatric disorders in a rural area in Tamil Nadu. *Indian Journal of Psychiatry* 1985;**27:**153–8.

Menon S. Psychosocial rehabilitation: Current trends. *NIMHANS Journal* 1996;**14:**295–305.

Mitchell PB, Pavlavic DH. Lithium treatment for bipolar disorder. *Bulletin of the World Health Organization* 2000;**78:**515–17.

Mohan D, Chopra A, Sethi H. A rapid assessment study on prevalence of substance abuse disorders in metropolis Delhi. *Indian Journal of Medical Research* 2001;**114:**107–14.

Mohan D, Chopra A, Sethi H. Incidence estimates of substance use disorders in a cohort from Dellhi, India. *Indian Journal of Medical Research* 2002;**115:**128–35.

Mohan D, Desai NG, Chopra A, Sethi H. Rapid survey and substance abuse disorder in the urban slums of New Delhi. *Indian Journal of Medical Research* 1992;**96:**122–7.

Mueser KT, McGurk SR. Seminar on schizophrenia. *Lancet* 2004;**363:**2063–72.

Murthy RS, Kala R, Wig NN. Mentally ill in a rural community—some initial experiences in case identification and management. *Indian Journal of Psychiatry* 1978;**20:**143–7.

Murthy RS. Disaster and mental health: Responses of mental health professionals. *Indian Journal of Social Work* 2000;**61:**675–92.

Murthy RS. Integration of mental health with primary health care—Indian experience. In: Murthy RS, Burns BJ (eds). *Community mental health*. Proceedings of the Indo–US symposium. National Institute of Mental Health and Neurosciences, Bangalore, 1987b.

Murthy RS. Integration of molecules and milieu: Towards an understanding of the mind. Souvenir of the 57th Annual National Conference of the Indian Psychiatric Society, 29 January–1 February 2005, Chandigarh.

Murthy RS. Mental health in the new millennium: Research strategies for India. *Indian Journal of Medical Research* 2004;**120:**63–6.

Murthy RS. Overview of psychiatric epidemiology in India. Workshop on research issues in psychiatric epidemiology in India, 1987a (unpublished).

Murthy RS. *Schizophrenia. Country Report, India.* Bangalore: Department of Psychiatry, NIMHANS; 1999.

Nambi SK, Prasad J, Singh D, Abraham V, Kuruvilla A, Jacob KS. Explanatory models and common mental disorders among patients with unexplained somatic symptoms attending a primary care facility in Tamil Nadu. *National Medical Journal of India* 2002;**15:**331–5.

Nandi DN, Ajmany S, Ganguli H, Banerjee G, Boral GC, Ghosh A, *et al.* The incidence of mental disorders in one year in a rural community in West Bengal. *Indian Journal of Psychiatry* 1976;**18:**79–87.

Nandi DN, Ajmany S, Ganguli H, Banerjee G. Psychiatric disorders in a rural community in West Bengal: An epidemiological study. *Indian Journal of Psychiatry* 1975;**17:**87–9.

Nandi DN, Banerjee G, Chowdhury AN, Banerjee T, Boral GC, Biswajit S. Urbanisation and mental morbidity in certain tribal communities in West Bengal. *Indian Journal of Psychiatry* 1992;**34:**334–9.

Nandi DN, Banerjee G, Boral GC, Ganguli H, Ajmany S, Ghosh A, *et al.* Socio-economic status and prevalence of mental disorders in certain rural communities in India. *Acta Psychiatrica Scandinavica* 1979;**59**:276–93.

Nandi DN, Banerjee G, Ganguli H, Ajmany S, Boral GC, Ghosh A, *et al.*The natural history of mental disorders in a rural community—longitudnal study. *Indian Journal of Psychiatry* 1978;**21:**390–6.

Nandi DN, Banerjee G, Mukherjee SP, Ghosh A, Nandi AS, Nandi S. Psychiatric morbidity of a rural Indian community—changes over a 20-year interval. *British Journal of Psychiatry* 2000;**176:**351–6.

Nandi DN, Banerjee G, Mukherjee SP, Sarkar S, Boral GC, Mukherjee A, *et al.* A study of psychiatric morbidity of a rural community at an interval of 10 years. *Indian Journal of Psychiatry* 1986;**28:**179–94.

Nandi PS. A study of psychiatric morbidity of the elderly population of a rural community in West Bengal. *Indian Journal of Psychiatry* 1997;**39:**122–9.

NIMHANS. Evaluation of the district mental health programme. Report submitted to the Ministry of Health and Family Welfare, Government of India, 2004.

NSSO. Disabled persons in India. New Delhi: NSSO, 58th round; July–December 2002; Report no 485, Government of India.

NSSO. Morbidity and treatment of ailments. New Delhi: NSSO, 52nd round; November 1998.

Padmavathi R, Rajkumar S, Narendra Kumar, Manoharan A, Kamath S. Prevalence of schizophrenia in an urban community in Madras. *Indian Journal of Psychiatry* 1987;**31:**233–9.

Parkar SR, Dawani VS, Apte JS. History of psychiatry in India. *Journal of Postgraduate Medicine* 2001;**47:**73–6.

Patel V, Kleinman A. Poverty and common mental disorders in developing countries. *Bulletin of the World Health Organization* 2003;**81:**609–15.

Patel V, Marilyn R, D'Souza N. Gender poverty and postnatal depression: A study of mothers in Goa, India. *American Journal of Psychiatry* 2002;**40:**364–9.

Patel V, Pereira J, Mann AH. Somatic and psychological models of common mental disorder in primary care in India. *Psychological Medicine* 1998;28:135–43.

Peng FC. Is dementia a disease? *Gerontology* 2003;**49:**384–91.

Ponnudarai R, Jayakar J, Raju B, Pattamuthu R. An epidemiological study of alcoholism. *Indian Journal of Psychiatry* 1991;**33:**176–9.

Pothen M, Kuruvilla A, Philip K, Joseph A, Jacob KS. Common mental disorders among primary care attenders in Vellore, south India: Nature, prevalence and risk factors. *International Journal of Social Psychiatry* 2003;**49:**119–25.

Prabhu GG, Raghuram A, Verma N, Maridass AC. Public attitudes toward mental illness: A review. *NIMHANS Journal* 1984;**2:**1–14.

Prabhu GG. Child and adolescent mental health research in India: An overview. *NIMHANS Journal* 1987;**5:**79–87.

Premarajan KC, Danabalan M, Chandrashekar R, Srinivasa DK. Prevalence of psychiatry morbidity in an urban community of Pondicherry. *Indian Journal of Psychiatry* 1993;**52:**99–102.

Puri DK. Depression in general clinical practice—a diagnostic problem. *Journal of the Indian Medical Association* 1995;**93:**103–4.

Rajkumar S, Kumar S, Thara R. Prevalence of dementia in a rural setting: A report from India. *International Journal of Geriatric Psychiatry* 1997;**12:**702–7.

Rajkumar S, Padmavathi R, Thara R, Menon S. Incidence of schizophrenia in an urban community in Madras. *Indian Journal of Psychiatry* 1993;**35:**18–21.

Rajkumar S. Epidemiology and cause of schizophrenia in India. In: Koslow *et al.* (eds). *Decade of brain. India/USA research in mental health and neurosciences.* Rockville, MD: US Department of Health and Human Services, National Institute of Mental Health; 1987:95–100.

Rao V. Psychiatric morbidity in the aged. *Indian Journal of Medical Research* 1997;**106:**361–9.

Reddy GNN, Channabasavanna SM, Devi GM, Prabhu GG, Shariff IA, Kalipernumal VG, *et al.* Extension of mental health services by satellite clinics as a model. *NIMHANS Journal* 1986; **4:**71–5.

Reddy GNN. Innovations in neuropsychiatric services. *NIMHANS Journal* 1983;**1:**1–14.

Reddy MV, Chandrashekar CR. Prevalence of mental and behavioral disorders in India: A meta-analysis. *Indian Journal of Psychiatry* 1998;**40:**149–57.

Reddy PR, Murthy KK, Anand B. An interval study of mental morbidity in a south Indian rural community in 1981–91. *Indian Journal of Social Psychiatry* 1994;**10:**11–19.

Rose S. Moving on from old dichotomies: Beyond nature–nurture towards a lifeline perspective. *British Journal of Psychiatry* 2001;**178:**S3–S7.

Roth M. The diagnoses of dementia in late and middle age of life. In: Mortimer JA (ed). *The epidemiology of dementia.* Oxford: Oxford University Press; 1980.

Sachdeva JS, Singh S, Sidhu BS, Goyal RKD, Singh J. An epidemiological study of psychiatric disorders in rural Faridkot (Punjab). *Indian Journal of Psychiatry* 1986;**28:**317–23.

Satija DC, Khatri JS, Satija YK, Nathawat SS. A study of prevalence and patterns of drug abuse in industrial workers. *International Journal of Social Psychiatry* 1997;**13:**47–52.

Sen B. Psychiatric phenomena in primary health care—their extent and nature. *Indian Journal of Psychiatry* 1987;**39:**200–7.

Seshadri S. An overview of child psychiatric epidemiology in India. In: Kapur M, Kellam S, Tarker R, Wilson R (eds). *Child mental health.* Proceedings of the Indo–US symposium, National Institute of Mental Health and Neuro Sciences, Bangalore, and Alcohol, Drug Abuse and Mental Health Administration, USA. 1993; 61–5.

Sethi BB, Gupta SC, Kumar R, Kumari P. A psychiatric survey of 500 rural families. *Indian Journal of Psychiatry* 1972;**14:**183–96.

Sethi BB, Gupta SC. An epidemiological and cultural study of depression. Paper presented at the 22nd Annual Conference of Indian Psychiatric Society, Hyderabad, 1970.

Sethi BB, Trivedi JK. Drug abuse in a rural population. *Indian Journal of Psychiatry* 1979;**21:**211–12.

Shah VA, Goswami UA, Maniar RC, Hajariwala DC, Sinha BK. Prevalence of psychiatric disorders in Ahmedabad (an epidemiological study). *Indian Journal of Psychiatry* 1980; **22**:384–9.

Shaji S, Promodu K, Abraham T, Roy KJ, Verghese A. An epidemiological study of dementia in a rural community in Kerala, India. *British Journal of Psychiatry* 1996;**168:**745–9.

Shaji S, Verghese A, Promodu K, George B, Shibu VP. Prevalence of priority psychiatric disorders in a rural area of Kerala. *Indian Journal of Psychiatry* 1995;**37:**91–6.

Sharma S, Singh MM. Prevalence of mental disorders: An epidemiological study in Goa. *Indian Journal of Psychiatry* 2001;**43:**118–26.

Sharma V, Murthy S, Kumar K, Agarwal M, Wilkinson G. Comparison of people with schizophrenia from Liverpool, England and Sakalwara, Bangalore, India. *International Journal of Social Psychiatry* 1998;**44:**225–30.

Shyamasundar C, Krishnamurthy S, Prakash O, Prabhakar N, Subbakrishna D. Psychiatric morbidity in a general practice in an Indian city. *British Medical Journal* 1986;292:1713–15.

Srinath S, Girimaji SC. Epidemiology of child and adolescent mental health problems and mental retardation. *NIMHANS Journal* 1999;**17:**355–66.

Sriram TG, Chandrashekar CR, Isaac MK, Murthy RS, Shanmugham V. Training primary care medical officers in mental care: An evaluation using a multiple-choice questionnaire. *Acta Psychiatrica Scandinavica* 1990;**81:**414–17.

Surya NC, Datta SP, Krishna GR, Sundaram D, Kutty J. Mental morbidity in Pondicherry. *Transactions of the All India Institute of Mental Health* 1962;51–61.

Thacore VR, Gupta SC, Suraiya M. Psychiatric morbidity in a north Indian community. *British Journal of Psychiatry* 1975;**126:**364–9.

Thara R, Srinivasn L. Disabilities in schizophrenia. *Indian Journal of Psychiatry* 1998;**40:**331–7.

Thara R. Focus of Psychiatry in India. *British Journal of Psychiatry* 2004a;**184:**366–73.

Thara R. Twenty-year course of schizophrenia. The Madras longitudinal study. *Canadian Journal of Psychiatry* 2004b; **49:**564–9.

United Nations and Government of India. The extent, pattern and trends of drug abuse in India – National Survey – 2004, Office of Drugs and Crime, United Nations and Ministry of Social Justice and Empowerment, Government of India, New Delhi, 2004.

Varma VK, Singh A, Singh S, Malhotra A. Extent and pattern of alcohol use and alcohol related problems in North India. *Indian Journal of Psychiatry* 1980;**22:**331–7.

Vas CJ, Pinto C, Panikker D, Noronha S, Deshpande N, Kulkarni L, *et al.* Prevalence of dementia in an urban Indian population. *International Psychogeriatrics* 2001;**13:**439–50.

Verghese A, Beig A, Senseman LA, Rao SSS, Benjamin. A social and psychiatric study of a representative group of families in Vellore town. *Indian Journal Medical Research* 1973;**61:**608–20.

Verma OP. Forty years after Bhore Committee. Dr BC Dasgupta memorial oration delivered at the 30th Annual Conference of the Indian Public Health Association, Calcutta, 1986.

Vohra AK, Yadav BS, Khurana HA. Study of psychiatric co-morbidity in alcohol dependence. *Indian Journal of Psychiatry* 2003;**45:** 247–50.

Vyas JN, Ahuja N (eds). *Textbook of postgraduate psychiatry.* New Delhi: Jaypee Publishers; 1999:1–2.

Walker E, Kestler L, Bollini A, Hochman KM. Schizophrenia: Course and etiology. *Annual Reviews in Psychiatry* 2004;**55:**401–30.

WHO and Ministry of Health and Family Welfare, Government of India. Epidemiological study of mental disorders; ongoing multicentric study, 2003.

WHO. *'Get high on alcohol without alcohol': Prevention of harm from alcohol use,* New Delhi: WHO, SEARO; 2003.

WHO. Child and adolescent mental health policies and plans: Mental Health Policy and service guidance package. Geneva: World Health Organization; 2005.

WHO. *From prejudice to hope: Epilepsy out of the shadows.* New Delhi: World Health Organization, SEARO; 2001b.

WHO. Schizophrenia: A multinational study: Public health paper No 63, World Health Organization, Geneva, 1975.

WHO. *The world health report. Mental health: New understanding, new hope.* Geneva: World Health Organization; 2001.

Wig NN, Murthy RS, Mani M, Arpan D. Psychiatric services through peripheral health centers. *Indian Journal of Psychiatry* 1980;**22:**311–16.

Wig NN, Murthy RS. Collaboration in mental health programmes: Need and scope in India. *Indian Journal of Psychiatry* 1977;**19:**60–8.

Wig NN, Vijoy KV, Mattoo SK, Behere PB, Misra AK, Srinivasa Murthy R, *et al.* An incidence study of schizophrenia in India. *Indian Journal of Psychiatry* 1993;**35:**27–39.

Appendix 1

Table A1.1 Prevalence rates of all mental disorders—combined rural and urban studies

Author and year	Place	Sample size	Screening instrument	Crude rate per 1000	Male rates	Female rates
Dube 1970	Agra	29,468	Prepared schedule	23.8 Rural: 18 Semi rural: 25 Urban: 25	15.8	33.3
Reddy *et al.* 1998	All India	33,572	NA	58.2 Urban: 79.1 Rural: 37.1	40.5	49.9
Ganguli 2000	All India	NA	NA	73 Urban: 73 Rural: 70.5	NM	NM
Sharma *et al.* 2000	Goa	4,022	Rapid Psychiatric Examination Schedule	60.2 Urban: 59 Rural: 61	85.7	35.6

NA: Not applicable; NM: not mentioned

Table A1.2 Prevalence rates of all mental disorders—urban studies

Author and year	Place	Sample size	Screening instrument	Crude rate per 1000	Male rates	Female rates
Surya *et al.* 1962	Pondicherry	2,731	Symptom checklist	9.5	NA	NA
Verghese *et al.* 1973	Vellore	26,039	Mental Health item sheet	66.5	60	73
Thacore *et al.* 1975	Lucknow	2,696	Health questionnaire	81.6	85	78
Shah *et al.* 1980	Ahmedabad	2,712	58 question symptom checklist	47.2	39	56
Banerjee *et al.* 1986	West Bengal	771	4 schedules prepared	51.9	54	49.7
Gopinath 1988	Bangalore	423	Mental illness questionnaire	16.54	NA	NA
Premarajan *et al.* 1989	Pondicherry	1,115	Modified IPSS	99.4	85.3	113.9

NA: not available; IPSS: Indian Psychiatric Survey Schedule

Table A1.3 Prevalence rates of all mental disorders—rural studies

Author and year	Place	Sample size	Screening instrument	Crude rate per 1000	Male rates	Female rates
Elnagar *et al.* 1971	Nasibpur village, Bengal	1,383	3 stage interview	27	36	19
Sethi and Gupta 1972	Lucknow	2,691	Questionnaire	39.4	50	23.5
Nandi *et al.* 1975	West Bengal	1,060	3 schedules prepared	103	91	115
Nandi *et al.* 1976	West Bengal	1,078	3 schedules prepared	108	101	114
Nandi *et al.* 1977	West Bengal	2,918	4 schedules prepared	58.3	57.5	59
Murthy *et al.* 1978	Haryana	2,500	Case vignettes	12.4	NM	NM
Nandi *et al.* 1978	West Bengal	1,259	3 schedules prepared	48	41	55
Nandi *et al.* 1979	West Bengal	3,718	4 schedules prepared	102	75.4	128.5
Mehta *et al.* 1985	Vellore	5,941	Symptoms in others of IPSS	14.5	16	13
Reddy *et al.* 1981	Andhra Pradesh	967	IPSS	59	52	67
Sachdeva *et al.* 1986	Faridkot	1,989	IPSS, symptoms in others	22	22	22.3
Nandi *et al.* 1986	West Bengal	1,539	4 schedules prepared	81.9	78.3	85.2
ICMR 1987	Calcutta	34,582	IPSS, symptoms in others	8.3	8.3	8.3
	Baroda	39,655	IPSS, symptoms in others	4.6	5.1	3.9
	Patiala	36,595	IPSS, symptoms in others	14.1	12.8	15.7
	Bangalore	35,548	IPSS, symptoms in others	11.1	12.8	9.3
Reddy *et al.* 1991	Andhra Pradesh	1,964	IPSS	58	47	70
Shaji *et al.* 1995	Ernakulam, Kerala	1,094	Symptoms in others, IPSS	14.6	12.3	16.87
Nandi *et al.* 2000	West Bengal	3,488	4 schedules prepared	105.2	73.5	138.3
Nandi *et al.* 2000	West Bengal	2,183	4 schedules prepared	116.8	86.9	146.8

IPSS: Indian Psychiatric Survey Schedule; NM: not mentioned

Economic burden of asthma

K.J.R. MURTHY, J.G. SASTRY

'Asthma' is a Greek word which means 'breathless' or 'to breathe with open mouth'. The Global Strategy for Asthma Management and Prevention Guidelines define asthma as 'a chronic inflammatory disorder of the airways associated with increased airway hyper-responsiveness, recurrent episodes of wheezing, breathlessness, chest tightness, and coughing, particularly at night/early morning'. Airway inflammation produces airflow limitation through acute bronchoconstriction, chronic mucus plug formation and airway wall swelling or remodelling. These symptoms may be relieved either spontaneously or after treatment. Asthma can occur at any age. However, in half the cases the onset is before 10 years of age. Asthma is thought to affect about 3% of the population in most countries. The highest prevalence (almost 30%) is found in New Zealand. The prevalence in a number of countries falls in the range of 10%–17%.[1]

Need to study the burden of the disease

A study of the burden of a disease is most needed by any health planner who is responsible for making specific plans to combat, contain and eliminate the disease in the population/community. As most planners are economists who prefer to optimize resource allocations based on the financial implications of the problem for the nation and the quantum of expenditure incurred by the population, this chapter documents the current prevalence of asthma in India and the amount of money presently spent by people for treatment, either to get cured or ameliorate suffering.

Review of the literature

Variation in the definition of asthma is likely to lead to inconsistent results among different studies. The following three principal methods have been used to define asthma in epidemiological studies:

- A positive answer to a question asking whether the subject has asthma, sometimes qualified by a further question whether this has been confirmed by a doctor
- A physiological measurement of increased bronchial responsiveness
- A positive answer to a question on symptoms, almost always a question on wheezing in the English language studies on asthma.

International database

- An overview of findings from several surveys investigating children and adults in Eastern and Western Europe shows a marked difference in the prevalence of asthma depending upon the criteria used to define asthma.[1]
- Two surveys conducted on British children 12 years of age between 1973 and 1988 showed that the prevalence of asthma increased from 5.6% to 10.3% in boys and from 2.7% to 7.9% in girls. The overall prevalence increased from 4.2% to 9.1%.[2]
- In New Zealand, the prevalence of asthma increased from 26.2% to 34.0% between 1975 and 1989 among adolescents (12–18 years of age).[3]
- From 1969 to 1982, the reported prevalence of ever having asthma among 11–13-year-old children in New Zealand increased from 7.1% to 13.5%.[4]
- In Finland, men of conscription age (20 years) were studied during 1926 and 1939 and the prevalence remained steady between 0.02% and 0.08%. Even in 1981, the rate plateaued at 0.08%. However, by 1989, it had reached 1.79%.[5]
- The increase in the prevalence of asthma was more in boys (2.8% in 1964 to 6.6% in 1989) than in girls (1.3% in 1964 to 3.8% in 1989) and it rose from 4.1% in 1964 to 10.2% in 1989 among schoolchildren of Aberdeen in the age group of 8–13 years.[6]
- Age-specific rates of admission to hospital for asthma in all the age groups and both the sexes in England and Wales showed an increasing trend during the period 1976 to 1991–92.[7]
- In England, from 1956–57 to 1968–69, the reported prevalence of currently diagnosed asthma among subjects

Mahavir Hospital and Research Centre
10/1/1, Mahavir Marg, A.C. Guards
Hyderabad 500004
e-mail: kollurijrm@hotmail.com; gjandhyala@hotmail.com

in the age group of 4–18 years increased from 1.8% to 2.3%.[8]

- A mixed longitudinal study of primary schoolchildren in England was conducted between 1973 and 1986. The results demonstrate that there is a true increase in morbidity.[9]
- The prevalence of asthma in children 6–17 years of age was studied through a nationwide survey in the USA which shows a higher prevalence in boys than in girls, and the prevalence increased from 1963–65 to 1976–80. No relationship between the socioeconomic status and asthma was seen.[10]
- The prevalence of asthma ranged from 1.7% in 0–9-year-old children in urban Ethiopia to 9.4% in those in the age group of 60–69 years; from 1.1% in 0–9-year-old children to 3.3% in those in the age group of 60–69 years in rural areas. The prevalence was higher in urban (3.6%) than in rural areas (1.3%).[11]
- A nationally representative sample of 0–17-year-old children in the USA was surveyed in 1981 and 1988. The results indicate that the prevalence of asthma is higher in boys than in girls (1981: boys 3.8%, girls 2.3%; 1988: boys 5.1%, girls 3.4%) and there was an increase in the prevalence during this period. Children born with normal weight (>2.5 kg) had a lower prevalence of asthma than those with low birth weight. Further, the risk of asthma in children from low-income families was high at both time points of the survey.[12]
- The prevalence of asthma has been increasing for the past two to three decades in the industrialized countries of the West. The overall current prevalence of asthma is 8%–10% in the USA, and 20%–25% in the UK, Australia and New Zealand.[13]
- In a study on students in the age group of 10–18 years in Chandigarh, 2.3% were diagnosed with asthma. The prevalence varied with age and the lowest prevalence was seen among those in the age group of 13–14 years.[14]
- Respiratory syncitial virus, parainfluenza virus, corona virus and adenovirus were the predominant ones isolated.[15]

Major observations of Indian studies

Barring the data collected by the National Family Health Survey-2 (NFHS-2) during 1998–99, there are no reports on the magnitude of the problem either at the national or at the State level. The results of several specific surveys/ investigations done on small samples at different places are documented in various scientific journals and some of these form the basis for the present review.

- According to the NFHS-2 report the estimated prevalence of asthma in India is 2468 per 100,000 persons.[16] The prevalence was higher in rural than in urban areas (2649 *v*. 1966). The prevalence among males was slightly higher (2561) than among females (2369). Among those below 15 years of age, asthma was seen in 950 per 100,000 persons. The prevalence rate was 2309 among those in the age group of 15–59 years, while it was 10,375 in those above 60 years of age.
- The prevalence of asthma in adult males (18 years and above) during 1995–97 was 3.94% in urban and 3.99% in rural areas. In females of the same age group, the prevalence was 1.27% in urban as well as rural areas. In earlier studies in the 1960s on adults (above 18 years of age), the prevalence of asthma in Delhi was 1.8% and 1.76% in Patna.[17]
- Among adults in the age group of 20–45 years, no specific age-related pattern in the prevalence was seen in Mumbai.[18] The study also revealed a ratio of 6.7 untreated cases to each treated case of asthma. A strong correlation of asthma with the family history was also seen.
- The following observations were made from studies on children below 18 years of age in Bangalore:[19]
  - —*Time trends:* The prevalence of asthma increased with time. The regression equation [authors' calculation] is:

    Asthma (%) = 18.4 + 1.1∗ (year – 1989); correlation coefficient = 0.9859. The prevalence of persistent asthma also increased from 20% to 27.5%, and that of persistent severe asthma increased from 4% to 6.5% between 1994 and 1999.
  - —*Urban and rural:* A prevalence of 5.7% among the rural and 16.64% among the urban groups showing that the prevalence of asthma is 2.9 times higher in urban children.
  - —*Effect of traffic:* Children attending schools located in areas with high- and low traffic zones have different levels of asthma prevalence (Table 1).
  - —Among those with asthma, 80% were suffering intermittently in 1994 and by 1999, this reduced to 74.3%. Among those with persistent asthma, 64% were suffering from mild asthma, 32% from moderate and the remaining 4% from severe asthma during 1994. The prevalence of severe asthma has shown an increase and reached 6.5% during 1999.
  - —*Age of onset:* The age of onset of asthma was 1 year in more than 26% of the cases; 52% had asthma when they were 1–5 years of age. In only 22% was the onset after 5 years of age.
  - —*Gender differences:* Among patients with asthma, 64% were males and 36% females. This sex ratio is dependent on the living conditions such as poor ventilation, living space and the type of cooking fuel used in their families.

Table 1. Prevalence of asthma in schoolchildren

Area	Socioeconomic status of children	Prevalence (%)
Heavy traffic zone	Affluent	19.34
Heavy traffic zone	Less affluent	31.14
Low traffic zone	Affluent	11.15

—*Role of family history:* The incidence of asthma was 18.79% if one of the parents had asthma, 1.65% if one of the siblings had asthma, and 4.12% if the grandparents had the disease.
—*Causes and symptoms:* Viral upper respiratory tract infections were identified as the triggering factor for asthma among 40% of children.
—*Seasonal variation:* More than one-third of the children had attacks of asthma during a specific season. Only 3% of these episodes occurred during the summer months.
—*Food item as a causal factor:* About one-fifth of the children seem to suffer from food-related asthma. The most blamed offenders are grapes (57%), bananas (53%), guavas (51%), citrus fruits (28%), ice cream (21.5%), fried foods (19%) and tomatoes (12.5%).

- Peak expiratory flow rate (PEFR) studies on 6–15-year-old schoolchildren from urban and rural areas of Bangalore and Karnataka have shown that 16.61% of them had respiratory symptoms and low values of PEFR. The PEFR values were similar for boys and girls from urban and rural areas.[20]
- The prevalence of bronchial asthma in women from villages near Chandigarh city was 0.6% and it varied from a low of 0.2% among LPG users to a high of 0.9% in those using stoves for cooking. However, 2.9% of those using a *chullah* for cooking had chronic bronchitis as compared to the overall prevalence of 1.9% in the community.[21]

The above studies show that in recent years, the number of asthma cases has increased rapidly in many parts of the world.[19,22,23] National surveys in some countries have shown that the increase is not due to a decrease in remissions. Thus, the proportion of people seeking medical help for asthma is increasing.[24]

Risk factors for asthma

Allergens

- The exposure of sensitive persons to allergens increases bronchial hyper-responsiveness. This increase is related to the occurrence of a late asthmatic response.[25]

Smoking

- Smoking has been associated with airway hyper-responsiveness in a number of surveys as well as in clinical studies.[26] The association is stronger in the elderly or in those with a greater lifetime exposure to cigarettes, which is strongly correlated with age in smokers. However, there is evidence suggesting that smoking among adults does not lead to any increased risk of developing asthma.[27]
- Children with asthma who are exposed to tobacco smoke at home are reported to require an increased use of emergency rooms for the management of asthma.[28]

Air pollution

- Although there is a strong reason to believe that air pollution could play an important role in asthma, it is difficult to document evidence suggesting that this is of primary importance. Studies done in the UK to compare the prevalence of asthma in areas with different levels of pollution provide no strong evidence that pollution levels are a determinant of asthma in the UK. The classic example is of high prevalence of asthma in different areas of New Zealand which do not have high levels of any pollutants.[29]
- The prevalence of asthma in children attending schools located in heavy traffic zones (with high air pollution) was higher compared to those attending schools in less polluted areas.
- The prevalence of asthma in traffic police personnel in Bangalore was 26.12% compared to 14.09% among non-traffic police personnel, indicating an 85% increase in their risk of developing asthma.[30]

Family history

- There is a strong correlation between diagnosis of asthma and the family history of the disease as seen in studies where the prevalence of asthma ranged from 2.1% to 16.2%. On applying gene segregation models to the data, a major gene which could be involved in the allergy was found to exist. However, asthma was not fully described by a single-gene model.[18]
- House dust mite (*Dermatophagoides pteronyssinus*) was found to play a significant role in asthma in Mumbai.[18]

Data for India

Information in the literature on the prevalence of asthma in India is inadequate as most of these studies lack the basic requirements needed for this type of exercise. These requirements are: adequate sample coverage representing all age groups, data from different regions in the country and adoption of uniform protocols. The NFHS-2 is the only study which meets these requirements. Hence, the results of NFHS-2 form the basis for estimation of the national prevalence of asthma. The following databases were used for the present exercise:

- The Registrar General of India estimated the population of India for the period 1996–2016. The urban–rural composition of the population is assumed to be undisturbed during this period, with 26.82% constituting the urban population. This database was used to arrive at the estimates of cases of asthma.[31]
- Prevalence levels of asthma in different population groups:
  —*Chronic asthma:* On examining the changes in the prevalence of asthma over a period of time in countries with comparable levels as India, an increase of up to

3% over a period of 10 years is seen.[32] This change of 3% per 10 years is used here as there is no such information at different time points for India. Table 2 gives the prevalence of chronic cases of asthma for the period 1996–2016.

—*Acute asthma:* Acute cases of asthma were calculated for the three age groups using the hospitalization rates observed in Hyderabad and surrounding municipalities (Table 3).[33]

—*Treatment and opportunity costs—current costs of treatment and those given by Guidelines for the treatment of asthma:* The results of a large-scale study on the population of Hyderabad and surrounding municipalities were used along with other relevant data to calculate the costs of treatment and opportunity losses incurred by patients for getting treatment for asthma.[33,34] The current costs for treatment of asthma (Rs per patient) used to arrive at the economic burden of the disease in India are given in Table 4.[33]

It may be noted that some chronic cases of asthma go into the acute stage for short periods of time and hence the chronic and acute groups are not considered as mutually exclusive.

The cost of treatment of asthma, as per the Guidelines,[35] was calculated using the costs (Rs/case/year) presented in Table 5.

Results

1. The estimated caseload of patients with asthma in urban and rural India in different years is given in Table 6.

It is evident that the majority (about 80%) of patients with asthma live in rural areas. As the magnitude of poverty is more in the rural set-up in India, there is an urgent need to create and strengthen health facilities for handling patients with asthma.

2. The age-wise distribution of the estimated numbers of patients with asthma in urban and rural areas from 1996 to 2016 is presented in Tables 7 and 8.

It is evident that a considerable proportion of the population in the active and economically productive age groups suffers from this chronic disease. Further, the elderly (above 60 years of age) constitute the majority of cases.

3. Acute attacks of asthma need hospitalization for immediate case management. These individuals are a part of the group of patients with chronic asthma. The estimated number of acute cases of asthma are presented in Table 9. As a large number of these patients live in rural areas, the basic equipment and skills for the management of patients with acute asthma should be made available to primary health care providers in the country. In remote and backward areas, the problem of shifting the patient to the nearest health facility is the major hindrance. Hence, the initiation of necessary developmental activities in such areas such as all-weather roads and transportation facilities would go a long way in improving the health status of the needy.

Table 2. Estimates of prevalence rates of chronic asthma by age (cases/100,000 persons)

Year	Locality	<15 years	15–59 years	60 years and above
1996	Urban	827	1790	8,279
	Rural	983	2784	11,272
2001	Urban	836	1811	8379
	Rural	995	2540	11,408
2006	Urban	849	1838	8,503
	Rural	1010	2577	11,577
2011	Urban	861	1865	8,628
	Rural	1024	2615	11,747
2016	Urban	874	1892	8,752
	Rural	1039	2653	11,917

Table 3. Prevalence of acute asthma in different age groups

Age (years)	Prevalence (per 100,000 population)
<15	19.25
15–59	40.01
≥60	107.58

Table 4. Current cost of treatment of asthma (Rs/patient) per year

Year	Chronic: Mild	Chronic: Moderate and severe	Hospitalization charges per episode
1996	303	11,250	3042
2001	436	16,200	4379
2006	569	21,141	5716
2011	702	26,087	7053
2016	835	31,045	8394

Table 5. Cost of treatment of asthma (Rs/case/year) as per the Guidelines

Year	Chronic: Mild	Chronic: Moderate and severe	Acute cases	Hospitalization charges per episode
1996	277	1825	4,867	3042
2001	400	2640	7,019	4379
2006	522	3447	9,163	5716
2011	644	4253	11,306	7053
2016	762	5060	13,454	8394

Table 6. Estimated number of chronic cases of asthma (in lakh)

	Urban			Rural		
Year	Males	Females	Total	Males	Females	Total
1996	24.18	22.56	46.73	92.18	83.42	175.60
2001	27.15	24.82	51.97	100.67	94.76	195.43
2006	30.76	28.30	59.05	114.34	108.34	222.68
2011	34.57	32.05	66.62	128.69	122.89	251.58
2016	37.30	35.97	73.27	139.50	137.99	277.49

Table 7. Estimates of the number of patients with asthma (in lakh) by age (in years) in urban areas

Year	<15	15–30	30–40	40–50	50–60	60+	Total
1996	7.74	11.80	6.14	4.39	2.96	13.69	46.73
2001	7.71	13.50	6.75	4.95	3.37	15.69	51.97
2006	7.54	15.64	7.58	5.83	4.00	18.46	59.05
2011	7.67	17.07	8.38	6.77	4.76	21.96	66.62
2016	6.86	17.11	9.83	7.60	5.63	26.23	73.27

Table 8. Estimates of the number of patients with asthma (in lakh) by age (in years) in rural areas

Year	<15	15–30	30–40	40–50	50–60	60+	Total
1996	25.47	45.80	23.97	17.18	11.58	51.60	175.60
2001	25.40	52.38	26.23	19.20	13.06	59.14	195.43
2006	24.84	60.69	29.41	22.63	15.53	69.58	222.68
2011	25.25	66.26	32.52	26.29	18.49	82.78	251.58
2016	22.64	66.42	38.13	29.53	21.87	98.90	277.49

Table 9. Estimates of the caseload of patients with acute asthma (in lakh)

	Urban			Rural		
Year	Males	Females	Total	Males	Females	Total
1996	0.478	0.433	0.911	1.304	1.218	2.522
2001	0.529	0.483	1.012	1.444	1.357	2.801
2006	0.587	0.539	1.126	1.602	1.515	3.117
2011	0.645	0.596	1.241	1.760	1.676	3.436
2016	0.674	0.653	1.326	1.838	1.834	3.672

4. The estimated total cost of treatment of chronic and acute cases of asthma according to current practices for 1996 to 2016 is presented in Fig. 1, Table 10 and Fig. 2.

5. The total cost of treatment of asthma if all cases are treated according to the procedures recommended in the Guidelines:[35] The problem of asthma can be easily managed when it is in the mild stage. In fact, 90% of these cases are mild and can be successfully handled at primary health care centres. This will reduce the financial burden on the family. The remaining 10% of cases need secondary care. The cost of treatment of these cases, according to the Guidelines is given in Table 5.[35]

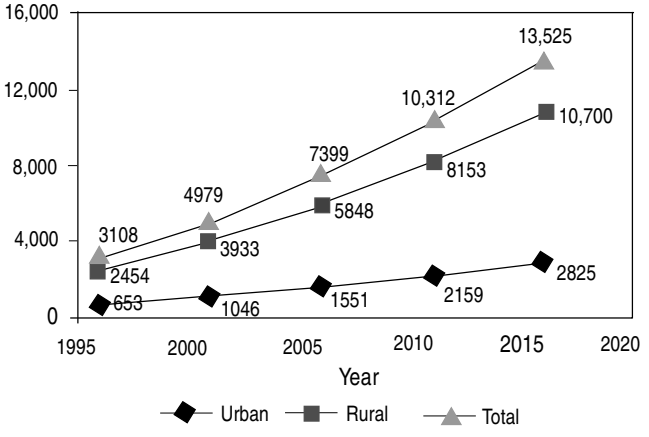

Fig. 1 Annual economic burden of chronic asthma (Rs in crore)

Table 10. Additional cost for patients with acute asthma (Rs in crore/year)

Year	Urban	Rural	Total
1996	27.72	76.71	104.42
2001	44.26	122.51	166.78
2006	64.37	178.19	242.56
2011	87.55	242.36	329.91
2016	111.32	308.26	419.59

The annual total economic burden of asthma (Rs in crore), according to the Guidelines, of chronic and acute case management has been worked out for urban and rural populations (Tables 11 and 12). Table 13 provides the economic burden on patients with chronic and acute asthma from 1996 to 2016.

6. Estimates of saving (avoidable costs) for patients with asthma for the period 1996–2016: The present exercise clearly brings out the amount of savings (Fig. 3) in the treatment costs that accrue to patients and their families *only if* all health providers in the country adopt the treatment protocols given in the Guidelines. As this approach will markedly reduce the magnitude and severity of the disease, medical personnel/health providers at PHCs and district-level hospitals/private clinics would be in a position to pay more attention and provide better care to those suffering from other diseases. Thus, the quality of life would improve.

Discussion

Asthma is associated with considerable patient morbidity, a diminution of productivity and an increase in health care utilization. The prognosis of asthma remains good with as many as 60%–80% of patients being able to lead normal lives without much disruption. However, 10%–20% of patients continue to have severe attacks throughout their lives.

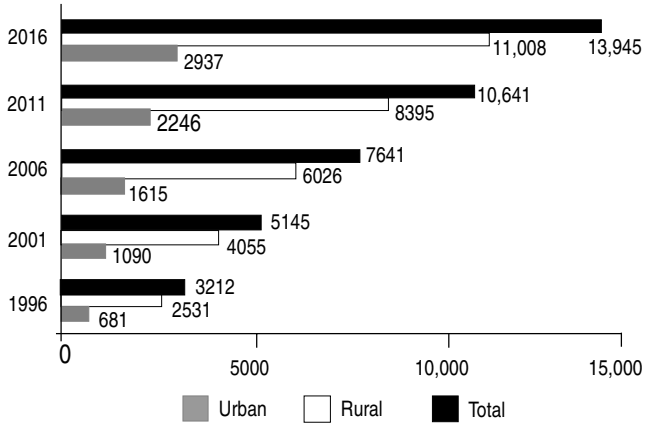

Fig. 2 Annual economic burden of asthma (Rs in crore)

Table 11. Cost of treatment of patients with chronic asthma

	Cases (in lakh)			Cost (Rs in crore)		
Year	Urban	Rural	Total	Urban	Rural	Total
1996	46.73	175.60	222.34	201.79	758.25	960.05
2001	51.97	195.43	247.39	324.27	1219.46	1543.74
2006	59.05	222.68	281.73	480.99	1813.74	2294.73
2011	66.62	251.58	318.20	669.44	2528.15	3197.60
2016	73.27	277.49	350.76	873.26	3307.09	4180.35

Table 12. Additional cost for patients with acute asthma

Year	Urban	Rural	Total
1996	44.34	122.73	167.07
2001	71.03	196.60	267.63
2006	103.20	285.65	388.84
2011	140.35	388.50	528.84
2016	178.43	494.09	672.52

Table 13. Economic burden of asthma (Rs in crore)

Year	Chronic	Acute	Total
1996	960.05	167.07	1127.12
2001	1543.74	267.63	1811.37
2006	2294.73	388.84	2683.57
2011	3197.60	528.84	3726.44
2016	4180.35	672.52	4852.86

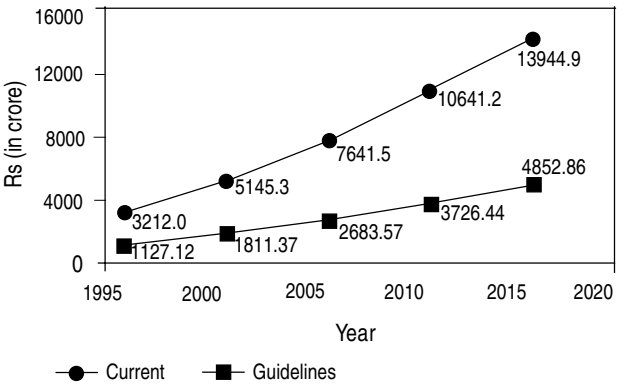

Fig. 3 Economic burden of asthma

International comparisons on prevalence rates suffer from lack of uniformity in definitions and the epidemiological survey tools used to monitor trends in the prevalence of asthma.[32] In the USA, between 1997 and 1999, the lifetime prevalence rate of asthma decreased by about 6% (96.6 per 1000 persons in 1997 to 90.9 per 1000 persons in 1999) but showed an increase of 25% by 2001 (113.4 per 1000 persons). Hence, in the USA the prevalence increased between 1980s and 1990s followed by a brief, but non-sustained, decline from 1997 to 1999. Recent studies in Mexico[36] suggest that control of asthma is achievable in large population groups with proper health care management and health insurance policies.

In a country such as India, unless we compile reliable databases on the prevalence of various important diseases on a regular basis at the national level, it is difficult to draw conclusions on whether there are any changes in the prevalence and, if so, in which direction. This paper suggests the requirements for undertaking such studies in the country. Irrespective of the cause, there is no doubt that asthma remains a major health care problem.[32] It is imperative that India make periodic efforts to monitor the situation and evolve new strategies to tackle and control asthma. It is suggested that asthma should be included as an important component of *all* further surveys of the NFHS whose primary emphasis is on children and women. The results of NFHS-2 on the prevalence of asthma adequately describe the national scene despite the reservations made in the NFHS-2 report.[16]

It was reported that in New Zealand 'high cost' asthma patients accounted for the use of 80% of the resources and their annual cost of treatment was US$ 2584 as compared to US$ 410 for others[37] and, accordingly, the cost of medication in India was estimated as US$ 30 per month.[38]

The mild and moderate forms of asthma can be managed successfully even by primary health care providers if they strictly follow the Guidelines in identifying cases at the early stages and prescribe medications (tablets) which are less expensive and ensure quick relief to the patient. Further, as the treatment is for a prolonged period, the compliance rates would also be high if the cost of treatment is affordable, resulting in a lesser number of patients getting into the acute phase.

In the costing according to the Guidelines approach, we have estimated average values of different medicines and avoided the expensive ones. However, as all asthmatics (mild and moderate cases) do not need medication all through the year, the present figures of cost of treatment would reflect on the higher side only.

Though the exact causes of the increasing prevalence rates of asthma across the globe are not fully understood, genetic and environmental factors are incriminated. There is evidence that environmental pollution has an indirect effect on the lung function levels of those exposed to it. Introduction of the Clean Air Act brought about substantial reduction in the pollution levels in Sheffield, UK and a better quality of life of asthmatics was recorded.[37] Hence, it is suggested that the Ministry of Health should make efforts for the inclusion of clean air as a fundamental right of citizens in the country.

The majority of patients with asthma live in rural areas. As poverty levels are higher in rural areas when compared to urban, it is imperative that primary health care providers should focus mainly on preventive rather than curative care of the disease and should focus on adopting the Guidelines for the treatment and management of asthma. The load on the present health staff in PHCs and their

subcentres can be easily reduced through redistribution of work load as mild and moderate cases, which form bulk of the caseload, by managing them at the local level itself by basic or peripheral health workers. The concept of 'shared care' between primary health workers and doctors in terms of handling mild and moderate cases by the former and acute cases by the latter, and following the principles of treatment provided in the Guidelines is the only approach that would reduce the economic burden of asthma in India, which can ill afford the present treatment protocols.[39] A practical approach at different levels of care has been worked out.[40]

Strengths and weaknesses of the present exercise

- More precise estimates of the magnitude of the problem and its financial implications can be calculated by developing appropriate databases in India through well-planned, properly conducted, large-scale surveys on the morbidity, health expenditure and opportunity costs borne by different members of the family in relation to utilization of health care facilities. Most of the existing studies are highly localized in terms of geographical location and case definition, and the type of population studied varies, which limits the purpose of the present exercise.
- One needs to be cautious in using prevalence figures based on large-scale field studies. Respondents may underreport diseases that carry stigma. Further, they may not be aware of the term used by the investigator to describe the condition. Errors can occur in situations where there is an overlap of symptoms between more than one disease. For example, chronic bronchitis may be reported as asthma or tuberculosis.
- Guidelines for the treatment of patients with chronic asthma are available to doctors. There are no such specific recommendations for the management of patients with acute asthma. This is possibly due to difficulties in controlling and/or recommending the management of care and its costing in hospitals/nursing homes, particularly in the private sector. Hence, we have used the current costs of treatment of acute cases, without any modifications for arriving at the economic burden of the disease according to the Guidelines.
- There is no information on the average number of times a patient with chronic asthma goes into the acute stage in a year and whether all acute cases get admitted to a hospital. Thus, we have assumed that the acute stage comes only once a year and all such patients get admitted to hospitals/nursing homes for treatment. Hence, all the costs arrived at reflect out-of-pocket expenditure incurred by patients for one year. It is possible that some of these chronic and/or acute cases are covered under different health insurance (public as well as private) schemes. No attempts were made to make the necessary corrections for such contingencies.

Recommendations

- Adoption of the Guidelines for case management by all health providers in the country would result in a drastic reduction of health care expenditure by families of asthma patients and would also help in reducing morbidity levels in a short span of time and improve their quality of life.
- Management protocols need to be prepared and disseminated for health providers at different levels. The existing Guidelines are mostly meant for doctors and similar guidelines should be developed for basic/primary health care workers. Primary health care workers should be empowered to detect and manage most of the mild or chronic cases of asthma. This would be the most cost-effective way to reduce the economic burden of the disease.
- Only acute cases and/or moderate and severe cases of asthma should be referred to or handled at the secondary care level of the present health set-up.
- Laws that would reduce smoking-related cases of asthma should be strictly enforced. Specific campaigns to create more awareness in this regard need to be undertaken on a periodic basis using all mass media approaches for this information, education and communication (IEC) activity. The focus should be more on the younger age groups so that in the long run new cases of asthma can be prevented.
- Pollution control through strict enforcement of laws passed by Parliament would go a long way in the overall improvement of morbidity rates including those for asthma. It would reduce allergy-related bronchial hyper-responsiveness.
- A public–private stakeholder participatory approach should be adopted to educate the general population on the preventive and curative aspects of asthma with simple and cheap drug regimens.
- A well-designed and need-based health database that would serve the needs of assessing the economic burden of disease in the country should be created. These studies need to be conducted on a periodic basis so that monitoring of disease profiles would help in making mid-course changes in various health strategies.

Acknowledgements

Our grateful thanks are due to Professor S.K. Jindal, Professor and Head, Department of Pulmonary Medicine, Postgraduate Institute of Medical Education and Research, Chandigarh; Dr H. Paramesh, Director and Paediatric Pulmonologist, Lakeside Medical Centre and Hospital, Bangalore; Dr Satish Kumar, Institute of Health Systems, Hyderabad; Dr G. Thippanna, Chest Physician, Hyderabad; and Dr Rohini V. Chowgule, Pulmonary Physician, Mumbai, for sharing their research publications and helpful suggestions.

We wish to thank the referees for their valuable suggestions and comments that helped in the presentation of this paper.

Out sincere thanks are also due to Ms Sujatha Rao, IAS, Government of India, for her encouragement and financial support to undertake this exercise.

References

1. Johnston SL, Holgate ST (eds). *Asthma: Critical debates.* London: Blackwell Science; 2003.
2. Burr ML, Butland BK, King S, Vaughan-Williams E. Changes in asthma prevalence: Two surveys 15 years apart. *Arch Dis Childhood* 1989;**64:**1452–6.
3. Shaw RA, Crane J, O'Donnell TV, Porteons LE, Coleman ED. Increasing asthma prevalence in a rural New Zealand adolescent population: 1975–89. *Arch Dis Childhood* 1990;**65:**1319–23.
4. Mitchell EA. Increasing prevalence of asthma in children. *NZ Med J* 1983;**96:**463–4.
5. Haahtela T, Lindholm H, Bjorksten F, Koskenvuo K, Laitinen LA. Prevalence of asthma in Finnish young men. *BMJ* 1990;**301:**266–8.
6. Ninan TK, Russell G. Respiratory symptoms and atopy in Aberdeen school children: Evidence from 2 surveys 25 years apart. *BMJ* 1992;**304:**873–5.
7. Hyndman SJ, Williams DRR, Merrill SL, Lipscombe JM, Palmer CR. Rates of admission to hospital for asthma. *BMJ* 1994;**308:** 1596–1600.
8. Smith JM, Harding LK, Cumming G. The changing prevalence of asthma in school children. *Clin Allergy* 1971;**1:**57–61.
9. Burney PGJ, Chinn S, Rona RJ. Has the prevalence of asthma increased in children? *BMJ* 1990;**300:**1306–10.
10. Gergen PJ, Mullally DI, Evans R III. National Survey of Prevalence of Asthma among children in United States: 1976 to 1980. *Pediatrics* 1988;**81:**1–7.
11. Yemaneberhan H, Bekele Z, Venn A, Lewis S, Parry E, Britton J. Prevalence of wheeze and asthma and relation to atopy in urban and rural Ethiopia. *Lancet* 1997;**350:**85–90.
12. Weitzman M, Gortmaker SL, Perrin JM. Recent trends in the prevalence and severity of childhood asthma. *JAMA* 1992;**268:** 2673–7.
13. Woolcock AJ, Peat JK. Evidence for increase in asthma worldwide. *Ciba Found Symp* 1997;**206:**122–39.
14. Gupta D, Aggarwal AN, Kumar R, Jindal SK. Prevalence of bronchial asthma and association with environmental tobacco smoke exposure in adolescent school children in Chandigarh, North India. *J Asthma* 2001;**38:**501–7.
15. Cypcar D, Stark J, Lemansko RF. The impact of respiratory infection on asthma. *Ped Clin North Amer* 1992;**39:**1259–73.
16. National Family Health Survey-2 (NFHS-2). India: 1998–99. International Institute of Population Studies; 2000.
17. Jindal SK, Gupta D, Aggarwal AN, Jindal RC, Singh V. Study of the prevalence of asthma in adults in north India using a Standardized Field Questionnaire. *J Asthma* 2000;**37:**345–51.
18. Chowgule RV, Shetye VM, Parmar JR, Bhosale AM, Khandagale MR, Phalnitkar SV, *et al.* Prevalence of respiratory symptoms, bronchial hyperreactivity, and asthma in a megacity—results of the European community respiratory health survey in Mumbai [Bombay]. *Am J Respir Crit Care Med* 1998;**158:**547–54.
19. Paramesh H. Epidemiology of asthma in India. *Indian J Ped* 2002;**69:**309–12.
20. Paramesh H. Normal peak expiratory flow rate in urban and rural children. *Indian J Ped* 2003;**70:**375–8.
21. Behera D, Jindal SK. Respiratory symptoms in Indian women using domestic cooking fuels. *Chest* 1991;**100:**385–8.
22. Mitchell EA. Increasing prevalence of asthma in children. *NZ Med J* 1983;**96:**463–4.
23. Morrison SJ. The prevalence of asthma and wheezing in children. *Br J Dis Chest* 1976;**70:**73–7.
24. Mitchell EA. Increasing prevalence of asthma in children. *NZ Med J* 1983;**96:**463–4.
25. Burney PGJ, Britton JR, Chinn S, *et al*. Descriptive epidemiology of bronchial reactivity in an adult population: Results from a community study. *Thorax* 1987;**42:**38–44.
26. Morrison Smith J. The prevalence of asthma and wheezing in children. *Br J Dis Chest* 1976;**70:**73–7.
27. Vesterinen E, Kaprio J, Koskenvuo M. Prospective study of asthma in relation to smoking habits among 14729 adults. *Thorax* 1988;**43:**534–9.
28. Evans D, Levinson MJ, Feldman CH, *et al.* The impact of passive smoking and emergency room visits of urban children with asthma. *Am Rev Respir Dis* 1987;**135:**567–72.
29. Burney PGJ. Epidemiology. In: Clark TJH, Godfrey S, Lee TH, Thomsom NC (eds). *Asthma.* London: Panther Publishers Private Limited; 2002:197–223.
30. Paramesh H. Air pollution and child health. *Academy Today*, January 2001.
31. Registrar General of India. Population Projections for 1996 to 2016.
32. Matricardi PM, Bonini S. Why is the incidence of asthma increasing? In: Johnston SL, Holgate ST (eds). *Asthma: Critical debates.* London: Blackwell Science; 2002:3–17.
33. Institute of Health Systems. *Health effects analysis* (unpublished data). Hyderabad, 2001.
34. Shafazand S, Colice G. Asthma: The epidemic has ended, or has it? *Chest* 2004;**125:**1969–70.
35. *Monthly Index of Medical Specialities (MIMS)*—India, New Delhi: AE Morgan Publications (India) Private Limited; 2004.
36. Vargas MH, Diaz-Mejia GS, Furuya MEY, Salas J, Lugo A. Trends of asthma in Mexico: An 11-year analysis in a nationwide institution. *Chest* 2004;**125:**1993–7.
37. Holt S. In: The burden of asthma. Wellington: Asthma & Respiratory Foundation of New Zealand; 2001.
38. Singh RB. Asthma in India: Applying science to reality. *Clin Exptl Allergy* 2004;**34:**686.
39. Howard P. The changing face of chronic bronchitis with airways obstruction. *BMJ* 1974;**2:**89.
40. Jindal SK, Gupta D, Aggarwal AG. Guidelines for management of chronic obstructive pulmonary disease (COPD) in India: A guide for physicians. *Indian J Chest Dis Allied Sci* 2004;**46:**137–53.

Appendix 1

Guidelines for the management of asthma

- Minimize exposure to known environmental triggers, particularly tobacco smoke.
- Approach treatment in a step-wise manner, commencing at a level appropriate to initial severity of symptoms.
- Ensure compliance, check whether the inhaler used is appropriate and is being used correctly before stepping up treatment.
- Ensure that patients and their families understand and are actively involved in the management of symptoms.
- Aims

Steps 1–3

—Minimize frequency of exacerbations and chronic or nocturnal symptoms
—Minimize the need for bronchodilators
—Remove limits on physical activity (or childhood activities)
—Maintain PEFR at 80%+ of predicted best, circadian variation <20%
—Minimize the side-effects of medicines

Steps 4–5

—Least possible symptoms are limits on activity
—Least possible need for bronchodilators for relief of symptoms
—Best possible PEFR with least circadian variation
—Least adverse effects from medicine

Table A1.1 Treatment of asthma in children under 5 years of age

Step	Symptom relief	Maintenance	Additional/alternative therapies
1	Occasional bronchodilator inhaled short-acting beta-2 agonist as required (not more than once daily)		Mild cases may respond to oral beta-2 agonists, but they are less effective and have more side-effects than when inhaled. If inhaled beta-2 agonists are needed more than once daily, proceed to Step 2.
2a	Inhaled short-acting beta-2 agonist as required	Regular inhaled dose of cromoglycate such as 20 mg of cromoglycate powder 3–4 times daily or 10 mg 3 times daily via metered dose inhaler (MDI) and large-volume spacer. Assess after 4–6 weeks.	To gain rapid control, consider a 5-day course of soluble prednisolone or doubling the dose of inhaled steroids for one month. Alternatively, give a short course of prednisolone or add other treatments before increasing the dose of inhaled steroids for prolonged periods.
2b	Inhaled short-acting beta-2 agonist as required	Regular low-dose inhaled steroids (up to 400 μg) beclomethasone or budesonide daily or up to 200 μg fluticasone daily.	
3	Inhaled short-acting beta-2 agonist as required	Regular high-dose inhaled steroids (up to 800 μg) beclomethasone or budesonide or up to 500 μg fluticasone daily via large-volume spacer device.	Additionally, consider adding regular long-acting beta-2 agonist twice daily before proceeding to Step 4. Alternatively, slow-release xanthine may be added, particularly for nocturnal symptoms, but side-effects may be troublesome.
4	Inhaled short-acting beta-2 agonist as required	Regular high-dose inhaled steroids (up to 2000 μg) beclomethasone or budesonide or up to 1000 μg fluticasone daily via large-volume spacer device.	Additionally, consider adding regular long-acting beta-2 agonist twice daily or slow-release xanthine as in Step 3, or nebulized beta-2 agonists.

Table A1.2 Treatment of asthma in children over 5 years of age and adults

Step	Symptom relief	Maintenance	Additional/alternative therapies
1	Occasional bronchodilators inhaled short-acting beta-2 agonist as required (not more than twice daily)		If inhaled beta-2 agnoists are needed more than twice daily, move to Step 2.
2	Inhaled short-acting beta-2 agonist as required	Regular low-dose inhaled steroids, e.g. 100–400 μg of beclomethasone or budesonide, or 50–200 μg fluticasone; all twice daily.	Alternatively, regular inhaled cromoglycate or nedocromil may be used. However, if control is not achieved, inhaled steroids should be started.
3	Inhaled short-acting beta-2 agonist as required	Regular high-dose inhaled steroids, e.g. 800–2000 μg beclomethasone or budesonide, or 400–1000 μg fluticasone daily via large-volume spacer, or regular low-dose inhaled steroids in addition to long-acting beta-2 agonists: 100–400 μg of beclomethasone or budesonide twice daily or 50–200 μg fluticasone twice daily plus 50 μg salmeterol twice daily.	Additionally, in patients experiencing problems with high-dose steroids or with persisting nocturnal symptoms, slow-release theophylline may be added to Step 2 doses of inhaled steroids. Regular inhaled cromoglycate or nedocromil may also be tried.
4	Inhaled short-acting beta-2 agonist as required	Regular high-dose inhaled steroids, e.g. 800–2000 μg beclomethasone or budesonide, or 400–1000 μg fluticasone daily via large-volume spacer device.	Additionally, one or more of the following long-acting bronchodialators may be sequentially added: —long-acting inhaled beta-2 agonist —sustained-release theophylline —inhaled ipratropium or oxitropium —long-acting beta-2 agonist tablets —high-dose inhaled bronchodialators —cromoglycate or nedocromil
5	Inhaled short-acting beta-2 agonist as required	High-dose inhaled steroids, e.g. 800–2000 μg beclomethasone or budesonide or 400–1000 μg fluticasone daily via large-volume spacer.	Additionally, give one or more of the long-acting bronchodilators as in Step 4 plus regular prednisolone tablets in a single daily dose.

- Use short-course steroid tablets to control acute exacerbations at any stage of treatment (prednisolone—children: 1–2 mg/kg/day for 1–5 days; adults: 30–60 mg/day continued 2 days after recovery).
- Review therapy and condition every 3–6 months.
- Reduce treatment, if appropriate, step-wise and gradually.
- Withdraw anti-inflammatory treatment used for seasonal symptoms at the end of that season.
- See prescribing notes for the management of children under 2 years of age.

Appendix 2

Suggested protocol for the management of asthma and COPD

Rural set-up: Public health

Health visitor

- The health visitor will be made aware of the hazards of smoking through IEC materials.
- The health visitor during regular visits to the village will identify smokers with breathlessness and refer them to the nearest medical facility (PHC/CHC).
- The health visitor will also ensure compliance of patients under treatment.
- The health visitor will identify patients who become breathless while on treatment and refer them to the nearest PHC/CHC.

Medical officer

- Doctors in PHCs/CHCs will use the Guidelines for management while prescribing drugs to patients with asthma.
- Doctors will also advise smoking cessation.

Health care facilities

- Nebulization facilities should be made available in all PHCs and CHCs.

Private health care

- Private health care providers to adopt the Guidelines for management of all grades of COPD and asthma.
- They should be actively involved in smoking cessation by patients.
- Nebulization facility to be available for patients with acute asthma.

Urban set-up: Public health

Medical officer

- Doctors in all clinics will use the Guidelines for management while prescribing medication to patients.
- Medical officers will also advise smoking cessation to all patients.

Health care facilities

- Nebulization facilities to be available in all clinics.

Private health care

- Private health providers to adopt the Guidelines for management for all grades of COPD and asthma.
- Private practitioners should be actively involved in smoking cessation by patients.
- Nebulization facility to be available for patients with acute asthma.

Appendix 3

Table A3.1 Causal analysis of COPD and asthma

Direct (medical)	Indirect/distant (non-medical)
COPD	
Delayed treatment seeking	• Tobacco smoking • Sex—more common in females • Atmospheric pollution • Lack of awareness of the symptoms of COPD • Cooking fuel
Asthma	
Viral upper respiratory tract infections	• Family history • More common in rural than urban areas • Sex—slightly higher prevalence in males than females • Poor housing conditions—poor ventilation, crowded living space • Season: more common in winter • Type of cooking fuel used—firewood/chullah, stove • Smoking • Air pollution • Occupational asthma—industry-related • Lack of awareness of the symptoms or causes of asthma

Table A3.3 Requirements for medical interventions for COPD and asthma

Skills/manpower needs	Equipment	Drugs	Tests
Public health worker	Peak flow gauge	Salbutamol/ terbutaline/ theophylline (tablets)	Peak flow gauge
Medical officer	Spirometer	• Ipratropium • Beclomethasone • Aminophylline • Prednisolone	• Spirometry • X-ray of the chest

Table A3.2 Causes of COPD and asthma

	Direct	Indirect	Distant
Asthma			
Main causes	Genetic	Smoking worsens it	—
Interaction with other causes	—	Pollution	—
References (from the paper on Economic burden of asthma)	17	47	
COPD			
Main causes	Smoking	Air pollution (atmospheric and due to cooking fuels)	Winter and smog
Interaction with other causes	—	Coronary heart disease	—
References (from the paper on Economic burden of COPD)	5,19	18	32

Table A3.4 Interventions for the management of COPD and asthma

Activity	Community	Community-based worker	Subcentre	PHC	CHC	District hospital
Smoking	Yes	Yes	Yes	Yes	Yes	Yes
Air pollution	Yes	Yes	Yes	Yes	Yes	Yes
Identification of symptomatics		Training	Testing	Testing	Testing	Testing
Diagnosis		Peak flow gauge (PFG)	PFG	PFG	PFG	Spirometry
Treatment		Oral and inhalation devices	Oral and inhalation devices	Oral and inhalation devices and nebulization and oxygen	Oral and inhalation devices and nebulization and oxygen	Nebulization and oxygen

Table A3.5 Interventions at different levels for COPD and asthma

Level	Intervention	Skills/manpower required	Equipment required	Tests required	Drugs required
Subcentre/solo practitioner	Recognition and peak flow gauge (PFG)	Training ANM/ practitioners	PFG	PFG	Inhalers
PHC/a hospital with less than 10 beds	Inhalation device/ nebulization	ANM/nurse/doctor	PFG, nebulizer	PFG	Inhalers, aerosol solutions, oxygen
CHC/a 30–50-bed hospital	Inhalation device/ nebulization	ANM/nurse/doctor	PFG, nebulizer	PFG	Inhalers, aerosol solutions, oxygen
District-level hospital/ a hospital with more than 50 beds	Inhalation device/ nebulization	ANM/nurse/doctor	PFG, nebulizer, spirometer	PFG, spirometry	Inhalers, aerosol solutions, oxygen

ANM: auxiliary nurse–midwife

Table A3.6 Interventions for the treatment of COPD and asthma

		Non-medical interventions/prevention		
Condition	Medical interventions	Exercise	Nutrition	Others
Asthma				
Mild	Beta-2 agonists (tablets/inhalers)	—	—	Keeping house clean—regular dusting
Moderate	Beta-2 agonists (tablets/inhalers); theophylline (oral); steroid inhalers	—	—	
Severe	Hospitalization; nebulization; beta-2 agonists and/or systemic steroids (parenteral); oxygen inhalation	—	—	
References (from the paper on Economic burden of asthma)	51			17, 51
COPD				
Mild	Beta-2 agonists (tablets/inhalers); theophylline; ipratropium bromide inhalation	—	—	Smoking cessation
Moderate	Beta-2 agonists (tablets/inhalers); theophylline; ipratropium bromide inhalation; steroid inhalers	—	—	
Severe	Hospitalization + nebulization; beta-2 agonists; ipratropium bromide; theophylline (parenteral); antibiotics (oral/parenteral); oxygen inhalation	—	—	
Reference (from the paper on Economic burden of COPD)	27			27

Table A3.7 Standard treatment protocols for asthma and COPD

	Personnel	Tests	Drugs	Inpatient stay
Asthma				
Type I (mild)	Peripheral health workers	Simple peak flow measurement	Inhalers (seasonal)	—
Type II (moderate)	Peripheral health workers	Simple peak flow measurement; spirometry	Inhalers (seasonal/life-long)	—
Type III (severe)	Nurses, doctors		Inhalations and parenteral steroids; theophylline; oxygen	Yes
COPD				
Type I (mild)	Peripheral health workers	Simple peak flow measurement; spirometry	Inhalers (life-long)	—
Type II (moderate)	Peripheral health workers	Simple peak flow measurement; spirometry	Inhalers (life-long)	—
Type III (severe)	Nurses, doctors	X-ray; blood gases; ECG	Nebulization; beta-2 agonists; ipratropium; theophylline (parenteral); antibiotics (oral/parenteral); oxygen inhalation; ventilatory management, if necessary	Yes

Needs for personnel

Training for peripheral health workers should be done
—to identify cases of asthma and chronic bronchitis
—to use simple peak flow gauge (PFG)
—to interpret the results of PFG
—to use inhalation devices including nebulization.

Economic burden of chronic obstructive pulmonary disease

K.J.R. MURTHY, J.G. SASTRY

Chronic obstructive pulmonary disease (COPD) refers to a group of disorders characterized by chronic airflow obstruction/limitation. The airway obstruction is persistent and largely irreversible. It includes two distinct patho-physiological processes—chronic bronchitis and emphysema. It is associated with an abnormal inflammatory response of the lungs to noxious particles or gases, especially tobacco smoke and air pollution—both indoor and outdoor.

Chronic bronchitis manifests as chronic cough and sputum production for more than 3 months in a year for at least 2 consecutive years. Exacerbations are frequent, more so during winter, resulting in progressive loss of the functional capacity of the lungs. It is of great public health importance, because it is largely preventable if identified in the early stages and treated properly. In the initial stages, no abnormal signs are seen. Most often, COPD is interpreted as smoker's cough with little or no breathlessness. If not detected and attended to with proper medication, deterioration slowly sets in as it progresses into the moderate form with breathlessness and/or wheezing on moderate exertion. Most importantly, the patient's inability to exert results in reduced ability to work and loss of wages. The patient not only suffers from physical discomfort but also undergoes medical treatment resulting in financial and psychological distress. The disease is gradually progressive with each episode of exacerbation leading to further respiratory disability and, ultimately, death.

COPD is currently the fourth leading cause of death worldwide.[1] The Global Burden of Disease Study estimated that in 1990, the worldwide prevalence of COPD was 9.34 per 1000 men and 7.33 per 1000 women. As these estimates are based on the total population, and the problem is seen mostly among older adults, the true age-specific prevalence will be much higher, especially in countries where cigarette smoking is common.

Mahavir Hospital and Research Centre
10/1/1, Mahavir Marg, A.C. Guards
Hyderabad 500004
e-mail: kollurijrm@hotmail.com; gjandhyala@hotmail.com

This paper attempts to quantify the magnitude of COPD and its economic implications for society.

Need to study the burden of disease

The definition of health has undergone major changes during the past 50 years and concurrently all countries have realized the need for developing their nations along a pre-planned path that includes provision of basic health needs. Such a planning operation requires prioritization of needs for monetary allocation. Economists who most often manage decision-making bodies such as planning commissions would look for the burden of disease (BOD) in terms of the caseload and quantum of economic burden borne by the society. These estimates of BOD help to plan priorities and allocate funds to suit strategies to control and eliminate the disease to the extent possible. The larger the economic burden, the more will be the role of interventions by health planners/managers of the State.

Review of the literature

A review of the scientific literature on COPD from India and other countries reveals the following:

- The spectrum of clinical manifestations of COPD is wide. There are great variations in the reported morbidity, which could partly be due to differences in the definition of a 'case'.[2] The data on mortality also underestimate COPD as a cause of death because the disease is more likely to be cited as a contributory rather than an underlying cause of death, or may not be cited at all.[3] Depending on the severity of the disease, the 5-year mortality rate for patients with COPD varies from 40% to 70%. The three major causes of death have been identified as COPD itself, lung cancer and cardiovascular disease.[4]
- The studies were confined to limited areas and do not represent the general population of that State or region. Table 1 presents the variation in prevalence rates reported by different researchers in India during 1964–1995.[5]

Table 1. Prevalence of COPD and its association with smoking as shown by the various population studies from India

Author/s (year)	Population	Prevalence of COPD (%)		M:F ratio	Smoker:non-smoker ratio
		Men	Women		
Wig *et al.* (1964)[6]	Delhi (rural)	3.36	2.54	1.3	2.0
Sikand *et al.* (1966)[7]	Delhi	7.0	4.3	1.6	2.5
Viswanathan (1966)[8]	Patna	2.12	1.33	1.6	
Bhattacharya *et al.* (1975)[9]	UP (rural)	6.67	4.48	1.6	
Radha *et al.* (1977)[10]	New Delhi	8.1	4.6	1.8	1.8
Thiruvengadam *et al.* (1977)[11]	Madras (now Chennai)	1.9	1.2	1.6	10.2
Viswanathan and Singh (1977)[12]	Delhi (rural)	4.7	3.5	1.3	9.6
	Delhi (urban)	8.0	4.3	1.9	4.0
Charan (1977)[13]	Punjab (rural)	2.28	1.63	1.4	
Malik (1986)[14]	North India (rural)	9.4	4.9	1.9	5.5
	North India (urban)	3.7	1.6	2.3	7.0
Jindal (1993)[15]	North India (rural)	6.2	3.9	1.6	
	North India (urban)	4.2	1.6	2.6	9.6
Ray *et al.* (1995)[16]	South India	4.08	2.55	1.6	1.6

- The prevalence of COPD is confined to adults 30 years of age and above.
- Table 2 shows that the prevalence of COPD did not change much over a period of time.
- The prevalence rates of COPD in males varied from 2.12% to 9.4% in studies conducted in north India and from 1.4% to 4.08% in south India. The respective ranges for females were 1.33%–4.9% in north India and 2.55%–2.7% in south India. The median values of these prevalence rates are 5% for males and 2.7% for females. Thus, COPD is more common among males than females. The male to female ratio varied from 1.32:1 to 2.6:1 with median ratio of 1.6:1.[5]
- A strong association exists between tobacco smoking and the occurrence of COPD. The reported smoker: non-smoker prevalence ratio varied from 1.6 to 10.2. Thus, smoking has been identified as a high-risk factor for COPD.[5] Surveys in India have revealed that 29.4% of males and 2.5% of females are current smokers.[17] However, in those 30 years of age and above, the prevalence of smoking in India is 40.9% for males and 3.9% for females. The difference in the prevalence of COPD among males and females could be due to the differences in their levels and type of smoking. Further, it is seen that the magnitude of smoking increases with age (Table 3).
- Indoor air pollution due to traditional domestic fuels was considered an important factor affecting the lung function of females in rural areas in prevalence studies of COPD.[18] However, no consistent evidence was observed in this direction.
- The occurrence of severe bronchitis among non-smokers was mainly due to their exposure to tobacco smoke either at home or at the workplace.[19] The prevalence of COPD was much higher in heavy smokers than among those who smoked a lesser number of cigarettes. The odds ratio was 2.4 for the total population—4.7 for females and 1.5 for males.[2] Among British male doctors, deaths due to chronic bronchitis were found to increase directly with the number of cigarettes smoked per day (Table 4). A decrease in mortality was seen among ex-smokers. However, the mortality was much higher than

Table 2. Prevalence of COPD—changes over the years (%)

Period	Males	Females
Up to 1970	4.2	2.7
1971–1990	5.7	2.6
After 1990	4.9	2.7
Average	5.0	2.7

Table 3. Prevalence of smoking among different age groups by sex (%)

Age group (years)	Males	Females
15–19	4.4	0.2
20–24	13.7	0.6
25–29	25.1	1.1
30–39	37.6	2.2
40–49	45.0	4.0
50–59	45.3	5.7
60+	38.2	5.3
Average	29.4	2.5

Table 4. Mortality due to chronic bronchitis/100,000 British male doctors[20]

No. of cigarettes/day	Mortality
Nil	3
1–14	51
15–24	78
≥25	114
Ex-smokers	44

Source: Doll and Peto (1976)

that seen among non-smokers.[20] Similar observations were also made in large-scale studies conducted in the USA and Canada.

- An association was seen between the method of smoking and the occurrence of chronic bronchitis. Pipe and cigar smokers were found to have a lower prevalence of the disease and less impairment of lung function even though the inhalation of cigar smoke is as irritative as that of cigarette smoke.[21–23] Introduction of the Clean Air Act 1981 has drastically reduced the morbidity due to lung dysfunction, if not COPD. In areas with high levels of industrial pollution, the effect of enforcement of anti-pollution laws was not clearly seen because of the increase in the cigarette smoking habits of the population. In other words, the beneficial effects of control of atmospheric pollution on bronchial health are difficult to demonstrate at a macro level. However, its indirect impact on the prevalence of COPD can be expected.
- A majority of cases with chronic COPD (57.4%) were found to suffer from a mild form and only 16% had severe COPD. The remaining 26.6% formed the moderate category. In fact, 81.4% of cases from the mild category (i.e. 46.7% of the total number of cases) were not aware that they had the disease and hence did not seek any medical advice.[2] The remaining 18.6% of these mild cases sought treatment as outpatients.
- A large-scale study in Hyderabad city and its surrounding municipalities, covering a population of more than 54 lakh and 28 hospitals/health posts, was done in 2001 to collect cause-specific morbidity data. The rates of hospital admissions of cases with COPD showed an age differential. While the rate was 47.84/100,000 persons at the community level, it was 57.28 for those 18–64 years of age and 546.17 for those above 65 years of age.[24] Similar differences among the various age groups have been reported earlier.[12]
- The unit values of hospital admission for COPD were US$ 122.23 towards medical costs and US$ 14.30 for opportunity loss; outpatient visits cost the patients US$ 8.26 for medicines and another US$ 1.43 because of opportunity losses. These costs (reported for the year 2001) were used to arrive at the current cost of COPD.[24]
- It was noted that, on an average, a person with COPD spent Rs 11,952 per year in 1992 and the same treatment cost Rs 23,300 in 1999.[25] This increment in the cost of treatment was calculated on a pro rata basis for the period 1996–2016. In the present exercise, the same rate of change was applied for all other costs.

Data used for the present exercise

The number of cases with chronic and severe COPD was estimated by using the projected population figures for the period 1996–2016.[26] The expected changes in the mortality figures in India were considered in this exercise.

Table 5. Treatment cost of a patient with COPD per year (in Rs)—current level

Year	Chronic case	Hospitalization cost (acute case)	Total cost
1991	10,331	2,390	12,721
1996	18,436	4,167	22,603
2001	26,541	6,144	32,685
2006	34,646	8,018	42,664
2011	42,751	9,894	52,645
2016	50,856	11,774	62,630

Only the population of those 30 years of age and above was considered, and a constant percentage (26.82%) for the urban population was operated till 2016.

To calculate the economic cost of COPD, the following factors were taken into account:

- A conversion factor of US$ 1 = Rs 45 was applied to costs that were provided in dollars. Table 5 gives information on the economic costs (in Rs) in one year for each patient with COPD.[12,25]
- Treatment guidelines for COPD were drawn up (Appendix 1) and the cost of treatment (per year) was arrived at to evaluate the potential savings that would accrue to the patient.[27] The cost of treatment (per year) adopted to arrive at the economic burden of COPD when the treatment was according to the guidelines is provided in Table 6.

The costs of medicines adopted from the guidelines' approach are based on the least expensive and effective drugs available in the market. The cost of treatment of a patient with acute COPD per year has two components—cost of regular medication and cost of hospitalization. The cost of regular medication was taken as that for a mild case and was applied to all acute cases of COPD, while the cost of hospitalization was the same as that of the current levels. As an example, the cost of Rs 4444 for the year 1996 is the sum of the cost of treatment (Rs 277) and that of hospitalization (Rs 4167). All these rates of prevalence (given per 100,000 population) and costs of treatment of COPD have been applied on the estimated number of cases among the projected population figures for the period 1996–2016.[26]

Table 6. Treatment cost of a patient with COPD per year (in Rs)—according to Guidelines*

Year	Mild	Moderate and severe	Acute case
1996	277	913	4,444
2001	400	1320	6,544
2006	522	1723	8,540
2011	644	2126	10,538
2016	766	2529	12,540

*Guidelines given in the *Monthly Index of Medical Specialities*

Table 7. Estimated number of patients with chronic COPD (in lakh)

	Males		Females		Total	
Year	Urban	Rural	Urban	Rural	Urban	Rural
1996	23.4	63.8	11.2	31.6	34.6	95.4
2001	26.6	72.6	13.2	37.0	39.8	109.6
2006	30.2	82.3	15.1	42.6	45.3	124.9
2011	34.3	93.5	17.2	48.3	51.5	141.9
2016	39.4	107.6	19.7	55.4	59.1	163.0

Results

The estimated number of patients with chronic and acute COPD and their distribution by sex and residence are given in Tables 7 and 8.

Health providers/planners need to get ready to face a caseload of COPD of about 222.16 lakh in 2016—a majority of this would be from rural areas where the poverty levels are high.

Table 8. Estimated number of patients with acute COPD (in lakh) by sex and residence

	Males		Females		Total no. of acute cases		
Year	Urban	Rural	Urban	Rural	Urban	Rural	Total
1996	0.69	1.89	0.62	1.75	1.31	3.64	4.95
2001	0.78	2.13	0.72	2.02	1.50	4.15	5.65
2006	0.89	2.44	0.83	2.35	1.73	4.78	6.51
2011	1.03	2.82	0.97	2.72	2.00	5.54	7.54
2016	1.20	3.28	1.13	3.18	2.34	6.47	8.81

Table 9 provides information on the estimated caseload according to the severity of COPD. In addition, there would be patients with acute COPD who need hospitalization and expert care.

The current annual cost of treatment was arrived at and the results are presented in Table 10.

It is estimated that a staggering amount of more than Rs 48,000 crore will be spent by patients and their families on the treatment of COPD alone in the year 2016. The total amount of money spent by these patients can be drastically reduced by adopting a number of strategies such as controlling various contributory factors, e.g. tobacco smoking, providing hospital care at cheaper rates and marketing the medicines at lower costs. These strategies are considered to be difficult to implement. However, if all patients with COPD are treated according to the suggested guidelines, the estimated cost of treating COPD can be reduced considerably (Table 11).

Table 9. Estimated number of patients with COPD by severity of the disease (in lakh)

	Severity of COPD			
Year	Mild	Moderate	Severe	Total
1996	75.67	33.28	21.06	130.01
2001	86.92	38.23	24.19	149.35
2006	99.04	43.57	27.57	170.18
2011	112.52	49.49	31.32	193.33
2016	129.30	56.87	35.99	222.16

Table 10. Total cost of treatment for COPD (Rs in crore)—current level

Year	Urban	Rural	Total
1996	2,729.1	7,519.4	10,248.5
2001	4,512.5	12,435.5	16,948.0
2006	6,711.5	18,497.7	25,209.1
2011	9,408.1	25,929.6	35,337.7
2016	12,860.9	35,445.2	48,306.1

The present exercise provides an opportunity to compare the current cost of COPD (Table 10) with that of the guidelines (Table 11). The difference between these two estimates of costs provides valuable information on the notional savings that accrue to patients and their families; a large number of them come from rural areas and are poor (Table 12).

Table 11. Total cost of treatment for COPD according to the guidelines* (Rs in crore)

Year	Urban	Rural	Total
1996	254.73	702.69	957.42
2001	525.41	1449.30	1974.70
2006	782.68	2159.22	2941.90
2016	1507.23	4158.10	5665.33

*Guidelines given in the *Monthly Index of Medical Specialities*

Discussion

The present exercise cannot be used to precisely estimate the economic burden of COPD in India because of inadequate information. Unlike asthma, the prevalence of COPD has not shown any change during the past 30 years. Further, there is a dearth of data on the frequency of medical consultation, hospitalization, and opportunity losses to the patient and attendants. Mortality rates among patients with COPD are not available for India. In the present exercise, the costing is mostly based on the large-scale investigation carried out in and around Hyderabad. In the absence of such data on costing from other parts of the country, the results were extrapolated for the entire country.

Table 12. Total cost of treatment of a patient with COPD (Rs in crore)

Year	Guidelines	Current cost
1996	957.42	10,248.5
2001	1974.70	16,948.0
2006	2941.90	25,209.1
2011	4135.17	35,337.7
2016	5665.33	48,306.1

The differences between the current costs and those according to guidelines were enormous. We have considered the average cost of the medicines (guidelines) that are mostly prescribed. Only a common pro rata change was applied to all costs in the present study. The cost of medicines was calculated for a calendar year, and all patients with acute COPD were assumed/expected to be admitted in a hospital once a year.

Exacerbations with increased cough, sputum and, often, wheezing are frequent in patients with chronic bronchitis and emphysema, though progressive deterioration in respiratory function occurs with very little cough or sputum and no exacerbations in some smokers. However, each infective exacerbation produces further lung damage and adds to the permanent impairment of respiratory function.

A number of studies have shown a correlation between the deterioration of respiratory function and frequency of exacerbations.[28] However, correlation does not necessarily imply causation. A number of confounding factors may be operating on both these variables, such as hereditary factors or living conditions congenial for acquiring different infections, as well as smoking habits. The prevalence of chronic bronchitis among the rural population was 57 per 1000 population. The type of smoking influenced the magnitude of prevalence.[9] The prevalence of chronic bronchitis in *hookah* smokers was 85/1000 population, in *beedi* smokers 31/1000, and in *chillum* smokers 17.5/1000. It was seen that 13.5% of *beedi* smokers had chronic bronchitis.[29]

The strong association between cigarette smoking and COPD points out the need to bring about drastic changes in the smoking habits of the population through legislation and increase in the cost of cigarettes. However, it is known that such a strategy would take 10–15 years to bring about a reduction in the prevalence rate; the higher prevalence rates seen in males as compared to females could be due to the higher levels of smoking among them. The results of the National Family Health Survey (NFHS)-2 show that around 4.5% of males in the age group of 15–19 years and about 14% of those 20–24 years of age are current smokers.[17] However, most field-based studies have observed that the problem of COPD exists only in adults 30 years of age and above. Does this mean that it takes about 10–15 years for COPD to develop after the initiation of cigarette smoking? In fact, the data presented in the NFHS-2 on current smokers in India from different age groups and by sex, show the prevalence of smoking to be 40.9% among males and 3.91% among females 30 years of age and above (23.06% for both the sexes).[17]

Population studies have found that chronic bronchitis is almost confined to smokers. In males 55–64 years of age, the prevalence of chronic bronchitis was found to be 17.6% in heavy smokers, 13.9% in light smokers, 4.4% among ex-smokers and nil among non-smokers.[22,30] The effect of cigarette smoking may begin at an early age, as indicated by increased respiratory illness and diminished pulmonary function in children passively exposed to cigarette smoke at home. After adjusting for age and frequency of smoking, it was found that women who smoke heavily were more susceptible than men to acquire smoking-related COPD (odds ratio [OR]=4.7).[14] Significant regional differences in the prevalence of COPD were seen in Greece.[2] Reports from northern India, which incidentally has cooler climatic conditions accompanied by smog during winter, also suggest that the prevalence may be higher than that in south India. The range of prevalence rates reported in males from north India (2.12%–9.4%) is generally higher than that reported from south India (1.4%–4.08%).[31] At the all-India level, the male to female smoking ratio was seen to be 11.76:1, which strongly suggests that males should be the focus group for information, education and communication (IEC) activities. While the rural/urban coverage among males and females in the survey is comparable (males: 2.48 and females: 2.58), these ratios among smokers are 3.78 for males and 8.89 for females. This observation indicates that females cannot be ignored (more so in rural areas) for IEC activities. The ratio of males to females among current smokers is 25.44:1 in urban areas and 10.81:1 in rural areas.[17] A review of the literature shows the prevalence of COPD to be around 5% among males and 2.7% among females, with a male to female ratio of 1.85:1.[5] A ban on cigarette smoking in public places is a step in the right direction. Even if attempts are made to control the younger generation from getting into this habit, its impact will not be seen for another 10–15 years.

The impact of air pollution on the occurrence of chronic bronchitis was studied in workers from a machine tools factory and woollen hosiery mills. It was found that chronic bronchitis was mainly related to smoking, with air pollution playing a minor role.[32]

The present study highlights the need to focus on the health infrastructure so that the magnitude of COPD cases in rural areas can be handled.[33] It is known that methodological differences are also responsible for differences in the prevalence rates. The assessment of severity is based on the degree of the spirometric abnormality. Based on the results of spirometry, COPD can be categorized into five stages: at risk, mild, moderate, severe and very severe (Table 13).[34]

It is a common observation that such objective measurements to categorize a patient by the severity of the disease are not possible under field conditions, particularly in rural and tribal areas of India. Further, adoption of such techniques needs highly trained manpower to collect the information, while the operational costs of the survey increase enormously. Hence, there is a need to adopt simple criteria to define a case, which brings in the factor of trade-off between the quality of the data and cost of data collection.

More than 80% of cases with mild COPD among the Greek population were found to be ignorant of their problem and hence did not seek any medical assistance/advice.[2] It is most likely that a similar situation prevails in India. If we could only have peripheral health workers constantly

Table 13. Global initiative for chronic obstructive lung disease (GOLD): Classification of severity[34]

Stage	Characteristics	Symptoms
0: At risk	• Normal spirometry • Chronic symptoms (cough, sputum production)	Normal
1: Mild	• FEV_1/FVC <70% • FEV_1 >80% predicted • With or without chronic symptoms (cough, sputum production)	Patient not aware of the problem
2: Moderate	• FEV_1/FVC <70% • 50% ≤FEV_1 ≤80% predicted • With or without chronic symptoms (cough, sputum production)	Shortness of breath on exertion. All patients seek medical attention.
3: Severe	• FEV_1/FVC<70% • 30% ≤FEV_1 ≤50% predicted • With or without chronic symptoms (cough, sputum production)	Increased breathlessness; quality-of-life affected because of repeated exacerbations.
4: Very severe	• FEV_1/FVC <70% • FEV_1 <30% predicted or FEV_1 <50% predicted + chronic respiratory failure • With or without chronic symptoms (cough, sputum production)	Quality-of-life appreciably affected; exacerbations may be life-threatening.

Source: National Institutes of Health 2003

monitoring the situation through IEC activities, all these mild cases could have the necessary medical intervention, and avoid suffering and deterioration of their health because of exacerbations, with simple and cheap medications. In other words, it emphasizes the need for the existing health care system to identify this 'silent' cohort of patients with COPD to prevent further progression of the disease. The following issues need serious consideration:

- COPD should be considered in any patient with a history of exposure to risk factors (especially tobacco smoking) and suggestive symptoms.
- Investigations should be done for patients presenting with chronic cough and expectoration not cured with first-level drugs. The use of a nebulizer would help the patient; this facility should be made available in all primary health centres (PHCs) to start with. It is expected that other private medical practitioners would also initiate the process depending upon the local needs. The medical officer at the primary care level has to be satisfied before a request for a chest X-ray is made to exclude and/or recognize alternate diagnoses and problems. It should always be done at the secondary care level. This facility should be available in all PHCs.
- Spirometry should be attempted if there is a doubt about the diagnosis. Proper equipment should be added with adequately trained manpower and UPS for computers located at the secondary care/district hospital or local private nursing home, if such a facility is available.
- Influenza vaccines can reduce serious illness and death in patients with COPD by about 50% and should be given once (in autumn) or twice (in autumn and winter).[35,36]
- Other diseases, especially tuberculosis, should be excluded. If the sputum is positive for acid-fast bacilli (AFB), the patient should be referred to the nearest Directly Observed Treatment, Short-course (DOTS) Centre under the Revised National Tuberculosis Control Programme. If a DOTS Centre is not available, anti-tuberculosis treatment should be started as per standard guidelines.
- Through proper registration and follow-up protocols, the auxiliary nurse–midwife (ANM) and medical officer should keep a watch on the progress of the patient, which includes improvement in the symptoms of cough and breathlessness, reduction in sputum production and increased exercise tolerance (e.g. six-minute walking). Periodic visits by the ANM would go a long way in this process. The key role of the family doctor should not be ignored. It is highly recommended to dovetail the efforts of all those concerned with the health of the patient.
- The following patients need to be referred to the secondary/tertiary care level health centre:
  —those with symptoms of cardiac or respiratory failure
  —those not responding to treatment at the primary level
  —those in whom alternate diagnoses are strongly suspected
  —those requiring assistance in tobacco cessation and/or respiratory rehabilitation.

  The patient should be referred to a centre where diagnostic and other facilities, including spirometry and inpatient services, are available.

Since the 1960s, beta-2 agonists are the mainstay of therapy for obstructive lung diseases with studies demonstrating sustained improvement in peak flows and respiratory symptoms with their use. However, the literature seems to be accumulating against their regular use as it results in tolerance to their bronchodilator and non-bronchodilator effects, and may lead to exacerbations of asthma and an increase in the number of deaths. A meta-analysis of a number of studies reinforces the accumulating evidence that the use of beta-2 agonists leads to an increased risk for cardiovascular disease. This is of special concern for patients with underlying cardiac conditions. In contrast, cardio-selective beta-blocker therapy is safe in patients with obstructive lung disease and associated with considerable reduction in cardiovascular mortality. As a word of caution, long-term trials need to be undertaken to evaluate the safety and efficacy of beta-2 agonists as against the use of other substances such as ipratropium, corticosteroids or beta-blockers. Until then, a careful, constant watch for any complication in patients with COPD is a must.[37]

Cochrane's review on COPD

Despite the lack of reversibility, patients often report symptomatic improvement with short-acting beta-2 bronchodilators. These are used for the management of both stable and acute exacerbations of COPD. A meta-analysis of 13 studies showed a slight but significant increase in FEV_1 and FVC when compared to placebo (weighted mean difference [WMD]=0.14 L; 95% CI=0.04, 0.25 and WMD=0.30 L; 95% CI=0.02, 0.58, respectively). In addition, both morning and evening peak expiratory flow rates (PEFR) were significantly better during active treatment than during placebo use (WMD=29.17 L/min; 95% CI=0.25, 58.09 and WMD=36.75 L/min; 95% CI=2.56, 70.94, respectively). A significant improvement in the daily breathlessness score was observed during treatment with beta-2 agonists when compared to placebo (SMD=1.33; 95% CI=1.0, 1.65). The risk of dropping out of the study (treatment failure) was almost double in patients on treatment with placebo as compared to those on treatment with beta-2 agonists (relative risk [RR]=0.49; 95% CI=0.33, 0.73). Patients preferred beta-2 agonists almost 10 times more often than placebo (OR=9.04; 95% CI=4.64, 17.61). One study that used a validated questionnaire for 'quality-of-life' assessment found highly significant improvements in the scores for dyspnoea (p=0.003) and fatigue (p=0.0003) during treatment with salbutamol. No studies have reported serious side-effects during treatment with inhaled beta-agonists. Hence, the use of short-acting beta-2 agonists on a regular basis for at least seven days in stable COPD is associated with improvements in post-bronchodilator lung function and a decrease in breathlessness. This review indicates that treatment with these older, inexpensive drugs is beneficial in patients with COPD. A practical approach at different levels of care has been worked out.[31]

Recommendations

- Organizing mass awareness programmes for the public and health providers to ensure early detection and initiation of treatment with low-cost, effective drugs would go a long way in controlling COPD. As cigarette smoking is the most important risk factor (Table 14), all efforts must be made to reduce and discourage this habit, particularly among the youth and young adults, to achieve overall reduction in the general morbidity, particularly that of COPD.
- The economic burden of COPD on families can be reduced if and only if all health providers strictly adopt the guidelines for early case detection, management and medication with simple and less expensive drugs to start with (Appendix 2).
- The detection of mild cases of COPD and initiation of basic treatment by peripheral health workers/providers with inexpensive drugs would go a long way in early detection and prevent disease progression to moderate and/or severe forms. Higher compliance rates for drug use (because the treatment is for a prolonged duration) and referring acute cases to the PHC/local medical doctors would reduce the financial burden of COPD on society. This act in itself would reduce the caseload on the medical doctors at the PHC/subcentre level and enable them to provide better service to other patients. This approach would help to achieve the goal of 'Health for all' in a more effective way.
- Massive training programmes for basic health workers/providers in the periphery to efficiently detect and manage the cases of COPD by adopting the guidelines would ensure success of the programme.
- Efforts should be made to reduce the burden of COPD among patients from rural areas who constitute the bulk of cases and are economically poor. One medical or a trained senior paramedical person should always be available at the PHC to attend to emergencies.
- A public–private stakeholder approach would be the most effective way to combat COPD in India. The involvement of all local medical practitioners/family doctors/pharmacists through trust and ensuring that this approach would not, in any way, affect their practice, would help this approach to succeed.

Table 14. Risk factors for COPD[38]

Factor	Comments
Age	Mainly due to association with cumulative exposure indices
Gender	More common in males, probably due to association with other risk factors
Smoking	Most important factor
Environmental pollution	Specific occupational groups
Economic status	In association with other risk factors

Source: Pande and Khilnani 2001

Acknowledgements

We gratefully thank Professor S.K. Jindal, Department of Pulmonary Medicine, Postgraduate Institute of Medical Education and Research, Chandigarh; Dr Paramesh, St John's Hospital, Bangalore; Dr K. Satish Kumar, Institute of Health Systems, Hyderabad, and Dr G. Thippanna, Chest Physician, Hyderabad, for sharing their publications and experiences.

We gratefully acknowledge the financial support and guidance of Ms Sujatha Rao, IAS. The critical reviews and suggestions made by the reviewers of the initial draft of the paper are gratefully acknowledged.

References

1. Murray CJL, Lopez AD. Evidence-based health policy—lessons from the Global Burden of Disease Study. *Science* 1966;**274:**740–3.
2. Tzanakis N, Anagnostopoulou U, Filaditaki V, Christaki P, Siafakas N. Prevalence of COPD in Greece. *Chest* 2004;**125:**892–900.

3. Mannino DM, Brown C, Giovino GA. Obstructive lung disease deaths in the United States from 1979 through 1993: An analysis using multiple-cause mortality data. *Am J Respir Crit Care Med* 1977;**156:**814–18.
4. Nishimura K, Tsukino M. Clinical course and prognosis of patients with COPD. *Curr Opin Pulm Med* 2000;**6:**127–32.
5. Jindal SK, Aggarwal AN, Gupta D. A review of population studies from India to estimate national burden of COPD and its association with smoking. *Indian J Chest Dis Allied Sci* 2001;**43:**139–47.
6. Wig KL, Guleria JS, Bhasin RC, Holmes E Jr, Vasudeva YL, Singh H. Certain clinical and epidemiological patterns of chronic obstructive lung disease as seen in northern India. *Indian J Chest Dis* 1964;**6:**183–94.
7. Sikand BK, Pamra SP, Mathur GP. Chronic bronchitis in Delhi as revealed by mass survey. *Indian J Tuberc* 1966;**13:**94–101.
8. Viswanathan R. Epidemiology of chronic bronchitis: Morbidity survey in Patna urban area. *Indian J Med Res* 1966;**54:**105–11.
9. Bhattacharya SN, Bhatnagar JK, Kumar S, Jain PC. Chronic bronchitis in rural population. *Indian J Chest Dis* 1975;**17:**1–7.
10. Radha TG, Gupta GK, Singh A, Mathur N. Chronic bronchitis in an urban locality of New Delhi: An epidemiological survey. *Indian J Med Res* 1977;**66:**273–95.
11. Thiruvengadam KV, Raghava TP, Bharadwaj KV. Survey of prevalence of chronic bronchitis in Madras city. In: Viswanathan R, Jaggi OP (eds). *Advances in chronic obstructive lung disease.* Delhi: Asthma and Bronchitis Foundation of India; 1977:59–69.
12. Viswanathan R, Singh K. Chronic bronchitis and asthma in urban and rural Delhi: In: Viswanathan R, Jaggi OP (eds). *Advances in chronic obstructive lung disease.* Delhi: Asthma and Bronchitis Foundation of India; 1977:44–58.
13. Charan NB. Chronic bronchitis in north India, Punjab. In: Viswanathan R, Jaggi OP (eds). *Advances in chronic obstructive lung disease.* Delhi: Asthma and Bronchitis Foundation of India; 1977:92–102.
14. Malik SK. Profile of chronic bronchitis in north India: The PGI experience (1972–1985). *Lung India* 1986;**4:**89–100.
15. Jindal SK. A field study on follow-up at 10 years of prevalence of COPD and PEFR. *Indian J Med Res* 1993;**98:**20–6.
16. Ray D, Abel R, Selvaraj KG. A 5-year prospective epidemiological study of COPD in rural south India. *Indian J Med Res* 1995;**101:**238–44.
17. International Institute for Population Sciences. *India—1998–99. National Family Health Survey (NFHS-2).* Mumbai, India: International Institute for Population Sciences; 2000.
18. Anderson HR. Respiratory abnormalities in Papua New Guinea children: The effect of locality and domestic wood smoke pollution. *Int J Epidemiol* 1978;**7:**63.
19. Charlton A. Children's coughs related to parental smoking. *Br Med J* 1984;**288**:1647.
20. Doll R, Peto R. Mortality in relation to smoking: 10 years' observation on British doctors. *Br Med J* 1976;**2:**1525.
21. Olson HC, Gilson JC. Respiratory symptoms, bronchitis and ventilatory capacity in men: An Anglo-Danish comparison, with special reference to differences in smoking habits. *Br Med J* 1960;**1:**450.
22. Higgins ITT. Tobacco smoking, respiratory symptoms and ventilatory capacity: Studies in random samples of the population. *Br Med J* 1959;**1:**325.
23. Robertson DG, Warrell DA, Newton-Howes JS, Fletcher CM. Bronchial reactivity to cigarette and cigar smoke. *Br Med J* 1969;**3:**269.
24. IES Project. *Health effects analysis.* Hyderabad: Institute of Health Systems; 2001 (unpublished).
25. RD's message in *Lifeline* for World Tobacco Day: 2004: *Tobacco Control in India:* Kishore Chaudhry: European Lung Whitebook Part-2, Reported by Susan Aldridge.
26. Registrar General of India. *Population projections for India and states: 1996–2016*.
27. *Monthly index of medical specialities*. New Delhi: AE Morgan Publications (India) Private Limited; 2004:24.
28. Lobowitz MD, Burrows B. The relationship of acute respiratory illness history to the prevalence and incidence of obstructive lung disorders. *Am J Epidemiol* 1977;**105:**544.
29. Malik SK. Chronic bronchitis and ventilatory impairment in *beedi* workers. *Indian J Chest Dis* 1977;**19:**21.
30. Higgins MW, Thom T. Incidence, prevalence and mortality: Intra and inter country differences. In: Hensley MJ, Saunders NA (eds). *Clinical epidemiology of COPD*. New York: Marcel Dekker; 1990:23–43.
31. Jindal SK, Gupta D, Aggarwal AN. Guidelines for management of COPD in India: A guide for physicians (2003). *Indian J Chest Dis Allied Sci* 2004;**46:**137–53.
32. Joshi RC, Madan RN, Brash AA. Prevalence of chronic bronchitis in an industrial population in north India. *Thorax* 1975;**30:**61–7.
33. Jindal SK. A field study on follow up at 10 years of prevalence of COPD and PEFR. *Indian J Med Res* 1993;**98:**20–6.
34. National Institutes of Health. *Global initiative for chronic obstructive lung disease. Global strategy for the diagnosis, management, and prevention of COPD. Executive summary.* National Institutes of Health; 2003:1–30.
35. Nichol KL, Margolis KL, Wuorenma J, Von Sternberg T. The efficacy and cost-effectiveness of vaccination against influenza among elderly persons living in the community. *N Engl J Med* 1994;**331:**778–84.
36. Edwards KM, Dupont WD, Westrich MK, Plummer WD Jr, Palmer PS, Wright PF. A randomized controlled trial of cold-adapted and inactivated vaccines for the prevention of influenza A disease. *J Infect Dis* 1994;**169:**68–76.
37. Salpeter SR, Ormiston TM, Salpeter EE. Cardiovascular effects of beta-agonists in patients with asthma and COPD: A meta-analysis. *Chest* 2004;**125:**2309–21.
38. Pande JN, Khilnani GC. Chronic obstructive airways disease—epidemiology and aetiology. In: Pande JN (ed). *Respiratory medicine in tropics.* New Delhi: Oxford University Press; 2001:316–22.

Appendix 1

Guidelines for the management of stable COPD[27]

Diagnosis Assess lung function (FEV_1 and vital capacity) using spirometry

Clinical features

- Mild: No abnormal signs; smoker's cough; little/no breathlessness
- Moderate: Breathlessness (with/without wheeze) on moderate exertion; cough (with or without sputum); variable abnormal signs—general reduction in breath sounds and presence of wheeze
- Severe: Breathlessness on minimal exertion or at rest; wheeze and cough often prominent; over-inflation of the lung usual; cyanosis, peripheral oedema and polycythaemia in advanced disease, especially during exacerbation

Aims

- To provide optimal symptom control
- To prevent deterioration of condition and complications
- To improve the quality of life

Additional supportive measures

- Cessation of smoking is absolutely essential to prevent accelerated decline in the condition.
- Encourage exercise to improve performance and reduce breathlessness.
- Obesity or poor nutrition should be addressed.
- Influenza vaccine is recommended particularly for those with severe COPD.
- Depression associated with severe COPD should be identified and treated.

Refer a patient with COPD to a specialist in the following situations:

- Suspected severe COPD
- Onset of cor pulmonale
- Assessment for oxygen nebulizer or oral corticosteroid therapy
- Bullous lung disease
- Rapid decline in FEV_1
- Under 40 years of age or having a family history of $alpha_1$-antitrypsin deficiency
- Uncertain diagnosis
- Symptoms disproportionate to lung function deficit
- Frequent infections

Table A1.1 Guidelines for the management of COPD

	Mild	Moderate	Severe
Predicted FEV_1	60%–80%	40%–60%	<40%
Bronchodilator therapy*	Use inhaled short-acting beta-2 agonist or inhaled anticholinergics, as required.	Regular use of inhaled short-acting beta-2 agonist and/or regular inhaled anticholinergics	Regular use of combined inhaled short-acting beta-2 agonist and anticholinergics High doses of bronchodilators (including the use of nebulizer) should only be prescribed after assessment by a specialist. Theophylline should be reserved for those in whom other treatments fail to control symptoms adequately.
Corticosteroid therapy*	Not required	Consider corticosteroid therapy	Perform corticosteroid trial. *Corticosteroid trial:* 30 mg oral prednisolone for 2 weeks; positive response to reversibility test—give regular inhaled corticosteroids.
Long-term oxygen therapy	Not required	Not required	Assessment of arterial blood gases by a specialist is required. Long-term oxygen therapy is prescribed if PaO_2 <7.3 kPa and FEV_1 <1.5 L

*No bronchodilator and/or corticosteroid therapy reversibility tests; a positive response is an increase in FEV_1, i.e. both ≥200 ml and a 15% increase from the baseline.
Note: Positive response to bronchodialtor therapy indicates improved likelihood of positive response to corticosteroids. The dose of the bronchodilator and/or corticosteroid may be increased during an acute exacerbation.

Appendix 2

Guidelines for the management of COPD in India: A guide for physicians[31,34]

Staging the severity of COPD

Table A2.1 Staging of COPD based on symptoms, signs, 6-minute walk test and peak expiratory flow rate (PEFR)

Stage	Symptoms (cough and sputum)	Signs	6-minute walk test	PEFR (optional)
At risk	No dyspnoea, hypersecretion+			
Mild	Dyspnoea on unaccustomed activity or climbing two flights of stairs	Mild hyperinflation	>200 m	50%–70%
Moderate	Dyspnoea on unaccustomed activity	Moderate hyperinflation	100–200 m	30%–50%
Severe	Dyspnoea at rest	Near absence of breath sounds, respiratory failure, polycythaemia and chronic heart failure (CHF)	<100 m	<30%

Table A2.2 Treatment guidelines depending upon the severity of COPD

Mild	Short-acting bronchodilators, when needed
Moderate	Regular treatment with one or more bronchodilators
Severe	As in moderate COPD + inhaled corticosteroids; treatment of complications

COPD should be considered and spirometry performed if any of these indicators are present (Table A2.3). The indicators are not diagnostic by themselves, but the presence of multiple indicators increase the probability of a diagnosis of COPD. Spirometry is needed to establish a diagnosis of COPD.

Table A2.3 Key indicators for considering a diagnosis of COPD

Chronic cough	Present intermittently or every day Often present throughout the day Seldom only nocturnal
Chronic sputum production	Any pattern of chronic sputum production may indicate COPD
Dyspnoea that is	Progressive (worsens over time); Persistent (present every day); Described by the patient as: 'increased effort to breathe', 'heaviness', 'air hunger', or 'gasping'; which is worse on exercise Worse during respiratory infections
History of exposure to risk factors, especially	Tobacco smoke Occupational dusts and chemicals Smoke from home cooking and heating fuels

The use of bronchodilators is the central point in the symptomatic treatment/management of COPD. These are given on an as-needed basis for the relief of persistent or worsening symptoms, or on a regular basis to prevent or reduce symptoms. Long-acting inhaled bronchodilators are more effective and convenient, but also more expensive. Combining various bronchodilators may improve the efficacy and decrease the risk of side-effects compared to increasing the dose of a single bronchodilator.

Guidelines for physicians on tobacco cessation

Follow the 5-A strategy

- Ask (about use)
- Assess (the status and severity of use)
- Advise (to stop)
- Assist (in smoking cessation)
- Arrange (the follow-up programme)

Details of advice to the patient

- Review your tobacco use. Accept that smoking is a problem and harmful for your health.
- Make a decision and be determined to quit. Don't be overconfident that you can quit any time you like.
- Share your decision with family, friends and your doctor. Accept their help.
- Fix a quit date. Don't postpone.
- Remove ashtrays and other objects that are reminders of the habit.
- Keep away from trigger situations.
- Adopt a healthy lifestyle such as relaxation, exercise, good diet with plenty of water, fruits, vegetables and avoid tea/coffee/alcohol.
- Take help from family, friends and your doctor.

First few steps of quitting tobacco smoking

I To reduce quantity

- Change to a non-preferred brand.
- Keep a record of the amount and frequency of tobacco use.
- Decrease the number of puffs when smoking.
- Leave large stubs.
- Don't inhale deeply.

II To deal with triggers when you have an urge to smoke (trigger coping)

- To overcome an extraordinary urge to take tobacco, try alternatives (chewing gum, toffee, peppermint, cardamom).
- Increase your water intake.

- Breathe deeply and quietly.
- Do some other work to engage and keep your mind off tobacco.
- Delay the act of smoking—count till 100 and think of pleasant situations.

Table A2.4 Commonly used bronchodilators in India

Drugs	Metered dose/dry powder inhalers (μg/dose)	Oral
Beta-agonists		
• Salbutamol	100–200	2–4 mg tid/qid
• Terbutaline	250–500	2.5–5 mg tid
• Salmeterol	25–50	
• Formoterol	6–12	
• Bambuterol		10–12 mg/day
Anticholinergics		
• Ipratropium	40–80	
• Tiotropium	18	
Methylxanthines		
• Aminophylline		225–450 mg/day
• Theophylline		200–600 mg/day

Note:
- A combination of a short-acting beta-agonist and the anticholinergic drug ipratropium in stable COPD produces greater and more sustained improvements in FEV_1 than either alone, and does not produce tachyphylaxis.
- The addition of oral theophylline should normally be considered only if inhaled treatments have failed to provide adequate relief.
- Don't use antibiotics except to control bacterial infections and infectious exacerbations.
- Regular use of antitussives should be discouraged in stable COPD.
- Respiratory stimulants, sedatives and narcotics should be avoided because of their respiratory depressant effects.
- Advise proper nutritional intake.
- Initiate rehabilitation programmes particularly for those quitting the tobacco habit.
- The common causes of an exacerbation are infection of the tracheobronchial tree and air pollution. The cause of a third of the exacerbations cannot be identified. Pneumonia, congestive heart failure, pneumothorax, pleural effusion, pulmonary embolism and arrhythmias, which also mimic COPD, need to be excluded. Bronchodilators are the cornerstone of managing exacerbations of COPD. The dose and/or frequency of use of the existing bronchodilator therapy need to be increased. Nebulizers may be used for drug administration. Systemic glucocorticoids should be used in acute exacerbations as they shorten the recovery time and help restore lung function more quickly.

III Once you quit

- Learn to say 'no' to tobacco offers from others. Don't take a single puff.
- Try to remain in smoke-free areas. Avoid the company of smokers and even tobacco-chewers.
- Form a group of people who have quit tobacco and share their experiences.
- Collect the money saved from each pack of cigarettes or *paan masala*. Buy gifts for your loved ones with that money.
- Try alternative ways to deal with mental stress and tension such as relaxation, deep breathing, listening to music.
- Remember that there can be some withdrawal symptoms after quitting, such as headache, irritability, lack of concentration, etc. But bear with them. These are temporary and will disappear in a few days.

Even if you fail to quit smoking
—don't get disheartened, try again.
—seek the help of those who have quit smoking.
—seek professional help and medical advice.

Reduction of other risk factors

- Avoid burning of crop residue.
- Suppress dust by the use of water.
- At the workplace, use a mask in areas of dust generation.
- Use smokeless *chullahs* to reduce the risk associated with solid fuel combustion. Use a thin cloth to cover the nose and mouth near sources of combustion.
- The kitchen should be adequately ventilated.
- Stop/minimize indoor smoking and in front of the children.

Drug treatment

Bronchodilators are central to the symptomatic treatment of COPD. Inhaled drugs are preferred to oral preparations. The availability of the patient and affordability of the drug need to be considered in the choice of drugs. Step-wise treatment is recommended.

See Appendix 2 in the paper on 'Economic burden of asthma' for the management of COPD. For causal analysis, and medical equipment and drug requirements for COPD, *see* Appendix 3 of the same paper.

Oral and dental diseases: Causes, prevention and treatment strategies

NASEEM SHAH

DENTAL CARIES

Dental caries is an infectious microbiological disease of the teeth that results in localized dissolution and destruction of the calcified tissues. It is the second most common cause of tooth loss and is found universally, irrespective of age, sex, caste, creed or geographic location. It is considered to be a disease of civilized society, related to lifestyle factors, but heredity also plays a role. In the late stages, it causes severe pain, is expensive to treat and leads to loss of precious man-hours. However, it is preventable to a certain extent. The prevalence of dental caries in India is 50%–60%.

Aetiology

An interplay of three principal factors is responsible for this multifactorial disease.

- Host (teeth and saliva)
- Microorganisms in the form of dental plaque
- Substrate (diet)

Thus, caries requires a susceptible host, cariogenic oral flora and a suitable substrate, which must be present for a sufficient length of time.

Host factors

Teeth[1–4]

- *Composition:* Deficiency in fluorine, zinc, lead and iron content of the enamel is associated with increased caries.
- *Morphological characteristics:* Deep, narrow occlusal fissures, and lingual and buccal pits tend to trap food debris and bacteria, which can cause caries. As teeth get worn (attrition), caries declines.
- *Position:* The interdental areas are more susceptible to dental caries. Malalignment of the teeth such as crowding, abnormal spacing, etc. can increase the susceptibility to caries.

Division of Conservative Dentistry and Endodontics
Centre for Dental Education and Research
All India Institute of Medical Sciences, New Delhi 110029
e-mail: nshah@aiims.ac.in

Saliva[5–8]

Saliva has a cleansing effect on the teeth. Normally, 700–800 ml of saliva is secreted per day. Caries activity increases as the viscosity of the saliva increases. Eating fibrous food and chewing vigorously increases salivation, which helps in digestion as well as improves cleansing of the teeth. The quantity as well as composition, pH, viscosity and buffering capacity of the saliva plays a role in dental caries.

- *Quantity:* Reduced salivary secretion as found in xerostomia and salivary gland aplasia gives rise to increased caries activity.
- *Composition:* Inorganic—fluoride, chloride, sodium, magnesium, potassium, iron, calcium and phosphorus are inversely related to caries.
  Organic—ammonia retards plaque formation and neutralizes the acid.
- *pH:* A neutral or alkaline pH can neutralize acids formed by the action of microorganisms on carbohydrate food substances.
- *Antibacterial factors:* Saliva contains enzymes such as lactoperoxidase, lypozyme, lactoferrin and immunoglobulin (Ig)A, which can inhibit plaque bacteria.

Dental plaque[9–12]

Dental plaque is a thin, tenacious microbial film that forms on the tooth surfaces. Microorganisms in the dental plaque ferment carbohydrate foodstuffs, especially the disaccharide sucrose, to produce acids that cause demineralization of inorganic substances and furnish various proteolytic enzymes to cause disintegration of the organic substances of the teeth, the processes involved in the initiation and progression of dental caries. The dental plaque holds the acids produced in close contact with the tooth surfaces and prevents them from contact with the cleansing action of saliva.

Table 1. Causes of dental caries

Direct	Indirect	Distant
1. Tooth • Structure—fluoride content and other trace elements such as zinc, lead, iron • Morphology—deep pits and fissures • Alignment—crowding 2. Microorganisms—dental plaque accumulation due to poor oral hygiene 3. Diet • Intake of refined carbohydrates such as sucrose, maltose, lactose, glucose, fructose, cooked sticky starch, etc. —quantity; frequency, physical form; oral clearance rate • Saliva (quantity and quality) —reduced secretion (xerostomia) increases caries —Viscosity: more viscous, more caries —pH: alkaline pH neutralizes acid, less caries —enzymes: lactoperoxidase, lysozyme lactoferrins —immunoglobulins IgA	• Poor contact between the teeth resulting in food impaction and caries due to the following causes —malalignment of the teeth (crowding) —loss of some teeth and failure to replace them • Gingival recession leading to root caries	• Socioeconomic status • Literacy level • Location—urban, rural • Age • Sex • Dietary habits • Climatic conditions and soil type • Social and cultural practices • Availability/access to health care facility • Health insurance

Substrate[13–16]

The role of refined carbohydrates, especially the disaccharide sucrose, in the aetiology of dental caries is well established. The total amount consumed as well as the physical form, its oral clearance rate and frequency of consumption are important factors in the aetiology. Vitamins A, D, K, B complex (B6), calcium, phosphorus, fluorine, amino acids such as lysine and fats have an inhibitory effect on dental caries.

Indirect causes[17,18]

- Loss of some natural teeth and failure to replace them results in drifting of the teeth in the edentulous space. This leads to increased food impaction between the teeth and formation of new carious lesions.
- Malalignment of the teeth, especially crowding, does not allow proper cleaning between the teeth and leads to an increased incidence of caries.
- Gingival recession, abrasion and abfraction defects at the neck of the tooth increase root caries.
- Selenium in the soil increases the formation of caries while molybdenum and vanadium decrease it.
- A high temperature is associated with a lower prevalence of caries. Water has a cleansing effect on the teeth. If the fluoride content of the water is at an optimum concentration, it will also exert an anticaries effect.

Distant causes[19,20]

- A low socioeconomic and literacy status is associated with caries.
- Urbanization is linked to an increased incidence of caries.
- Caries is more common in childhood and adolescence, and after 60 years of age, when the incidence of root caries is higher.
- Females develop caries more often than males.
- Non-vegetarians develop caries more often than vegetarians.
- Availability/access to a health care facility can affect utilization of health care services.
- Lack of oral health insurance promotes oral neglect and increases disease levels.

Table 1 summarizes the causes of dental caries.

Prevention and control of dental caries

1. Increase the resistance of the teeth.[21–25]

Systemic use of fluoride: (i) Fluoridation of water, milk and salt; (ii) fluoride supplementation in the form of tablets and lozenges; and (iii) consuming a fluoride-rich diet such as tea, fish, etc.

Topical: (i) Use of fluoridated toothpaste and mouth wash; (ii) use of fluoride varnishes (in-office application, longer duration of action, high fluoride content); (iii) use of casein phosphopeptide–amorphous calcium phosphate (CPP–ACP), which is available as tooth mousse, helps to remineralize the soft initial carious, demineralized areas of the teeth.

2. Combat the microbial plaque by physical and chemical methods.

(i) *Physical methods*[26–30]

The correct method and frequency of brushing should be followed—in the morning and before going to bed and preferably after every major meal.

Tongue cleaning and the use of indigenous agents such as the bark of neem or mango (where toothbrush and paste are unaffordable) should be encouraged. The use of coarse

toothpowder and tobacco-containing dentifrices should be avoided.

The use of various interdental cleaning aids such as dental floss, interdental brush, water pik, etc. supplements the cleansing effect of a toothbrush. Use of an electronic toothbrush in children and persons with decreased manual dexterity is recommended.

(ii) *Chemical methods*

These include the use of a fluoride-containing toothpaste, mouth rinses and 0.2% chlorhexidine and povidine–iodine mouthwash. These should be used on prescription of a dental surgeon.

3. Modify the diet.[31–34]

Reduce the intake and frequency of refined carbohydrates. Avoid sticky foods and replace refined with unrefined natural food. Increase the intake of fibrous food to stimulate salivary flow, which is protective against caries. Consume caries-protective foods such as cheese, nuts, raw vegetables, fruits, etc. Stimulate salivary flow with sugar-free chewing gum. Xylitol (a sugar substitute)-containing chewing gum, if chewed between meals, produces an anti-caries effect by stimulating salivary flow.

Preventive interventions[35–43]

The use of pit and fissure sealants[35,36] and application of fluoride varnish[37,38] help in slowing down the development of caries.

Preventive restorations should be carried out[39,40] and atraumatic restorative treatment (ART) should be used as a community-based approach for the treatment and prevention of dental caries.[41–43]

Treatment of dental caries

Treatment comprises removal of decay by operative procedures and restoration with appropriate materials such as silver fillings, gold inlays, composite resin, glass ionomer cement, full metal or porcelain crowns, etc. In advanced cases, where the pulp of the tooth is involved, endodontic treatment may be required. Where there is extensive destruction of the tooth structure or when endodontic treatment is not feasible, extraction of the tooth and replacement by an artificial prosthesis may be required.

Miscellaneous measures

These include the following:

- Prevention of malocclusion (especially crowding of the teeth)
- Prevention of premature loss of deciduous teeth
- Restoration of missing permanent teeth by prostheses (dentures)
- Making sugar-free chewing gum freely available and affordable in the country

Table 2. Prevention and treatment of dental caries

Medical interventions	Non-medical interventions	Other interventions
• Use of systemic and topical fluorides • Use of pit and fissure sealants • Preventive restorations • Different types of restorations and endodontic treatment • Regular dental check-up	• Oral health education • Nutrition and diet • Proper methods of maintaining oral hygiene —use of fluoride tooth-paste and brush —use of dental floss and interdental brushes, etc. —antiseptic mouth washes (under prescription)	• Make oral health care more accessible and affordable • Improve the socioeconomic and literacy level of the population • Include oral health care in general health insurance

- Using sugar substitutes such as saccharine, xylitol, mannitol, aspartame, etc. in paediatric medicinal syrups, bakery products, jams, marmalade, etc.
- Making toothbrushes and fluoridated toothpaste available to the masses at low cost. Regular use of fluoridated toothpaste is proven to reduce the incidence of dental caries by 30%.

Table 2 summarizes the prevention and treatment strategies for dental caries.

References

1. Babaahmady KG, Marsh PD, Challacombe SJ, Newman HN. Variations in the predominant cultivable microflora of dental plaque at defined subsites on approximal tooth surfaces in children. *Arch Oral Biol* 1997;**42:**101–11.
2. Liu F. [The relation between the resistance distribution on crown surface and caries.] *Zhonghua Kou Qiang Yi Xue Za Zhi* 1993;**28:**47–9.
3. Marcucci M, Bandettini MV. Dental caries in the rat in relation to the chemical composition of the teeth and diet. Variations in the diet of the Ca/P ratio obtained by changes in the phosphorus content. *Minerva Stomatol* 1981;**30:**17–20.
4. Haldi J, Wynn W, Bentley KD, Law ML. Dental caries in the albino rat in relation to the chemical composition of the teeth and of the diet. IV. Variations in the Ca/P ratio of the diet induced by changing the calcium content. *J Nutr* 1959;**67:**645–53.
5. Daniels TS, Silverman S, Michalski JP, Greenspan JS, Sylvester RA, Talal N. The oral component of Sjogren's syndrome. *Oral Surg* 1975;**39:**875–85.
6. Finn SB, Klapper CE, Voker JF. Intra-oral effects upon experimental hamster caries. In: RF Sognnaes (ed). *Advances in experimental caries research*. Washington, DC: American Association for the Advancement of Sciences; 1955:155–68.
7. Frank RM, Herdly J, Phillippe E. Acquired dental defects and salivary gland lesions after irradiation for carcinoma. *J Am Dent Assoc* 1965;**70:**868–83.
8. Kermiol M, Walsh RF. Dental caries after radiotherapy of the oral regions. *J Am Dent Assoc* 1975;**91:**838–45.
9. Fitzgerald RJ, Keyes PH. Demonstration of the etiologic role of streptococci in experimental caries in the hamster. *J Am Dent Assoc* 1960;**61:**9–19.
10. Keyes PH. The infection and transmissible nature of experimental dental caries. *Arch Oral Biol* 1960;**1:**304–20.

11. Orland FJ, Blayney JR, Harrison RW, Reyniers JA, Trexler PD, Ervin RF, *et al.* Experimental caries in germ-free rats inoculated with enterococci. *J Am Dent Assoc* 1955;**50:**259–72.
12. Rosen S, Kolstad RA. Dental caries in gnotobiotic rats inoculated with a strain of *Peptostreptococcus intermedius*. *J Dent Res* 1977;**56:**187.
13. Burt BA, Eklund Sa, Morgan KJ, Larkin FE, Guire KE, Brown LO, *et al.* The effects of sugar intake and frequency of ingestion on dental caries increment in a three-year longitudinal study. *J Dent Res* 1988;**67:**1422–9.
14. Caldwell RC. Physical properties of foods and their caries-producing potential. *J Dent Res* 1970;**49:**1293–8.
15. Harris RS. Minerals: Calcium and phosphates. In: RF Gould (ed). *Dietary chemicals vs. dental caries. Advances in chemistry services* 94. Washington, DC: American Chemical Society; 1970:116–22.
16. Nizel AE. *Nutrition in preventive dentistry*: *Sciences and practice*. 2nd ed. Philadelphia: WB Saunders; 1981:417–52.
17. Helm S, Petersen PE. Causal relation between malocclusion and caries. *Acta Odontol Scand* 1989;**47:**217–21.
18. Warren JJ, Slayton RL, Yonezu T, Kanellis MJ, Levy SM. Interdental spacing and caries in primary dentition. *Pediatr Dent* 2003;**25:**109–13.
19. Ellwood RP, Davies GM, Worthington HV, Blinkhorn AS, Taylor GO, Davies RM. Relationship between area deprivation and the anticaries benefit of an oral health programme providing free fluoride toothpaste to young children. *Commun Dent Oral Epidemiol* 2004;**32:**159–65.
20. Shah N, Sundaram KR. Impact of socio-demographic variables, oral hygiene practices, oral habits and diet on dental caries experience of Indian elderly: A community-based study. *Gerodontology* 2004;**21:**43–50.
21. Hicks J, Garcia-Godoy F, Flaitz C. Biological factors in dental caries: Role of remineralization and fluoride in the dynamic process of demineralization and remineralization (Part 3). *J Clin Pediatr Dent* 2004;**28:**203–14.
22. Kargul B, Caglar E, Tanboga I. History of water fluoridation. *J Clin Pediatr Dent* 2003;**27:**213–17.
23. Featherstone JD. Prevention and reversal of dental caries: Role of low level fluoride. *Commun Dent Oral Epidemiol* 1999;**27:**31–40.
24. Stephen KW. Systemic fluorides: Drops and tablets. *Caries Res* 1993;**27**(Suppl. 1):9–15.
25. Cai F, Shen P, Morgan MV, Reynolds EC. Remineralization of enamel subsurface lesions in situ by sugar-free lozenges containing casein phosphopeptide–amorphous calcium phosphate. *Aust Dent J* 2003;**48:**240–3.
26. Klock B. Krasse B. Effect of caries preventive measures in children with high numbers of *S. mutans* and lactobacilli. *Scand J Dent Res* 1978;**86:**221.
27. Krasse B. *Caries risk*: *A practical guide for assessment and control*. Chicago: Quintessence Publishing Co. Inc; 1985.
28. Loe H. Human research model for the production and prevention of gingivitis. *J Dent Res* 1971;**50:**256.
29. Emilson CG. Potential efficacy of chlorhexidine against mutant streptococci and human dental caries. *J Dent Res* 1994;**73:** 682–91.
30. Twetman S. Antimicrobials in future caries control? A review with special reference to chlorhexidine treatment. *Caries Res* 2004;**38:**223–9.
31. Marshall TA. Carries prevention in pediatrics: Dietary guidelines. *Quintessence Int* 2004;**35:**332–5.
32. van Loveren C, Duggal MS. Experts' opinions on the role of diet in caries prevention. *Caries Res* 2004;**38** (Suppl. 1):16–23.
33. Vanobbergen J, Declerck D, Mwalili S, Martens L. The effectiveness of a 6-year oral health education programme for primary schoolchildren. *Commun Dent Oral Epidemiol* 2004;**32:**173–82.
34. TanzerJM. Xylitol chewing gum and dental caries. *Int Dent J* 1995;**45**(Suppl. 1): 65–76.
35. Kumar J, Siegal MD. Workshop on guidelines for sealant use: Recommendations. *J Pub Health Dent* 1955;**5**(Special issue): 263–73.
36. Swift EJ Jr. The effect of sealants on dental caries: A review. *J Am Dent Assoc* 1988;**116:**700–4.
37. Beltran-Aguilar ED, Goldstein JW, Lockwood SA. Fluoride varnishes—a review of their clinical use, cariostatic mechanism, efficacy and safety. *J Am Dent Assoc* 2000;**131:**589–96.
38. Savanberg M, Westergren G. Effect of SnF2, administered as mouth rinses or topically applied, on *Streptococcus mutans*, *Streptococcus sanguis* and lactobacilli in dental plaque and saliva. *Scand J Dent Res* 1983;**91:**123.
39. Simonsen RJ. Preventive resin restoration. *Quintessence Int* 1978;**9:**69–76.
40. Simonsen RJ. Preventive resin restorations: Three year results. *J Am Dent Assoc* 1980;**100:**535–9.
41. Frencken JE. [Atraumatic restorative treatment (ART). A special tissue preservative and patient-friendly approach.] *Ned Tijdschr Tandheelkd* 2003;**110:**218–22.
42. Carvalho CK, Bezerra AC. Microbiological assessment of saliva from children subsequent to atraumatic restorative treatment (ART). *Int J Paediatr Dent* 2003;**13:**186–92.
43. Smales RJ, Gao W. *In vitro* caries inhibition at the enamel margins of glass ionomer restorations developed for the ART technique. *J Dent* 2000;**28:**249–56.

DENTOFACIAL ANOMALIES OR MALOCCLUSION

Dentofacial anomalies include hereditary, developmental and acquired malocclusion or malalignment of the teeth. Worldwide, the average prevalence of malocclusion in the 10–12 years' age group is reported to be 30%–35%.

Aetiology

Direct causes[1–17]

- *Heredity:* Hereditary factors play an important role in conditions such as cleft lip and palate, facial asymmetries, variations in tooth shape and size, deep bites, discrepancies in jaw size.[1–4]
- *Congenital:* These include cleft lip and palate, and syndromes associated with anomalies of craniofacial structures, cerebral palsy, torticollis, cleidocranial dysostosis, congenital syphilis, etc.[5,6]
- *Abnormal pressure habits and functional aberrations:* These include abnormal suckling, thumb and finger sucking, tongue thrusting and sucking, lip and nail biting, mouth breathing, enlarged tonsils and adenoids, trauma and accidents.[7–13]

Table 3. Causes of dentofacial anomalies and malocclusion

Direct	Indirect	Distant
• Hereditary/congenital • Abnormal pressure habits and functional aberrations —abnormal suckling —mouth breathing —thumb and finger sucking —tongue thrusting and sucking —abnormal swallowing • Trauma and accidents • Local factors —abnormalities of number (supernumerary teeth, missing teeth) —abnormalities of tooth size and shape —abnormal labial frenum and mucosal barriers —premature tooth loss —prolonged retention of deciduous teeth —delayed eruption of permanent teeth —abnormal eruptive path —untreated dental caries and improper dental restorations, especially on the proximal surfaces	• Environmental factors —prenatal causes such as trauma, maternal diet and metabolism, German measles, certain drugs, and position *in utero* —postnatal causes such as birth injury, cerebral palsy, temporomandibular joint injury	• Poor nutritional status—deficiency of vitamin D, calcium and phosphates • Endocrine imbalance such as hypothyroidism • Metabolic disturbances and muscular dystrophies • Infectious diseases such as poliomyelitis • Functional aberrations —psychogenic tics and bruxism —posture

- *Local factors:* These include abnormalities of number such as supernumerary and missing teeth, abnormalities of tooth size and shape, abnormal labial frenum causing spacing between the upper anterior teeth, premature tooth loss with drifting of the adjoining and opposite teeth, prolonged retention of the milk teeth, delayed eruption of the permanent teeth, abnormal eruptive path, dental caries, and improper dental restorations.[14–17]

Indirect causes[18–25]

Environmental

—Prenatal: trauma, maternal diet and metabolism, German measles, certain drugs and position *in utero*

—Postnatal: birth injury, cerebral palsy, temporomandibular joint injury

Distant causes[26,27]

- *Endocrine imbalance:* Hypothroidism is related to an abnormal resorption pattern, delayed eruption and gingival disturbances. Retained deciduous teeth may be due to hypothroidism.
- *Metabolic disturbance and infectious diseases:* Acute febrile conditions delay growth and development. Diseases such as poliomyelitis, muscular dystrophy and cerebral palsy have a characteristic deforming effect on the dental arch.
- *Nutritional:* Vitamin D, calcium and phosphorus are associated with bone metabolism and their deficiency could lead to growth disturbances.
- *Abnormal muscle function and posture:* Psychogenic tics and abnormal head posture can contribute towards malrelation of the jaws.

Factors responsible for causing dentofacial anomalies and malocclusion are summarized in Table 3.

Prevention and treatment[28–33]

The prevention and treatment of dentofacial anomalies can be undertaken at three levels (Table 4).

- Primary prevention—Preventive orthodontics
- Secondary prevention—Interceptive orthodontics
- Tertiary prevention—Corrective orthodontic treatment by removable and fixed appliances, and surgical orthodontics

Table 4. Strategies for the prevention and treatment of dentofacial anomalies and malocclusion

Medical interventions	Non-medical interventions
• Habit-breaking appliances • Serial extractions • Space-maintainers and -regainers • Functional appliances in developing malocclusion to correct jaw relations • Frenectomies and simple appliances to correct anterior cross-bites • Removable and fixed appliances • Orthognathic and plastic surgery • Speech therapy • Regular dental check-up for early intervention • Counselling • Preservation and restoration of primary and permanent teeth	• Control harmful oral habits • Prenatal and perinatal care • Genetic counselling

Primary prevention

This includes control of harmful oral habits, and preservation and restoration of primary and permanent dentition.

Secondary prevention

Habit-breaking appliances should be used. Serial extractions, space maintainers/regainers, and functional appliances to correct jaw relations are other modalities. Frenectomies and simple appliances can be used to correct anterior crossbites.

Tertiary prevention

Corrective orthodontic treatment includes the use of fixed and removal appliances and surgical orthodontics in cases of severe malocclsion.

References

1. Mossey PA. The heritability of malocclusion: Part 1. Genetics, principles and terminology. *Br J Orthod* 1999;**26:**103–13.
2. Mossey PA. The heritability of malocclusion: Part 2. The influence of genetics in malocclusion. *Br J Orthod* 1999;**26:**195–203.
3. Varrela J. Genetic and epigenetic regulation of craniofacial development. *Proc Finn Dent Soc* 1991;**87:**239–44.
4. Moss ML. Genetics, epigenetics, and causation. *Am J Orthod* 1981;**80:**366–75.
5. Golan I, Baumert U, Hrala BP, Mussig D. Early craniofacial signs of cleidocranial dysplasia. *Int J Paediatr Dent* 2004;**14:**49–53.
6. Ortiz-Posadas MR, Vega-Alvarado L, Toni B. A similarity function to evaluate the orthodontic condition in patients with cleft lip and palate. *Med Hypotheses* 2004;**63:**35–41.
7. Chen QR, Zhong HL. [Lower lip biting habits and malocclusions.] *Shanghai Kou Qiang Yi Xue* 1994;**3:**3–6.
8. Yamaguchi H, Sueishi K. Malocclusion associated with abnormal posture. *Bull Tokyo Dent Coll* 2003;**44:**43–54.
9. daCosta OO, Orenuga OO. Dentofacial anomalies related to the digit sucking habit. *Afr J Med Med Sci* 2002;**31:**239–42.
10. Massler M. Oral habits: Development and management. *J Pedod* 1983;**7:**109–19.
11. Popovich F. The prevalence of sucking habit and its relationship to oral malformations. *Appl Ther* 1966;**8:**689–91.
12. Hatzakis S, Toutountzakis N. Speech defects and malocclusion. *Hell Stomatol Chron* 1984;**28:**97–106.
13. Hawkins AC. Mouth breathing and its relationship to malocclusion and facial abnormalities. *N M Dent J* 1969;**20:**18–21.
14. Nik-Hussein NN. Supernumerary teeth in the premaxillary region: Its effects on the eruption and occlusion of the permanent incisors. *Aust Orthod J* 1990;**11:**247–50.
15. Northway WM, Wainright RL, Demirjian A. Effects of premature loss of deciduous molars. *Angle Orthod* 1984;**54:**295–329.
16. Basdra EK, Kiokpasoglou MN, Komposch G. Congenital tooth anomalies and malocclusions: A genetic link? *Eur J Orthod* 2001;**23:**145–51.
17. Forsberg CM, Tedestam G. Etiological and predisposing factors related to traumatic injuries to permanent teeth. *Swed Dent J* 1993;**17:**183–90.
18. Proffit WR. On the aetiology of malocclusion. The Northcroft lecture, 1985 presented to the British Society for the Study of Orthodontics, Oxford, 18 April, 1985. *Br J Orthod* 1986; **13:**1–11.
19. Defabianis P. Post-traumatic TMJ internal derangement: Impact on facial growth (findings in a pediatric age group). *J Clin Pediatr Dent* 2003;**27:**297–303.
20. Schoenwetter RF. A possible relationship between certain malocclusions and difficult or instrumental deliveries. *Angle Orthod* 1974;**44:**336–40.
21. Vittek J, Winik S, Winik A, Sioris C, Tarangelo AM, Chou M. Analysis of orthodontic anomalies in mentally retarded developmentally disabled (MRDD) persons. *Spec Care Dentist* 1994;**14:**198–202.
22. Strodel BJ. The effects of spastic cerebral palsy on occlusion. *ASDC J Dent Child* 1987;**54:**255–60.
23. Matsumoto S, Morinushi T, Ogura T. Time dependent changes of variables associated with malocclusion in patients with Duchenne muscular dystrophy. *J Clin Pediatr Dent* 2002;**27:**53–61.
24. Singh GD, Rivera-Robles J, de Jesus-Vinas J. Longitudinal craniofacial growth patterns in patients with orofacial clefts: Geometric morphometrics. *Cleft Palate Craniofac J* 2004;**41:** 136–43.
25. Mg'ang'a PM, Chindia ML. Dental and skeletal changes in juvenile hypothyroidism following treatment: Case report. *Odontostomatol Trop* 1990;**13:**25–7.
26. Gola G. [Dietetic factors in the development of the facial bones and in the etiology of malocclusion.] *Riv Odontostomatol Implantoprotesi* 1983;**3:**25–9, 31.
27. Iwamoto J, Takeda T, Ichimura S, Sato Y, Yeh JK. [Differential effect of vitamin K and vitamin D supplementation on bone mass in young rats fed normal or low calcium diet.] *Yonsei Med J* 2004;**45:**314–24.
28. Kerosuo H. The role of prevention and simple interceptive measures in reducing the need for orthodontic treatment. *Med Princ Pract* 2002;**11:**16–21.
29. Varrela J, Alanen P. Prevention and early treatment in orthodontics: A perspective. *J Dent Res* 1995;**74:**1436–8.
30. Sapino S. Space maintenance devices. *Minerva Stomatol* 1989;**38:**981–7.
31. Binder RE. Serial extraction in preventive dentistry. *Clin Prev Dent* 1979;**1:**21–2.
32. Richard JP. Superior labial frenectomies in the child. *Pedod Fr* 1977;**11:**171–6.
33. Taylor PM, Mason RM. An orthodontist's perspective on the use of habit appliances. *Int J Orofacial Myology* 2002;**28:**3–4.

PERIODONTAL DISEASES

Periodontal diseases are one of the major causes of tooth loss in India. These include pathological conditions of the supporting structures of the teeth, i.e. gingiva, alveolar bone, periodontal ligament and cementum. Gingival and periodontal diseases affect 90% of the population. Gingival disease progresses to periodontal disease, if not checked in time.

Aetiology

Direct causes[1–6]

These include poor oral hygiene leading to accumulation of dental plaque and calculus, and traumatic occlusion.

Distant causes[19–25]

These include low socioeconomic and literacy level, difficult access to an oral health care facility, poor oral health awareness, and lack of oral health insurance. Stress is known to predispose to acute necrotizing ulcerative gingivitis.

Table 5. Causes of periodontal diseases

Direct	Indirect	Distant
• Poor oral hygiene resulting in accumulation of dental plaque and calculus • Traumatic occlusion	• Food impaction • Chewing and smoking of tobacco • Malnutrition—deficiency of vitamins A and C • Endocrine disturbances —physiological (puberty, pregnancy and the menopause) —pathological (hyperthyroidism, hyperparathyroidism and diabetes mellitus) • Decreased immunity—HIV infection, persons on immunosuppressive drugs • Blood disorders—anaemia, leukaemia • Idiopathic—gingival fibromatosis • Drug induced—phenytoin sodium, nifedipine, etc.	• Socioeconomic status • Literacy level • Access to oral health care facility • Oral health knowledge and awareness • Health insurance

Indirect factors[7–18]

- Malnutrition (deficiency of vitamins A and C, niacin and protein) is associated with a higher prevalence of periodontal diseases.
- Endocrine disturbances including physiological causes such as puberty, pregnancy, menopause, and pathological causes such as hyperthyroidism, hyperparathyroidism and diabetes may aggravate existing periodontal disease.
- Decreased immunity as in persons with HIV and those on immunosuppressive drugs.
- Blood disorders such as acute monocytic leukaemia and pernicious anaemia can lead to periodontal diseases.
- Malalignment of the teeth interferes with proper plaque control.
- Tobacco smoking and chewing reduce tissue resistance and increase the susceptibility to periodontal diseases.
- An improper brushing technique, besides resulting in inadequate plaque removal, can also cause gingival recession.
- Drugs—certain drugs such as phenytoin sodium and nifedipine can cause gingival hyperplasia.

The various causes of periodontal diseases are summarized in Table 5.

Prevention and treatment

These are the same as for dental caries.[26–38] Oral health education is required for the maintenance of oral hygiene (brushing, flossing, rinsing, etc.). The use of chemical mouthwashes (under prescription) and improved nutrition, as well as removal or treatment of aggravating factors are additional strategies. Interventions for the prevention and treatment of periodontal diseases are given in Table 6.

Table 6. Prevention and treatment of periodontal diseases

Medical interventions	Non-medical interventions	Other interventions
• Scaling and polishing of teeth • Oral and systemic antibiotics • Use of mouth washes • Gingival and periodontal surgery —gingivoplasty, gingivectomy, flap surgery, mucogingival surgeries, guided tissue regeneration, synthetic bone grafts, etc.	• Oral health education • Nutrition and diet • Proper methods of oral hygiene maintenance —use of toothpaste and tooth brush —use of inter-proximal cleaning devices such as interdental brushes, dental floss and water pik, etc. • Regular dental check-up	• Make oral health care more accessible and affordable • Improve the socioeconomic and literacy level of the population • Include oral health care in general health insurance

References

1. Lovegrove JM. Dental plaque revisited: Bacteria associated with periodontal disease. *J N Z Soc Periodontol* 2004;**87:**7–21.
2. Listgarten MA. Pathogenesis of periodontitis. *J Clin Periodontol* 1986;**13:**418–30.
3. Higgins TJ, Hunter N, Knox KW. Current concepts in periodontal diseases. *Med J Aust* 1985;**142:**590–4.
4. Overman PR. Biofilm: A new view of plaque. *J Contemp Dent Pract* 2000;**1:**18–29.
5. Genco RJ. Current view of risk factors for periodontal diseases. *J Periodontol* 1996; **67**(Suppl.):1041–9.
6. Checchi L, D'Achille C, Montella A. Tartar and periodontal disease—a cofactor in etiopathogenesis. *Dent Cadmos* 1991;**59:**80–4, 87–90, 93–5.
7. Verma S, Bhat KM. Diabetes mellitus—a modifier of periodontal disease expression. *J Int Acad Periodontol* 2004;**6:**13–20.
8. Genco RJ, Grossi SG. Is estrogen deficiency a risk factor for periodontal disease? *Compend Contin Educ Dent Suppl* 1998;**22:**S23–S29.
9. Genco R, Offenbacher S, Beck J. Periodontal disease and cardiovascular disease: Epidemiology and possible mechanisms. *J Am Dent Assoc* 2002;**133**(Suppl.):14S–22S.
10. Gera I. [Osteoporosis: A risk factor for periodontal disease (literature review).] *Fogorv Sz* 2002;**95:**49–54.
11. Kinane DF, Marshall GJ. Periodontal manifestations of systemic disease. *Aust Dent J* 2001;**46:**2–12.
12. Johnson GK, Slach NA. Impact of tobacco use on periodontal status. *J Dent Educ* 2001;**65:**313–21.
13. Slots J, Contreras A. Herpesviruses: A unifying causative factor in periodontitis? *Oral Microbiol Immunol* 2000;**15:**277–80.
14. Seymour RA, Ellis JS, Thomason JM. Risk factors for drug-induced gingival overgrowth. *J Clin Periodontol* 2000;**27:**217–23.
15. Hennig BJ, Parkhill JM, Chapple IL, Heasman PA, Taylor JJ. Association of a vitamin D receptor gene polymorphism with localized early-onset periodontal diseases. *J Periodontol* 1999;**70:**1032–8.
16. Enwonwu CO. Interface of malnutrition and periodontal diseases. *Am J Clin Nutr* 1995;**61:**430S–436S.
17. Turnbull B. Smoking and periodontal disease. A review. *J N Z Soc Periodontol* 1995;**79:**10–15.
18. Murray PA. HIV disease as a risk factor for periodontal disease. *Compendium* 1994;**15:**1052, 1054–63; quiz 1064.
19. Schenkein HA, Burmeister JA, Koertge TE, Brooks CN, Best AM, Moore LV, *et al.* The influence of race and gender on periodontal microflora. *J Periodontol* 1993;**64:**292–6.
20. Dougherty MA, Slots J. Periodontal diseases in young individuals. *J Calif Dent Assoc* 1993;**21:**55–69.
21. Genco RJ. Host responses in periodontal diseases: Current concepts. *J Periodontol* 1992;**63** (Suppl.):338–55.
22. Hung HC, Willett W, Ascherio A, Rosner BA, Rimm E, Joshipura KJ. Tooth loss and dietary intake. *J Am Dent Assoc* 2003;**134:** 1185–92.
23. Page RC. Current understanding of the aetiology and progression of periodontal disease. *Int Dent J* 1986;**36:**153–61.
24. Forrest JL, Miller SA. Manual versus powered toothbrushes: A summary of the Cochrane Oral Health Group's Systematic Review. Part II. *J Dent Hyg* 2004;**78:**349–54.
25. Borrell LN, Burt BA, Neighbors HW, Taylor GW. Social factors and periodontitis in an older population. *Am J Public Health* 2004;**94:**748–54.
26. Bsoul SA, Terezhalmy GT. Vitamin C in health and disease. *J Contemp Dent Pract* 2004;**5:**1–13.
27. Nield-Gehrig JS, Daniels AH. Improving awareness and dental care of diabetic patients. *Pract Proceed Aesthet Dent* 2004;**16:**85–7.
28. Deery C, Heanue M, Deacon S, Robinson PG, Walmsley AD, Worthington H, *et al.* The effectiveness of manual versus powered toothbrushes for dental health: A systematic review. *J Dent* 2004;**32:**197–211.
29. Glassman P, Miller CE. Preventing dental disease for people with special needs: The need for practical preventive protocols for use in community settings. *Spec Care Dentist* 2003;**23:**165–7.
30. Glassman P. Practical protocols for the prevention of dental disease in community settings for people with special needs: Preface. *Spec Care Dentist* 2003;**23:**157–9.
31. Kendall KH, Marshall RI. Antibiotics for periodontal therapy—where are we now and where are we going? Prophylaxis and systemic antibiotics. *Ann R Australas Coll Dent Surg* 2002;**16:**93–4.
32. Venezia E, Shapira L. Use of antimicrobial agents during supportive periodontal therapy. *Oral Dis* 2003;**9**(Suppl. 1):63–70.
33. Baehni PC, Takeuchi Y. Anti-plaque agents in the prevention of biofilm-associated oral diseases. *Oral Dis* 2003;**9**(Suppl. 1):23–9.
34. Haffajee AD, Arguello EI, Ximenez-Fyvie LA, Socransky SS. Controlling the plaque biofilm. *Int Dent J* 2003;**53**(Suppl. 3): 191–9.
35. Ower P. The role of self-administered plaque control in the management of periodontal diseases. 2: Motivation, techniques and assessment. *Dent Update* 2003;**30:**110–16.
36. Frentzen M, Ploenes K, Braun A. Clinical and microbiological effects of local chlorhexidine applications. *Int Dent J* 2002;**52:**325–9.
37. Hancock EB, Newell DH. The role of periodontal maintenance in dental practice. *J Indiana Dent Assoc* 2002;**81:**25–30.
38. Cobb CM. Clinical significance of non-surgical periodontal therapy: An evidence-based perspective of scaling and root planing. *J Clin Periodontol* 2002;**29**(Suppl. 2):6–16.

ORAL CANCER

India has the highest prevalence of oral cancer in the world (19/100,000 population). It is the most common cancer in men and the fourth most common cancer in women, and constitutes 13%–16% of all cancers. Of all the oral cancers, 95% are related to the use of tobacco.

Oral cancer has a high morbidity and mortality. The 5-year survival rate is 75% for local lesions but only 17% for those with distant metastasis. Therefore, early diagnosis of oral cancer is important. Since the oral cavity is easily accessible for examination and the cancer is always preceded by some pre-cancerous lesion or condition such as a white or red patch, an ulcer or restricted mouth opening, it is preventable to a great extent. Unfortunately, in India, most cancers are diagnosed at a very late stage, when treatment not only becomes more expensive, but the morbidity and mortality also increase.

Aetiology

Direct causes

- Tobacco—Many forms of tobacco are used in India—smoking (78%); chewing of betel quid, *paan masala*, *gutka*, etc. (19%); inhalation of snuff (2%); and dentifrices (>1%)

Table 7. Causes of oral cancer

Direct	Indirect	Distant
• Tobacco smoking/chewing • *Paan masala/gutka* chewing • Infections—HPV, HSV, AIDS, syphilis, candidiasis • Chronic irritation—faulty prosthesis, sharp teeth • Exposure to radiation	• Industrial pollution—asbestos, lead, leather and textile industries • Compromised immune status • Nutritional deficiencies (vitamins A and B complex, and zinc)	• Low socioeconomic and literacy level • Poor access to oral health care facilities for prevention and early detection • Poor oral health awareness

- Alcohol[6,7]
- Bacterial infections such as syphilis, and fungal (candidiasis) and viral (HPV, HSV, AIDS) infections[8–10]
- Chronic irritation due to sharp teeth and faulty prosthesis[11,12]
- Exposure to radiation[16,17]

Indirect causes

- Industrial pollution due to asbestos, lead[13–15]
- Nutritional deficiencies such as those due to vitamins A, B complex, and iron deficiency[18–19]

Distant causes

- Low socioeconomic and literacy level
- Poor oral health awareness
- Poor access to oral health care facilities for prevention and early detection

Table 7 lists the direct, indirect and distant causes of oral cancer.

Prevention and treatment

Strategies for prevention and treatment of oral cancer are summarized in Table 8.

References

1. Gupta PC. *Gutka*: A major new tobacco hazard in India. *Tob Control* 1999;**8:**134.
2. Dharkar D. Oral cancer in India: Need for fresh approaches. *Cancer Detect Prev* 1988;**11:**267–70.
3. Shiu MN, Chen TH. Impact of betel quid, tobacco and alcohol on three-stage disease natural history of oral leukoplakia and cancer: Implications for prevention of oral cancer. *Eur J Cancer Prev* 2004;**13:**39–45.
4. Gerson SJ. Oral cancer. *Crit Rev Oral Biol Med* 1990;**1:**153–66.
5. Maier H, Weidauer H. Alcohol drinking and tobacco smoking are the chief risk factors for ENT tumors. Increased incidence of mouth cavity, pharyngeal and laryngeal carcinomas. *Fortschr Med* 1995;**113:**157–60.
6. Kabat GC, Wynder EL. Type of alcoholic beverage and oral cancer. *Int J Cancer* 1989;**43:**190–4.
7. Weinstein RL, Francetti L, Maggiore E, Marchesi G. Alcohol and smoking. The risk factors for the oral cavity. *Minerva Stomatol* 1996;**45:**405–13.
8. Dickenson AJ, Currie WJ, Avery BS. Screening for syphilis in patients with carcinoma of the tongue. *Br J Oral Maxillofac Surg* 1995;**33:**319–20.
9. Iamaroon A, Pongsiriwet S, Mahanupab P, Kitikamthon R, Pintong J. Oral non-Hodgkin lymphomas: Studies of EBV and p53 expression. *Oral Dis* 2003;**9:**14–18.
10. Glick M, Muzyka BC, Lurie D, Salkin LM. Oral manifestations associated with HIV-related disease as markers for immune suppression and AIDS. *Oral Surg Oral Med Oral Pathol* 1994;**77:**344–9.
11. Llewellyn CD, Linklater K, Bell J, Johnson NW, Warnakulasuriya S. An analysis of risk factors for oral cancer in young people: A case–control study. *Oral Oncol* 2004;**40:**304–13.
12. Lockhart PB, Norris CM Jr, Pulliam C. Dental factors in the genesis of squamous cell carcinoma of the oral cavity. *Oral Oncol* 1998;**34:**133–9.
13. Mose E, Lee WR. Occurrence of oral and pharyngeal cancers in textile workers. *Br J Ind Med* 1974;**31:**224.
14. Varghese I, Rajendran R, Sugathan CK, Vijayakumar T. Prevalence of oral submucous fibrosis among the cashew workers of Kerala, South India. *Indian J Cancer* 1986;**23:**101–4.
15. Kennedy AR, Billings PC, Maki PA, Newberne P. Effects of various preparations of dietary protease inhibitors on oral carcinogenesis in hamsters induced by DMBA. *Nutr Cancer* 1993;**19:**191–200.
16. Pogoda JM, Preston-Martin S. Solar radiation, lip protection, and lip cancer risk in Los Angeles County women (California, United States). *Cancer Causes Control* 1996;**7:**458–63.
17. Elwood JM. Epidemiological studies of radiofrequency exposures and human cancer. *Bioelectromagnetics* 2003;Suppl. 6:S63–S73.
18. McLaughlin JK, Gridley G, Block G, Winn DM, Preston-Martin S, Schoenberg JB, *et al.* Dietary factors in oral and pharyngeal cancer. *J Natl Cancer Inst* 1988;**80:**1237–43.
19. La Vecchia C, Negri E, D'Avanzo B, Boyle P, Franceschi S. Dietary indicators of oral and pharyngeal cancer. *Int J Epidemiol* 1991;**20:**39–44.

Table 8. Prevention and treatment of oral cancer

Medical interventions	Non-medical interventions	Other interventions
• Biopsy of pre-malignant lesions • Surgery • Radiotherapy • Chemotherapy • Combination treatment	• Stop all oral abusive habits such as tobacco smoking and chewing • Improve oral hygiene • Remove all irritants from the mouth • Improve the nutritional status • Undergo regular oral check-up	• Self-examination of the oral cavity • Prevent initiation of harmful habits • Industrial safety legislation and protection of the health of workers

DENTAL FLUOROSIS[1–6]

Fluorine is a trace element which has a caries-preventive effect. The optimum level of fluorine in drinking water is 0.75–1 ppm. A fluoride content higher than 1 ppm is known to cause dental and skeletal fluorosis. Dental fluorosis is also known as 'mottled enamel'. It manifests as unsightly, chalky white or yellowish-brownish discoloration of the teeth, sometimes with structural defects in the enamel such as pitting of the surface. Table 9 lists the direct, indirect and distant causes of dental flourosis.

Fluoride toxicity depends upon several factors such as (i) the total quantity of ingested fluoride from all sources—water, food and drugs with a high fluoride content, (ii) climatic conditions of the region—in tropical countries such as India, water consumption can be high causing higher ingestion of fluoride-containing water, (iii) nutritional status of the individual—deficiency of vitamin D, calcium and phosphate can aggravate the manifestations of fluoride toxicity, (iv) presence of advanced kidney disease and hyperthyroidism are associated with manifestations of fluoride toxicity.

The prevention of dental fluorosis can be undertaken at three levels (Table 10).

References

1. Hodge HC. The concentration of fluoride in drinking water to give the point of minimum caries with maximum safety. *J Am Dent Assoc* 1950;**40:**436.
2. Dean HT, Jay P, Arnold FA, Elvove E. Domestic water and dental caries including certain epidemiological aspects of oral *L. acidophilus*. *Public Health Rep* 1939;**54:**862–88.
3. Beltran-Aguilar Ed, Goldstein JW, Lockwood SA. Fluoride varnishes—a review of their clinical use, cariostatic mechanism, efficacy and safety. *J Am Dent Assoc* 2000;**131:**589–96.
4. Svanberg M, Westergren G. Effect of SNF_2, administered as mouth rinses or topically applied, on *Streptococcus mutans*, *Streptococcus sanguis* and lactobacilli in dental plaque and saliva. *Scand J Dent Res* 1983;**91:**123.
5. Moudgil A, Srivastava RN, Vasudev A, Bagga A, Gupta A. Fluorosis with crippling skeletal deformities. *Indian Pediatr* 1986;**23:**767–73.
6. Susheela AK. Treatise on fluoride. Project report, sponsored by Task Force on Safe Drinking Water, Government of India. 2003.

Table 9. Causes of dental fluorosis

Direct	Indirect	Distant
• Exposure to high levels of fluorides: >1 ppm of fluoride in drinking water • Airborne fluoride from industrial pollution (aluminium factories, phosphate fertilizers, glass-manufacturing industries, ceramic and brick products) • Fluoride-rich dietary intake—sea food, poultry, grain and cereal products (especially sorghum), tea, rock salt, green leafy vegetables, etc.	• Tropical climate—excess ingestion of water and beverages with a high fluoride content • Presence of kidney diseases affecting the excretion of fluoride • Thyroid and thyrotrophic hormones have a synergistic effect on fluoride toxicity	• Poor nutritional status—deficiency of vitamin D, calcium and phosphates • Decreased bone phosphatase activity is linked to fluoride toxicity

Table 10. Strategies for the prevention of dental fluorosis

Primary prevention	Secondary prevention	Tertiary prevention
• Specific guidelines on the use and appropriate dose levels of fluoride supplements, and use of fluoride toothpaste for young children • In high fluoride areas —provide an alternate supply of drinking water —employ defluoridation techniques at the community or individual level	• Improve the nutritional status, especially of expecting mothers, newborns and children up to the age of 12 years. • Treat other causes of fluoride toxicity such as kidney and thyroid diseases, etc.	Treat the discoloured/disfigured dentition by appropriate aesthetic treatment such as bleaching, micro-abrasion, laminate veneers, etc.

Table 11. Equipment, minimum manpower required and approximate cost for medical interventions for oral and dental diseases

Medical interventions	Equipment/instruments required	Time required	Personnel	Set-up	In dental schools (in Rs)	In private clinics* (in Rs)
Dental check-up	Gloves, face mask, head light, mouth mirror, explorer, tweezers, cotton/ gauze, etc.	5 minutes	Dental surgeon	At all levels	Nil	100–300
ART restorations	All the above + set of 8–10 hand instruments, glass–ionomer cement type IX, vaseline, etc.	15 minutes/ filling	Dental surgeon/ health care workers/ dental hygienist (after adequate training; controversial)	At the PHC and community level	50/- per filling	250–500
Silver filling	Dental clinic set-up with micro-motor/air-rotor and inventory of cutting and filling instruments Cost of clinic set-up, excluding the place, is minimum 2.5 lakh	30 minutes/ filling	Dental surgeon	At the CHC level and upwards	100/- per filling	250–1000 (depending on simple or complex restoration)
Aesthetic fillings	As for silver filling + aesthetic restorative materials (composite resins, compomers, glass–ionomers) + light cure units	30 minutes/ filling	Dental surgeon	At district hospital and upwards	100/- per filling	400–1000
Indirect restorations (full crowns, inlays, veneers, etc.)	Dental clinic supported by well-equipped dental laboratory	Minimum 2 sittings of 30 minutes–1 hour each	Specialist dental surgeon/dental surgeon	At dental colleges, tertiary care hospitals and in private clinics	250/- per restoration	1500–2000
Root canal treatment	Dental clinic as in (3) Instruments for root canal treatment, sealants, gutta-percha, medicaments and irrigants	3–4 sittings of 30 minutes each	Specialist dental surgeon (endodontist)	At district hospital and above	150/-	1500–3000
Scaling and polishing of teeth	Hand scalers/ultrasonic scalers	2–3 sittings of 20 minutes each	Dental hygienist/ dental surgeon	At the CHC level and above	Nil or 50/-	800–1000
Surgical procedures (gingivectomy, flap operation, mucogingival surgery and endodontic surgery)	Dental surgery set-up as in (3) + all surgical instruments and retro-filling materials	45–60 minutes	Dental surgeon/ specialist dental surgeon (periodontist, endodontist, oral surgeon)	At district hospital and above	100/-	1500–5000
Orthodontics—removable	Dental surgery clinic set-up	6–12 months	Orthodontist/ dental surgeon	At district hospital and above	200/- per appliance	2500–3000 per appliance
Orthodontics—fixed appliances	Dental surgery clinic set-up with extraoral radiographic facility and inventory of all orthodontic instruments and supply of brackets, arch wires, elastics, etc.	One year to two-and-a-half years	Orthodontist	At dental schools, tertiary care hospitals and private clinics	2000–3000	15,000–30,000
Complete dentures	Dental surgery clinic set-up supported by a dental laboratory	5–7 sittings at intervals of 2–7 days	Specialist dental surgeon/dental surgeon	At CHC level and upwards	350–500	5000–10,000
Partial denture (removable)	Dental surgery clinic set-up supported by a dental laboratory	3–4 sittings	Dental surgeon	At CHC and upwards	100/- 25/- per additional tooth	300–1000
Partial denture (fixed)	Dental surgery clinic set-up supported by a dental laboratory	3–4 sittings	Specialist dental surgeon (prosthodontist)	At dental schools, tertiary care hospitals and private clinics	250/- per unit	2000 per unit

(Cont.)

Table 11 *(cont.).* Equipment, minimum manpower required and approximate cost for medical interventions for oral and dental diseases

Medical interventions	Equipment/instruments required	Time required	Personnel	Set-up	In dental schools (in Rs)	In private clinics* (in Rs)
Biopsy	Dental surgery set-up	15–30 minutes	Dental surgeon	At the CHC level and upwards	Nil	500–1000
Surgical extraction (impaction)	Dental surgery set-up + all surgical instruments and retro-filling materials	1 hour	Oral surgeon/dental surgeon	At district hospital and above	100	2000–3000
Fracture reduction/ cyst enucleation/ benign growth excision	Dental surgery set-up as for silver filling + all surgical instruments and retro-filling materials	1 hour	Oral surgeon/dental surgeon	At district hospital and above	Nil	5000–8000

PHC: primary health centre; CHC: community health centre; ART: atraumatic restorative treatment
*These rates are common in Delhi; may vary from State to State.

EPIDEMIOLOGY OF ORAL AND DENTAL DISEASES

Oral and dental diseases are widely prevalent in India. Though not life-threatening, these diseases are often very painful, expensive to treat and cause loss of several man-days. On the other hand, they are, to a great extent, preventable. It has now been recognized that oral and general health are closely interlinked. Periodontal (gum) diseases are found to be closely associated with several serious systemic illnesses such as cardiovascular and pulmonary diseases, stroke, low birth-weight babies and preterm labour. Besides, poor oral health affects the functions of mastication and speech, and ultimately the overall well-being of an individual.

The major oral and dental diseases/disorders are (i) dental caries, (ii) periodontal diseases, (iii) dentofacial anomalies and malocclusion, (iv) edentulousness (tooth loss), (v) oral cancer, (vi) maxillofacial and dental injuries, and (vii) fluorosis.

Dental caries

Dental caries is a universal disease affecting all geographic regions, races, both the sexes and all age groups. The prevalence of dental caries is generally estimated at the ages of 5, 12, 15, 35–44 and 65–74 years for global monitoring of trends and international comparisons. The prevalence is expressed in terms of point prevalence (percentage of population affected at any given point in time) as well as DMFT index (number of decayed, missing and filled teeth in an individual and in a population).

As per the WHO Oral Health Surveillance 1992, the DMFT index in 12-year-old Indian is 0.89. In India, different investigators have studied various age groups, which can be broadly classified as below 12 years, above 12 years, above 30 years and above 60 years (Tables 12–15). Based on the analysis of all these tables, the prevalence of dental caries in urban and rural populations at various specified age groups has been calculated (Table 16).

Table 12. Incidence of caries in the age group of less than 12 years

Investigator and year	Index used	State	Place	Sample size	Point prevalence	Mean DMFT
Shourie 1941	Day and Sadwick 1934	Delhi	Delhi (Urban)	69	50.8	2.83
			Delhi (Rural)	54	31.5	1.0
Chopra *et al.* 1985	WHO 1987		Delhi (Urban)	381	34.1	1.14
Gautam *et al.* 2001	WHO 1997		Delhi (Urban)	2366	35.12	1.18
Shourie 1947	Day and Sadwick 1934	Rajasthan	Ajmer (Urban)	178	50.0	2.1
Thapar 1989	Mollers (1966)		Jaipur	?	31.4	0.5
Sehgal 1960		Maharashtra	Bombay	69	39.36	5.9
Anita 1962			Bombay	504		6.64
Tewari *et al.* 1985	WHO 1983		Bombay	220	89.0	5.3
Damle and Patel 1993	WHO 1983		Fishermen community around Mumbai (11–15 years)	431	61.5	1.9

(Cont.)

Table 12 *(cont.).* Incidence of caries in the age group of less than 12 years

Investigator and year	Index used	State	Place	Sample size	Point prevalence	Mean DMFT
Gaikwad 1993			Aurangabad	1995 (5–14 years)	57.89 (M) 45.2 (F)	0.55
Dutta 1965		West Bengal	Dumdum	180	67.1	2.96
Sarkar and Chowdhary 1992	WHO 1971			40	0.0 (1 year)	
				40	13.2 (3 years)	
				50	25.5 (4 years)	
				50	? (5 years)	
Chowdhary 1967		Uttar Pradesh	Lucknow (Urban)	107	32.7	
Gill and Prasad 1968			Lucknow (Rural)	138	44.0	1.1
Kavita *et al.* 1987	WHO 1983		Dehradun (Urban)		54.7	2.1
			Meerut (Urban)		57.4	1.9
			Lucknow (Urban)		89.0	4.4
			Banaras (Urban)		53.0	1.3
Kavita *et al.* 1987	WHO 1983		Dehradun (Rural)		42.4	1.2
			Meerut (Rural)		50.0	1.4
			Lucknow (Rural)		63.6	1.7
			Banaras (Rural)		54.0	1.5
Mishra and Shee 1979		Orissa	Behrampur		56.6	
Sahoo *et al.* 1986	WHO 1983		Orissa (Urban)	170	58.82	2.52
			Orissa (Rural)	160	57.5	2.66
Damle *et al.* 1982	Mollers 1966	Haryana	Haryana (Rural)	123	74.0	3.3
Gathwala *et al.* 1993	1993		Rohtak	501 (5–13 years)		?
Tiwari 1999	WHO 1987		Haryana	113	36.3 (5 years)	0.87
				157	38.2 (6 years)	0.9
Thapar 1953	?	Punjab	Moga	70	47.7	?
Chopra *et al.* 1983	WHO 1962		Punjab (Urban)	141	61.1	1.72
Chopra *et al.* 1985	WHO 1987		Jalandhar (Urban)	?	46.8	1.5
			Jalandhar (Rural)	151	39.7	1.0
			Abohar (Urban)	145	27.6	0.6
			Abohar (Rural)	150	24.7	0.6
Norboo *et al.* 1998	WHO 1987	Jammu and Kashmir	Leh (Urban)	62	74.6	4.3
			Leh (Rural)	72	63.9	2.3
			Kargil (Urban)	63	70.7	2.9
			Kargil (Rural)	71	63.4	2.2
Tewari *et al.* 1985	WHO 1983	Union Territory	Chandigarh (Urban)	204	59.0	2.26
			Chandigarh (Rural)	197	60.0	2.21
Chawla *et al.* 1993	WHO 1983		Chandigarh (Urban)	?	?	1.2
Goyal *et al.* 1997	WHO 1983		Chandigarh (Rural)	135	1.5 (1 year)	
				144	7.0 (2 years)	2.0
				154	19.4 (3 years)	2.35
				137	28.5 (4 years)	
				95	1.0 (1 year)	
				128	12.0 (2 years)	
				120	23.0 (3 years)	
				11	32.0 (4 years)	
				124	48.0 (5 years)	
Tewari *et al.* 1985	WHO 1983	Bihar	Bihar (Urban)	212	54.0	1.5
			Bihar (Rural)	99	35.0	1.1
Tewari and Mandal 1985	WHO 1983	Madhya Pradesh	Indore (Urban)	147	52.4	2.3

(Cont.)

Table 12 *(cont.).* Incidence of caries in the age group of less than 12 years

Investigator and year	Index used	State	Place	Sample size	Point prevalence	Mean DMFT
Virjee Shankar Aradhya 1987	Johnsen *et al.* 1984	Karnataka	Bangalore (Urban)	673	66.3 (4.5 years)	2.9
			Chickballapur (Rural)	394	58.4 (4.5 years)	2.3
Gupta *et al.* 1987	WHO 1983		Davengere (Rural)	100	25.0	0.6
			Davengere (Urban)	100	53.0	1.68
			Bangalore (Urban)	100	70.0	1.66
Sethi and Tandon 1996	William 1994		Udupi	404	65.5 (3–5years)	
Menon and Indushekhar 1999	WHO 1987		Dharwad	624	2.56	0.03
			Gadag	256	1.17	0.01
Rao *et al.* 1999	WHO 1987		Modbidri	550	75.3	0.2
Sharma *et al.* 1988	WHO 1983	North-east	Shillong	180	88.33	6.36
			Imphal	199	88.44	5.53
			Guwahati	199	80.90	5.35
			Kohima	198	90.40	6.4
Mandal *et al.* 1994	WHO 1983		Sikkim (Urban)	10	61.8	2.50
			Sikkim (Rural)	109	22.02	0.70
Mandal *et al.* 1994	WHO 1983		W. Bengal (Urban)	124	52.42	1.86
			W. Bengal (Rural)	20	48.33	1.48
Gupta *et al.* 1987	WHO 1983	Andhra Pradesh	Hyderabad (Rural)	187	50.8	1.63
Gupta *et al.* 1987	WHO 1983	Kerala	Calicut (Urban)	156	56.41	2.1
			Trivandrum (Urban)	103	51.46	1.81
Kuriarose and Joseph 1999	WHO		Trivandrum	600	57	2.28
Gopinath *et al.* 1999	WHO 1987	Tamil Nadu	Tamil Nadu	97	36.0	36 (M) 17 (F)

DMFT: number of decayed, missing and filled teeth

Table 13. Incidence of dental caries in children above 12 years of age

Investigator and year	Index used	State	Place	Sample size	Point prevalence	Mean DMFT
Shourie 1941	Day and Sadwick 1934	Delhi	Delhi (Urban)	95 (12 years)	54.8	5.7
Shourie 1941	Day and Sadwick 1934		Delhi (Urban)	19	52.7	1.2
			Delhi (Rural)	40 (15 years)	42.5	1.1
Gupta *et al.* 1993	WHO 1983		New Delhi	(12 years)	87.0	0.86
Chopra *et al.* 1995	WHO 1987		Delhi (Urban)	392 (15 years)	20.9	0.42
Shourie 1942	Day and Sadwick 1934	Tamil Nadu	Tamil Nadu (Urban)	42	57	2.0
Gopinath *et al.* 1999	WHO 1987		Tamil Nadu	232 (12 years)	61.2	3.2 (M) 3.7 (F)
Shourie 1947	Day and Sadwick 1934	Rajasthan	Ajmer (Urban)	(15 years)	56.3	
Thapar *et al.* 1989	Moller 1966		Rajasthan (Rural)	(12 years)	31.4	0.5
Chaudhary *et al.* 1957	Own criteria	Uttar Pradesh	Lucknow	368 (12 years)	32.0	1.15
Chaudhary *et al.* 1957	Own criteria		Lucknow	107 (5 years)	32.7	
Gill *et al.* 1968	WHO 1962		Lucknow (Urban)	99 (12 years)	99.0	43.3
Gill *et al.* 1968	WHO 1962		Lucknow (Urban)	23 (15 years)	66.8	0.7
Mehta *et al.* 1987	WHO 1983		Dehradun (Urban)	202 (15 years)	45.0	1.0
			Meerut (Urban)		42.0	1.1
			Lucknow (Urban)		42.6	1.0
			Banaras (Urban)		38.4	1.0
Mehta *et al.* 1987	WHO 1983		Dehradun (Rural)	112 (15 years)	38.2	0.8
			Meerut (Rural)		38.4	0.8
			Lucknow (Rural)		20.5	0.4
			Banaras (Rural)		41.0	0.8
Singh *et al.* 1999	WHO 1987		Faridabad (Rural)	233 (12 years)	33.1	0.79

(Cont.)

Table 13 *(cont.)*. Incidence of dental caries in children above 12 years of age

Investigator and year	Index used	State	Place	Sample size	Point prevalence	Mean DMFT
Singh *et al.* 1999	WHO 1987		Faridabad (Rural)	207 (15 years)	42.5	1.29
Anita 1962		Maharashtra	Bombay	503 (15 years)		2.5
Damle *et al.* 1982	Moller 1966		Naraingarh (Rural)	230 (15 years)	77.2	2.4
Damle and Patel 1984	WHO 1983		Bombay	(15 years)	78.0	3.6
Tiwari *et al.* 1985	WHO 1983		Bombay (Urban)	202 (15 years)	96.0	4.7
Damle and Ghonmode 1993	WHO 1983		Nagpur	(12 years)	82.6	4.0
Damle and Ghonmode 1993	WHO 1983		Nagpur	(15 years)	82.6	4.0
1993?			Nagpur	1811 (12–18 years)	81.3	>3
1994?	WHO 1983		Bombay (Urban)	367 (12 years)	80.0	3.8
Rodrigues and Damle 1998			Mumbai		68.02	
Ali *et al.* 1998	WHO 1987		Akola	508 (5–6 years)	61.4% 2.75+3.98	
Rodrigues and Damle 1998	WHO 1997		Bhiwandi	256 (12 years)	55.5	1.08
Tiwari and Chawla 1977	WHO 1971	Uttaranchal	Chandigarh (Urban)	82 (15 years)	86.6	4.7
Tiwari *et al.* 1983	WHO 1966		Chandigarh			
			(Urban)	217	51.1	1.38
			(Rural)	205 (15 years)	47.5	1.30
Chawla *et al.* 1993	WHO 1983		Chandigarh	(12 years)		1.2
Damle *et al.* 1982	Moller 1966	Haryana	Haryana (Rural)	152 (12 years)	89.5	3.2
Tiwari *et al.* 1985	WHO 1983		Haryana (Urban)	229	50.0	1.35
			Haryana (Rural)	200 (15 years)	47.5	1.30
Sharma *et al.* 1998	WHO		Haryana District (Gurgaon and Mahendragarh)	3031 (12–16 years)	36.7	0.67
Gauba *et al.* 1983	Moller 1966	Punjab	Punjab (Rural)	173 (12 years)	86.1	3.9
Gauba *et al.* 1983	Moller 1966		Ludhiana (Rural)	101 (15 years)	88.1	5.0
Chopra *et al.* 1983	WHO 1962		Punjab (Urban)	255 (12 years)	67.2	1.3
Chopra *et al.* 1995	WHO 1987		Jalandhar (Urban)	150	42.0	0.9
			Jalandhar (Rural)	146	24.7	0.46
			Abohar (Urban)	46	21.0	0.43
			Abohar (Rural)	46	24.0	0.43
Mishra and Shee 1985		Orissa	Orissa	(12 years)	61.1	
Tiwari *et al.* 1985	WHO 1983		Orissa (Rural)	174	63.8	2.1
			Orissa (Urban)	159 (12 years)	63.1	2.1
Sahoo *et al.* 1986	WHO 1983		Orissa Urban)	(12 years)	63.8	2.1
			Orissa (Rural)		67.9	2.0
Sahoo *et al.* 1986	WHO 1983		Orissa (Urban)	175 (15 years)	62.3	2.0
Mandal *et al.* 1994	WHO 1987		Orissa (Rural)	121 (15–16 years)	19.8	0.3
Mandal *et al.* 1994	WHO 1987		Bhubaneshwar (Urban)	120	18.3	0.3
Mandal *et al.* 2001	WHO 1983		Orissa (Urban)	702		
			Orissa (Rural)	351	56.0	
				351	48.7	
Tiwari *et al.* 1985	WHO 1983	Himachal Pradesh	Himachal (Urban)	178	50.0	1.2
			Himachal (Rural)	191 (15 years)	49.0	1.3
Tiwari *et al.* 1985	WHO 1983	Bihar	Bihar (Urban)	160	42.5	1.2
			Bihar (Rural)	202 (15 years)	49.5	1.3
Tiwari and Mandal 1985	WHO 1983	Madhya Pradesh	Indore (Urban)	162 (15 years)	68.0	2.8
Sharma *et al.* 1988	WHO 1983	North-east	Shillong (Urban)	183	60.1	2.1
			Imphal (Urban)	197	63.45	1.76
			Guwahati (Urban)	200	83.5	3.13
			Kohima and Mokokochung (Urban)	195 (15 years)	63.08	2.36
Mandal *et al.* 1994	WHO 1987		Gangtok (Urban)	106 (15 years)	30.2	0.5

(Cont.)

Table 13 *(cont.).* Incidence of dental caries in children above 12 years of age

Investigator and year	Index used	State	Place	Sample size	Point prevalence	Mean DMFT
Mandal *et al.* 1994	WHO 1987		Sikkim (Rural)	106 (15–16 years)	17.9	0.3
Mandal *et al.* 2001	WHO 1983		Sikkim	644		
			(Urban)	323	61.8	
			(Rural)	321	22.0	
Mandal *et al.* 1994	WHO 1987	West Bengal	Calcutta (Urban)	119	21.0	0.3
Mandal *et al.* 1994	WHO 1987		West Bengal (Rural)	118	15.2	0.3
Mandal *et al.* 2001	WHO 1983		West Bengal	720	52.4	5.6
			(Urban)	361	48.3	
			(Rural)	359		
Norboo *et al.* 1998	WHO 1987	Jammu and	Leh (Rural)	74	43.2	0.87
		Kashmir	Kargil (Rural)	69 (12 years)	29.0	0.68
Norboo *et al.* 1998	WHO 1987		Leh (Urban)	65	47.7	1.01
			Kargil (Urban)	73 (12 years)	35.8	0.63
Norboo *et al.* 1998	WHO 1987		Leh (Urban)	70	60.0	1.01
			Leh (Rural)	60	45.0	1.15
			Kargil (Urban)	79	47.5	1.2
			Kargil (Rural)	69 (15 years)	39.7	1.0
Nagaraga Rao 1980		Karanataka	Udupi	(2 years)		4.1
Gupta *et al.* 1987	WHO 1983		Davangere (Rural)	98 (15 years)	42.86	1.07
Menon and Indushekhar 1999	WHO 1987		Dharwad	300	31.0	0.78
			Gadag	488 (12 years)	24.6	0.6
Rao *et al.* 1999	WHO 1987		Moodbidri (Urban)	771 (12 years)	67.1	1.29
Menon and Indushekhar 1999	WHO 1987		Dharwad	106	55.7	1.09
			Gadag	127 (15 years)	30.3	0.75
Sogi and Bhasker 2001			Davangere			3.12
Javali and Prasad 2001	WHO		Karnataka	8152 (12–17 years)	43.5 (M) 50.5 (F)	1.57
Kulkarni and Deshpande 2002	WHO 1987		Belgaum	2005 (11–15 years)	45.12%	1.18
Gupta *et al.* 1987	WHO 1983	Andhra Pradesh	Hyderabad	85 (15 years)	34.12	0.96
Retnakumari 2000	WHO 1997		Varkala	119 (12 years)	67.2	2.067
Goel *et al.* 2000	WHO 1987		Puttur	203 (12 years)	59.6	1.87

DMFT: number of decayed, missing and filled teeth

Table 14. Incidence of dental caries in the age group of above 30 years

Investigator and year	Index used	State	Place	Sample size	Point prevalence	Mean DMFT
Barreto *et al.* 1953		Maharashtra	Bombay	331		1.50
Mangi and Jalili 1967		Madhya Pradesh	Madhya Pradesh	331		4.10
Ramachandran *et al.* 1973		Tamil Nadu	Tamil Nadu (Urban)	NA		2.88
			Tamil Nadu (Rural)			2.10
Damle *et al.* 1982	Mollers 1966	Haryana	Haryana (Rural)	667	61	1.70
Tewari *et al.* 1985	WHO 1983		Haryana (Urban)	101	46.5	1.5
			Haryana (Rural)	200	68.0	3.04
Tewari *et al.* 1985	WHO 1983	Uttaranchal	Chandigarh (Urban)	156	81.4	4.38
			Chandigarh (Rural)	196	82.1	4.38
Tewari *et al.* 1985	WHO 1983	Uttar Pradesh	Lucknow (Urban)	199	47.0	1.13
			Lucknow (Rural)	118	45.8	1.22
Tewari *et al.* 1985	WHO 1983	Jammu and	J&K (Urban)	NA		4.9
		Kashmir	J&K (Rural)	NA		5.8
Chopra *et al.* 1985	WHO 1987	Punjab	Jalandhar (Urban)	144	34.72	1.08

(Cont.)

Table 14 *(cont.).* Incidence of dental caries in the age group of above 30 years

Investigator and year	Index used	State	Place	Sample size	Point prevalence	Mean DMFT
			Jalandhar (Rural)	145	30.34	0.76
			Abohar (Urban)	140	20.0	0.42
			Abohar (Rural)	149	24.16	0.41
Sharma *et al.* 1985	WHO 1983	North-east	Meghalaya (Urban)	196	54.6	1.18
			Manipur (Urban)	199	63.82	1.86
			Assam (Urban)	244	66.0	1.86
			Nagaland (Urban)	202	62.4	2.13
Mandal *et al.* 1994	WHO 1983		Sikkim (Urban)	107	29.91	0.62
			Sikkim (Rural)	107	24.53	0.60
Tewari and Mandal 1985	WHO 1983	Madhya Pradesh	Indore	66	70.0	3.80
Tewari and Damle 1985	WHO 1983			201	80.0	3.57
Gupta *et al.* 1985	WHO 1983	Kerala	Trivandrum (Urban)	103	79.61	2.21
			Calicut (Urban)	104	78.9	2.16
			Calicut (Rural)	90	47.8	1.2
Gupta *et al.* 1985	WHO 1983	Andhra Pradesh	Hyderabad (Urban)	111	64.86	2.16
			Hyderabad (Rural)	87	44.83	1.16
Gupta *et al.* 1985	WHO 1983	Karnataka	Bangalore (Urban)	98	73.47	2.17
			Davengere (Urban)	102	68.63	2.29
			Davengere (Rural)	102	48.04	1.07
Mandal *et al.* 1994	WHO 1987	Orissa	Orissa (Urban)	5	24.35	0.47
			Orissa (Rural)	114	20.17	0.48
Mandal *et al.* 1994	WHO 1987	West Bengal	West Bengal (Urban)	18	19.49	0.47
			West Bengal (Rural)	20	18.18	0.40
Chopra *et al.* 1995	WHO 1987	Delhi	Delhi (Urban)	388	24.5	0.50
Tewari *et al.* 1995	WHO 1983	Bihar	Bihar (Urban)	149	69	1.75
			Bihar (Rural)	193	63.2	1.85

DMFT: number of decayed, missing and filled teeth; NA: not available

Table 15. Incidence of dental caries in those above 60 years of age

Year	State	Place	Index used	Sample size	Point prevalence	Mean DMFT
1994	Karnataka			300		13.51
2004	Delhi	New Delhi	WHO 1987	1052	72.4	—

DMFT: number of decayed, missing and filled teeth

Table 16. Prevalence of dental caries in different age groups

Age group (years)	Urban	Rural	Average	DMFT
5–6	67.23	46.22	56.72	2.1
12	57.94	36.90	47.39	1.6
15	55.97	43.28	49.59	1.37
30–35	45.21	39.27	42.24	1.39
60–75	79.40	61.90	70.65	—

DMFT: number of decayed, missing and filled teeth

Periodontal diseases

Periodontal diseases affect the supporting structures of teeth, i.e. the gingiva (gums), periodontal ligament, alveolar bone and cementum (covering the roots of the teeth) and are the commonest cause of tooth loss in India. A thin, adherent microbial film on the tooth surfaces, called dental plaque, is the main pathological cause of gingival and periodontal inflammation. Poor oral hygiene, faulty food habits, poor nutrition, presence of metabolic diseases such as diabetes, use of tobacco, etc. are the major contributory factors for periodontal diseases.

Periodontal diseases are common in the adult population, but not very common in children. Several indices are used to measure periodontal diseases, such as plaque index, oral hygiene index, bleeding index, community periodontal index (CPI), etc. A scoring system to score the gradation from mild to severe forms of the disease is also available. Therefore, there is no uniformity in data on the prevalence of periodontal diseases and hence, it is difficult to compare the data. However, it is widely accepted that periodontal

diseases affect over 90% of the Indian population, but the majority of them may have only mild gingivitis and bleeding from the gums, which is reversible with proper oral hygiene measures. More advanced periodontal disease with pocket formation and bone loss, which could ultimately lead to tooth loss if not treated properly, may affect 40%–45% of the population. It is also known that use of tobacco, especially habitual chewing of tobacco, presence of metabolic diseases such as diabetes, nutritional deficiencies, compromised immune status and increasing age are associated with an increase in periodontal diseases.

Table 17 documents only some studies, and highlights totally incoherent data. Moreover, most of the studies have been conducted on the child population, in whom periodontal diseases are not widely prevalent.

Table 17. Periodontal diseases

Investigator and year	State	Place	Index	Sample size	Prevalence
Anuradha *et al.* 2002	Karnataka	Davangere	CPI and plaque index	NA	Decrease with increase in the fluoride content of water
Sogi and Bhasker 2001		Davangere	Oral hygiene index (OHI)	2007 (13–14 years)	NA
Doifode *et al.* 2000	Maharashtra	Nagpur		5061 (all age groups)	Periodontal diseases 34.8% total <15 years 18.4% 15–30 years 36.4% 30–60 years 50.2% 60+ years 54.4%
Rao and Bharambe 1993		Wardha		778 (5–12 years) (Rural) (Urban)	17.8% 22.6% 10.5% 15.0%
Gathwala 1993	Haryana	Rohtak		501 (5–13 years)	36.3% (gingivitis)
Rao and Bharambe 1993	Maharashtra	Wardha		778 (5–12 years)	4.8% (bleeding/abscess)
Shah 2003	Delhi	South Delhi	CPI index	1052 (above 60 years)	100% mild 9.1% moderate 19% severe 71.9%

CPI: community periodontal index

Dentofacial anomalies and malocclusion

The prevalence of malocclusion in India is estimated to be 30% in school-age children (Table 18). Malocclusion may vary from mild to severe, causing aesthetic and functional problems, and may also predispose to dental caries, periodontal diseases as well as increased susceptibility to trauma, especially to excessively proclined teeth. The major dentofacial deformity is cleft lip and palate, which is seen in 1.7/1000 live-births (Table 19).

Table 18. Prevalence of dentofacial anomalies and malocclusion

Author and year	State	Place	Age group (years)	Prevalence (%)
Shourie 1952	Punjab	Punjab	13–16	50
Guaba *et al.* 1998		Ambala	6–15	29.2
Shaik and Desai 1966	Tamil Nadu	Madras	15–25	19.6
Jacob 1969	Kerala	Trivandrum	12–15	44.97
Jose and Joseph 2003		Kerala	12–15	NA
Prasad and Savadi 1971	Karnataka	Bangalore	5–15	51.5
Nagaraja Rao 1980		Udupi	5–15	28.8
Gardiner 1989		South Kanara	10–12	42
Jalili 1989	Madhya Pradesh	Mandu (Tribal area)	6–14	14.4
Kharbanda 1991	Delhi	Delhi	5–13	10–18
Kharbanda 1995		Delhi	10–13	45.7
Goel *et al.* 2000	Andhra Pradesh	Puttur	5–6	1.79
			12–13	36.95

Table 19. Incidence of cleft lip and cleft palate in India (hospital-based studies)

Location of the hospital	Incidence (%)	
	Cleft lip	Cleft lip and palate
Delhi	2.21	0.71
Delhi (AIIMS)	1.40	0.30
Chandigarh	1.0	—
Jaipur	1.12	0.35
Patiala	1.5	—
Lucknow	1.09	—
Ajmer	0.90	—
Mumbai	1.30	0.20
Ahmedabad	1.06	0.24
Chennai	1.60	0.10
Kolkata	0.63	0.16
Hyderabad	1.90	1.90

AIIMS: All India Institute of Medical Sciences

Edentulousness (tooth loss)

Tooth loss results from dental caries, periodontal diseases and trauma. Tooth loss increases with advancing age (Table 20). Loss of the teeth results in decreased masticatory efficiency, causing a shift in dietary practices. This may result in nutritional deficiencies. Tooth loss may also cause problems in speech and affect aesthetics, causing an overall loss of self-esteem and confidence. Very little data are available on tooth loss.

Table 20. Tooth loss (edentulousness)

Age group (years)	Number of missing teeth	Edentulousness (%)
60–64	8.5	11.1
65–74	10.9	19.4
75+	18.1	32.3

Dental fluorosis

In India, a high fluoride content in ground water is endemic in some areas. The states that are most affected are Andhra Pradesh, Gujarat and Rajasthan. Table 21 shows the distribution of fluoride in different states. It has been estimated that about 666.2 lakh people are at risk for fluoride toxicity of which children below the age of 14 years constitute 60 lakh.

Data available from a field survey in Gujarat, Haryana and Delhi are presented in Tables 22, 23 and 24, respectively.

Table 21. Distribution of fluoride analysis of ground water samples from different States of India

States		Number of water samples	Fluoride <1.0 mg/L	Fluoride 1.0–1.5 mg/L	Fluoride >1.5 mg/L	Maximum fluoride value (mg/L)
Uttar Pradesh	No.	502	398	62	42	15.0 (Marksnagar, Unnao district)
	%		79.2	12.4	8.4	
Andhra Pradesh	No.	786	752	19	15	7.90 (Nalgonda district)
	%		95.7	2.4	1.9	
Rajasthan	No.	780	403	114	263	22.0 (Nagaur district)
	%		51.7	14.6	33.7	
Maharashtra	No.	161	156	—	5	5.0 (Chandrapur district)
	%		96.9	—	3.1	
Madhya Pradesh (West)	No.	749	678	51	20	4.5 (Sirohi, Bhind district)
	%		90.5	6.8	2.7	
Karnataka	No.	773	634	91	48	8.3 (Kulgeri, Bijapur district)
	%		82.0	11.8	6.2	
Chandigarh	No.	1	—	1	—	—
	%		—	100	—	
Punjab	No.	332	232	46	54	11.7 (Bathinda district)
	%		69.9	13.9	16.2	
Haryana	No.	306	134	48	124	21.0 (Hissar district)
	%		43.8	15.7	40.5	
Delhi	No.	38	31	4	3	3.25 (Palam)
	%		81.6	10.5	7.9	
Orissa	No.	83	69	5	9	11.0 (Balasore and Bolangir district)
	%		83.1	6.0	10.8	
Bihar	No.	328	313	5	10	4.2
	%		95.4	1.5	3.1	

(Cont.)

Table 21 *(cont.).* Distribution of fluoride analysis of ground water samples from different States of India

States		Number of water samples	Fluoride <1.0 mg/L	Fluoride 1.0–1.5 mg/L	Fluoride >1.5 mg/L	Maximum fluoride value (mg/L)
Tamil Nadu	No.	464	398	53	213	6.8 (Madurai district)
	%		85.8	11.4	2.8	
Gujarat	No.	589	554	15	20	11.0 (Amreli district)
	%		94.1	2.5	3.4	
West Bengal	No.	466	454	—	12	16.0 (Birbhum district)
	%		97.4	—	2.6	
Kerala	No.	676	669	3	4	4.6 (Konnakuzhill district, Trichur)
	%		99.0	0.4	0.6	
Madhya Pradesh (East)	No.	346	340	—	6	—
	%		98.3	—	1.7	
Jammu and Kashmir	No.	117	117	4	1	0.78 (Dablehar)
	%		100	—	—	
Himachal Pradesh	No.	79	74	4	1	9.5 (Dhaulakuwan district)
	%		93.7	5.0	1.3	
States in the North-east	No.	295	295	—	—	0.5 (Darang district)
	%		100	—	—	
Total	No.	7871	6701	521	649	—

Source: Ground Water Authority, India

Table 22. Dental fluoride survey in schoolchildren from 18 districts in Gujarat

District	No. of schools surveyed	No. of students examined in the schools (8 years and above)			No. of students with dental fluorosis	Percentage affected with fluorosed teeth
		Boys	Girls	Total		
Ahmedabad	199	27,947	20,123	48,070	8,537	17.75
Gandhinagar	29	4,436	4,023	8,459	967	11.43
Mehsana	415	62,322	38,912	101,234	25,307	24.90
Banaskantha	367	36,463	20,925	57,388	10,032	17.78
Sabarkantha	278	21,000	18,405	39,405	5,728	14.50
Baroda	240	13,826	11,825	25,651	4,329	16.87
Kheda	210	24,064	19,219	43,283	5,266	12.16
Panchmahal	311	34,603	25,729	60,332	5,207	8.40
Bharuch	42	4,781	4,459	9,240	1,378	14.90
Surat	19	1,697	1,581	3,278	260	7.90
Valsad	14	1,939	1,889	3,828	101	2.60
Junagadh	50	7,075	5,314	12,389	4,097	33.00
Amreli	75	9,159	7,975	17,134	2,855	16.60
Surendranagar	71	7,442	6,010	13,452	2,961	22.00
Jamnagar	28	3,070	2,316	5,386	838	15.50
Bhavnagar	77	10,667	8,472	19,139	2,714	14.10
Rajkot	44	6,065	7,320	13,385	1,971	14.70
Kutch	13	1,599	1,561	3,160	640	20.25
Total	2482	278,155	206,058	484,213	83,188	% range: 2.6–33.0

Source: Gujarat Health Department, 1996–97 (From: Susheela AK. *Treatise on fluoride*. Project report. Sponsored by the Task Force on Safe Drinking Water, Government of India, 2003)

Table 23. Incidence of dental fluorosis in two villages in Haryana

Village	Drinking water fluoride level (mg/L)	Incidence of dental fluorosis (%)
Sotai	1.89–3.83	77
Machgar	0.64	13

Source: MD Thesis of Gajender Singh Meena, AIIMS 1983 (From: Susheela AK. *Treatise on fluoride*. Project report. Sponsored by the Task Force on Safe Drinking Water, Government of India, 2003)

Table 24. Incidence of dental fluorosis in children of 6 schools and status of contamination of drinking water with fluoride in the Palam area of NCTD

School	Total no. of students in the school	No. of students examined for dental fluorosis	Total no. of water samples collected through afflicted students	No. of students afflicted	Percentage afflicted	No. of fluoride contaminated sources (above 1.0 mg/L)	Range of fluoride contamination (mg/L)	No. of safe sources (fluoride below 1.0 mg/L)
1	237	67	44	25	37	17	1.10–2.0	27
2	1017	578	81	98	17	Nil	—	81
3	2100	745	98	119	16	33	1.13–5.01	65
4	1956	1037	86	140	13	4	1.15–2.88	72
5	2000	1290	86	144	11	4	1.62–9.30	82
6	1700	1200	48	55	4.5	20	1.1–12.45	28
Total	9010	4917	443	581	4.5–37	88	1.1–12.45	355

NCTD: National Capital Territory of Delhi
Source: Water Foundation Survey, 2002 (From: Susheela AK. *Treatise on fluoride*. Project report. Sponsored by the Task Force on Safe Drinking Water, Government of India, 2003)

Oral cancer

In India, the incidence of oral cancer is the highest in the world and is preceded by some premalignant lesion. The most important of all premalignant lesions is oral submucous fibrosis. It is characteristically found in people of South-East Asian origin and is associated with the chewing of betel nut. Its prevalence has increased manifold in the past three decades due to increased consumption of *paan masala* and *gutka* by persons of all age groups, including children (Table 25).

The condition has a high malignant potential, 7.5% of the lesions become malignant over a 10-year period and more than one lesion may develop at different sites in the oral cavity.

Data from specialized cancer hospitals across the country over a period of 7 years (1993–2000) are shown in Table 26. The prevalence of oral cancer reported by Population-based Cancer Registries is given in Table 27. A summary of annual incidence of oral cancer of different sites from National Cancer Registries in Mumbai and Chennai for the period 1988–92 is shown in Tables 28 and 29, respectively. It shows the age-standardized incidence rate for different sites. Overall, the incidence per 100,000 population is 29 for males and 14.3 for females, the average for the population being 21.65. When these data are compared with data from other parts of the world (US 4.4, Japan 1.6, UK 2), it is evident that the prevalence in India is much higher. Given the large population of India, the actual number of cases of oral cancer is gigantic.

Table 25. Oral submucous fibrosis in India (1990)

	Incidence 100,000/year	
Sex	North India	South India
Males	5–8	9
Females	2–6	20

Table 26. Number of treated cases in cancer hospitals

ICD 140–149	Number	Data not available from
1993	6209	Bihar, Gujarat and Himachal Pradesh
1994	5961	Bihar, Gujarat, Himachal Pradesh and Maharashtra
1995	6794	Bihar, Gujarat and West Bengal
1996	9444	Bihar, Gujarat, Tripura and West Bengal
1997	9165	Andhra Pradesh, Bihar, Gujarat and West Bengal
Number of hospitals—25		
2000	9430	Bihar, Gujarat and Orissa
Number of hospitals—35		

ICD Code: 140 lip; 141 tongue; 142 salivary gland; 143–145 mouth; 146 oropharynx; 147 nasopharynx; 148 hypopharynx and 149 pharynx
Source: Health Information of India (1993–2000)

Table 27. Population-based Cancer Registry (PBCR) report

PBCR	Site of cancer		Bangalore No.	Bangalore %	Barshi No.	Barshi %	Bhopal No.	Bhopal %	Chennai No.	Chennai %	Delhi No.	Delhi %	Mumbai No.	Mumbai %
1990–96	Tongue	M	348	3.4	30	4.7	206	8.1	535	4.7	1242	4.7	1456	5.0
		F	88	0.8	8	1.0	26	1.2	176	1.4	334	1.3	448	1.8
	Oral cavity	M	284	2.9	33	5.2	182	7.2	671	5.9	854	3.3	1601	5.5
		F	726	6.2	11	1.4	104	4.6	610	4.9	427	1.7	919	3.4
	Hypopharynx		560	5.5	68	10.7	166	6.5	550	4.8	555	2.1	1519	5.3
1997–98	Lip		5	3.16	1	3.52	4	3.46	10	3.27	24	3.27	24	3.25
	Tongue		136	3.44	5	2.59	70	8.04	192	5.26	384	4.2	417	4.84
	Oral cavity		101	3.28	13	3.52	82	9.41	190	5.20	333	3.69	480	5.34
	Oropharynx		75	2.45	1	10.36	17	1.95	80	2.19	212	2.35	164	1.90
	Hypopharynx		171	5.55	20	0.00	50	5.74	167	4.57	193	2.14	362	4.20

Table 28. Oral cancer in Mumbai (1988–1992)

Age group (years)	Sex	Site of cancer: Lip	Tongue	Salivary gland	Mouth
0–4	M	—	—	0.0	—
	F	0.0	0.0	—	0.0
5–9	M	0.0	—	—	0.0
	F	—	—	—	—
10–14	M	—	—	0.0	—
	F	—	—	0.1	—
15–19	M	—	—	—	0.1
	F	—	—	—	0.0
20–24	M	—	0.1	0.0	0.3
	F	—	0.1	0.0	0.2
25–29	M	—	0.3	0.3	0.4
	F	—	0.4	0.1	0.4
30–34	M	0.1	0.9	0.2	1.4
	F	0.1	0.5	0.2	0.8
35–39	M	0.1	2.4	0.3	3.9
	F	—	1.2	0.7	2.6
40–44	M	0.1	4.8	0.7	6.1
	F	0.1	2.2	0.4	5.1
45–49	M	0.7	9.6	0.6	12.3
	F	0.7	2.9	0.4	6.6
50–54	M	0.7	13.3	1.1	16.7
55–59	F	0.4	5.2	0.8	13.1
	M	1.4	21.5	0.9	22.3
60–64	F	1.5	10.9	—	14.9
	M	1.4	27.5	2.7	23.2
	F	1.7	9.6	1.4	21.3
65–69	M	1.1	38.2	5.7	32.5
	F	2.0	8.3	1.2	18.9
70–74	M	2.4	36.6	4.1	23.0
	F	—	11.6	0.6	23.8
75+	M	3.2	45.0	1.9	30.4
	F	2.0	13.6	2.5	26.2

Note: Annual incidence per 100,000 by age group

Source: Parkin *et al. Cancer incidence in five continents, Vol. VII.* Lyon: IARC Scientific Publications No. 143; 1997

Table 29. Oral cancer in Chennai (1988–1992)

Age group (years)	Sex	Site of cancer: Lip	Tongue	Salivary gland	Mouth
0–4	M	—	0.1	—	—
	F	—	—	0.1	0.1
5–9	M	—	—	—	—
	F	—	—	—	0.1
15–19	M	—	0.1	—	—
	F	—	—	0.2	0.2
20–24	M	—	0.1	0.1	0.3
	F	—	0.2	0.1	0.3
25–29	M	—	0.3	0.2	0.2
	F	—	0.3	0.3	0.4
30–34	M	0.3	2.0	0.3	0.4
	F	—	0.2	0.5	2.2
35–39	M	—	2.7	0.5	3.2
	F	—	0.8	—	2.8
40–44	M	0.5	5.1	0.7	4.0
	F	—	1.3	0.2	9.2
45–49	M	1.4	9.0	0.2	11.7
	F	0.5	3.2	1.0	10.3
50–54	M	1.1	15.3	0.5	20.9
	F	0.9	7.6	1.8	30.1
55–59	M	0.7	23.0	1.4	29.3
	F	1.3	7.3	1.3	29.2
60–64	M	2.6	25.9	1.3	34.2
	F	2.6	8.3	—	43.0
65–69	M	2.4	35.6	7.1	47.5
	F	3.0	8.3	0.8	37.5
70–74	M	1.0	23.7	2.1	22.7
	F	3.9	8.9	3.0	29.6
75+	M	—	19.0	1.2	40.4
	F	3.2	6.5	1.1	29.2

Note: Annual incidence per 100,000 by age group

Source: Parkin *et al. Cancer incidence in five continents, Vol. VII.* Lyon: IARC Scientific Publications No. 143; 1997

Tobacco-related cancers

Sites of cancer that have been associated with the use of tobacco (tobacco-related cancers [TRCs]) include the lip, tongue, oral cavity, pharynx (including oropharynx and hypopharynx), oesophagus, larynx, lungs and urinary bladder.

The total proportion of these sites of cancer relative to all sites in males and females is given in Table 30. In males, this proportion varies from 36.1% in Bangalore to 54.6% in Bhopal, whereas in females, Bangalore and Mumbai have the highest proportion of 16.2% and 16.3%, respectively.

Bibliography

1. WHO Oral Health Country/Area Profile Programme. WHO Collaborating Centre, Malmo University, Sweden; 1992.
2. National Cancer Registry Programme, Indian Council of Medical Research. Biennial report 1988–89, Consolidated report of PBCR 1990–96, 1997–98, HBCR 1984–93 and 1994–98. New Delhi: Indian Council of Medical Research.
4. Damle SG. *Pediatric dentistry*. 1st ed. New Delhi: Arya (Medi) Publishing House; 2000.
5. John J. *Textbook of preventive and community dentistry*. New Delhi: CBS Publishers and Distributors; 2003.
6. Singh G (ed). *Textbook of orthodontics*. New Delhi: Jaypee Brothers.
7. Susheela AK. *Treatise on fluoride. Task force on safe drinking water*. Government of India, New Delhi, 2003.

Table 30. Number and relative proportion (%) of specific sites of cancer related to the use of tobacco relative to all sites of cancer

	Bangalore		Barshi		Bhopal		Chennai		Delhi		Mumbai	
Site of cancer	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Males												
Lip	5	0.16	1	0.52	4	0.46	10	0.27	24	0.27	24	0.28
Tongue	106	3.44	5	2.59	70	8.04	192	5.26	384	4.26	417	4.84
Oral cavity	101	3.28	13	0.52	82	9.41	190	5.20	333	3.69	460	5.34
Oropharynx	75	2.43	1	10.36	17	1.95	80	2.19	212	2.35	164	1.90
Hypopharynx	171	5.55	20	0.00	50	5.74	167	4.57	193	2.14	362	4.20
Pharynx, etc.	34	1.10	0	8.81	2	0.23	18	0.49	37	0.41	92	1.07
Oesophagus	221	7.17	17	2.59	70	8.04	295	8.08	393	4.36	564	6.55
Larynx	111	3.60	5	3.63	44	5.05	165	4.52	575	6.37	492	5.71
Lung	218	7.08	7	2.59	104	11.94	370	10.13	897	9.94	783	9.09
Urinary bladder	70	2.27	5	6.74	33	3.79	84	2.30	382	4.23	277	3.21
TRC	1112	36.09	162	38.34	476	54.65	1571	43.02	3430	38.01	3635	42.18
All sites	3081	100.0	193	100.0	871	100.0	3652	100.0	9023	100.0	8617	100.0
Females												
Lip	4	0.11	1	0.47	1	0.13	7	0.17	8	0.09	11	0.13
Tongue	33	0.93	2	0.95	14	1.80	52	1.29	116	1.32	166	1.95
Oral cavity	197	5.54	7	3.32	47	6.04	166	4.12	140	1.59	279	3.28
Oropharynx	13	0.37	0	0.00	1	0.13	16	0.40	46	0.52	27	0.32
Hypopharynx	44	1.24	1	0.47	4	0.51	59	1.47	31	0.35	81	0.95
Pharynx, etc.	9	0.25	0	0.00	0	0.00	3	0.07	10	0.11	29	0.34
Oesophagus	186	5.23	10	4.74	28	3.60	195	4.84	194	2.20	367	4.32
Larynx	13	0.37	2	0.95	5	0.64	20	0.50	75	0.85	75	0.88
Lung	60	1.69	4	1.90	15	1.93	73	1.81	172	1.95	267	3.14
Urinary bladder	21	0.59	2	0.95	5	0.64	37	0.92	93	1.06	72	0.85
TRC	580	16.32	29	13.74	120	15.42	628	15.60	885	10.05	1374	16.16
All sites	3554	100.0	211	100.0	778	100.0	4026	100.0	8805	100.0	8504	100.0

TRC: tobacco-related cancer

Source: National Cancer Registry Programme. *Two-year report of the population-based cancer registries 1997–1998*. New Delhi: Indian Council of Medical Research; 2002

Appendix 1

Baseline and projected scenario for dental health in India, 2000–2015

Based on the prevalence data compiled in this paper, the table below assesses the trends of different oral and dental diseases and gives projection for the next 10 years.

Categories	Prevalence (%)	Age group (years)	Prevalence (in lakh)			
			2000	2005	2010	2015
Dental caries	50.00	All	5084.7	5484.6	5869.0	6231.8
Periodontal diseases (relatively severe)	45.00	15+	2957.6	3190.2	3413.8	3624.8
Malocclusion	32.50	9–14	401.4	433.0	463.3	491.9
Oral cancer	0.03	35+	NA	0.6	NA	0.8
Fluorosis	5.50	All	559.3	603.3	645.6	685.5
Severe fluorosis	1.0	All	101.7	109.7	117.4	124.6

Note: It is assumed that the prevalence rate will remain unchanged over the period of projections, except for oral cancer and peridontal diseases, due to the rampant use of *paan masala* and *gutka* by persons of all age groups and both the sexes. If minor periodontal diseases are included, the proportion of population above the age of 15 years with this disease could be 80%–90%. The projections may best be viewed as upper bound except for severe periodontal diseases and oral cancers, which are lower bound.
Source: Shah 2004a and 2004b

Acknowledgement

The author gratefully acknowledges the AIIMS authority for allowing to undertake the study. The author also wishes to put on record the contributions made by Dr Ajay Mahal for preparation of the projections table given in the Appendix and Dr Puneet Batra for collection of data during the initial stages.

National Programme for Control of Blindness

AVTAR SINGH DUA

Magnitude of the problem

In earlier days, trachoma was the leading cause of blindness. With socioeconomic development and increased longevity, cataract was the leading cause of blindness in 1986–89. According to surveys on the magnitude and causes of blindness, and surgical outcomes of cataract carried out in 1999–2002 and in 2003, the estimated prevalence of blindness was found to be 1.1% in the major States and 1.38% in the north-eastern States. Females were found to have a higher prevalence of blindness as compared to men, and rural respondents as compared to urban respondents. Cataract was the commonest cause of blindness (62.6%) followed by uncorrected refractive errors (19.7%); 16.6% individuals went blind after cataract surgery. Visual outcomes after cataract surgery were poorer among females, rural residents and those who underwent surgery at an older age (more than 70 years). An analysis of data from Sentinel Surveillance Units (SSUs) for the year 2002–03 showed that nearly 39% of patients treated were bilaterally blind, about half the beneficiaries were SC/ST/OBC and most SSUs reported a higher number of female beneficiaries.

National Programme for Control of Blindness

The National Programme for Control of Blindness (NPCB), launched in 1976, was a 100% centrally-sponsored Programme. In 1983, the National Health Policy of India reiterated that blindness was an important public health problem and set a target to reduce the blindness prevalence rate from 1.4% to 0.3% (Government of India 1983). The 1986–89 survey showed that cataract was the major cause of blindness in the country. The Government of India has now laid down a target for reduction in the prevalence of blindness to 0.8% by the end of the Tenth Five-Year Plan (Ministry of Health and Family Welfare 2004 (Government of India 2004b) and to 0.5% by 2010 (Government of India 2002c).

Member, National Commission on Macroeconomics and Health, Government of India, New Delhi
e-mail: avtarsinghdua@yahoo.co.in

In the early 1990s there was a considerable backlog of clients needing cataract surgery because the number of cataract surgeries performed was not sufficient to compensate for the increase in incident cases and targets were allocated on the basis of previous performance, not on the basis of the prevalence of cataract blindness or capacity of the surgical services (Limburg *et al.* 1996). The World Bank-financed Cataract Blindness Control Project came into effect from 31 January 1995 and covered 7 States—Andhra Pradesh, Madhya Pradesh, Maharashtra, Orissa, Rajasthan, Tamil Nadu and Uttar Pradesh—which accounted for over 70% of cataract blindness in India. The seven-year Project sought to reduce the prevalence of blindness by more than 50% in these States by conducting 110.3 lakh cataract surgeries and eliminating the backlog. It included four components:

- Enhancing the quality of care and expanding service delivery through regularizing of camps, involvement of NGOs and the private sector, and strengthening service capacity and improving efficiency;
- Developing human resources for eye care through the training of ophthalmologists, OT personnel, ophthalmic assistants, and management training;
- Promoting outreach activities and public awareness through outreach and screening camps organized by NGOs and government teams, facilitating transportation and compensation to patients from remote areas, and enabling promotion through schools by creating awareness among teachers and students;
- Building institutional capacity for eye management at the Central, State and district levels, and improving cooperation between the government and private/voluntary sectors.

A National Programme Management Cell (NPMC) was made responsible for designing and communicating programme strategies and for the overall implementation of the Programme. About 278 District Blindness Control Societies were created, covering all the Project districts. State Blindness Control Societies were also set up to decentralize monitoring and ensure the smooth flow of funds. New operation theatres and eye wards were constructed under

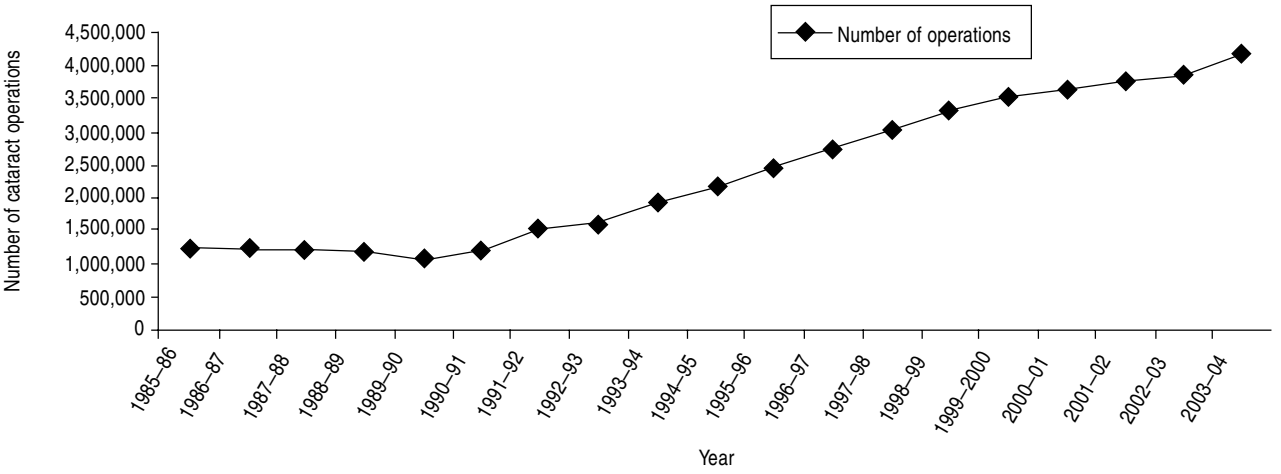

Fig. 1 Progress in cataract surgeries

the project and high-quality goods and equipment were supplied including operating microscopes, slit-lamps, A-scans, keratometers, YAG lasers, intraocular lenses (IOLs) and ophthalmic sutures.

Against a target of 110 lakh operations during the Project period, 153.5 lakh cataract surgeries (139%) were performed (Ministry of Health and Family Welfare 2002b). However, the performance was far less than desired in Orissa and Rajasthan, where 73% and 91% of the targeted cataract operations were performed. The number of cataract operations performed has steadily increased from 15.11 lakh operations in 1991–92 to 42.0 lakh operations in 2003–04 (Fig. 1). The State-wise performance of cataract operations conducted is given in Table 1. The proportion of patients getting operated before the age of 50 years increased from 10% to 17%. In 2003–04, the proportion of cataract surgeries with IOL implants had increased to 83%; in two States it was quite low—Rajasthan (63%) and Uttar Pradesh (57%). Consequently, surgical outcomes improved—postoperative visual acuity improved to 82% following intracapsular cataract extraction (ICCE) surgery compared with 75% before 1994, and in the case of IOL implants it improved to 95% compared with the earlier 84%. Initially, about 40% of cataract operations were being performed in camps while at the end of the Project more than 90% of them are being conducted in institutions, which have been strengthened.

NGOs have been actively involved in the implementation of the NPCB. In a study on the cost–benefit analysis of the World Bank-assisted Cataract Blindness Control Project, it was found that NGO-organized screening camps supplemented by surgery of screened persons at base hospitals was the most cost-effective method for conducting cataract operations (Rs 1128 per operation). This was low compared to that in private hospitals (Rs 5331 per operation), NGO hospitals (Rs 4977 per operation) or in government camps (Rs 2143 per operation) (MoHFW 2002).

As a result of interventions and better management of the NPCB, the prevalence of blindness in India has come down from 1.49% in 1986–89 to 1.1% in 2001–03. The State-wise prevalence of blindness is given in Table 2. The prevalence of visual disability/handicap in the population was found to be 1.06% in the 2001 Census, which compares well with 1.1% found in the sample survey conducted during 2001–03. It was earlier thought that the prevalence of blindness would start declining once a figure of about 40 lakh cataract operations per year was achieved and sustained. However, over 40 lakh operations per year could be performed only for the first time in 2003–04, but 2001–03 surveys showed a decline in the prevalence of blindness in the general population. The probable reason for this might be that the incidence of cataract in India (and also other tropical countries) is lower than that in temperate areas. Another factor that could possibly explain this phenomenon to some extent is that not all private practitioners might be reporting cataract operations under the Programme.

A disturbing observation, however, is that against a target of 13.20 lakh operations for the Project period for Andhra Pradesh, 24.0 lakh operations (182%) were performed. Yet, according to the latest survey, the prevalence of blindness in AP reduced only marginally from 1.50% in 1986–89 to 1.42% in 2001–03. This could be due to the poor quality of cataract operations resulting in no improvement in visual acuity in many cases. Thus, while increasing the number of operations for cataract is important, the quality of services also needs to be continuously monitored.

Since cataract was the major cause of blindness according to the 1986–89 survey, the focus of the NPCB in the initial years was on promotion and rationalization of targets for cataract operations. Having achieved substantial progress in this area, attention was then given to the second most important cause of blindness—refractory errors, primarily in schoolchildren. Schoolteachers were involved in screening schoolchildren for refractive errors and corrective glasses were provided to children to improve their visual acuity.

Table 1. State-wise performance of cataract surgeries from 1996 to 2004

	1996–1997		1997–1998		1998–1999		1999–2000		2000–2001		2001–2002		2002–2003		2003–2004	
State	Target	Achv.	Target	Achv.	Target	Achv.	Target	Achv.	Target	Achv.	Target	Achv.	Target	Achv.	Target	Achv.
Andhra Pradesh	220,000	275,163	246,400	295,735	271,050	343,680	296,000	337,980	320,000	358,799	350,000	371,949	350,000	404,002	350,000	443,091
Bihar	175,000	127,450	175,000	124,586	192,500	110,121	210,000	138,277	170,000	90,430	140,000	81,104	140,000	63,927	140,000	87,876
Chhattisgarh									76,000	51,961	80,000	52,224	80,000	56,451	80,000	64,196
Goa	5,000	4,093	5,600	4,767	6,150	4,472	6,500	4,743	6,700	2,982	7,000	5,044	7,000	5,294	7,000	5,497
Gujarat	168,000	248,681	229,160	274,243	252,000	291,030	290,000	414,580	300,000	405,386	400,000	414,580	400,000	436,740	400,000	449,234
Haryana	80,000	70,063	89,600	78,505	98,600	87,757	100,000	89,000	110,000	91,515	110,000	102,171	110,000	90,665	110,000	89,706
Himachal Pradesh	10,000	9,813	11,200	13,075	12,300	12,652	14,500	14,213	15,000	14,172	16,000	16,843	16,000	16,226	16,000	18,343
Jammu and Kashmir	9,000	6,332	10,080	7,109	11,100	10,646	12,500	8,314	13,000	10,092	13,000	10,503	13,000	11,553	13,000	10,412
Jharkhand									40,000	32,552	70,000	29,510	70,000	295,44	70,000	28,054
Karnataka	150,000	134,553	168,000	165,000	184,800	172,569	200,000	164,033	210,000	164,693	220,000	202,851	220,000	244,699	220,000	263,613
Kerala	55,000	50,140	61,600	59,358	67,800	65,728	80,000	79,446	80,000	72,169	90,000	69,028	90,000	83,345	90,000	79,696
Madhya Pradesh	250,000	212,954	280,000	254,138	308,000	287,201	300,000	275,108	244,000	241,314	240,000	234,527	240,000	224,049	240,000	233,870
Maharashtra	300,000	357,407	336,000	389,701	369,600	404,738	400,000	381,929	420,000	459,721	420,000	473,145	420,000	480,356	420,000	519,561
Orissa	100,000	60,641	112,000	74,713	123,200	79,271	125,000	63,391	130,000	84,231	130,000	86,386	130,000	81,619	130,000	82,652
Punjab	120,000	119,354	134,400	126,182	147,850	144,885	150,000	108,240	160,000	140,735	160,000	120,504	160,000	122,670	160,000	133,376
Rajasthan	160,000	136,103	179,200	157,243	197,200	176,955	194,000	188,417	210,000	185,036	220,000	196,835	220,000	188,747	220,000	226,829
Tamil Nadu	275,000	296,847	308,000	329,773	338,800	373,690	350,000	356,953	375,000	364,597	400,000	373,058	400,000	371,559	400,000	452,650
Uttar Pradesh	350,000	371,251	392,000	419,865	431,200	473,528	435,000	557,326	445,223	564,135	450,000	536,647	450,000	551,516	450,000	567,718
Uttaranchal									28,234	27,628	31,056	27,544	100,000	34,703	100,000	37,105
West Bengal	150,000	144,000	168,000	146,405	184,800	169,397	200,000	205,790	210,000	176,473	220,000	229,665	220,000	233,382	220,000	249,895
Total	2,577,000	2,624,845	2,906,240	2,920,398	3,196,950	3,208,320	3,363,500	3,387,740	3,563,157	3,538,621	3,767,056	3,634,118	3,836,000	3,731,047	3,836,000	4,043,374

Funding for the NPCB

The allocation of funds for the NPCB increased with the start of the World Bank-financed Project in 1995. The total outlay for the NPCB in the Ninth Five-Year Plan was Rs 480 crore. In 2001–02, the outlay was Rs 127.57 crore, of which the major contribution came from the World Bank (75.7%) and 16.4% from the Government of India. After the end of the World Bank-financed Project in 2001–02, the NPCB is being sustained mainly through the domestic budget for which an allocation of Rs 445 crore has been made in the Tenth Five-Year Plan. The Government of India contributed 84.6% of the Rs 85.59 crore for the Programme in 2002–03 and 90.5% of the Rs 86.96 crore in 2003–04; the remaining funds come from Danish assistance (Table 3).

In 2001–02, 17.5% of the total expenditure was on capital expenses (10.6% on medical equipment and 6.9% on civil works/furniture), and 82.5% on recurring costs (32% on medical supplies/consumables for cataract operations and 26.9% on salaries) (Table 4). The Government of India purchased and supplied medical equipment to the States, which had the twin benefits of lowered procurement costs as well as assured delivery to the facilities. Earlier experience showed that funds released to the States for procurement of equipment would be diverted to other areas such as payment of salaries. Of the expenditure, 18.2% was incurred through NGOs which were provided grants-in-aid for performing free cataract surgery. The Government of India also released non-recurring grants-in-aid to NGOs to strengthen/expand eye care services in rural areas.

The major part of the funding went to the districts (87.52%), thereby indicating that a large quantum of the earmarked funds go directly for patient care. Of the funds utilized at the Central and State levels, the major part is spent on information, education and communication (IEC) activities, training, review, monitoring and evaluation.

Table 2. Prevalence of blindness in the 50+ population in 15 major States and in north-eastern States

State/Union Territory	Bilateral blind (%)
Andhra Pradesh	10.9
Chhattisgarh	12.4
Madhya Pradesh	8.9
Maharashtra	7.3
Orissa	10.8
Rajasthan	11.9
Tamil Nadu	6.0
Uttar Pradesh	7.2
Bihar	6.0
Gujarat	8.2
Himachal Pradesh	5.4
Karnataka	13.7
Kerala	4.3
Punjab	7.8
West Bengal	9.2
Arunachal Pradesh	17.56
Assam	23.48
Manipur	10.66
Meghalaya	5.72
Mizoram	6.03
Nagaland	8.09
Sikkim	4.99
Tripura	5.96
All India	8.5

Note: Extrapolating the results of the survey to the general population, it is estimated that 1.1% of the general population is blind (1.38% in the north-eastern region)

Table 3. Abstract summary of funding of NPCB for the years 2001–02, 2002–03 and 2003–04 (Rs in lakh)

			From external agencies			
Year		Domestic budget	World Bank	WHO	DANIDA	Total
2001–02	In cash	2078.09	9601	80.06	998.23	12757.38
	In kind	0	0	0	0	0
	Grants-in-aid to societies/NGOs	0	0	0	0	0
	Total	2078.09 (16.4%)	9601 (75.7%)	80.06 (.63%)	998.23 (7.9%)	12677.32
	Grant/loan	Not applicable	Loan	Grant	Grant	
2002–03	In cash	7237.62	0	96.3	1224.73	8558.65
	In kind	0	0	0	0	0
	Grants-in-aid to societies/NGOs	0	0	0	0	0
	Total	7237.62 (84.6%)	0 (0%)	96.3 (1.1%)	1224.73 (14.3%)	8558.65
	Grant/loan	Not applicable	Not applicable	Grant	Grant	
2003–04	In cash	7870	0	96.3	730	8696.3
	In kind	0	0	0	0	0
	Grants-in-aid to societies/NGOs	0	0	0	0	0
	Total	7870 (90.5%)	0	96.3 (1.1%)	730 (8.4%)	8696.3
	Grant/loan	Not applicable	Not applicable	Grant	Grant	

WHO: World Health Organization; DANIDA: Danish International Development Agency

Table 4. Expenditure (by level of utilization) under the National Programme for Control of Blindness, 2001–02 (Rs in lakh)

Expenditure head	Centre	States* (DBCS/NGOs)	Districts	Total	%
Recurrent expenditure					
01. Salaries	24.38	123.6	3,115.68	3,263.66	27.7
1 Regular staff	20.78	123.6	2,953	3,097.38	26.2
2 Contractual staff	3.12	0	162.68	165.8	1.4
3 Daily wages	0.48†	0	0	0.48	0.0
02. Maintenance	3.45	3.5	120.2	127.15	1.1
1 Of medical equipment	0	0	120.2	120.2	1.0
2 Of office equipment	0.23	3.5	0	3.73	0.0
3 Civil works	3.22	0	0	3.22	0.0
03. Medical supplies/consumables	0	0	3,886.42	3,886.42	32.9
1 IOLs	0	0	1,554.71	1,554.71	13.2
2 Sutures	0	0	899.53	899.53	7.6
3 Drugs and medicines	0	0	1,002.5	1,002.5	8.5
4 Spectacles	0	0	429.68	429.68	3.6
04. Office expenses	9.75	14.95	552.43	552.43	4.7
1 Stationery	0.6	3.5	0	4.1	0.0
2 POL	1.31‡	0	186.32	187.63	1.6
3 TA	7.84	11.45	0	19.29	0.2
4 Others (contingencies)	0	0	366.11‡	366.11	3.1
05. Training	202.28	158.5	45.58	406.36	3.4
06. IEC	379.74	304	274.52	958.26	8.1
07. Review and monitoring	98.05	11.45	0	109.5	0.9
08. Research	138.49	0	0	138.49	1.2
Total	856.14	616	8,263.82	9,735.96	82.5
Capital expenditure					
10. Civil works	0.42	0	818.2	818.62	6.9
1 New construction	0	0	488.5	488.5	4.1
2 Furniture	0.42	0	329.7	330.12	2.8
11. Equipment	0.8	0	1,246.85	1,247.65	10.6
1 Medical equipment	0	0	1,246.85	1,246.85	10.6
2 Office equipment	0.8	0	0	0.8	0.0
12. Vehicle	0	0	0	0	0.0
Total	1.22	0	2,065.05	2,066.27	17.5
Grand total	857.36	616	10,328.87	11,802.23	100.0
%	7.26	5.22	87.52	100	

*Based on allocation, detailed expenditure not available

†Extrabudgetary support from WHO

‡Including maintenance of vehicles

Expenditure under the NPCB could be disaggregated into administration costs (comprising salaries of Programme Managers and contractual staff for management of the Programme at Central, State and district levels, office expenses, office equipment and its maintenance, purchase of furniture/civil works), patient care costs (comprising salaries of ophthalmic surgeons, paramedical ophthalmic assistants, medical equipment/consumables used during cataract operations, maintenance of medical equipment), development costs (comprising IEC activities and training) and assessment costs (comprising review, monitoring and evaluation and research studies). In 2001–02, under the Programme, Rs 8.96 crore (7.6%) were spent on administration, Rs 90.25 crore (76.5%) on patient care, Rs 13.65 crore (11.6%) on IEC activities and training, and Rs 2.48 crore (2.1%) on assessment of the Programme.

Sustainability

Since 2001–02, the NPCB is being funded primarily through

the domestic budget, with Rs 445 crore being provided for the Programme in the Tenth Five-Year Plan. However, it needs to be kept in mind that for strengthening the institutional capacity to manage the Programme, funds were provided under the NPCB for the salaries of Programme Managers at the State level. Similarly, posts of District Programme Managers in District Blindness Control Societies were filled on a contractual basis. However, with the ending of the World Bank-financed Project, services of District Programme Managers hired on contract are no longer available, and the responsibility of managing the Programme at the district level has been given to a Deputy CMO who is provided an honorarium equivalent to 20% of the basic salary (subject to a maximum of Rs 2000 per month). In many States there are vacancies at the district level and the effectiveness of this strategy will be clear only after some time.

Similarly, under the NPCB, the States created posts of ophthalmic surgeons in the Eighth and Ninth Five-Year Plans and their salaries were provided from Project funds. The salaries of paramedical ophthalmic assistants were also paid from Project funds. In the subsequent Plans, however, central support for the salaries of these categories of personnel was stopped and the States were asked to meet these expenses. With most of the States reeling under a financial crisis, it might be difficult to provide the salaries of these categories of health personnel. In that case, it might be difficult for the States to maintain the pace of cataract operations in the coming years, let alone scale up cataract operations.

Recommendations

It would be desirable if the Centre would continue to support the States in the payment of salaries not only to maintain the current level of services but also to up-scale them. Increasing attention needs to be given for improving the quality of cataract surgery services because, for example, in Andhra Pradesh, against the set target of cataract operations, 182% operations were performed and yet the prevalence of blindness reduced only marginally, indicating that the restoration of sight following surgery was probably not good. The scope of eye care surgeries under the NPCB needs to be increased beyond cataract surgeries and correction of refractive errors to address other causes of blindness as well. Since corneal opacities also account for a significant proportion of blindness, eye banking services need to be provided an impetus.

Blindness estimations, projections and service delivery

MURALIKRISHNAN R.*, PRAVEEN KRISHNA R.†, THULASIRAJ R.D.*, DAMODAR BACHANI‡, SANJEEV GUPTA§, G.V.S. MURTHY§

This paper has the following sections:

1. Estimated prevalence and incidence
2. Reported cases
3. Projections
4. Causes, interventions and standard treatment protocols
5. References
6. Appendices

The scope of estimations applied in items 1–3 contains all States and Union Territories listed as per the Census of India 2001. The causes, interventions and treatment protocols have been limited to those diseases which have been prioritized for the World Health Organization—Vision 2020 Programme for India.

Estimated prevalence and incidence

The prevalence and incidence of blindness refer to the year 2004. The prevalence has been estimated for all the States and Union Territories listed in the Census of India Report, 2001. Incidence has been estimated as the number of new cases in the year 2004 for all the States. State-wise prevalence and incidence data per 1000 population—age-wise, sex-wise and consolidated—are presented in Table 1. The methods of estimation are detailed below.

Methods of estimation

State-wise population projections

The Census of India for the years 1991 and 2001 along with projected population as per the Registrar General[1] were used in the estimation and projection of prevalence and incidence in this paper.

*Lions Aravind Institute of Community Ophthalmology
72, Kuruvikaran Salai, Gandhi Nagar, Madurai 625020
e-mail: murali@aravind.org

†Aravind Eye Hospital, Madurai

‡Directorate General of Health Services, Ministry of Health and Family Welfare, New Delhi e-mail: adgo17@yahoo.com

§RP Centre for Ophthalmic Sciences, All India Institute of Medical Sciences, New Delhi

Estimates of prevalence

The data used in this paper pertain to the 37 districts covered by various surveys,[2–4] i.e. the 1999–2001 survey conducted by the National Programme for Control of Blindness (NPCB) in 15 States (15 districts), the rapid assessment in the north-eastern States of India, Goa, Haryana, Delhi, Jammu and Kashmir, Jharkand and Uttaranchal, Andhra Pradesh (2 districts), Madhya Pradesh (2 districts), Maharashtra (2 districts), Orissa (2 districts), Rajasthan (2 districts), Tamil Nadu (2 districts) and Uttar Pradesh (2 districts). The prevalence of blindness (visual acuity <6/60) in the general population was estimated for each State (Table A1.1 of Appendix 1). For the Union Territories where prevalence estimates were not available, estimates from the nearest State were used (e.g. Tamil Nadu rates for Pondicherry). For the Andaman and Nicobar Islands, the overall Indian prevalence rate was applied.

Using the estimated population who are blind, the age–sex-wise population was further estimated. As projected population-based data on age- and sex-wise distribution of blindness in the general population were not adequate, the results of the National Survey done in 1986–89 along with some population-based studies conducted in Andhra Pradesh in 2000–2001[5–8] were used to estimate the same for other States. The age–sex distribution of blindness estimated from the population-based studies is given in Table A1.2 of Appendix 1. This is the best available estimate for India. It was assumed that the age–sex distribution would remain the same over the years and hence was used for the projected population of 2005, 2010 and 2015.

However, the prevalence rate of blindness as per the NPCB surveys in 1986–89 and 2001–02 showed a reduction of 26.2%. This reduction has occurred over a period of 13 years. Based on this, it was assumed that the prevalence rate of blindness would reduce by 2% every year. This assumption was also applied while estimating the prevalence of blindness for the projected years (Table A1.1 of Appendix 1).

Table 1. Estimated prevalence and incidence of blindness (2004)

State/Union Territory	Estimated prevalence per 1000 population (by age)			Estimated prevalence per 1000 population (by sex)		Estimated number of new cases per year (incidence by age)			Estimated number of new cases per year (incidence by sex)		Estimated prevalence per 1000 population	Estimated number of new cases per year
	0–14	15–49	50+	Male	Female	0–14	15–49	50+	Male	Female	All	All
Andaman and Nicobar Islands	0.1	0.5	117.9	9.1	11.8	3	23	832	403	455	10.3	858
Andhra Pradesh	0.1	0.7	89.2	12.4	14.3	453	4,075	146,404	70,938	79,994	13.3	150,932
Arunachal Pradesh	0.2	1.2	209.1	19.3	23.8	8	72	2,574	1,247	1,407	21.4	2,654
Assam	0.3	1.4	236.9	26.1	31.5	165	1,486	53,399	25,874	29,177	28.7	55,050
Bihar	0.1	0.4	55.6	6.6	8.1	438	3,946	141,750	68,683	77,451	7.3	146,134
Chandigarh	0.1	0.4	94.1	8.2	11.0	6	53	1,921	931	1,049	9.5	1,980
Chhattisgarh	0.1	0.8	111.2	13.7	16.6	175	1,571	56,450	27,352	30,844	15.1	58,196
Dadra and Nagar Haveli	0.1	0.5	89.6	8.5	9.4	1	11	411	199	225	8.9	424
Daman and Diu	0.1	0.5	80.8	9.6	10.5	1	8	301	146	164	10.1	310
Delhi	0.1	0.5	102.5	9.5	11.9	96	866	31,129	15,083	17,009	10.6	32,092
Goa	0.1	0.7	98.7	13.6	15.0	11	95	3,430	1,662	1,874	14.3	3,536
Gujarat	0.1	0.5	72.1	9.2	11.0	306	2,757	99,058	47,997	54,125	10.1	102,122
Haryana	0.2	0.9	133.8	15.8	20.0	126	1,134	40,740	19,740	22,260	17.8	42,000
Himachal Pradesh	0.1	0.4	43.7	6.3	6.8	44	393	14,104	6,834	7,706	6.6	14,540
Jammu and Kashmir	0.1	0.9	130.4	14.2	16.1	64	579	20,789	10,073	11,359	15.1	21,432
Jharkhand	0.1	0.7	100.6	11.9	14.7	206	1,857	66,705	32,321	36,447	13.3	68,768
Karnataka	0.2	0.8	107.9	15.5	18.0	328	2,953	106,104	51,411	57,975	16.7	109,386
Kerala	0.1	0.2	27.8	5.0	5.5	200	1,802	64,728	31,363	35,367	5.3	66,730
Lakshadweep	0.04	0.3	43.2	5.0	5.5	0	4	153	74	84	5.3	158
Madhya Pradesh	0.1	0.6	80.1	9.9	12.0	339	3,050	109,577	53,094	59,872	10.9	112,966
Maharashtra	0.1	0.4	57.0	8.2	9.8	569	5,121	183,993	89,151	100,533	8.9	189,684
Manipur	0.1	0.7	105.1	12.3	13.7	17	151	5,428	2,630	2,966	13.0	5,5596
Meghalaya	0.05	0.4	73.4	6.6	7.3	16	146	5,248	2,543	2,867	7.0	5,410
Mizoram	0.1	0.4	70.3	6.8	7.8	6	57	2,058	997	1,1125	7.3	2,122
Nagaland	0.1	0.5	91.1	9.0	10.8	11	102	3,655	1,771	1,997	9.9	3,768
Orissa	0.1	0.6	84.6	12.2	14.2	223	2,003	71,957	34,866	39,316	13.2	74,182
Pondicherry	0.1	0.4	51.3	7.0	7.6	7	67	2,398	1,162	1,310	7.3	2,472
Punjab	0.1	0.5	63.5	8.5	10.6	147	1,325	47,600	23,064	26,008	9.5	49,072
Rajasthan	0.1	0.8	109.5	13.1	16.1	345	3,103	111,480	54,016	6,912	14.6	111,928
Sikkim	0.05	0.3	60.2	5.6	6.7	4	34	1,207	558	659	6.1	1,244
Tamil Nadu	0.1	0.3	38.3	6.8	7.9	383	3,443	123,689	59,932	67,582	7.3	127,514
Tripura	0.1	0.4	56.6	6.8	7.7	25	227	8,152	3,950	4,454	7.2	8,404
Uttar Pradesh	0.1	0.5	66.3	7.9	9.9	1,118	10,060	361,416	175,119	197,475	8.8	372,594
Uttaranchal	0.04	0.3	39.5	4.7	5.9	56	503	18,069	8,755	9,873	5.3	18,628
West Bengal	0.1	0.5	76.2	10.1	12.3	498	4,486	161,177	78,096	88,066	11.2	166,162
All India	0.1	0.6	77.3	10.2	12.2	6,396	57,565	2,068,087	1,002,063	1,129,985	11.2	2,132,048

Estimates of incidence

Incidence rates for blindness are not available and there is only one published study from Raipur district that reported the incidence of age-specific cataract in 1990.[9] Limburg *et al.* have used these incidence figures, which were adjusted by means of a factor making the age-specific prevalence rates for Raipur at par with those for India in 1996.[10] The important assumption made was that the incidence of cataract blindness would remain constant for the next 16 years. For this report, we used the WHO estimates of incidence of cataract (not cataract blindness) of 2 per 1000 per year (i.e. 0.2%) as the basis for calculating blindness. If it is presumed that half of these persons will have bilateral cataract leading to blindness, the incidence of cataract blindness would be 0.1%. In the absence of a larger cohort study, the best estimate used was 0.2% per year, as cataract accounts for nearly 50% of the cases of blindness.[7] The major assumptions used in this estimation are as follows:

1. The incidence of blindness would remain the same over the years, similar to what Limburg *et al.*[10] have used in their estimations.
2. The age- and sex-wise breakdown for the incidence of blindness was calculated using similar estimates as for prevalence. The assumption used for the projected population is that the age- and sex-wise proportion of blindness would remain the same over the years.

Reported cases

The reported cases include only one major blindness-related disease (cataract) for the period April 2003 to March 2004. The reported cases per 1000 population have also been estimated using the same data. Due to non-availability of data, the estimations could not be presented age-wise but have been presented sex-wise based on the NPCB survey results. The methods of estimation are detailed below.

Methods of estimation

The lacuna in data on diseases other than blindness due to cataract is the major reason for the non-inclusion of other diseases. Cataract was considered as the major problem as per the national surveys of 1986–89 and 1999–2001[1,5] and the priority of service delivery was towards this. This has led to a situation where only cataract surgery rates (CSRs) per million population are monitored.

For this estimation, the CSRs (Appendix 2) for every State for the period April 2003 to March 2004 (*Source:* NPCB) were used as a basis to estimate the reported new cases the preceeding year for cataract. The secondary data available include only those cases that were operated. There could have been many patients who were advised cataract surgery and refrained from undergoing surgery, due to various barriers such as fear, cost, lack of attenders, superstitious beliefs, etc.[11] Hence, based on previous experience it was assumed that around 60% had accepted and undergone cataract surgery. This would mean that the reported cases of cataract would also constitute those who did not accept surgery even after advice. Based on this assumption, the reported cases were calculated using the formulae

Reported cases (cataract) = Total cataract surgeries performed + those who did not accept surgery (surgery acceptance rate: 60%)

Reported cases (cataract) per 1000 population = Number of reported cases/population × 1000

The reported cases have been presented per 1000 population in Table 2. However, one of the major limitations is the estimation of the reported cases in terms of age break-up because the CSR refers to the number of cataract surgeries done per million population and the available data are for surgeries performed on people above 50 years of age. This constraints the presentation of the data age-wise. Based on the gender-wise break-up of cataract surgeries as reported in the NPCB 2001–02 for various States, the reported cases have been presented on the basis of gender break-up.

Since there are no data available for other diseases, estimations for these were not made. However, there were some reports available from the sentinel centres in different States but as these reports are hospital based they cannot be taken as the actual reported cases. Moreover, the proportion of eye diseases varies from centre to centre. For example, the proportion of refractive errors among patients attending the sentinel centre varies from 11.80% in Maharashtra (Nagpur) to 53.78% in Gujarat (Surat).

Projections

The estimations of prevalence and incidence calculated for the year 2000 and for the project years 2005, 2010 and 2015 have been combined and presented as 'cases'. Though most eye-related diseases do not cause 'deaths' directly, the number of deaths due to blindness has not been estimated. The estimated 'cases' have been presented in Tables 3a–3f. The methods of estimation are detailed below.

Methods of estimation

There is evidence that the blind in India and Nepal have higher mortality rates than the non-blind. According to the Nepal Blindness Survey, 5-year survival rates of less than 50% have been reported in some areas.[12] A 1992 longitudinal study of a cohort of 1020 people aged 40–64 years in central India with cataract reported a mortality rate that was double that of the normal population.[13] However, there are no reports on blindness as a cause of death. The probable chance of death due to blindness would be due to accidents.

Table 2. Reported cases of cataract

State/Union Territory	Reported cases per 1000 population		Reported new cases last year (April 2003–March 2004)		Reported cases per 1000 population	Reported new cases last year (April 2003–March 2004)
	Male	Female	Male	Female	All	All
Andaman and Nicobar Islands	2.32	2.66	531	624	2.69	1,155
Andhra Pradesh	9.27	9.52	354,473	384,012	9.79	738,485
Arunachal Pradesh	0.76	0.83	524	616	0.86	1,140
Assam	1.51	1.62	21,525	16,913	1.40	38,438
Bihar	2.03	2.23	77,624	68,836	2.00	146,460
Chandigarh	8.45	10.01	4,537	6,014	10.66	10,550
Chhattisgarh	3.41	3.66	51,357	55,637	3.68	106,993
Dadra and Nagar Haveli	NA	NA	NA	NA	NA	NA
Daman and Diu	3.59	3.45	273	347	4.00	620
Delhi	7.74	8.61	65,392	68,061	8.32	133,453
Goa	5.24	5.12	4,581	4,581	5.18	9,162
Gujarat	12.55	13.28	329,438	419,285	14.66	748,723
Haryana	6.59	7.41	73,260	76,250	7.12	149,510
Himachal Pradesh	4.29	4.12	15,286	15,286	4.21	30,572
Jammu and Kashmir	1.48	1.50	7,983	9,371	1.62	17,353
Jharkhand	1.38	1.51	24,781	21,976	1.36	46,757
Karnataka	6.16	6.37	171,348	268,007	8.03	439,355
Kerala	3.56	3.45	58,444	74,383	3.98	132,827
Lakshadweep	NA	NA	NA	NA	NA	NA
Madhya Pradesh	4.67	5.01	136,424	253,359	6.90	389,783
Maharashtra	7.09	7.53	346,374	519,561	9.13	865,935
Manipur	0.37	0.37	516	406	0.33	922
Meghalaya	0.80	0.79	1,073	990	0.76	2,063
Mizoram	1.29	1.31	690	637	1.25	1,327
Nagaland	0.39	0.42	379	336	0.38	715
Orissa	4.25	4.37	79,897	57,856	3.71	137,753
Pondicherry	11.53	11.06	6,978	9,637	13.44	16,615
Punjab	7.41	8.21	95,586	126,707	9.06	222,293
Rajasthan	5.80	6.33	173,902	204,146	6.58	378,048
Sikkim	0.83	0.88	266	156	0.68	422
Tamil Nadu	9.84	10.04	316,855	437,562	11.83	754,417
Tripura	2.96	2.95	6,208	7,288	3.21	13,497
Uttar Pradesh	4.72	5.27	463,636	482,560	5.08	946,197
Uttaranchal	6.17	6.89	30,302	31,539	6.64	61,842
West Bengal	4.83	5.21	208,246	208,246	5.01	416,492
All India	5.68	7.44	3,128,690	3,831,183	6.53	6,959,873

Projection of blindness and cataract surgeries

As cataract is the major cause of blindness, it was assumed that an increase in cataract surgery would reduce the blindness problem as well. Based on the previous years' cataract surgeries performed it was estimated that there was an increase of almost 50% between 1995 and 2000. An increase of 50% is expected every five years in the future. As the projected cases have been estimated till 2015, the projected cataract surgeries were also estimated and both the data have been presented in Fig. 1. The figure is expected to show the huge gap between the need and what is delivered for policy decisions.

Causes, interventions and standard treatment protocols

At the global level, from among the many causes of avoidable blindness, five conditions have been identified as immediate priorities within the framework of Vision 2020. The choice of these conditions is based on the burden of blindness they represent and the feasibility and affordability of interventions to prevent and treat them. These are: cataract, trachoma, onchocerciasis, childhood blindness and refractive errors, and low vision.

In India, during the 'Launch Workshop on Vision 2020: Right to Sight' at Goa between 10 and 13 October 2001, it

Table 3a. Projections for blindness (all combined)

State/Union Territory	Cases			
	2000	2005	2010	2015
Andaman and Nicobar Islands	5,090	5,333	5,367	5,049
Andhra Pradesh	1,243,982	1,136,820	1,029,658	922,496
Arunachal Pradesh	30,105	31,316	31,272	29,079
Assam	867,515	840,357	813,460	768,301
Bihar	676,620	681,538	680,563	673,262
Chandigarh	10,924	11,484	11,608	10,969
Chhattisgarh	499,513	497,753	490,827	477,732
Dadra and Nagar Haveli	2,233	2,331	2,350	2,219
Daman and Diu	1,808	1,883	1,907	1,786
Delhi	188,877	205,873	215,541	210,646
Goa	27,936	28,995	28,885	26,859
Gujarat	623,204	613,365	596,352	569,208
Haryana	422,049	414,186	404,799	387,141
Himachal Pradesh	61,339	62,566	61,316	56,179
Jammu and Kashmir	183,188	183,419	175,750	156,566
Jharkhand	523,929	523,301	517,322	505,523
Karnataka	1,050,047	1,018,527	978,716	920,370
Kerala	248,828	241,323	233,291	220,765
Lakshadweep	548	579	593	567
Madhya Pradesh	727,992	728,311	721,526	706,296
Maharashtra	1,065,169	1,031,427	994,664	942,512
Manipur	40,495	42,192	42,225	39,448
Meghalaya	23,240	24,451	24,745	23,442
Mizoram	9,478	9,993	10,142	9,627
Nagaland	21,404	22,585	22,886	21,682
Orissa	583,801	557,286	528,200	491,487
Pondicherry	11,061	11,644	11,788	11,165
Punjab	289,589	280,956	271,990	257,261
Rajasthan	953,886	951,730	944,279	922,806
Sikkim	4,824	5,083	5,175	4,923
Tamil Nadu	615,062	590,476	563,321	528,223
Tripura	37,268	39,179	39,625	37,486
Uttar Pradesh	1,873,559	1,924,211	1,955,987	1,977,929
Uttaranchal	65,629	68,023	69,902	71,612
West Bengal	1,116,987	1,090,351	1,056,068	1,004,774
All India	14,107,178	13,878,846	13,542,100	12,995,390

Table 3b. Projections for blindness (males)

State/Union Territory	Cases			
	2000	2005	2010	2015
Andaman and Nicobar Islands	2,392	2,506	2,522	2,373
Andhra Pradesh	584,671	534,305	483,939	433,573
Arunachal Pradesh	14,149	14,719	14,698	13,667
Assam	407,732	394,968	382,326	361,102
Bihar	318,011	320,323	319,865	316,433
Chandigarh	5,134	5,397	5,456	5,155
Chhattisgarh	234,771	233,944	230,689	224,534
Dadra and Nagar Haveli	1,049	1,095	1,104	1,043
Daman and Diu	850	885	896	839
Delhi	88,772	96,760	101,304	99,004
Goa	13,130	13,628	13,576	12,624
Gujarat	292,906	288,282	280,285	267,528
Haryana	198,363	194,667	190,255	181,956
Himachal Pradesh	28,829	29,406	28,819	26,404
Jammu and Kashmir	86,099	86,207	82,603	73,586
Jharkhand	246,247	245,952	243,142	237,596
Karnataka	493,522	478,707	459,996	432,574
Kerala	116,949	113,422	109,647	103,760
Lakshadweep	257	272	279	266
Madhya Pradesh	342,156	342,306	339,117	331,959
Maharashtra	500,630	484,771	467,492	442,981
Manipur	19,033	19,830	19,846	18,541
Meghalaya	10,923	11,492	11,630	11,018
Mizoram	4,455	4,697	4,767	4,525
Nagaland	10,060	10,615	10,756	10,191
Orissa	274,386	261,924	248,254	230,999
Pondicherry	5,199	5,473	5,540	5,248
Punjab	136,107	132,049	127,835	120,913
Rajasthan	448,326	447,313	443,811	433,719
Sikkim	2,267	2,389	2,432	2,314
Tamil Nadu	289,079	277,524	264,761	248,265
Tripura	17,516	18,414	18,624	17,618
Uttar Pradesh	880,573	904,379	919,314	929,627
Uttaranchal	30,846	31,971	32,854	33,658
West Bengal	524,984	512,465	496,352	472,244
All India	6,630,374	6,523,058	6,364,787	6,107,833

Table 3c. Projections for blindness (females)

State/Union Territory	Cases			
	2000	2005	2010	2015
Andaman and Nicobar Islands	2,698	2,826	2,844	2,676
Andhra Pradesh	659,310	602,515	545,719	488,923
Arunachal Pradesh	15,956	16,598	16,574	15,412
Assam	459,783	445,389	431,134	407,200
Bihar	358,608	361,215	360,698	356,829
Chandigarh	5,790	6,087	6,152	5,813
Chhattisgarh	264,742	263,809	260,138	253,198
Dadra and Nagar Haveli	1,183	1,235	1,245	1,176
Daman and Diu	958	998	1,011	946
Delhi	100,105	109,113	114,237	111,643
Goa	14,806	15,367	15,309	14,235
Gujarat	330,298	325,084	316,066	301,680
Haryana	223,686	219,518	214,543	205,185
Himachal Pradesh	32,509	33,160	32,498	29,775
Jammu and Kashmir	97,090	97,212	93,148	82,980
Jharkhand	277,682	277,350	274,181	267,927
Karnataka	556,525	539,819	518,719	487,796
Kerala	131,879	127,901	123,644	117,006

Table 3c. (*cont.*) Projections for blindness (females)

State/Union Territory	Cases			
	2000	2005	2010	2015
Lakshadweep	290	307	314	301
Madhya Pradesh	385,836	386,005	382,409	374,337
Maharashtra	564,540	546,656	527,172	499,531
Manipur	21,463	22,362	22,379	20,908
Meghalaya	12,317	12,959	13,115	12,424
Mizoram	5,023	5,296	5,375	5,102
Nagaland	11,344	11,970	12,129	11,492
Orissa	309,414	295,361	279,946	260,488
Pondicherry	5,862	6,172	6,248	5,917
Punjab	153,482	148,907	144,155	136,348
Rajasthan	505,559	504,417	500,468	489,087
Sikkim	2,557	2,694	2,743	2,609
Tamil Nadu	325,983	312,952	298,560	279,958
Tripura	19,752	20,765	21,001	19,868
Uttar Pradesh	992,986	1,019,832	1,036,673	1,048,303
Uttaranchal	34,783	36,052	37,048	37,954
West Bengal	592,003	577,886	559,716	532,530
All India	7,476,804	7,355,789	7,177,313	6,887,557

Table 3d. Projections for blindness by age (0–14 years)

State/Union Territory	Cases			
	2000	2005	2010	2015
Andaman and Nicobar Islands	15	16	16	15
Andhra Pradesh	3,732	3,410	3,089	2,767
Arunachal Pradesh	90	94	94	87
Assam	2,603	2,521	2,440	2,305
Bihar	2,030	2,045	2,042	2,020
Chandigarh	33	34	35	33
Chhattisgarh	1,499	1,493	1,472	1,433
Dadra and Nagar Haveli	7	7	7	7
Daman and Diu	5	6	6	5
Delhi	567	618	647	632
Goa	84	87	87	81
Gujarat	1,870	1,840	1,789	1,708
Haryana	1,266	1,243	1,214	1,161
Himachal Pradesh	184	188	184	169
Jammu and Kashmir	550	550	527	470
Jharkhand	1,572	1,570	1,552	1,517
Karnataka	3,150	3,056	2,936	2,761
Kerala	746	724	700	662
Lakshadweep	2	2	2	2
Madhya Pradesh	2,184	2,185	2,165	2,119
Maharashtra	3,196	3,094	2,984	2,828
Manipur	121	127	127	118
Meghalaya	70	73	74	70
Mizoram	28	30	30	29
Nagaland	64	68	69	65
Orissa	1,751	1,672	1,585	1,474
Pondicherry	33	35	35	33
Punjab	869	843	816	772
Rajasthan	2,862	2,855	2,833	2,768
Sikkim	14	15	16	15
Tamil Nadu	1,845	1,771	1,690	1,585
Tripura	112	118	119	112
Uttar Pradesh	5,621	5,773	5,868	5,934
Uttaranchal	197	204	210	215
West Bengal	3,351	3,271	3,168	3,014
All India	42,322	41,637	40,626	38,986

Table 3e. Projections for blindness by age (15–49 years)

State/Union Territory	Cases			
	2000	2005	2010	2015
Andaman and Nicobar Islands	137	144	145	136
Andhra Pradesh	33,588	30,694	27,801	24,907
Arunachal Pradesh	813	846	844	785
Assam	23,423	22,690	21,963	20,744
Bihar	18,269	18,402	18,375	18,178
Chandigarh	295	310	313	296
Chhattisgarh	13,487	13,439	13,252	12,899
Dadra and Nagar Haveli	60	63	63	60
Daman and Diu	49	51	51	48
Delhi	5,100	5,559	5,820	5,687
Goa	754	783	780	725
Gujarat	16,827	16,561	16,101	15,369
Haryana	11,395	11,183	10,930	10,453
Himachal Pradesh	1,656	1,689	1,656	1,517
Jammu and Kashmir	4,946	4,952	4,745	4,227
Jharkhand	14,146	14,129	13,968	13,649
Karnataka	28,351	27,500	26,425	24,850
Kerala	6,718	6,516	6,299	5,961
Lakshadweep	15	16	16	15
Madhya Pradesh	19,656	19,664	19,481	19,070
Maharashtra	28,760	27,849	26,856	25,448
Manipur	1,093	1,139	1,140	1,065
Meghalaya	627	660	668	633
Mizoram	256	270	274	260
Nagaland	578	610	618	585
Orissa	15,763	15,047	14,261	13,270
Pondicherry	299	314	318	301
Punjab	7,819	7,586	7,344	6,946
Rajasthan	25,755	25,697	25,496	24,916
Sikkim	130	137	140	133
Tamil Nadu	16,607	15,943	15,210	14,262
Tripura	1,006	1,058	1,070	1,012
Uttar Pradesh	50,586	51,954	52,812	53,404
Uttaranchal	1,772	1,837	1,887	1,934
West Bengal	30,159	29,439	28,514	27,129
All India	380,894	374,729	365,637	350,876

was recommended that diseases that are at the global level should be concentrated on and a few more diseases were also included as the task force felt that these were important problems as far as India is concerned. Thus the diseases that fall under the priority of the India Vision 2020 are:

1. Cataract
2. Childhood blindness
3. Refractive errors and low vision
4. Corneal blindness
5. Glaucoma
6. Diabetic retinopathy
7. Trachoma (focal)

In this paper, the causes (Tables 4a–4f), interventions (Tables 5a–5f) and standard treatment protocols (Tables 6a–6f) pertain to diseases that are of priority in the NPCB Vision 2020 National Action Plan.

Table 3f. Projections for blindness by age (50 years and above)

State/Union Territory	Cases			
	2000	2005	2010	2015
Andaman and Nicobar Islands	4,937	5,173	5,206	4,898
Andhra Pradesh	1,206,662	1,102,715	998,768	894,821
Arunachal Pradesh	29,202	30,377	30,334	28,206
Assam	841,490	815,147	789,056	745,252
Bihar	656,321	661,092	660,146	653,064
Chandigarh	10,596	11,139	11,260	10,640
Chhattisgarh	484,527	482,820	476,102	463,400
Dadra and Nagar Haveli	2,166	2,261	2,279	2,152
Daman and Diu	1,754	1,827	1,850	1,732
Delhi	183,211	199,697	209,075	204,327
Goa	27,098	28,125	28,018	26,053
Gujarat	604,508	594,964	578,461	552,131
Haryana	409,388	401,760	392,655	375,527
Himachal Pradesh	59,498	60,689	59,477	54,494
Jammu and Kashmir	177,693	177,916	170,478	151,869
Jharkhand	508,211	507,602	501,803	490,358
Karnataka	1,018,546	987,971	949,354	892,759
Kerala	241,363	234,083	226,292	214,142
Lakshadweep	531	562	575	550
Madhya Pradesh	706,152	706,461	699,880	685,107
Maharashtra	1,033,214	1,000,484	964,824	914,237
Manipur	39,281	40,926	40,958	38,265
Meghalaya	22,543	23,717	24,002	22,739
Mizoram	9,194	9,693	9,838	9,338
Nagaland	20,762	21,908	22,199	21,032
Orissa	566,287	540,567	512,354	476,742
Pondicherry	10,729	11,295	11,434	10,830
Punjab	280,901	272,528	263,830	249,543
Rajasthan	925,269	923,178	915,951	895,121
Sikkim	4,679	4,931	5,020	4,775
Tamil Nadu	596,610	572,761	546,422	512,376
Tripura	36,150	38,004	38,436	36,362
Uttar Pradesh	1,817,353	1,866,484	1,897,308	1,918,591
Uttaranchal	63,660	65,982	67,805	69,464
West Bengal	1,083,477	1,057,641	1,024,386	974,631
All India	13,683,962	13,462,481	13,135,837	12,605,528

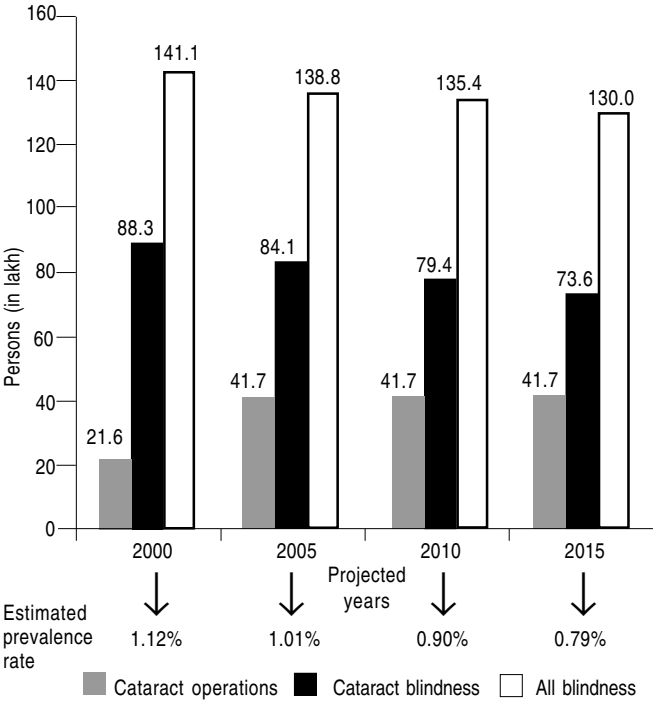

Fig. 1 Projection of blindness cases and cataract surgeries

Table 4a. Causes of cataract

	Direct (normally clinical)	Indirect (diet, exercise, alcohol)	Distant (sociopolitical, economic, empowerment, gender, literacy, etc.)
Main causes	Senile, inflammation (intraocular), traumatic, metabolic, congenital	Sun exposure, poor nutrition, acute diarrhoea in early life, smoking, alcohol	Poor socioeconomic status, feminine gender, low literacy, older age
Interaction with other causes	Corticosteroids (systemic and topical), Metallic foreign body, inflammation, intrauterine infections, rubella during pregnancy		

Sources: 1. *Quality assurance in cataract surgery, Clinical practice module.* New Delhi: NPCB, Ministry of Health and Family Welfare, Government of India; 2001. 2. Ughade SN, Khanolkar VA. Risk factors for cataract: A case–control study. *Indian J Ophthalmol* 1998;**46:**221–7.

Table 4b. Causes of refractive errors and low vision

	Direct (normally clinical)	Indirect (diet, exercise, alcohol)	Distant (sociopolitical, economic, empowerment, gender, literacy, etc.)
Main causes	Hypermetropia, myopia, astigmatism, presbyopia	Axial length of eyeball, curvature of cornea, refractive index of lens, trauma, surgery	Non-utilization of spectacles associated with lower level of education, living in rural areas, cost
Interaction with other causes	Diabetes, cataract		

Sources: 1. Quality assurance in refraction, Clinical practice module. New Delhi: NPCB, Ministry of Health and Family Welfare, Government of India; 2001.
2. Dandona R, Dandona L, Kovai V, Giridhar P, *et al.* Population-based study of spectacles use in southern India. *Indian J Ophthalmol* 2002;**50:**145–55.

Table 4c. Causes of corneal blindness

	Direct (normally clinical)	Indirect (diet, exercise, alcohol)	Distant (sociopolitical, economic, empowerment, gender, literacy, etc.)
Main causes	Trachoma (infection by *Chlamydia trachomatis*)	Poor hygiene	Poverty, poor sanitation, feminine gender, rural areas, illiteracy, low socioeconomic status
	Ocular trauma and corneal ulceration	Traditional medication, work environment, war and social unrest	
	Xerophthalmia	Measles infection, malnourishment, vitamin A deficiency, traditional eye medication	
	Ophthalmia neonatorum	Herpes simplex virus, trachoma, gonococcal infection, traditional eye medication	

Source: Whitcher JP, Srinivasan M, Upadhyay MP. Corneal blindness: A global perspective. *Bull World Health Organ* 2001;**79:**214–21.

Table 4d. Causes of glaucoma

	Direct (normally clinical)	Indirect (diet, exercise, alcohol)	Distant (sociopolitical, economic, empowerment, gender, literacy, etc.)
Main causes	Primary open-angle (intraocular pressure change, susceptibility of optic nerve head), primary angle-closure (intraocular pressure change, shallow anterior chamber angle)	Myopia, hyperopia, cataract, diabetes, thyroid dysfunction	Age, race, family history

Source: Quality assurance in management of glaucoma (primary open-angle and angle-closure), Clinical practice module. New Delhi: NPCB, Ministry of Health and Family Welfare, Government of India; 2001

Table 4e. Causes of diabetic retinopathy

	Direct (normally clinical)	Indirect (diet, exercise, alcohol)	Distant (sociopolitical, economic, empowerment, gender, literacy, etc.)
Main causes	Duration of diabetes, lack of control of blood sugar	Hypertension, hyperlipidaemia, renal and cardiac problems, smoking and alchohol, family history	Age, ethnicity, increased life expectancy, food habits, urbanization, lifestyle changes, improved socioeconomic conditions

Source: Manual on diabetic retinopathy. New Delhi: RP Centre, NPCB, Ministry of Health and Family Welfare, Government of India; 2001

Table 4f. Causes of childhood blindness

	Direct (normally clinical)	Indirect (diet, exercise, alcohol)	Distant (sociopolitical, economic, empowerment, gender, literacy, etc.)
Main causes	Corneal scar, cataract, glaucoma, optic atrophy	Premature birth, measles, congenital rubella syndrome, vitamin A deficiency, ophthalmia neonatorum	Traditional eye medication

Source: Gilbert C, Foster A. Childhood blindness in the context of Vision 2020—the right to sight. *Bull World Health Organ* 2001;**79:**227–32.

Table 5a. Interventions for cataract

Disease/condition	Medical intervention	Non-medical intervention (exercise, nutrition, others)
Senile	IOL microsurgery	Nil
Inflammation	Corticosteroids (oral and topical) and cycloplegics (topical), IOL microsurgery	Nil
Traumatic	IOL microsurgery	Protective glasses for at-risk workers
Metabolic	IOL microsurgery	Good metabolic control
Congenital	Rubella vaccination in mothers, IOL microsurgery, spectacles and review at appropriate intervals to prevent amblyopia	Nil

Source: Quality assurance in cataract surgery, Clinical practice module. New Delhi: NPCB, Ministry of Health and Family Welfare, Government of India; 2001.

Table 5b. Interventions for refractive errors and low vision

Disease/condition	Medical intervention	Non-medical intervention (exercise, nutrition, others)
Hypermetropia Myopia Astigmatism Presbyopia	Spectacle correction, contact lens, refractive surgery, low-vision aids for low-vision patients, ocular physiotherapy	Nil

Sources: 1.Quality assurance in refraction, Clinical practice module. New Delhi: NPCB, Ministry of Health and Family Welfare, Government of India; 2001.
2. Dandona R, Dandona L, Kovai V, Giridhar P, *et al.* Population-based study of spectacles use in southern India. *Indian J Ophthalmol* 2002;**50:**145–55.

Table 5c. Interventions for corneal blindness

Disease/condition	Medical intervention	Non-medical intervention (exercise, nutrition, others)
Trachoma	Azithromycin 20 mg (single dose), surgery for trichiasis	Facial cleanliness, improvement in environmental conditions
Ocular trauma and corneal ulceration	Antibiotics, corneal wound closure, keratoplasty or corneal transplant	Protective eye wear
Xerophthalmia	Vitamin A supplementation	Dietary fortification, nutritional education
Ophthalmia neonatorum	Prevention of sexually transmitted disease, antenatal screening of pregnant women, ocular prophylaxis at birth	Nil

Source: Whitcher JP, Srinivasan M, Upadhyay MP. Corneal blindness—a global perspective. *Bull World Health Organ* 2001;**79:**214–21.

Table 5d. Interventions for glaucoma

Disease/condition	Medical intervention	Non-medical intervention (exercise, nutrition, others)
Primary open-angle	Medical therapy, laser trabeculoplasty, trabeculectomy	Nil
Primary angle-closure	Medical therapy, laser iridotomy, trabeculectomy	Nil

Source: Quality assurance in management of glaucoma (primary open-angle and angle-closure), Clinical practice module. New Delhi: NPCB, Ministry of Health and Family Welfare, Government of India; 2001.

Table 5e. Interventions for diabetic retinopathy

Disease/condition	Medical intervention	Non-medical intervention (exercise, nutrition, others)
NPDR PDR (laser) PDR (surgery)	Early detection by periodic check-up, laser treatment, vitrectomy surgery and low-vision aids	Control of diabetes mellitus, regular exercise, proper food intake

NPDR: non-proliferative diabetic retinopathy; PDR: proliferative diabetic retinopathy
Source: Manual on diabetic retinopathy. New Delhi: RP Centre, NPCB, Ministry of Health and Family Welfare, Government of India; 2001.

Table 5f. Interventions for childhood blindness

Disease/condition	Medical intervention	Non-medical intervention (exercise, nutrition, others)
Corneal scar Cataract Glaucoma Optic atrophy	Refer to the respective interventions table	School vision screening, ROP screening, health and nutritional education to parents, fortification of food, vitamin A supplementation, promotion of primary eye care

Source: Gilbert C, Foster A. Childhood blindness in the context of Vision 2020—the right to sight. *Bull World Health Organ* 2001;**79:**227–32.

Table 6a. Standard treatment protocols for cataract

Condition	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay
Senile Inflammation Traumatic Metabolic Congenital	Ophthalmologist trained in microsurgery, paramedical staff (time: 1/2 hour) + Anaesthetist	Slit-lamp examination, intraocular tension, nasolacrimal duct examination, BP, urine sugar, keratometry, A-scan	Preoperative: Eye drops (antibiotic and cycloplegic), xylocaine injection Postoperative: Eye drops (antibiotic, steroids and cycloplegic), paracetamol tablets	Routine: 2 days

Source: Quality assurance in cataract surgery, Clinical practice module. New Delhi: NPCB, Ministry of Health and Family Welfare, Government of India; 2001.

Table 6b. Standard treatment protocols for refractive errors and low vision

Condition	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay
Hypermetropia Myopia Astigmatism Presbyopia	Ophthalmologist trained in low-vision aids, refractionist or optometrists, paramedical staff (time: 1/2 hour)	Visual acuity measurement, pinhole visual acuity, verification of existing glasses, retinoscopy, subjective refraction, interpupillary distance measurement	Eye drops (cycloplegic, atropine 1%, homatropine 2%, cyclopentolate 1%, tropicamide 1%)	Nil

Source: 1. Quality assurance in refraction, Clinical practice module. New Delhi: NPCB, Ministry of Health and Family Welfare, Government of India; 2001.
2. Dandona R, Dandona L, Kovai V, Giridhar P, *et al.* Population-based study of spectacles use in southern India. *Indian J Ophthalmol* 2002;**50:**145–55.

Table 6c. Standard treatment protocols for corneal blindness

Condition	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay
Trachoma Ocular trauma and corneal ulceration Xerophthalmia Ophthalmia neonatorum	Ophthalmologist trained in corneal diseases, paramedical staff (time: 1 hour)	Slit-lamp examination, intraocular tension, nasolacrimal duct examination, BP, urine sugar, posterior segment evaluation	Preoperative: Eye drops (antibiotic and cycloplegic), xylocaine injection Postoperative: Eye drops (antibiotic, steroids and cycloplegic), paracetamol tablets	5–7 days

Source: Whitcher JP, Srinivasan M, Upadhyay MP. Corneal blindness—a global perspective. *Bull World Health Organ* 2001;**79:**214–21.

Table 6d. Standard treatment protocols for glaucoma

Condition	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay
Primary open-angle Primary angle-closure	Ophthalmologist trained in diagnosis and treatment of glaucoma, paramedical staff (time: 1/2 hour)	Slit-lamp examination, intraocular tension, gonioscopy, field test	Eye drops (beta-blockers, miotics), tablets (carbonic anhydrase inhibitors), prostaglandin analogue, alpha-I receptor agonist	Nil except in case of surgery (2 days)

Source: Quality assurance in management of glaucoma (primary open-angle and angle-closure), Clinical practice module. New Delhi: NPCB, Ministry of Health and Family Welfare, Government of India; 2001.

Table 6e. Standard treatment protocols for diabetic retinopathy

Condition	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay
NPDR PDR (laser) PDR (surgery)	Ophthalmologist trained in retinal diseases, indirect ophthalmoscopy and laser photocoagulation, paramedical staff (time: 1/2 hour)	Slit-lamp examination, intraocular tension, indirect ophthalmoscopy, FFA, gonioscopy, field test	Eye drops (cycloplegic), paracetamol tablets Preoperative: Cycloplegic eye drops, xylocaine injection Postoperative: Eye drops (antibiotic and cycloplegic), paracetamol tablets	Nil except in case of surgery (3–5 days)

NPDR: non-proliferative diabetic retinopathy; PDR: proliferative diabetic retinopathy; FFA: fundus fluorescein angiogram
Source: Manual on diabetic retinopathy. New Delhi: RP Centre, NPCB, Ministry of Health and Family Welfare, Government of India; 2001

Table 6f. Standard treatment protocols for childhood blindness

Condition	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay
Corneal scar Cataract Glaucoma Optic atrophy	Ophthalmologist trained in paediatric eye care and surgery, anaesthetist for surgeries, paramedical staff (time: refer to the respective condition)	Slit-lamp examination, examination under general anaesthesia	Refer to the respective condition	Refer to the respective condition

Source: Gilbert C, Foster A. Childhood blindness in the context of Vision 2020—the right to sight. *Bull World Health Organ* 2001;**79:**227–32.

References

1. Registrar General of India. *Population projections for India and States 1996–2016*. New Delhi: Office of the Registrar General, Government of India.
2. Murthy GVS, Gupta SK, Bachani D, Jose R, John N. Current estimates of blindness in India. *Br J Ophthal* 2005;**89:**257–60.
3. National Programme for Control of Blindness, Directorate General of Health Services. *Rapid assessment of blindness in north-eastern states.* New Delhi: Ministry of Health and Family Welfare, Government of India; 2003.
4. National Programme for Control of Blindness, Directorate General of Health Services. *Rapid assessment of cataract blindness.* New Delhi: Ministry of Health and Family Welfare, Government of India; 1998.
5. Mohan M. Survey of blindness, India (1986–89): Summary and results. In: *Present status of National Programme for Control of Blindness.* New Delhi: Directorate General of Health Services, Ministry of Health and Family Welfare, Government of India; 1992.
6. Dandona L, Dandona R, Srinivas M, *et al*. Blindness in the Indian state of Andhra Pradesh. *Invest Ophthalmol Vis Sci* 2001;**42:**908–16.
7. Dandona L, Dandona R, John RK. Estimation of blindness in India from 2000 through 2020: Implications for the blindness control policy. *Natl Med J India* 2001;**14**:327–34.
8. Dandona R, Dandona L. Childhood blindness in India: A population based perspective. *Br J Ophthalmol* 2003;**87**:263–5.
9. Minasian DC, Mehra V. 38 lakh blinded by cataract each year: Projections from the first epidemiological study of incidence of cataract blindness in India. *Br J Ophthalmol* 1990;**74**:341–3.
10. Limburg H, Kumar R, Bachani D. Forecasting cataract blindness—and planning to combat it. *World Health Forum* 1996;**17**:15–20.
11. Fletcher AE, Donoghue M, Devavaram J, *et al*. Low uptake of eye services in rural India: A challenge for programs of blindness prevention. *Arch Ophthalmol* 1999;**117**:1393–9.
12. Brilliant L, Pokhrel RP, Grasset NC, Brilliant LB. *The epidemiology of blindness in Nepal*. San Rafael, CA: Seva Foundation; 1981:225–6.
13. Minassian DC, Mehra V, Johnson GJ. Mortality and cataract: Findings from a population-based longitudinal study. *Bull World Health Organ* 1992;**70**:219–23.

Appendix 1

Table A1.1 Estimated and projected prevalence rates of blindness

State/Union Territory	Estimated prevalence (Visual acuity < 6/60) in the general population (%)					
	2000	2002	2004	2005	2010	2015
Andaman and Nicobar Islands[1]	1.12	1.10	1.03	1.01	0.90	0.79
Andhra Pradesh	1.45	1.42	1.33	1.31	1.16	1.02
Arunachal Pradesh	2.33	2.28	2.14	2.10	1.87	1.64
Assam	3.11	3.05	2.87	2.81	2.50	2.20
Bihar	0.80	0.78	0.73	0.72	0.64	0.56
Chandigarh[2]	1.03	1.01	0.95	0.93	0.83	0.73
Chhattisgarh	1.64	1.61	1.51	1.48	1.32	1.16
Dadra and Nagar Haveli[3]	0.97	0.95	0.89	0.87	0.78	0.68
Daman and Diu[4]	1.09	1.07	1.01	0.98	0.88	0.77
Delhi	1.15	1.13	1.06	1.04	0.93	0.81
Goa	1.55	1.52	1.43	1.40	1.25	1.09
Gujarat	1.09	1.07	1.01	0.98	0.88	0.77
Haryana	1.93	1.89	1.78	1.74	1.55	1.36
Himachal Pradesh	0.71	0.70	0.66	0.64	0.57	0.50
Jammu and Kashmir	1.64	1.61	1.51	1.48	1.32	1.16
Jharkhand	1.44	1.41	1.33	1.30	1.16	1.02
Karnataka	1.82	1.78	1.67	1.64	1.46	1.28
Kerala	0.57	0.56	0.53	0.52	0.46	0.40
Lakshadweep[5]	0.57	0.56	0.53	0.52	0.46	0.40
Madhya Pradesh	1.18	1.16	1.09	1.07	0.95	0.84
Maharashtra	0.97	0.95	0.89	0.87	0.78	0.68
Manipur	1.41	1.38	1.30	1.27	1.13	0.99
Meghalaya	0.75	0.74	0.70	0.68	0.61	0.53
Mizoram	0.80	0.78	0.73	0.72	0.64	0.56
Nagaland	1.07	1.05	0.99	0.97	0.86	0.76
Orissa	1.43	1.40	1.32	1.29	1.15	1.01
Pondicherry[6]	0.80	0.78	0.73	0.72	0.64	0.56
Punjab	1.03	1.01	0.95	0.93	0.83	0.73
Rajasthan	1.58	1.55	1.46	1.43	1.27	1.12
Sikkim	0.66	0.65	0.61	0.60	0.53	0.47
Tamil Nadu	0.80	0.78	0.73	0.72	0.64	0.56
Tripura	0.79	0.77	0.72	0.71	0.63	0.55
Uttar Pradesh	0.96	0.94	0.88	0.86	0.77	0.68
Uttaranchal	0.57	0.56	0.53	0.52	0.46	0.40
West Bengal	1.21	1.19	1.12	1.09	0.98	0.86
India	1.12	1.10	1.03	1.01	0.90	0.79

Note: 1. Andaman and Nicobar Islands (All-India prevalence %)
2. Chandigarh (Punjab prevalence %)
3. Dadra and Nagar Haveli (Maharashtra prevalence %)
4. Daman and Diu (Gujarat prevalence %)
5. Lakshadweep (Kerala prevalence %)
6. Pondicherry (Tamil Nadu prevalence %)

Table A1.2 Age- and sex-specific proportion of blindness prevalence

Age group (years)	Males (%)	Females (%)
0–14	0.10	0.20
15–59	1.30	1.40
60+	45.60	51.40
Total	47.00	53.00

Source: National Blindness Survey (1986–1989)[5] and Andhra Pradesh Eye Diseases Study (APEDS)[6–8]

Appendix 2

Number of cataract surgeries performed between April 2003 and March 2004

State/Union Territory	Census population 2001	Growth rate	Estimated population 2003	Total cataract operations performed	Cataract surgery rate (per 1,000,000 population)	IOL operations performed	% IOL
Andaman and Nicobar	356,265	2.39	373,294	693	1,856	657	94.8
Andhra Pradesh	75,727,541	1.30	77,696,457	443,091	5,703	409,808	92.5
Arunachal Pradesh	1,091,117	2.33	1,141,963	684	599	608	88.9
Assam	26,638,407	1.73	27,560,096	23,063	837	11,509	49.9
Bihar	82,878,796	2.50	87,022,736	87,876	1,010	30,757	35.0
Chandigarh	900,914	3.39	961,996	6,330	6,580	5,850	92.4
Chhattisgarh	20,795,956	1.66	21,486,382	64,196	2,988	53,574	83.5
Dadra and Nagar Haveli	220,451	4.65	240,953	0	NA	0	NA
Daman and Diu	158,059	4.42	172,031	372	2,162	298	80.1
Delhi	13,782,976	3.81	14,833,239	80,072	5,398	76,229	95.2
Goa	1,343,998	1.39	1,381,361	5,497	3,979	0	NA
Gujarat	50,596,992	2.03	52,651,230	449,234	8,532	426,772	95.0
Haryana	21,082,989	2.47	22,124,489	89,706	4,055	62,902	70.1
Himachal Pradesh	6,077,248	1.62	6,274,151	18,343	2,924	13,917	75.9
Jammu and Kashmir	10,069,917	2.55	10,583,483	10,412	984	0	NA
Jharkhand	26,909,428	2.09	28,034,242	28,054	1,001	8,416	30.0
Karnataka	52,733,958	1.59	54,410,898	263,613	4,845	243,657	92.4
Kerala	31,838,619	0.90	32,411,714	79,696	2,459	74,619	93.6
Lakshadweep	60,595	1.59	62,522	0	NA	0	NA
Madhya Pradesh	60,385,118	2.18	63,017,909	233,870	3,711	197,176	84.3
Maharashtra	96,752,247	2.04	100,699,739	519,561	5,160	484,558	93.3
Manipur	2,388,634	2.63	2,514,276	553	220	517	93.5
Meghalaya	2,306,069	2.62	2,426,907	1,238	510	1,214	98.1
Mizoram	891,058	2.56	936,680	796	850	687	86.3
Nagaland	1,988,636	4.97	2,186,306	429	196	326	76.0
Orissa	36,706,920	1.48	37,793,445	82,652	2,187	78,590	95.1
Pondicherry	973,829	1.87	1,010,250	9,969	9,868	9,888	99.2
Punjab	24,289,296	1.80	25,163,711	133,376	5,300	75,722	56.8
Rajasthan	56,473,122	2.49	59,285,483	226,829	3,826	143,560	63.3
Sikkim	540,493	2.85	571,301	253	443	0	NA
Tamil Nadu	62,110,839	1.06	63,427,589	452,650	7,136	439,194	97.0
Tripura	3,191,168	1.46	3,284,350	8,098	2,466	5,858	72.3
Uttar Pradesh	166,052,859	2.30	173,691,291	567,718	3,269	322,861	56.9
Uttaranchal	8,479,562	1.76	8,778,043	37,105	4,227	31,317	84.4
West Bengal	80,221,171	1.64	82,852,425	249,895	3,016	233,057	93.3
Total	1,027,015,247	1.93	1,067,062,942	4,175,924	3,913	3,444,098	82.5

Source: National Programme for Control of Blindness

Appendix to Section II: Services, equipment, personnel and drugs required at different levels of health care for non-communicable diseases

BARIDALYNE NONGKYNRIH*, CHERIAN VARGHESE†

The following schemes are indicative and may be modified as per local requirements and available resources.

Appendix A1

Services, equipment, drugs and personnel required at the district level

Table A1.1 Services to be provided at the district level

Services	Stroke	CVD	Cancer	COPD
Screening and diagnosis	• Identification of signs and symptoms of acute stroke, transient ischaemic attack • Screening for hypertension, diabetes, use of oral contraceptive pills, etc. • Detailed investigation: CT scan in all cases, ECG, pulse oximetry, 2D-ECHO, X-ray, lipid profile	• Non-invasive screening (history, tobacco use, BMI, waist circumference, etc.) • Screening for hypertension, diabetes mellitus • Investigations: ECG, X-ray, lipid profile, ECHO	• Opportunistic screening for oral, breast and cervical cancers • Cytology—FNAC, Pap smear • Haematology, biochemistry, ultrasound • Endoscopy, colposcopy, X-ray, mammogram	• Screening by suggestive respiratory signs and symptoms • Rule out TB—examine the sputum for AFB • X-ray spirometry
Management				
Acute/emergency	• Inpatient care • Management of blood pressure with parentral agents • Supportive care • Prophylaxis for DVT • Acute rehabilitation • Refer to a tertiary care centre in case of significant, pressure effects, or surgical candidates with haemorrhage	• Evaluate the haemodynamic status • Thrombolysis • Inpatient care for uncontrolled hypertension with end-organ complications	• Control of bleeding and pain • Superior vena caval obstruction	• Intensive care for acute exacerbations • Antibiotics for suspected infection • Controlled oxygen therapy
Routine	• Prescription of multiple drugs and anticoagulants • Tobacco cessation for users	• Tobacco cessation for users • Treatment of hypertension, diabetes	• Tobacco cessation for users • Minimal surgical interventions • Chemotherapy • Pain relief and palliative care	• Start on bronchodilators • Give antibiotics, if needed
Follow-up	Health education, follow-up for compliance, investigations and change of prescriptions if needed, referral of complicated cases to a tertiary-level centre, and rehabilitation			

CVD: cardiovascular disease; COPD: chronic obstructive pulmonary disease; CT: computerized tomography; ECG: electrocardiogram; FNAC: fine-needle aspiration cytology; Pap: Papanicolaou; TB: tuberculosis; AFB: acid-fast bacilli; DVT: deep vein thrombosis

Source: Background papers submitted to NCMH 2005

*Centre for Community Medicine, All India Institute of Medical Sciences, New Delhi; e-mail: bari_111@rediffmail.com
†WHO India Office, New Delhi; e-mail: varghesec@whoindia.org

The material presented in this Annexure are the views of the authors and do not imply the expression of any opinion whatsoever on the part of the organization they belong to.

Table A1.2 Equipment required for NCD services at the district level

General

- Thermometer
- Weighing machine
- Height measuring board
- Stethoscope
- Sphygmomanometer
- Oxygen mask
- Oxygen cylinder
- Ambu bag
- Infusion sets
- Sterilizer
- Spotlight
- Laryngoscope
- Endotracheal tube
- Ophthalmoscope
- Otoscope
- Surgical tools
- Electrocardiogram (ECG)
- X-ray machine
- Ultrasound machine

Specialized

- Nebulizer and spacer
- Ventilator
- Cardiac monitor
- External pacemaker
- Defibrillator
- ECHO cardiogram machine
- Mammogram
- Spirometer
- Peripheral Doppler
- Sigmoidoscope
- Proctoscope
- Punch biopsy forceps
- Nasal speculum
- Cusco speculum
- Sim speculum
- Bronchoscope
- Upper gastrointestinal endoscope
- Colonoscope
- Dental chair and accessories

Laboratory equipment

- Microscope
- Glucometer
- Uristix
- Haematolgy analyser
- Biochemistry autoanalyser

Source: Background papers submitted to NCMH 2005

Table A1.4 Personnel required at the district level

- Specialists (medicine, obstetrics and gynaecology, anaesthesiologist, surgery, ENT, dental surgeon, radiologist, pathologist)
- General duty medical officers
- Technicians: Laboratory technicians, ECG, X-ray
- Cytotechnologist
- Nurses
- Counsellor, dietician, physiotherapist

Source: Background papers submitted to NCMH 2005

Table A1.3 Drugs required at the district level

- 5-Flurouracil
- ACE inhibitors
- Acid-inhibiting drugs
- Aldactone
- Aminophylline
- Analgesics
- Antibiotics
- Anticoagulants
- Antiepileptics
- Antispastic drugs—Baclofen, Tizanide
- Aspirin
- Atenolol
- Atorvastatin
- Atropine
- Benzathine penicillin
- Biguanides
- Bleomycin
- Calcium-channel blockers
- Chlorambucil
- Cisplatin
- Clopidogrel
- Corticosteroids
- Cyclophosphamide
- Digoxin
- Dobutamine
- Doxorubicin
- Etoposide
- Folic acid
- Formoterol
- Frusemide
- Heparin (Inj.)
- Insulin
- Ipratropium
- Low molecular-weight heparin
- Methotrexate
- Methycobol with alpha lipoic acid
- Metoprolol
- Morphine
- Nitrates (oral, Inj.)
- Nitroglycerine
- Oral anticoagulants
- Salbutamol
- Salmeterol
- Statins
- Streptokinase (Inj.)
- Sulphonylureas
- Tamoxifen
- Terbutaline
- Theophylline
- Thiazides (oral)
- Tiotropium
- Vinblastine
- Vincristine

Source: Background papers submitted to NCMH 2005

Appendix A2
Services, equipment, drugs and personnel required at the subdistrict level

Table A2.1 Services to be provided at the subdistrict level

Services	Stroke	CVD	Cancer	COPD
Screening and diagnosis	• Identification of signs and symptoms of acute stroke, transient ischaemic attack • Screening for hypertension, diabetes, use of oral contraceptive pills, etc. • Investigations: ECG, X-ray, lipid profile	• Non-invasive screening (history, tobacco use, BMI, waist circumference, etc. • Screening for hypertension, diabetes • Investigations: ECG, X-ray, lipid profile	• Opportunistic screening for oral, breast and cervical cancers • Investigations: Haematology, biochemistry, X-ray	• Screening by suggestive respiratory signs and symptoms • Rule out TB—examine the sputum for AFB • Investigation: X-ray
Management				
Acute/emergency	ABC of resuscitation If not equipped to carry out acute management or in case of unstable/deteriorating condition, refer immediately	• Evaluate the haemodynamic status • Thrombolysis • Inpatient care for uncontrolled hypertension	Pain relief and palliative care	• Intensive care for acute exacerbations • Antibiotics for suspected infection • Controlled oxygen therapy
Routine	• Prescription of multiple drugs and anticoagulants • Tobacco cessation for users	• Tobacco cessation for users • Treatment of hypertension, diabetes	• Tobacco cessation for users • Minimal surgical interventions • Pain relief and palliative care	• Start on bronchodilators • Give antibiotics, if needed
Follow-up	Health education, follow-up for compliance, investigations and change of prescriptions if needed, referral of complicated cases to a tertiary-level centre, and rehabilitation			

CVD: cardiovascular disease; COPD: chronic obstructive pulmonary disease; ECG: electrocardiogram; BMI: body mass index; AFB: acid-fast bacilli
Source: Background papers submitted to NCMH 2005

Table A2.2 Equipment required for NCD services at the subdistrict level

General
- Thermometer
- Weighing machine
- Height measuring board
- Stethoscope
- Sphygmomanometer
- Oxygen mask
- Oxygen cylinder
- Ambu bag
- Infusion sets
- Sterilizer
- Spotlight
- Laryngoscope
- Endotracheal tube
- Ophthalmoscope
- Otoscope
- Surgical tools
- Electrocardiogram (ECG)
- X-ray machine

Specialized
- Nebulizer and spacer
- Spirometer
- Proctoscope
- Indirect laryngoscope (IDL) with light source
- Punch biopsy forceps
- Nasal speculum
- Cusco speculum
- Sim speculum
- Dental chair and accessories

Laboratory
- Microscope
- Sahli haemoglobinometer
- Glucometer
- Semi-autoanalyser for biochemistry

Source: Background papers submitted to NCMH 2005

Table A2.3 Drugs required at the subdistrict level

- ACE inhibitors
- Acid-inhibiting drugs
- Aldactone
- Aminophylline
- Analgesics
- Antibiotics
- Anticoagulants
- Antiepileptics
- Antispastic drugs—Baclofen, Tizanide
- Aspirin
- Atenolol
- Atorvastatin
- Atropine
- Benzathine penicillin
- Biguanides
- Calcium-channel blockers
- Clopidogrel
- Corticosteriods
- Digoxin
- Dobutamine1
- Folic acid
- Frusemide
- Heparin (Inj.)
- Insulin
- Metoprolol
- Morphine
- Nitrates (oral and injectables)
- Nitroglycerine
- Oral anticoagulants
- Salbutamol
- Salmeterol
- Statins
- Streptokinase (Inj.)
- Sulphonylureas
- Terbutaline
- Theophylline
- Thiazides (oral)

Source: Background papers submitted to NCMH 2005

Table A2.4 Personnel required at the subdistrict level

- Specialists (medicine, obstetrics and gynaecology, anaesthesiologist, surgery, ENT, dental surgeon)
- General duty medical officers
- Technicians: Laboratory technician, ECG, X-ray
- Cytotechnologist
- Nurses
- Counsellor, dietician, physiotherapist

Source: Background papers submitted to NCMH 2005

Appendix A3
Services, equipment, drugs and personnel required at the primary health care level

Table A3.1 Services to be provided at the primary health care level

Services	Stroke	CVD	Cancer	COPD
Screening and diagnosis	• Identification of signs and symptoms of acute stroke, transient ischaemic attack • Screening for hypertension, diabetes, use of oral contraceptive pills, etc.	• Non-invasive screening (history, tobacco use, BMI, waist circumference, etc. • Screening for hypertension, diabetes	Cancer-related physical examination and appropriate referral	• Screening by suggestive respiratory signs and symptoms • Rule out TB
Management				
Acute/emergency	ABC of resuscitation If not equipped to carry out acute management or in case of unstable/deteriorating condition, refer immediately	• Evaluate the haemodynamic status • Oral nitrates • Aspirin	Pain relief and palliative care	• Management of acute exacerbations • Antibiotics for suspected infection
Routine	Prescription for secondary prevention	• Secondary prophylaxis for rheumatic heart disease • Tobacco cessation for users	Tobacco cessation for users	Bronchodilators and antibiotics
Follow-up	Health education, follow-up for compliance along with refill of medicines, referral of complicated cases, and rehabilitation			

CVD: cardiovascular disease; COPD: chronic obstructive pulmonary disease; BMI: body mass index; TB: tuberculosis
Source: Background papers submitted to NCMH 2005

Table A3.2 Equipment required at the primary health care level

General

- Thermometer
- Weighing machine
- Height measuring board
- Stethoscope
- Sphygmomanometer
- Oxygen mask
- Oxygen cylinder
- Ambu bag
- Infusion sets
- Sterilizer
- Spotlight
- Laryngoscope
- Endotracheal tube
- Ophthalmoscope
- Otoscope
- Electrocardiogram (ECG), if possible

Specialized

- Nebulizer and spacer
- Cusco speculum
- Sims speculum

Laboratory

- Microscope
- Glucometer
- Uristix

Source: Background papers submitted to NCMH 2005

Table A3.3 Drugs required at the primary health care level

- ACE inhibitors
- Acid-inhibiting drugs
- Aldactone
- Aminophylline
- Analgesics
- Antibiotics
- Anticoagulants
- Antiepileptics
- Antispastic drugs—Baclofen, Tizanide
- Aspirin
- Atenolol
- Atropine
- Benzathine penicillin
- Biguanides
- Calcium channel blockers
- Corticosteriods
- Digoxin
- Dobutamine
- Folic acid
- Frusemide
- Insulin
- Metoprolol
- Nitrates (oral and injectables)
- Nitroglycerine
- Oral anticoagulants
- Salbutamol
- Sulphonylureas
- Terbutaline
- Theophylline
- Thiazides (oral)

Source: Background papers submitted to NCMH 2005

Table A3.4 Personnel required at the primary care level

- General duty medical officer
- Multipurpose health workers
- Laboratory technician

Source: Background papers submitted to NCMH 2005

Note:

Primary level: Health posts, clinics, primary health centres and other basic health units.

Secondary level: Hospitals that deal more with difficult cases and those referred by primary health care centres.

Tertiary level: Hospitals where complicated cases are referred from the secondary level centres and which have more advanced levels of diagnostic and treatment facilities.

Section III

Injury

Injuries in India: A national perspective

GURURAJ G.

India is passing through a major sociodemographic, epidemiological, technological and media transition. The political, economic and social changes have altered the health scenario. In the past two decades, India has witnessed rapid urbanization, motorization, industrialization and migration of people resulting from socioeconomic growth and development. With mechanization and revolution in technology, traditional ways of living and working are being altered. Injuries are a major public health problem in India. Lack of reliable and good quality national or regional data has thwarted their recognition. Many injuries are linked to social, environmental, cultural and biological issues in causation; recognized as man-made and behaviour-linked disorders and linked to sociodemographic transition. Prevention, acute and long-term care, and rehabilitation are the major challenges faced today.

Motorization in India

The rapid and unprecedented motorization in India combined with the lack of a safety environment has been a noticeable feature. Figure 1 shows that the number of vehicles has grown from a mere 306,000 in 1951 to 58,863,000 by 2002 (Ministry of Road Transport and Highways, Transport Research Wing 2001–02). The 23 metropolitan cities account for 33% of total vehicles in India. Two-wheelers; cars, jeeps and taxis; buses; goods vehicles; and others account for 71%, 13%, 1%, 5% and 10%, respectively of the total vehicle population (Fig. 2). While the total number of buses increased from 331,000 in 1991 to 669,000 in 2002 (an increase of 102%), two-wheelers increased from 14,200,000 to 41,478,000 (an increase of 300%). Rapid and accelerated motorization has been witnessed in some States such as Andhra Pradesh (4,336,000), Gujarat (6,008,000), Madhya Pradesh (7,414,000), Tamil Nadu (5,658,000) and Uttar Pradesh (5,171,000) as compared with other States. During 2001–02, nearly 3,473,401 two-wheelers were added on the roads of Andhra Pradesh, while the number of cars added was 279,903, with similar patterns across other States. Correspondingly, in Bangalore, nearly 1,253,408 two-wheelers were added in 2001–02 as against 11,267 buses and 234,888 cars. Nearly 35/1000 persons own a two-wheeler while only 6/1000 are in possession of a car (CSO 2004).

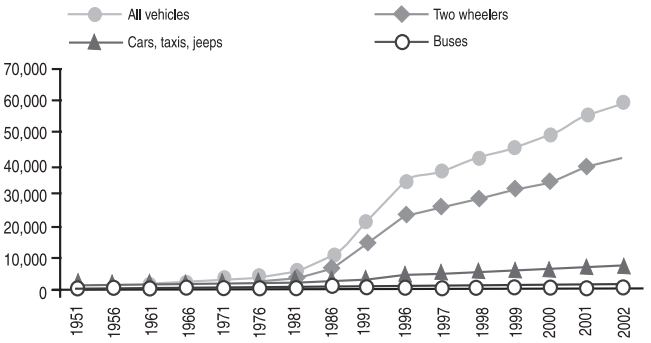

Fig. 1 Motorization pattern in India, 1951–2002

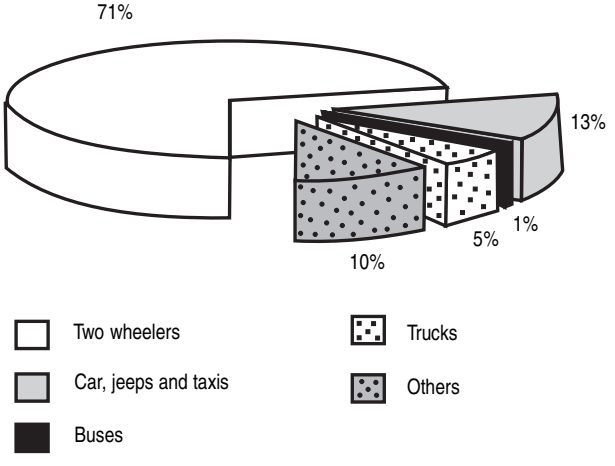

Fig. 2 Distribution of vehicles in India 2002

Department of Epidemiology
WHO Collaborating Centre for Injury Prevention and Safety Promotion
National Institute of Mental Health and Neuro Sciences
Bangalore 560029
e-mail: guru@nimhans.kar.nic.in

Injuries: An emerging public health problem

The term 'injury' by definition means that there is a body lesion due to an external cause, either intentional or unintentional, resulting from a sudden exposure to energy (mechanical, electrical, thermal, chemical or radiant) generated by agent–host interaction. This leads to tissue damage, when it exceeds the physiological tolerance of the individual (Robertson 1983; Baker 2000). On the contrary, injury can also occur due to the sudden withdrawal of a vital requirement of the body, e.g. withdrawal of air in drowning. Thus, an injury is damage to a body organ which occurs rapidly and is visible, with the causative mechanism being sudden energy transfer (Barss *et al.* 1998). Four factors that differentiate injury from other health conditions are: (i) a definite interaction between agent–host and environment, (ii) acuteness of the event, (iii) varying severity, and (iv) chances of repetitiveness (Bangdiwala 2000).

Information from around the world indicates that injuries account for more than half the deaths in the age group of 5–44 years. An examination of 'years of potential life lost' indicates that injuries are the second most common cause of death after 5 years of age in India (Mohan and Anderson 2000). Like any other health problem, injuries also have a definitive causative pattern and mechanism in terms of agent (product/vehicle), host (human beings) and environmental (roads, homes, workplaces) factors along with system-related issues. A precise understanding of this mechanism is crucial to develop and implement mechanisms for prevention and control of injuries. Every year, injuries contribute to a significant number of deaths, hospitalizations (for short and long periods), emergency care, disabilities (physical, social and psychological), amputations, disfigurement, pain, suffering and agony. Many children become orphans, women become destitute and the elderly grieve in isolation. In addition, injuries also result in disruption of several activities leading to loss of work, income, education and other social activities, causing long-term suffering among survivors and families. The extent of economic loss is yet to be recognized due to lack of systematic research. As India moves forward in its quest for growth, development and economic prosperity, the dark and ugly side of this progress is rapidly emerging due to the absence of accompanying safety systems.

Global burden of injuries

Nearly 50 lakh people lost their lives due to injury as per WHO estimates during the year 2002 (WHO 2004a). Injuries caused 9% of the total deaths. The global injury mortality rate is estimated to be 98/100,000 population, with male and female rates of 128/100,000 (38 lakh deaths) and 67/100,000 (19 lakh deaths), respectively (WHO 1999). Five of the top ten causes of death globally are due to injuries. Among the total disability-adjusted life-years (DALYs), 13% were due to injuries. Unintentional and intentional injuries contributed to three-fourth and one-fourth of total DALYs, respectively. Among unintentional injuries, road traffic injuries (RTIs), falls and burns resulted in, respectively, 29%, 12% and 9% of total DALYs. In the intentional group, suicide and violence accounted for 41% and 43% of total DALYs, respectively (WHO 2003a). The WHO–World Bank Report, which reviewed the disease transformation scenarios, indicates that RTIs will be the third leading cause of mortality by 2020, moving up from their present ninth position. Similarly, suicide and violence will move from the twelfth and sixteenth to tenth and fourteenth positions by 2020 (Murray and Lopez 1996).

Burden of injuries in India

The precise number of deaths and injuries due to specific causes, or any scientific estimates of injury deaths in India are not available from any single source. The National Crime Records Bureau (NCRB) is the principal nodal agency under the Ministry of Home Affairs, Government of India, and is responsible for the collection, compilation, analysis and dissemination of injury-related information (NCRB 2001a, 2001b). As per the report of 2001, 2,710,019 accidental deaths, 108,506 suicidal deaths and 44,394 violence-related deaths were reported in India. There has been an increase in accidental deaths from 122,221 to 188,003, from 40,245 to 78,450 for suicidal deaths and from 22,727 to 39,174 for violence-related deaths between 1981 and 1991. The injury mortality rate was 40/100,000 population during 2000. The number of deaths due to accidents increased by 47% during the period 1990–2000; 93% were due to unnatural causes and 7% (17,366) due to natural causes. The mortality rate among different age groups was: 8.2% (<14 years), 62% (15–44 years), 20% (45–59 years) and 9.2% (>60 years). Seventy-three per cent of total deaths occurred among men, with a ratio of 3:1 between men and women (Figs 3a–c). Significant regional variations were noticed across States. The 23 metropolitan cities (population of >10 lakh)

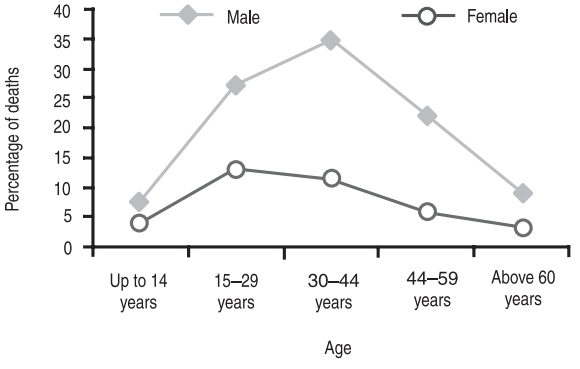

Fig. 3a Age–sex distribution of deaths due to injuries in Indian reports (%)
Source: National Crime Records Bureau (NCRB), 2000

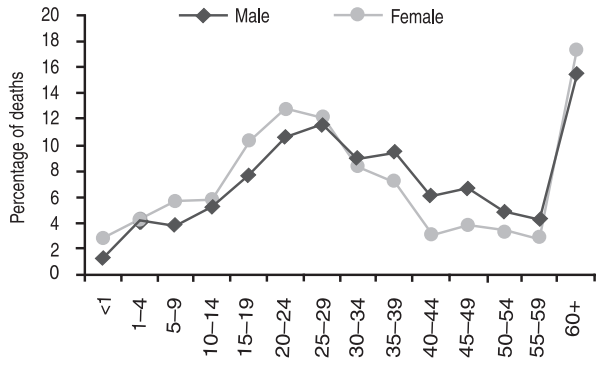

Fig. 3b Age–sex distribution of deaths due to injuries in Indian reports (%)

Source: Survey of Causes of Death (SCD), 1998

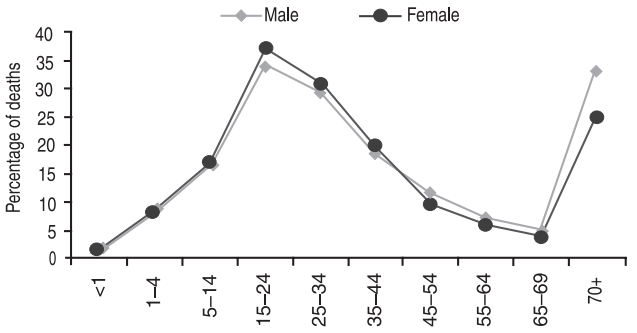

Fig. 3c Age–sex distribution of deaths due to injuries in Indian reports (%)

Source: Medical Certification of Causes of Death (MCCD), 1998
Note: Peaks in the 60+ years age group under SCD and MCCD are due to the combination of further age subgroups

accounted for 12% of deaths and 13% of injuries. The precise number of people hospitalized and injured is not available for intentional injuries. RTIs (20%), suicide (27%), violence-related deaths (11%), burns (9%), poisoning (6%) and drowning (6%) were the major causes of injury deaths (Table 1).

The Survey of Causes of Death (SCD) under the Sample Registration System (SRS; Registrar General of India 1998b) of India examined causes of death from 1602 of the 2059 (selected) primary health centres (PHCs) covering 40,351 deaths. The rate of injury-related deaths increased from 9% of total deaths to 11% between 1994 and 1998. Sixty-eight per cent of total deaths were in the age group of 5–44 years, with a male to female ratio of 1.5:1.

The Medical Certification of Causes of Death (MCCD; Registrar General of India 1998a) survey covered 15% of

Table 1. Distribution of external causes of injuries in national reports and population-based studies from India (%)

Year	Place	Author/ Agency	Source of data	RTIs	Domes-tic	Burn	Poison-ing	Fall	Animal-related	Work-related	Suicide	Vio-lence	Drown-ing	Other
2001	India	National Crime Records Bureau	National report based on police records	20	2	9	6	2	0.1	0.1	27	11	6	14.8
1998b	India	Registrar General of India (SRS)	National report based on cause of death by verbal autopsy	20	—	9	3	6	7	—	25	4	10	8
1998a	India	Registrar General of India (MCCD)	National reports based on medically certified death	22	—	37	9	6	—	2	10	12	3	—
Population-based studies														
1990	Haryana	Varghese	Rural, community-based study	14	33	—	—	—	—	36	—	6	—	10
2003b	New Delhi	WHO	Urban, community-based study	25	—	10	1	34	6	13	0.3	6	0.7	3
1998	Faridabad Haryana	Verma	Rural, community-based study	29	—	17	2	25	12	—	—	—	2	14
Hospital-based studies														
2004	Mumbai, Mahara-shtra	Murlidhar and Nobhojit	Urban, commu-nity-based study	39	—	—	—	29	—	1	—	3	—	1
2004b	Bangalore, Karnataka	Gururaj *et al.*	Urban, rural, urban slum, community-based study	52	12	3	—	13	7	4	2	3	—	4

RTIs: road traffic injuries; SRS: Sample Registration System; MCCD: Medical Certification of Causes of Death

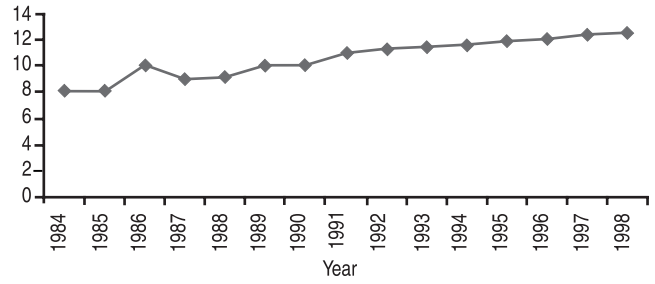

Fig. 4 Trends of death due to injuries, Medical Certification of Causes of Death (MCCD), 1984–98 (%)

total and 41% of urban deaths. Injuries accounted for 12.1% of total deaths in 1998 (*n*=60,577), an increase from 8% in 1984 (Fig. 4). Among total reported deaths (*n*=498,586), injuries and poisoning were the fourth leading causes of death. Sixty-nine per cent of deaths occurred in the age group of 5–44 years. Injuries were the leading cause of death in the age group of 15–24 years (13,309; 35%), second leading cause of death in 5–14 years (3003; 16.2%), and 25–34 years (15,330; 30%), third in 35–44 years (10,352; 19%) and fourth in 45–54 years (6238; 11%). RTIs (22%), burns (37%), violence (12%), suicide (10%) and falls (6%) were the leading causes of deaths.

A selective examination of injury studies from hospitals reveals that injuries account for 10%–30% of total registrations, with case-fatality rates of 5%–20% in different studies in India. A few population-based surveys with varying sample sizes reveal a significantly higher incidence and mortality rates of injuries compared with national figures (Table 2). Varghese *et al.* (1990), in a survey of 25,000 people from rural Haryana, revealed the incidence to be 8%. Sathyasekaran (1996) in a study of 4333 slum-dwellers in Chennai observed an incidence rate of 12.7%, with rates in males and females being 13.7% and 11.8%, respectively. Ashok *et al.* (2004), in a study of 720 households, noticed that nearly 5% of the population had suffered a hospital-registered injury in the past year. From Delhi, in a study of 30,554 individuals, an incidence rate of 11.3% was observed (WHO 2003b); 6% without long-term disabilities and 1% with disabilities. In the largest population-based survey from Bangalore, covering 96,569 individuals from 19,969 households, the injury incidence rate was 12% in the total population; 10% each in urban and slum areas and 14% in rural areas. The injury mortality rates were 5/1000, with rates in urban, slum and rural areas being 4, 5.1 and 7.4/1000, respectively (Gururaj and Suryanarayana 2004; Aeron Thomas *et al.* 2004). These studies (Table 2) clearly indicate that while mortality rates are 5–10/1000 population, the injury incidence rates vary from 70 to 140/1000, with a ratio of 1:14 in India. Due to recall bias, many of these surveys might have excluded minor injuries. In all studies, higher rates have been observed among men, with an overall injury ratio of 3:1. Few of the studies have reported higher rates from rural areas compared with urban areas (14% *v.* 10%) (Gordon *et al.* 1962; Varghese 1990; Gururaj and Suryanarayana 2004).

Road traffic injuries

On an average, 3242 persons die each day around the world in road crashes (Road Peace 2003). As per WHO estimates, nearly 12 lakh people died in road crashes in 2002 (WHO 2004a). The overall global mortality rate was 19/100,000 with nearly 90% of these occurring in low- and middle-income countries. The motorization of India, especially during the past two decades, has resulted in greater number of deaths and injuries due to absence of safety policies, programmes and environmental norms.

About 70%–80% of people in high-income countries (HICs) have cars and only 5%–10% have motorized two-wheelers. Whereas in India cars comprise around 10% of the total vehicles while 75% are motorized two-wheelers. Consequently, pedestrians, two-wheeler occupants and bicyclists are killed and injured in greater numbers in India (Table 3). Although buses and trucks constitute only 7%–10% of the total vehicles, they are associated with nearly 30%–50% of total deaths. The 23 metropolitan cities have an RTI mortality rate of 1000±200/lakh, which is higher than the national average of 800/lakh.

In 2001, road accidents caused 353,100 injuries and 80,262 deaths, with a male to female ratio of 5:1, and mortality and incidence rates of 8/100,000 and 34/100,000, respectively (NCRB 2001a). Deaths due to road accidents increased in India from 40,000 in 1986 to 85,000 by 2001 (Fig. 5). Nearly two-thirds of deaths occurred among those 16–44 years of age, with the highest rate among 30–44-year-olds (35%). The death rates among children and the elderly were 9% and 7%, respectively. The 10 States recording highest number of deaths caused by road accidents were Tamil Nadu (15.8%), Maharashtra (12.4%), Kerala (11.5%), Karnataka (10.1%), Andhra Pradesh (7.9%), Gujarat (7.2%), Madhya Pradesh (7.1%), Rajasthan (6.5%), Tripura (4.3%) and Uttar Pradesh (3.4%). The 23 metropolitan cities accounted for 12% of total deaths. Among cities, the highest number of deaths occurred in

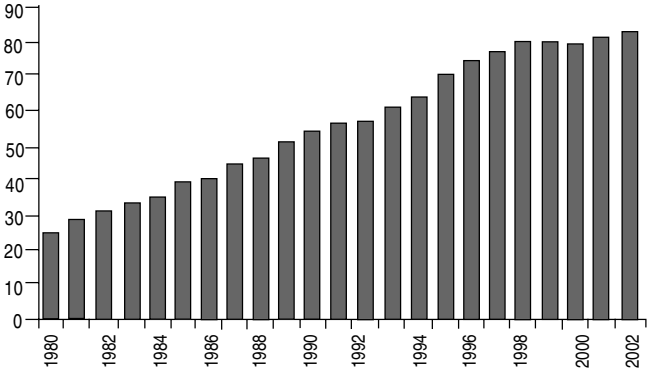

Fig. 5 Deaths due to road accidents in India, 1980–2002

Table 2. Incidence and mortality rates of injuries in India (includes all types of injuries)

Author/Agency	Place	Year	Total no. of subjects/Total population	Source of data	Injury incidence		Injury mortality	
					Number	Rate	Number	Rate
National Crime Records Bureau	India	2001	All India	Information from police records	870,839	87/100,000 per year	391,504	40/100,000 per year
Registrar General of India (SCD)	India	1998b	40,351 deaths from 1602 PHCs	Based on cause of death lay reporting	Injuries were responsible for 8% of total deaths (8.4% of deaths among males and 6.7% among females)*			
Registrar General of India (MCCD)	India	1998a	498,586 deaths from 2129 hospitals	Medically certified death	Injuries and poisoning contributed to 12% of total deaths (11.8% of deaths among males and 12.8% among females)			
Population-based studies								
Gordon *et al.*	Punjab	1962	4377 rural population	Rural, community-based study	—	115/1000	—	—
Varghese	9 villages of Haryana	1990	25,000 population from 3500 households	Rural, follow-up study	2164	80/1000	—	—
Sathyasekaran	Chennai, Tamil Nadu	1996	4333 slum dwellers	Urban slum, cohort study	542	127/1000 Men: 137; Women:118	—	—
Ashok *et al.*	Bangalore, Karnataka	2004	3538 population from 720 households	Urban, community-based study	210	51/1000	—	—
WHO	Delhi	2003b	30,554 population from 5412 households	Urban, community-based study	3566	Total 116/1000 62/1000 without disability 9/1000 with disability	46	2/1000
Gururaj *et al.*	Bangalore, Karnataka	2004b	96,569 population from 19,919 households	Urban, rural and urban slum, community-based study (hospitalized injured persons only)	Total=1112 Urban=324 Slum=343 Rural=445	Total =12/1000 Urban=10/1000 Slum=11/1000 Rural=14/1000	Total=53 Urban=13 Slum=16 Rural=24	(/100,000) Total=55 Urban=40 Slum=51 Rural=74
Verma	Faridabad, Haryana	1998	1095 population from 215 households	Rural, community-based study	102	93/1000	—	—
Hospital-based studies								
Sidhu *et al.*	Patiala, Punjab	1993	2482 cases	Urban, hospital-based study	2482	9% of total hospital admission	208	8%
Gururaj *et al.*	Bangalore, Karnataka	2000a	—	Urban, hospital-based study	—	23% of emergency room registrations	—	—
Goel *et al.*	Lucknow, Uttar Pradesh	2004	180 trauma cases	Urban, hospital-based study	—	—	55	31%

SCD: Survey of Causes of Death; MCCD: Medical Certification of Causes of Death
*No rates could be calculated due to the absence of population figures

Delhi (1736), Mumbai (1362), Chennai (761) and Bangalore (659). In these places both the human and the vehicle population have been growing at the rates of 39% (compared with 23% for India) and 15% (compared with 2.5/1000 for India), respectively.

The SCD (Registrar General of India 1998b) revealed that 2.6% of total deaths were due to vehicular accidents. The highest number of deaths were reported in those 25–34 years of age (21%), followed by 15–24-year-olds (19%), 35–44-year-olds (16%), 45–54-year-olds (15.3%), those above 60 years of age (14%), 5–14-year-olds (11.2%), 1–4-year-olds (2.5%) and those below 1 year of age (1.3%). Vehicular accidents were the tenth leading cause of death in the overall ranking. The MCCD (Registrar General of India 1998a) reported that vehicular accidents resulted in 1.5% of total (n=7258) and 16% of injury deaths. The distribution of RTIs revealed that 675 of 1161 deaths occurred in those 5–44 years of age. The incidence of RTIs in hospital emergency rooms varied from 23% to 53% in a few studies, with a male preponderance in every setting (Sidhu *et al.*1993; Verma *et al.* 2004; Gururaj 2000). The case-fatality rate varied from 5% to 10% depending on the available facilities. The incidence of RTIs in community-based studies varied from 649 in Bangalore to 2857/100,000

Burden of injuries 2000–2015

Estimating the burden of injuries is crucial for understanding the magnitude of the problem, developing mechanisms for intervention, allocating physical, human, financial resources for control of the problem, and for reducing the burden of injuries in the coming years. A review of Indian studies and observations by other agencies indicate the ratio of deaths to serious injuries needing hospitalization to minor injuries as 1:20:50. In Bangalore and Haryana this ratio was 1:18:50 and 1:29:70, respectively (Gururaj *et al.* 2000b; Varghese and Mohan 2003). A recent population-based survey of health behaviour surveillance in Bangalore has shown the ratio of completed:attempted:suicidal ideation to be 1:8:20 (Gururaj *et al.* 2004). A large-scale population-based survey of 96,569 individuals from Bangalore revealed a ratio of 1:20:40 for deaths:hospitalizations:injuries (Gururaj and Suryanarayana 2004). Thus, based on a conservative ratio of 1:20:50 for deaths, serious injuries and mild injuries, it is estimated that injuries will contribute to nearly 850,000 deaths during the year 2005, and nearly 17,000,000 persons would be hospitalized (for short or long periods). Further, nearly 42,500,000 persons would have minor injuries, incapacitating them for short or long periods during 2005 (detailed calculations are available in the original report [Gururaj 2005c]). Nearly 70% of these deaths and injuries would occur among men 15–44 years of age. Eighty per cent of these deaths and injuries would occur in rural areas, where health care is poor and deficient. One-third of disabilities are due to injuries with an estimated 70 lakh persons suffering from various disabilities. If no systematic efforts are introduced and implemented, the number of deaths due to injuries is likely to increase to 11 lakh by 2010 and 12 lakh by 2015.

India injury pyramid, 2005

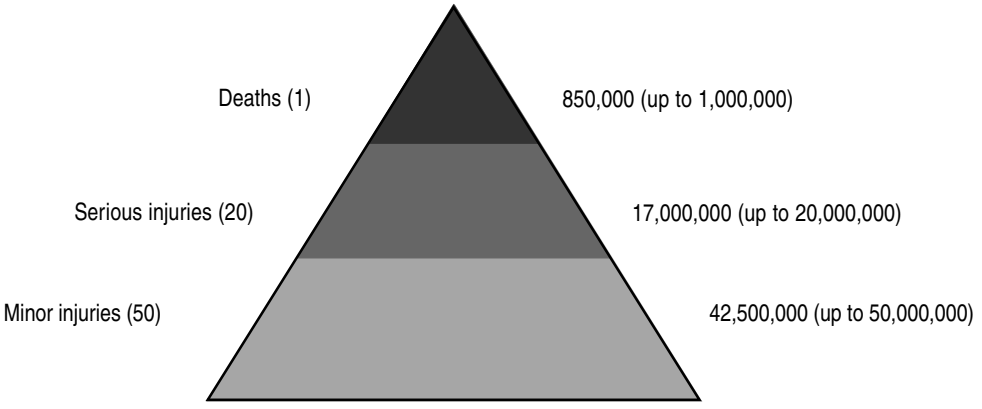

Table 3. Categories of road users injured and killed in road accidents in India

Author(s) and year	Place	Pedestrians	Two-wheeler occupants	Bicyclists	Three-wheeler occupants	Four-wheeler occupants	Heavy vehicle drivers and passengers	Others
National Crime Records Bureau 2001a and 2001b	New Delhi	9	11	3	4	17	46	7
Sidhu *et al.* 1993 (dead)	Patiala, Punjab	16	17	8		58		
Sidhu *et al.* 1993 (injured)	Patiala, Punjab	14	29	12		43		
Mohan and Bawa 1985	New Delhi	33	16	21	3	3	4	10
Jha *et al.* 2003	Pondicherry	23	23	23		10	15	7
Maheswari and Mohan 1989	New Delhi	26	39	12		1		14
Gururaj *et al.* 1993	Bangalore, Karnataka	31	35	10		1	21	3
Sathyasekaran 1991	Chennai, Tamil Nadu	28	15	29		8	4	4
Gururaj *et al.* 2000a	Bangalore, Karnataka	26	44	4		5	21	
Gururaj *et al.* 2005a	Bangalore, Karnataka	26	43	8		3	7	13
Colohan *et al.* 1989	New Delhi	20	22	1		25		32
Sahadev *et al.* 1994	New Delhi	33	40	6		4		17

in Delhi (Gururaj and Suryanarayana 2004; Jha *et al.* 2003; Varghese 1990; Sathyasekaran 1996; Gururaj 2004a; WHO 2003a). The RTI mortality varied from 33 to 56/100,000 population in different places. The ratio of deaths to injuries varied from 1:20 in Bangalore and Haryana to 1:67 in Delhi. In a recent population-based survey of RTIs and other injuries in Bangalore, the mortality and incidence rates were found to be 11 and 649/100,000, respectively. Nearly 60% of brain injuries are caused by RTIs as revealed by studies from Bangalore and Delhi (Gururaj *et al.* 1993, 2005a; Colohan *et al*. 1989). The incidence of traumatic brain injuries alone is 150/100,000, with a mortality rate of 20/100,000 and a case-fatality rate of 10% (Gururaj 2000).

Police reports capture nearly 90% of deaths and 50% of severe injuries. A recent report of the working group on RTIs estimated the ratio of deaths to serious injuries needing hospitalization to minor injuries registering in emergency rooms of hospitals to be 1:15:70 (Planning Commission 2003). This amounted to 80,000 deaths, 12 lakh hospitalized persons and 56 lakh sustaining minor injuries in the year 2000. If there is a 5% increase in RTIs every year, it is estimated that in 2004, nearly 100,000 persons would have died; 15 lakh sustained serious injuries and about 70 lakh received care for minor injuries (Mohan 2004; Gururaj 2005).

Severity and nature of RTIs

The severity, nature and outcome of road crashes is determined by the impact of the crash; the amount of energy transferred to the host; physiological factors such as age, sex, fragility of body organs; presence of protective devices such as helmets, seat belts, child restraints; nature and speed of vehicle-impacting crash; and availability, affordability and accessibility to health care. Rautji and Dogra in a study of 127 autopsy reports noticed that in a majority of cases, exsanguination (31%) and brain injury (11%) were the major causes of early deaths, while sepsis and multiorgan failure contributed to late deaths (Rautji and Dogra 2004). Sahadev *et al.* (1994) in an autopsy study of 177 RTI deaths noticed that neurological injury and haemorrhagic shock were responsible for 60% and 25% of deaths, respectively. It was concluded that 23% of deaths were preventable, 46% possibly preventable and 30% not preventable by any intervention. The average Injural Severity Score (ISS) for mortality was 37.8 in the series. As per the study of Sidhu *et al.* (1993) in Patiala, 41.3% of those dead had polytrauma and 25% had brain injury. Polytrauma was observed in 21% of those injured in road accidents (Gururaj *et al.* 2005a). Information from the Bangalore City Police for RTI deaths during 2002 reveals that 26% of victims died at the crash site, 12% during transit to hospital and 62% during or after their hospital stay (Gururaj, personal communication with the Bangalore City Police).

Varghese (1990), in a population-based survey, observed that only 11% had sustained injuries of Abbreviated Injury Score (AIS) category 2 or 3 while 87% were in AIS 1 category. Studies at NIMHANS (Gururaj *et al.* 1993; Gururaj *et al.* 2005a; Gururaj *et al.* 2000a), Bangalore revealed that minor, moderate and severe brain injuries (due to RTIs) were recorded in 60%–65%, 16%–20% and 15%–20% of cases as per the Glasgow Coma Scale (GCS) grading. Mortality was higher among those with severe brain injuries. Polytrauma was documented in 1%–21% of cases. Facial, chest, abdominal and limb injuries were documented in 48%, 3%, 1% and 10% of cases, respectively. Bharati *et al.* (1993) in Meerut reported that based on the GCS grading, 32% had severe, 25% moderate and 42% minor brain injuries at the time of admission. Mortality rates were higher among those with severe brain injuries (41%). The study at Chennai by Sathyasekaran (1991) revealed that among the victims of road accidents, 11% had life-threatening injuries, 11% had serious disabling injuries and 38% had mild disabling injuries. Thirty-eight per cent of injured persons had a serious injury to the head and face region.

Occupational injuries

The past two decades have witnessed the expansion of industries in India. Of the total employed population in the country during 2001, 17.8% (270 lakh) was in the organized sector (Registrar General of India 2001). Among the 830 lakh engaged in the unorganized sector (82.2%), agriculture was the major activity, followed by manufacturing, retail trade and other activities.

As per the International Labour Organization (ILO) (1994) estimates, nearly 2 lakh workers die annually and about 1200 lakh are injured. Nearly 50% of these deaths and injuries occur in developing countries. The fatality rates are estimated to be 30–43/100,000 in these countries, which are much higher than those in developed countries. In India, occupational injuries contributed to 2% of total deaths, 1.8% of total life-years lost due to disabilities and 2% of DALYs in 1990 (Sudhir 1998). It is estimated that 19 fatal and 1930 (1:100) non-fatal accidents occur annually per 100,000 workers (Nag and Patel 1998). The incidence of industrial injuries among employed workers was 9/1000, with a frequency of 2.6 per 100,000 man-days work (CSO 2004). As per the NCRB report of 2001, 667 people were killed in factory/machine accidents. Related deaths in other occupational categories include 446 deaths in mine/quarry disaster, 220 deaths due to leakage of poisonous gases and several work-related deaths in traffic accidents. According to the report, 2346 deaths occurred due to collapse of structures, the source of which could be work related.

A limited number of population-based epidemiological studies reveal that occupational injuries constitute approximately 10% of total deaths due to injuries and 20%–25% of all injuries. Mohan (1992) in a study of industrial workers

reported a death rate of 6/1000 workers. Varghese *et al.* (1990), in a cohort of 25,000 people from 9 villages of Haryana, observed the incidence rate of work-related injuries to be 31% over a one-year period. From a study of 2682 workers in Digboi, Assam, Sharma *et al.* (2001) reported that nearly 35% of total injuries occurred at the workplace. An incidence of 3.6/1000 workers/year was reported from Jaipur by Mathur and Sharma (1988). Malhotra *et al.* (1995) observed the incidence to be 4.1% among 2008 workers in a hydroelectric project in the Shivali range of Jammu and Kashmir. The injury incidence rate was 2% in a study in Chennai among 4333 slum dwellers (Sathyasekaran 1996). In a recent study by WHO in municipal areas of Delhi, it was seen that 2% of total injuries were work related (WHO 2003b).

Across studies, the highest number of injuries occur among men and in the economically productive age group of 21–49 years. In India, 25%–30% of injuries occur in those 16–20 years of age, 30%–45% in those 21–35 years of age and about 30% in those 36–49 years of age.

No information is available on agricultural injuries in India. Adarsh *et al.* (1998) revealed an incidence rate of 28% in phase 1 and 49% in phase 2 in a sample of 2635 workers from 9 villages of Uttar Pradesh and 30 villages of Haryana. In a review of equipment-related injuries in Indian agriculture, it was observed that 5% and 46% of injuries are caused by tractors and hand-held equipment (Adarsh *et al.* 2000). In a longitudinal study of 12,189 agricultural workers by Tiwari *et al.* (2002) in Madhya Pradesh during 1995–99, the incidence rate was 1.25/1000 workers/year. Nearly 9.2% of the incidents were fatal and 43% each were caused by tractors and snake bites. Seventy-eight per cent of all injuries were due to farm machinery, 12% due to hand tools and 11% due to other causes. Mohan and Patel (1992), in an epidemiological study in Haryana, identified 576 agricultural injuries in one year; of these, 87% were minor, 11% moderate and 2% severe injuries. It was estimated that agricultural activities caused 5000–10,000 deaths, 15,000–20,000 amputations and 150,000–200,000 serious injuries every year in Haryana, Punjab and Madhya Pradesh alone. Mittal *et al.* (1996) reported from Punjab that 9% of agricultural injuries were fatal and 91% non-fatal. The incidence rate (per 1000 machines/year) was highest for tractors (23.7), sprayers (15.5), electric motors (7.1), threshers (5.7) and cutters (2.2).

Children comprise nearly 10%–20% of the workforce in developing countries (ILO 1994). In India, 25% of children work in hazardous places especially in rural areas, slums and the unorganized urban labour sector. Both community- and hospital-based studies in India reveal that nearly 10%–15% of injuries occur among children (Mathur and Sharma 1988; Malhotra *et al.* 1995). Children are primarily involved in manual jobs exposing them to physical injuries. Banerjee (1993) in a study of 500 agricultural child workers reported an injury incidence rate of 57%.

There is no centralized agency in India to examine occupational injuries. Workers are exposed to many hazards resulting in musculoskeletal injuries. Occupational deaths are listed under general medical conditions and the underlying causes are not documented and reported; hence, the precise extent of occupational injuries is difficult to establish.

Burns and fire-related injuries

Burn injuries can be accidental, suicidal and homicidal (for details see under Injury causation, p. 337). Depending on the extent and severity of burns, and the availability and accessibility to health care, the impact of burns varies from superficial burns and scalds to damage of the internal body organs. Absence of facilities in district and peripheral hospitals, combined with traditional unscientific household practices and lack of safety systems result in high mortality and disability from burn injuries. Secondary complications of burns leading to contractures, deformities and disfigurement are extremely common. Secondary infections could lead to a number of complica-tions resulting in delayed recovery and death.

During 2001, 32,509 persons died in India due to burn injuries. This amounts to 15% of unnatural deaths (12,120 burns-related deaths classified as suicide are not included here). The various causes of burns were: electrocution (7%, *n*=5570), explosion (2%, *n*=666), fire (71%, *n*=23,043) and firearms (10%, *n*=3230). The total number of injured were 6030, indicating that burn injuries are highly under-reported (32,509 deaths *v.* 6030 injured persons). The mortality due to burn injuries was 3.5/100,000 population. The highest number of injuries occurred in the age group of 15–44 years (72%). More women suffered burn injuries compared to men (1.6:1) in all age groups, except among those 44–59 years of age (NCRB 2001).

Burns contributed to 1% of total deaths (*n*=398) and 15% of total injury deaths under the SCD, with a male to female ratio of 4:1. Under the MCCD, burns accounted for 12.5% of total deaths, with a male to female ratio of 1:2.2. Burns were responsible for 27% of total injury deaths; 15.6% were in males and 53.5% in females, respectively. Studies on hospital-based burns reveal that nearly 4%–12% of total trauma registrations are of patients with burns. The age distribution reveals that the incidence and mortality of burns are the highest in those 10–44 years of age, with 65% occurring in 15–39-year-olds. Studies undertaken in various hospitals have revealed higher admissions of women with burns, varying from 58% to 83% (Ghuliani *et al.* 1988 (83%); Singh *et al.* 1998 (62%); Kumar *et al.* 2000 (75%); Ahuja *et al.* 2002 (58%); Sharma *et al.* 2002 (75%); and Batra 2003 (80%). A population-based survey of 30,554 people in New Delhi revealed the mortality and incidence due to burns to be 10/100,000 and 955/100,000 population/year, respectively (WHO 2003b). A recent population-based study from Bangalore covering 96,569 individuals from

19,919 households reported an incidence of 2500/100,000, with a higher rate in slum (4100/100,000) and rural areas (2300/100,000) (Gururaj and Suryanarayana 2004).

Mortality on account of burns is influenced by several factors such as severity, level of care and nature of study. Mortality due to burns has varied from 8% to 56% in hospital series. Based on data from police records, the case-fatality rates varied from 6.3% among men to 44% among women in Wardha, Maharashtra. Sharma *et al.* (2002) reported a case-fatality rate of 25% in a study of burn injuries during 1994–2001. Subramanyam (1996) reported a case-fatality rate of >70% in severe burns, which is similar to that of other studies.

One of the major determinants of outcome of burn injuries is the severity of body involvement. In Indore, Madhya Pradesh, the mortality rate was 22% among hospitalized subjects with burns (Mukerji *et al.* 2001). In a study of burn injuries at Solhapur, Maharashtra, 70% of patients with >70% burns died, while only 6% died among those with <40% burns (Subramanyam 1996). Singh *et al.* (1998) from Chandigarh observed that 56% of cases had >80% burns. Septicaemia, neurogenic shock and hypovolaemic shock caused death in 55%, 28% and 15% of cases, respectively. Kumar (2000) observed that among those with burn injuries, 63% had an involvement of <20% of the body surface area. Sepsis (35%) and multiorgan failure (26%) were the major causes of death (Kumar 2000). Ahuja and Bhattacharya (2002) in New Delhi noticed that 47% of patients had >50% burns. Even among those with 60% burns, the mortality was only 6% if they reached a hospital early and received good-quality first aid at the site of injury. The major causes of death were resuscitation failure, inhalation injury or infections.

Burns-related injuries are frequent during the festival of lights (Diwali) in India. A study from two hospitals in New Delhi revealed that children were injured in greater numbers while lighting crackers. In addition, many of the injured were unaware that the application of cold water soon after suffering burns was helpful (Mohan and Varghese 1990).

Poisoning

Poisoning may be accidental, commonly suicidal and sometimes homicidal, the precise distinction of which is based on the intent and skills of investigating agencies. With the usage of pesticides since the beginning of the 1950s in agriculture, deaths and injuries due to poisoning have increased. Poisoning commonly occurs with adulterated food and alcohol, pesticides, herbicides, rodenticides, a variety of drugs such as sedatives, anxiolytics, hypnotics and barbiturates. Some types of poisoning such as those due to aluminium phosphide and plant extracts result in deaths and injuries in specific parts of India. Leakage of various poisonous gases and chemicals in industrial settings also results in a large number of cases of accidental poisoning. Poisoning by animal bites, especially snake and scorpion bites, is extremely common in rural India.

As per the NCRB reports (2001a and b), 24,775 persons lost their lives due to poisoning and only 1989 were reported as injuries (underreporting!). There were 15,304 (63.6%) deaths among males and 8773 (26.4%) among females, with a ratio of 1.8:1. Two-thirds of the total poisoning deaths were in the age group of 15–44 years. Food poisoning/accidental intake of insecticides (40.3%), consumption of spurious liquor (2.4%), leakage of poisonous gases (0.5%), snake/animal bite (27.6%) and others (29.4%) were the commonest types of poisoning. As per the SCD (Registrar General of India 1998b), poisoning caused 5% of total injury deaths, and varied from State to State. Poisoning was more common among men (63%) and in younger age groups. The MCCD report (1998a) examined 498,586 deaths from urban India and noticed that poisoning contributed to 8331 deaths among males and 4307 females, respectively, accounting for 2.5% of total deaths (2.7% among males and 2.3% among females). Within this group, poisoning and toxic effects accounted for 22.7% of deaths among males and 18% among females. Some of the hospital- and community-based studies (Gururaj 2005) reveal that those in the age group of 15–44 years are involved in higher numbers. Nearly 10%–25% of emergency room registrations (Sharma 2002) and 2%–3% of total admissions are poisoning-related. Case-fatality rates vary from 2% to 5%, with very high rates in case of aluminium phosphide poisoning.

Drowning

Drowning in India commonly occurs in rivers, ponds, lakes and wells and can be accidental, suicidal or sometimes homicidal in nature. The entire coastal belt of India is a risk-prone area. Owing to easy access to water bodies, the occupation of individuals, occurrence of natural calamities at frequent intervals, the risk-prone nature of young children and adolescents, drowning is common in India. Drowning as a suicidal method is also responsible for a significant number of deaths and is discussed under suicide.

According to the NCRB (2001a and b), 20,739 deaths (5.6% of total injury deaths) and 355 injuries (under-reporting!) were reported due to drowning in 2001; the male and female rates were 71% and 29%. Eighty-two per cent of deaths were in the age group of 15–59 years. The SCD report (1998b) reveals that 1.1% of total deaths and 18% of total injury deaths were due to drowning. Drowning was one of the top 10 killers among children 5–14 years of age (7.2%). A recent study from Vellore, Tamil Nadu among 106,000 people, reported sex-specific rates of 37 and 14 per 100,000 population among men and women, respectively (Bose *et al.* 2000). The incidence of drowning increased from 31/100,000 in 1991 to 44/100,000 population by 1997.

Falls and domestic injuries

Studies on domestic injuries are virtually non-existent in India (underreporting!). The type of domestic injury is often determined by several host factors (age, sex, residence, co-morbidity, alcohol and drugs, etc.), agent factors (a number of domestic products which are commonly used by people for day-to-day activities) and environmental factors (type of housing, flooring, roofing, safety environment, etc.). Studies on traumatic brain injuries at NIMHANS reveal that falls are the second leading cause of deaths and injuries contributing to 20%–30% of total traumatic brain injuries (Gururaj *et al*. 1993; Gururaj 2005). Nearly two-thirds of falls occur at home. Children and the elderly account for 30%–40% and 10%–20% of the total falls. Falls often result in variety of musculoskeletal injuries including fractures. The outcome of the fall is mainly dependent on the nature of the landing surface, height of fall and use of any protective devices.

As per the MCCD reports, fractures constituted 7.5% of total injuries; fractures of the skull and face, and lower limbs accounted for 52% and 24%, respectively. Seventy-seven per cent of these occurred in the age group of 15–44 years, with a male to female ratio of 3:1. Sathyasekaran (1996) noticed that the incidence of domestic injuries was 55/1000, 52/1000, 61/1000 and 56/1000 in the age groups of 0–14 years, 16–30 years, 31–45 years and 45+ years, respectively. A survey of 759 households in Bangalore revealed that domestic injuries accounted for 6% of total injuries (Ashok *et al*. 2004). A large-scale community-based survey in Bangalore revealed that domestic injuries accounted for 14% of total injuries, with the majority occurring among children and the elderly (Gururaj and Suryanarayana 2004). Common household objects were responsible for all injuries.

Animal-related injuries

Injuries due to dog-, scorpion- and snake-bites are common. In a recent WHO-sponsored community-based study of 52,731 people from urban and rural areas, the incidence of rabies was 1.4/1000 and 1.8/1000 in urban and rural areas, respectively. The prevalence of rabies was 2/100,000 in the study population. The highest incidence of rabies was in those 10–44 years of age (72%) (Sudarshan 2004). In a community-based study in Delhi of 30,554 individuals, the incidence of animal bites was 2.5/1000 for minor injuries and 5.3/1000 for major injuries, with an overall rate of 8/1000/year (WHO 2003b).

Suicide

Suicide is defined as 'the human act of self-inflicting one's own life cessation' (Shneidman 1985). Due to complex medicolegal associations and stigma, suicide has always been concealed in Indian society, severely underreported and misclassified in official reports.

The NCRB (2001a and b) reported the death of 108,506 persons due to suicide, with an incidence rate of 11/100,000/year during 2000 (Fig. 6). From nearly 46,008 suicides in 1974, the number of cases of suicide have nearly tripled during the past three decades. Suicide accounted for 28% of total injury deaths in 2001, with a male to female ratio of 2:1. The State-wise distribution revealed that Pondicherry (50), Kerala (29), Karnataka (24), Tripura (25), Tamil Nadu (18), Maharashtra (15), West Bengal (17), Goa (17), Andhra Pradesh (13) and Sikkim (15) reported higher than the national average rate of 11/100,000. The 23 metropolitan cities of India contributed to 9% of total suicides, revealing that a large majority of suicides occur in rural areas, *taluks* and districts of India. The cities of Bangalore, Chennai, Delhi and Mumbai recorded nearly half (49%) the number of suicides reported from other cities. In the age groups of 15–29 years and 30–44 years, the rates of suicides were 36% and 34%, respectively. Hanging (26%), poisoning (38%), fire/self-immolation (11%) and drowning (8%) were the commonest methods of committing suicide in India.

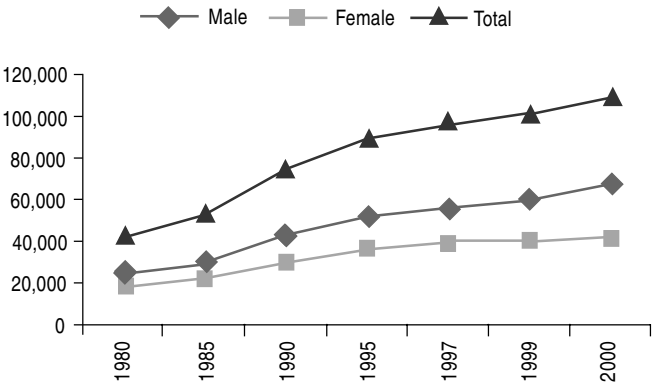

Fig. 6 Changing pattern of suicide in India, 1980–2000

The SCD, covering 26 States, revealed that suicide accounted for 3.2% of total deaths, 25% of total injury deaths, and is the ninth leading cause of death in India. Of the total of 1107 suicides, 28.8%, 26.8%, 16.3%, 14.2% and 11.4% were in the age groups of 15–24 years, 25–34 years, 35–44 years, 45–59 years and 60+ years, respectively. Suicide was the leading cause of death among women 15–44 years of age (11.3%). The MCCD survey, covering 498,586 medically certified deaths, revealed that suicide (n=3032) accounted for 8% of total injury deaths (n=54,709).

Several researchers in India have examined suicide based on analyses of police data. These studies reveal regional differences, with suicide rates, which vary from 8/100,000 to 95/100,000 population (Gururaj and Isaac 2001a). The majority of studies are based on analyses of police or hospital records with the assumption that all suicides are reported to the police and misclassifications do not exist. An in-depth community-based study in Vellore, Tamil Nadu (Aaron *et al*. 2004) reported the incidence to be 95/100,000 population based on verbal autopsy methods. The male and female rates were 58 and 148/100,000, indicating a three-fold higher rate among women. A recent study from

Bangalore observed that the incidence was 35/100,000 population for completed suicide and 250–300/100,000 for attempted suicide, with 60% of deaths occurring in the age group of 15–34 years (Gururaj and Isaac 2001a). These studies clearly indicate that many cases of suicide may not be reported to the police. Several hospital-based reports have revealed the incidence of suicide among the hospital-based population to vary from 5% to 15% of the total emergency and medical admissions (Gururaj *et al.* 2005b and c).

Violence

The WHO (2002b) defines violence as 'the intentional use of physical force or power, threatened or actual, against oneself, another person, or against a group or community, which either results in or has a high likelihood of resulting in injury, death, psychological harm, maldevelopment or deprivation'. The types of violence are broadly classified as self-directed violence (deliberate self-harm or suicide), interpersonal violence (family and intimate partner violence and communal violence) and collective violence (social, political and economic violence). The nature of violent acts could be physical, sexual, psychological and involving deprivation or neglect. In 2000, an estimated 16 lakh people died as a result of violence globally, with an age-adjusted rate of 29/100,000 population. Half of these were suicides, one-third homicides and one-fifth war-related deaths (WHO 2002b).

The epidemiology of violence in India has been least understood, since violence is not considered a health problem. As per the NCRB reports, 44,394 persons were killed and 191,340 injured, with an annual mortality and incidence of 5/100,000 and 20/100,000 population, respectively during 2001. The problem, pattern and causes of violence vary significantly across rural/urban areas, between ages and sexes, and in different socioeconomic groups. The incidence of crimes as per the Indian Penal Code (IPC) in Kerala, Rajasthan, Madhya Pradesh, Tamil Nadu, Gujarat and Karnataka was higher than the national average of 177/100,000 population. Similarly, 15 of the 23 metro-politan cities reported higher rates of crime compared with the national average.

Indian cities contributed to only 10% of violence-related deaths and injuries. Among the total crimes, 13% were violent and nearly 50% of these had affected an individual's life directly and indirectly. The age groups of 18–30 years and 31–50 years accounted for 42% and 40% of deaths, respectively. Homicides registered an increase from 22,149 in 1980 to 44,394 by 2001. In 2001, an increase of 4.1% in the crime rate against women was officially recorded in India. Nearly 142,375 episodes of violent acts were reported among women during 2001. Rape (11%), dowry deaths (5%) and cruelty by husbands and relatives (35%) were the common patterns. The rate in urban India, especially in cities, was 19/100,000 which is much higher than the national figures. The offenders were known to victims in 87% of instances, 30% of them being neighbours. In the SCD (1998b), homicides constituted 1% of total deaths (n=476) and 7% of total injury deaths. The highest number of deaths (82%) occurred in the age group of 15–44 years, with a male to female ratio of 4:1. As per the MCCD report, 3535 deaths were related to acts of violence during 1998, resulting in 8% of injury deaths. The male to female ratio was 3:2. The highest number of deaths occurred in the age group of 15–44 years (79%).

In a recent examination of domestic violence in the cities of Thane, Bangalore and Mumbai (International Center for Research on Women 2000), it was observed that women subjected to violence approached official agencies only as a last resort (Haq 2000). As per a pioneering study undertaken in seven India cities (n=9938) during 1997–99, domestic violence is common in India. Overall, 45% of women reported experiencing at least one physical or psychological violent act in their lifetime. Thirteen per cent reported being subjected to physical violence (kicking, beating, burning) more than three times. In the total sample, 15% reported sexual abuse in the past 12 months. Among women subjected to violence, 30% felt ashamed to seek help, 30% were managed at home and 30% did not have economic access to help (International Clinical Epidemiologists Network 2000). In a recent study undertaken by Yuganatar Education Society, Nagpur (Planning Commission 2004), 1250 families were studied from the States of Andhra Pradesh, Chhattisgarh, Gujarat, Madhya Pradesh and Maharashtra; 250 families were selected from each State on a random basis. Seventy-eight per cent of women were in the age group of 18–40 years. Shockingly, 84% had experienced violence in one form or the other; 74% had experienced physical violence and 10% sexual abuse. Nearly 90% had undergone emotional abuse regularly. Seventy-three per cent had been subjected to violence for more than a year (Planning Commission 2004).

The extent of violence in population-based studies is observed to vary from 10% to 20% across studies (Gururaj *et al.* 2005c). However, studies using qualitative research methods, which are focused and in-depth in nature, indicate that nearly one-third to half of women reported experiencing violence at regular intervals. Even studies among men have revealed that nearly 20%–30% had agreed that they had inflicted violence on their spouses (Narayana 1996).

Some hospital-based studies have reported the rate of violence to vary from 8% to 77% in different States. Nearly 5% of hospital-based mortality was due to violence as per the study conducted by Sidhu *et al.* 1993 in Patiala. The only autopsy study from Manipal, Karnataka revealed that violence was the cause of death in 31% of total autopsies (Kumar *et al.* 2000). Studies on traumatic brain injuries have reported that nearly 10%–15% of hospital admissions and 5% of mortality are related to violence (Gururaj *et al.* 1993, 2005a).

Impact of injuries

Economic impact

An injured person has to spend resources for care at different levels—before reaching the hospital and after discharge for transport, drugs, admission, investigation and interventions, depending on the place of care. The rehabilitation costs can be huge in certain types of injuries such as RTIs, burn injuries, violence and work-related injuries. Loans taken or savings spent put a strain on the resources of the family. With much of the care in public hospitals being subsidized, the costs to the public health care system can be enormous. The majority of the survivors with moderate and severe grades of injuries and their families experience life-long psychosocial impact and have a poor quality of life. Damage to goods, property and vehicles lead to repair costs based on the extent of damage. Work absenteeism leads to loss of productivity and indirect losses to the employer. In addition, related costs in medicolegal cases can be huge. Even costs towards funeral expenses may be substantial for a family with no income. Health care system costs in terms of investments in manpower, equipment, infrastructure and supplies are phenomenal and occupy 70%–80% of the allocated budget under trauma care. Due to lack of research, no comprehensive data are available from India on the impact of injuries.

Recent estimates from Global Road Safety Programme indicate the costs of RTIs alone to be 1% of the total GDP. Nearly 75% of RTI victims were earning members, with 9%–45% being sole earners (Ghee *et al.* 1997). In a recent study by Mohan (2004), the economic impact of RTIs was estimated to be Rs 55,000 crore, nearly 3% of the GDP, much higher than the 2% in high-income countries. In a population-based survey of 96,569 individuals from 19,919 households in Bangalore, the incidence of mortality due to RTIs was 23/100,000, with rates in poor and non-poor communities being 31 and 17/100,000, respectively (Aeron Thomas *et al.* 2004). The incidence of serious injuries was 212/100,000, with a higher rate among poor communities (238 *v.* 186). The majority of households reported a decline in earnings after injury; further, many had to borrow money from external sources for survival and only 5% received compensation from insurance agencies or their employers. The poor spent Rs 6000–25,000 (average Rs 18,000), while the non-poor spent Rs 32,000 (average Rs 27,000) on medical costs. The costs of property and vehicle damage varied from Rs 10,000 to Rs 25,000 across different groups. The economic loss due to nearly 108,595 suicides, and attempted suicide and suicidal ideators (10 and 100 times the number of suicides) has never been examined in a comprehensive way (Gururaj and Isaac 2001a and b). Similarly, the economic impact of violence resulting in nearly 40,000 homicides and a large number of other acts of violence is also not clearly known. It can be 'guesstimated' that injuries cost nearly 5% of the GDP for India.

Injury and disabilities

Disabilities due to injuries will increase significantly in the coming years (Suresh 1997). Disabilities of speech, hearing, neurological functions, vision, locomotor activities and psychosocial functioning are direct outcomes of injury. The pattern of injury, nature of body organs injured, extent of damage, and availability and utilization of rehabilitation services influence and determine the type and extent of disability. There is no information available in this area from India, except isolated studies in individual settings. However, nearly 100% of those with severe injuries, 50% of those with moderate injuries and 10%–20% of those with minor injuries carry disabilities of a physical and/or psychological nature requiring long-term rehabilitation services (Gururaj 2000). Using the Glasgow Outcome Scale, it was observed that nearly 1%–2% of persons with brain injury leave the hospital in a persistent vegetative state (Gururaj *et al.* 2005a). Many patients with fractures and bone injuries need short- to medium-term rehabilitation services. Disfigurement, chronic physical pain, loss of vision and hearing, neuropsychological disabilities such as memory problems, information processing problems, post-traumatic headache, post-traumatic epilepsy, post- traumatic syndrome are some common complications of RTIs.

The disability rate in India was 1.9% of its population in 1991; it increased to 2.1% in 2001, as per the 2001 Census. Injuries of various types are directly responsible for one-third of disabilities (National Sample Survey Organization (NSSO) 1994; Suresh 1997). Based on this estimate, nearly 70 lakh people are disabled due to injuries. As per the 47th round of the National Sample Survey (NSS) (NSSO 1981), the prevalence of injury-related visual disability was 35/1000 in rural and 32/1000 in urban areas. Similarly, the prevalence of injury-related speech disability was 32 and 47/1000 in urban and rural areas, respectively. Burns caused locomotor disability in 211 and 225/1000 population in urban and rural areas, respectively. The survey revealed that disabilities of all types were higher in rural areas, and more among men.

As per the NSSO (2003) report, the prevalence of mental retardation, mental illness, visual, hearing, speech and locomotor disabilities was 4%, 7%, 11%, 10%, 5% and 53%, respectively. Injuries caused the above-mentioned disabilities in 30 (head injuries only), 50, 51, 56 and 279 per 1000 population, respectively (mental retardation has not been included as most of the causes are congenital). The incidence of disability (in the past 365 days) was 69/100,000, with almost similar rates for urban and rural areas.

In a study of 425 persons with brain injury, it was observed that at 4 months' follow-up 42% had not recovered totally and 6% had died due to complications. Many disabilities in physical and cognitive areas were observed, which required rehabilitation (Gururaj *et al.* 1993). In a two-year follow-up of brain injuries by domiciliary visits in Bangalore, nearly 35% had problems in the health, social

and economic spheres of life, and more than 30% continued to have problems at the end of the second year. The quality of life was poor in 30% of persons with brain injury at 2 years' post-discharge (Gururaj *et al*. 2005a). From a hospital-based study at NIMHANS on persons with brain injuries caused by RTIs, falls and violence, it was observed that 46%, 30% and 24% were suffering from severe, moderate and minor disabilities at the time of discharge (Taly *et al.* 1996).

Injury causation

It is believed that injuries are events due to risky behaviors and human beings do not care for their safety. Absence of research in injury causation and mechanism has resulted in the belief that human being alone is responsible for every injury, thus resulting in trying to change his/her behaviour by only increasing his/her awareness.

Research in the past two decades has revealed that injuries occur due to a complex interaction of human, agent (vehicle/products) and environmental factors in any socioeconomic and political context (Berger and Mohan 1996). A precise and scientific understanding of these problems has resulted in a dramatic and significant reduction of deaths and injuries in high-income countries. However, in India, no systematic and scientific studies are available to highlight specific human, agent and environmental factors responsible for several types of injuries. Limited data indicate that excessive speed, non-usage of helmets, driving under the influence of alcohol, poor road design and infrastructure-related factors, poor visibility and crashworthiness of vehicles are some major risk factors for the increasing number of RTIs. The causes of other injuries are not clearly understood in India. A substantial number of deaths (nearly 30%–40%) occur at the injury site and before the injured persons get definitive care. Comprehensive trauma care in India (including emergency, acute care and rehabilitation services) is in total disarray amid disparities of high technology and sophistication in urban areas and non-availability of care elsewhere. Deficient emergency care, non-availability of physical and medical resources and lack of skilled staff lead to sub-standard care and unnecessary referrals resulting in an increase in secondary injuries. As nearly 60% of care is provided by the private sector, affordability of care is a matter of concern for the poor and middle-income groups.

Road traffic injuries

The WHO report on road traffic injury prevention has outlined four sets of risk factors contributing to RTIs (WHO 2004a). These are (i) factors related to exposure (economic and demographic factors; land-use planning practices; mix of motorized and non-motorized traffic; and lack of focus on integrating road functions with speed, design and layout), (ii) factors influencing crash involvement (excessive speed; use of alcohol and drugs; young male; being vulnerable road users (VRUs); poor visibility; vehicle factors and poor eyesight of the road user), (iii) factors influencing severity of the crash (human tolerance; high speed; non-usage of seat belts; non-usage of helmets; presence of objects on the road; consumption of alcohol; and insufficient vehicle protection against crash), and (iv) factors influencing the severity of post-crash injuries (inadequate prehospital and emergency care; deficient trauma care in care facilities and delays in care).

A review of the road safety scenario in India and other developing countries undertaken by Mohan and Bawa (1985), Mohan (1992, 2002, 2004) and Tiwari *et al.* (1998) at Transportation Research and Injury Prevention Programme (TRIPP), Delhi has identified various issues and factors for the increase in road deaths and injuries in India. Some reviews by the road safety cell of the Ministry of Transport have also raised areas of critical concern (Sarin *et al.* 2000). Studies undertaken by NIMHANS during the past decade have identified non-usage of helmets, drinking and driving, speeding, two-wheeler safety, pedestrian safety and trauma care as the key issues (Gururaj *et al.* 1993; Channabasavanna and Gururaj 1994; 2000a; 2004b and 2004c.

Some important issues concerning increase in RTI deaths in India can be summarized as follows:

- States with rapid motorization rates have experienced greater deaths and injuries indicating that higher exposure combined with the absence of safety norms are a major factor.
- Even though Indian highways comprise only 2% of the total road network, they account for more than 25% of fatal injuries, indicating specific causative factors in national highways.
- Significant differences exist in RTI patterns between cities and rural areas (only 11% occur in cities), highway and non-highway injuries, arterial and non-arterial roads, and different environments.
- VRUs in India are pedestrians, riders and pillion riders of motorized two-wheelers and bicyclists. Nearly 75% of deaths and injuries occur among them.
- Thirty to forty per cent of total deaths and injuries occur among motorized two-wheeler riders and pillion riders due to absence and poor enforcement of laws related to helmet use.
- Nearly one-third of deaths and disabling injuries in India occur among pedestrians due to lack of safe walking spaces, crossing facilities and visibility factors.
- High speeds along with the absence of traffic coordination and calming facilities are major factors responsible for the increase in RTI deaths.
- Alcohol consumption by road users is a major risk factor and causes nearly 30%–40% of night-time road crashes.
- More RTIs occur during night and early morning hours due to poor visibility of vehicles and roads. Poor, inappropriate design and maintenance of roads is a significant factor.

- Deteriorating traffic law enforcement due to the absence of enforcing teams, skills, facilities and resources is a contributory factor.
- Emphasis on road user education is not accompanied by changes in road engineering, enforcement, trauma care and systems improvement. Thus, there has been no decline in the number of deaths and injuries.
- Limited trauma care facilities in cities/towns, absence of trauma care in rural areas accompanied by a lack of human and physical resources in health care facilities are major factors for the increase in deaths and disabilities.
- Scientific crash investigation, analysis, dissemination of information are absent in India.

Work-related injuries

The causes of work-related injuries are not clearly known in India. Various machines and equipment (old and unsafe/new and unknown), factors linked to workers (age, sex, co-morbidity, use of protective devices, alcohol, lack of experience), complex and unsafe work environment (exposure to extremes of heat, chemicals, fumes, ill-ventilated places, etc.) combined with a lack of safety systems (lack of inspection, safety audits) contribute to injuries and deaths. Common causes are falls, being struck or hit by objects, impact of hot fumes and chemicals, inhalation of toxic fumes, etc. Absence of emergency and trauma care, inadequate care provided by local practitioners combined with various dangerous home remedies aggravate injuries and complications, especially in rural areas and districts.

Burn injuries

Burns are caused by a number of agent factors such as chemicals, hot liquids, fumes and electrical items. Leakage of kerosene stoves, the practice of low-level cooking, use of synthetic, loose-fitting garments have been cited as major causes of burns at home by a number of authors (Ghuliani 1988; Subramanyam 1996; Singh *et al.* 1998; Kumar *et al.* 2000; Ahuja and Bhattacharya 2002; Gupta *et al.* 1996; Bhalla *et al.* 2000). Unsafe crackers used during festivals result in death, blindness, disfigurement among a number of children, though no official figures are available (Mohan and Varghese 1990).

Suicides

The causes of suicide have their roots in the social, economic, cultural, psychological and health status of an individual. In many cases the causes of suicide are multifactorial, cumulative and progressive (Gururaj and Isaac 2001b). The causes of completed and attempted suicide are different, though some commonalities are observed. As per the NCRB report (2001b), family problems (21%) and presence of illness (21%) were the two major causes. Unknown and other causes accounted for nearly 50% of suicides indicating that suicides are not investigated in totality. The 'other' causes comprised disappointment in love affairs (3%), poverty (2.4%), economic bankruptcy (2.5%), unemployment (2.4%) and dowry dispute (2.3%). No clear conclusions can be drawn from such findings for developing specific and targeted interventions.

Among the various social factors, disturbed interpersonal relationships with family members, maladjustment with the spouse and in-laws, and broken relationships have emerged as major causes in nearly 10%–50% of suicide across independent studies. Marriage has been found to have both a protective and stressful role in the aetiology of suicide. Several economic factors such as unemployment, sudden economic bankruptcy and chronic economic deprivation are responsible factors in nearly 15%–25% of suicides, especially among men. Dowry disputes are responsible for more than 50% of suicides among young married women (NCRB 2001). Among major mental health problems, depression (14%–67%), schizophrenia (2%–12%), alcoholism in the self or spouse (7%–35%), affective disorders (10%–22%), drug dependence (3%–6%), adjustment disorders (13%–38%), obsessive–compulsive disorders (4%), and mood and personality disorders (11%–38%) have been identified among those with completed and attempted suicide (Tousignant *et al.* 1998; Venkoba Rao and Madhavan 1983; Patel and Gaw 1996; Vijay Kumar and Rajkumar 1999; Vahia *et al.* 2000; Krishnamurthy *et al.* 2000; Chandrashekaran *et al.* 2003; Gururaj and Isaac 2001a). In recent years, it has become known that genetic predisposition and vulnerability combined with neurotransmitter imbalance (often precipitated by social and economic factors) are the causes of suicide (Du *et al.* 2001; Diego De Leo 2002). A lower concentration of serotonin or 5-hydroxy indole acetic acid (HIAA) has been observed by Indian researchers. However, not much work has been carried out in India in this regard.

In a case–control study of 100 suicides (Vijay Kumar and Rajkumar 1999) in Chennai, it was observed that the presence of Axis I mental disorder in the individual (odds ratio [OR] = 19.5), family history of psychopathology (OR = 12.75), presence of negative life-events in the previous months (OR = 28.5), widow/separated/divorced status (OR = 12), personality disorder (OR = 9.5), previous suicidal attempts (OR = 5.2) and presence of medical illness (OR = 4.5) were the major risk factors. A recent case–control study at NIMHANS revealed that a combination of risk factors and absence of positive protective factors were responsible for suicide (Gururaj *et al.* 2005b). Unresolved family conflicts (OR = 22.7), presence of negative life-events (OR = 15.1), unemployment (OR = 6.2), sudden economic bankruptcy (OR = 7.1), domestic violence (OR = 6.9), alcohol consumption by self (OR = 23.4) and spouse (OR = 6.1), previous history of suicidal attempts (OR = 42.6), and presence of a mental illness (OR = 11.1) were major risk factors for suicide. Accompanying these factors were

absence of a positive outlook (OR = 269.1), problem-solving skills (OR = 56.1), coping abilities (OR = 46.1), help in crisis situation (OR = 10.1), and communication abilities (OR = 10.1). Srivastava *et al.* (2004) examined 137 cases of attempted suicide with matched controls in a hospital setting in Pondicherry and identified unemployment (OR = 15.82), lack of formal education (OR = 3.1), stressful life-events (OR = 3.95), physical disorders (OR = 3.12), and presence of idiopathic pain (OR = 6.78) as important risk factors. A recent study, using qualitative methods of psychological autopsy, revealed that causative factors are multiple and operate in an interactive, progressive and cumulative manner in the majority of suicides (Gururaj *et al.* 2005b).

Violence

The causes of violence could be broadly classified under individual, family (relationship), community and societal factors (WHO 2002b). Each of these are interlinked in a complex manner and precise distinctions are difficult. No systematic and carefully evaluated population-based data are available to quantify and qualify causes of violence in Indian studies. The causes vary among the sexes, age groups, location, and type and severity of violence based on the individual researcher's initiative and interest. National-level data are highly underreported as many do not seek help or register a case with the police due to fear, stigma and legal complications.

Among various social factors, age, gender, education, socioeconomic status and place of residence have been associated with violence (Vijayendra 1997). Violence is often found to be inflicted by the husband (54%), mother-in-law (37%) and other family members (10%) as reported by some of the studies. The occurrence is high among women, especially those belonging to lower socioeconomic groups and younger age groups (Rathod 2002; Jejeebhoy 1998). The role and status of women, lack of education and employment opportunities, and patriarchial nature of families have a major influence on violence against women (Datta and Misra 2000; Martin *et al.* 2002). Social status, dependence, responsibility and issues with regard to family and extramarital relations have also been found to be influencing factors (Unisa 1999). Property disputes are a major cause of violence among men. Male dominance, role and rights of women, sexual abuse of women, and their position within the family are also found to be associated with violence (Malhotra and Sood 2000; Vijayendra 1997; Segal 1999). Alcohol usage and dependency, personality and mood disorders, aggressive behaviours, stressful states and poor coping abilities have been incriminated in the aetiology of violence (Heise *et al.* 1994; Helen 1999).

Information from India on causative factors and mechanisms of poisoning, drowning and other injuries are not known.

Injury prevention and control

With the realization that injuries are caused by a complex interaction among agent (vehicle, product), human and environmental factors operating in complex sociopolitical and economic systems, injury prevention and control depending on evidence-based research is gaining momentum all over the world. High-income countries have made significant progress in the past 2–3 decades by developing comprehensive, integrated and intersectoral approaches based on scientific understanding. This has resulted in a decline in death and disability due to injuries. The lessons learnt so far reveal the following:

- Injury prevention and control is an intersectoral activity requiring inputs from different sectors such as the police, road transport, road engineering, health, education, the media and others.
- It is an integrated activity as multiple interventions need to be combined to obtain the best results and greater success.
- It is best developed by a systems approach by integrating several components for each intervention.
- It is in need of active inputs in terms of resources, support and cooperation of policy-makers, professionals, public and the press (media); political commitment is crucial in this process.
- It should be implemented according to a public health approach of identifying the problem, delineating risk factors and mechanisms, developing, prioritizing and implementing interventions, and evaluating them for cost-effectiveness, sustainability and culture specificity; ad hoc and crisis-oriented approaches do not lead to a real decline in deaths and disability.
- Injury prevention and control is possible only with the development of institutional mechanisms for research, policies and programmes.
- It is dependent on development, implementation and evaluation of programmes at local, State and national levels.
- It is based on the combined approaches of engineering, enforcement, education and emergency care (4 Es), resulting in economic benefits.
- It needs many passive countermeasures (requiring minimal or no action by the individual), as implementing active measures (requiring voluntary human efforts) is difficult and time-consuming.
- Investments made in prevention and control are beneficial to society in the long run.

To decrease the burden of death and disability from injury, a spectrum of activities ranging from surveillance and basic research to prevention programmes to trauma management is required. Large gains can be made from prevention and hence major emphasis should be placed on this approach (WHO 2004a).

Analysis of injury data is crucial for developing relevant, cost-effective, culture-specific and sustainable interventions. William Haddon Jr proposed a matrix for various strategies that have revolutionized injury prevention and control programmes all over the world (Haddon 1968). With this approach, causes can be identified before, during or after an injury at human, vehicle/product and environment levels, thus helping in developing interventions at different stages.

These strategies can be translated into action through the 4 Es of injury prevention and control, viz. Education, Engineering, Enforcement and Emergency care (Robertson 1983; Berger and Mohan 1996; Christopher and Gallanger 1999). Educational methods rely on provision of information to people with the premise that individuals will change their behaviour and take knowledge-based action on their own. Engineering approaches involve modifying injury-causing products to make them less injury prone or undertaking environmental modifications which make them safer. Enforcement strategies rely on application of various laws in society to control or regulate human behaviour for reducing risk. Emergency and trauma care aims to provide high-quality services to minimize injuries and death, in the event of an injury. A detailed discussion on the merits and demerits of these individual approaches is beyond the scope of this report; combined approaches yield better results. However, it is important to note that in India, injury prevention strategies focus mainly on educational approaches (Gururaj 1998). The recent WHO report on RTI prevention (2004a) highlights that 'when used in isolation, education, information and publicity do not generally deliver tangible and sustained reductions in deaths and serious injuries. Although such efforts can be effective in changing behaviour, there is no evidence that they have been effective in reducing rates of road traffic crashes'. The lessons learnt also reveal that passive interventions (requiring minimal action from individuals) are much more effective than active interventions. However, India has to do much more through a systems approach than merely trying to change people's behaviour by passive approaches.

Road traffic injuries

Road traffic injuries can be effectively reduced by several interventions such as reducing individual exposure by investing in and improving public transportation in all places; separation of slow- and fast-moving traffic on all possible roads; promoting traffic-calming measures by scientific methods; reducing speeds on roads, especially on highways and in all residential areas; mandatory helmet laws, seat-belt laws and their strict implementation in all States; implementing strict programmes on drinking and driving by the police; improving the visibility of vehicles (brighter, reflective colours) and roads in all places (at vehicle design and road formation levels and thereafter); applying international safety standards for all vehicles and roads; improving safety on existing roads, and incorporating road safety audits, on all newly built roads; and restrictions on motorcycle engine power. Scientific evidence exists for most of these interventions and they only need proper implementation (WHO 2004a).

Burn injuries

Burn injuries can be reduced by instituting fire safety systems and laws in all public places, and their strict implementation; developing safer stoves; products such as electrical items should come with appropriate safety criteria and standards; promoting less inflammable fabrics; educating the community on safer first-aid practices such as applying cold water soon after sustaining burns; and improving the quality, design and structure of buildings and houses.

Occupational injuries

Occupational (including agricultural) injuries can be prevented by ensuring industry-specific protective devices such as gloves, eye shields, facemasks, etc. for all workers and necessary enforcement of safety laws; strict and periodical inspection of all workplaces for safety norms and standards, and instituting remedial measures in hazardous places; all agricultural equipment should be made safer, especially fodder cutters and threshers; establishing national safety standards for all machines and tools in the manufacturing sector; and eliminating child labour and extending protection systems by all methods.

Poisoning

The measures that can reduce poisoning-related deaths are making available essential antidotes for treatment of poisoning at all levels of health system; promoting child-proof containers for all medicines, pesticides, kerosene, etc.; establishing guidelines and norms for the manufacture, distribution, promotion and sale of pesticides across the country (limiting easy availability of these should gain priority); establishing regulatory mechanisms for limiting the easy availability of drugs and organophosphorus compounds to prevent misuse by vulnerable people.

Suicide

Some strategies likely to reduce suicides in India are: limiting/regulating the easy availability of organophosphorus compounds and drugs; enhancing the skills of primary care and family physicians for the recognition and treatment of mental health problems and those experiencing violence; early recognition and treatment of those with depression, alcoholism and other mood and personality problems; increasing social support systems (especially for people in distress situations); educating media professionals to be more responsible in reporting suicide; and enhancing counselling facilities in all hospitals,

educational institutions and workplaces. Strengthening and supporting programmes to destigmatize suicide, facilitating mechanisms to decriminalize suicide by modifying existing laws, and promoting community awareness programmes on suicide prevention can also reduce the number of suicides. Broader mechanisms of social and economic security for distressed populations can be of considerable help.

Violence

Implementing health screening for violence in all health care institutions; early recognition of victims of violence; enhancing health and social support systems; promoting counselling mechanisms in hospitals, schools, colleges and workplaces; limiting access of weapons and alcohol to young people; and imposing meaningful restrictions on depiction of violence (and sex) in the media can reduce violence. Promoting violence reduction programmes in the community by greater awareness, facilitating conflict-resolution techniques at the family and interpersonal levels, enhancing educational and employment opportunities for women and children (especially in rural areas), and reducing gender inequalities across society will go a long way in reducing violence in India.

Trauma care system

A trauma care system encompassing prehospital (emergency), hospital (acute care) and post-hospital (rehabilitation services) care is an essential component of preventive and control strategies. With the organization and delivery of health care being a State subject, wide disparities in health care delivery are noticed in different parts of the country. Among the plethora of intervention strategies, selected ones need to be prioritized based on technological availability, cost-effectiveness and sustainability, along with professional support and people's participation. Political support for injury prevention is important in India. Injury prevention and control requires active participation of and inputs from professionals from the transport, police and road engineering departments; sectors such as health (public health specialists, physicians, surgeons, trauma care professionals, psychologists, paediatricians, etc.), law, education, social welfare, biomechanics, the media, agriculture, industry; vehicle and product manufacturers; industry and larger civil society.

In India, more than half the health care is provided by the private sector. Teaching, non-teaching and corporate hospitals are the major providers of secondary and tertiary health care in cities. So far, no attempt has been made to include these resources in the trauma care system, where appropriate. The private sector also includes family physicians, clinics and nursing homes, missionary hospitals and unqualified traditional healers, who provide varied care depending on the available facilities and paying power of people. Trauma care is in early stages of development, the budget for primary and secondary trauma care is grossly inadequate; health functionaries are unskilled; disparities exist within States. A strategy for integrated, coordinated trauma care and injury prevention activities needs to be developed in India.

Prehospital and emergency medical care

An injured patient needs (i) treatment for life threatening injuries to maximize the likelihood of survival, (ii) treatment for potentially disabling injuries to minimize disabilities and promote return to optimal functioning, and (iii) reduction in pain and suffering (Mock *et al.* 2004). Deficiencies in trauma care are due to lack of skilled human resources, physical resources in terms of infrastructure, equipment and supplies, and the process of organization and delivery. Often, there is a lack of evaluation and quality assurance mechanisms to monitor systems. Recent studies and an extensive review of the literature point to the fact that there has been nearly 15%–30% reduction in deaths in different parts of the world due to better organization of overall trauma care at different levels (WHO 2004a; Mock *et al.* 2004).

According to Trunkey (1983), 50% of deaths occur within the first hour, 30% between 1 hour and 1 week, and 20% occur after the first week. The 'golden hour' and 'platinum hour' highlight the importance of early trauma care. Important factors responsible for increasing secondary injuries and complications are non-availability of first aid, delay in transfer of patients from the injury site to a hospital, lack of definitive treatment in first-contact hospitals (such as first aid, recognition of internal body organ injury), absence of triage (matching patients to hospitals according to the severity of injury), and external medicolegal problems (waiting for the police to arrive and move the patient; legal problems; lack of provision of care by hospitals). In a recent study in Bangalore (Gururaj *et al.* 2005a), those who were provided first aid at or near the injury site, transported early to a definitive hospital for management, reached a definitive hospital directly on their own or after the first medical contact had better survival and outcome (lesser grades of disability measured on Glasgow Outcome Scale).

An emergency medical services (EMS) system is an integral part of a comprehensive health system, which provides for the organization of personnel, facilities, logistics and equipment for effective and coordinated delivery of health care services covering all geographic areas of the country under emergency health conditions (WHO 1983). First aid is defined as 'measures taken by lay people in cases of injury to prevent deterioration in condition and to maintain vital functions until definitive help becomes available' (Berger and Mohan 1996). Considerable good may be accomplished by ensuring that victims receive life-sustaining care within a few minutes of injury (Mock 2003). Even in countries with limited resources, many lives may

Role of the health sector

Health professionals have been responsible for providing curative and partial rehabilitative services to trauma patients. With the realization that 'injuries are no more accidents', the health sector and health professionals have a major role to play in the prevention, management, rehabilitation and development of injury prevention and control programmes. Injuries have been neglected events in India for a long time due to lack of initiatives in the area of prevention. The health sector can play an effective role in injury prevention and control by:

- Providing appropriate emergency and prehospital care programmes, especially in district, taluka and rural hospitals
- Introducing trauma audits at all levels by establishing minimal care guidelines
- Adequate provision of physical, technical and human resources at all levels of the health care delivery system
- Developing cost-effective, culture-specific and sustainable rehabilitation programmes, especially in rural areas.
- Promoting implementation of evidence-based interventions in societies and evaluating them for effectiveness
- Actively supporting and evaluating interventions such as helmet-use promotion, reducing alcohol drinking and driving, health screening for suicidal and violent persons, enhancing after-care services, etc.
- Strengthening capacity building and manpower development by improving the curricula of medical, nursing and allied education programmes
- Enhancing the skills of health professionals in trauma care to avoid time delays and inappropriate referrals
- Systematically collecting data on the injury problem, pattern, severity and outcome at all levels of the health care delivery system by injury surveillance and trauma registries
- Strengthening research on understanding injury/risk factors and mechanism
- Including injury prevention and control in all developmental programmes by integrating them with ongoing programmes
- Advocating for the scientific development of injury prevention programme at national and State levels
- Networking with related sectors of police, transport, social welfare, women and child development, NGOs, education, the media and others to develop an intersectoral approach by taking a leadership role
- Educating all sections of society, especially patients and families, on the use of safety approaches in day-to-day life.

be saved and many disabilities prevented by teaching motivated people what to do at the scene of accident. The foundation of an effective prehospital trauma care system may be laid by recruiting carefully selected volunteers and non-medical professionals to receive special training, and providing them with basic supplies and equipment needed for effective prehospital trauma care (Joshipura *et al.* 2004).

In 2004, WHO in association with the International Association for the Surgery of Trauma and Surgical Intensive Care and the International Society of Surgery published *Guidelines for essential trauma care* (Mock *et al.* 2004) (Available from URL: www.who.int/violence_injury_prevention/publications/services/en/). This publication outlines a list of essential trauma care services along with human and physical resources that are required to deliver such services. A series of resource tables are available in the document, which detail the vital interventions along with knowledge, skills and resources required at different levels of the health care delivery system. In addition, various resources required for the management of head injuries, neck injuries, spinal injuries, chest injuries, abdominal injuries, extremity injuries, burns and wounds, pain, and rehabilitation are outlined in the report. Further, several requirements of training, performance improvement, integration of systems and coordination are discussed in detail (Mock *et al.* 2004). Developing a trauma care system in India needs inputs from all concerned stakeholders and a systematic approach to evolve a consensus for the formulation of national guidelines.

Acute care in hospitals

In a study of trauma outcomes in three different settings, Mock *et al.* noticed that the mortality rate in a low-income setting was 63% compared with 55% in a middle-income setting and 35% in a high-income setting (Mock *et al.* 1998). Apart from high-cost technology, the decline in death rate has been due to improvements in the organization and delivery of trauma care. In a recent nationally representative survey of trauma care facilities in India (Joshipura *et al.* 2003), it was observed that several barriers exist in the delivery of appropriate trauma care services. While cities with hi-tech hospitals and advanced trauma centres are beyond the reach of many in India, injured persons in rural areas have limited or no access to good quality care.

Accident and trauma care services have been identified as an important area for growth and development during the Tenth Plan period (Planning Commission 2003). The report acknowledges that 'there are no organized comprehensive trauma care services either at the Centre or State level.' It specifies that 'services developed in the past have not been linked to an effective multidisciplinary trauma care system'. The report further highlights that during the Tenth Plan period emphasis will be laid on adequate training of medical and paramedical personnel, provision of facilities for transport of patients, suitable strengthening of existing emergency and casualty services, and improving referral linkages.

Both research and experience have proved that with existing resources, many activities can be performed at peripheral levels with adequate knowledge and skills. This implies that staff (medical/non-medical) require training to perform these tasks with basic and refresher programmes. Availability of equipment means that these facilities are not only available but also functional, and can be put to use throughout a 24-hour period. Organizational support must be provided for skills enhancement, equipment

functioning and drug availability. Guidelines, standards and protocols for the management of injuries at different levels of the health care system need to be developed to deliver appropriate trauma care.

Rehabilitation services

Rehabilitation forms an integral part of overall trauma care. Restoring an individual to his optimum level of functioning, reducing impairments and handicaps, and improving the quality of life are the essential goals of this programme. Rehabilitation services have not developed uniformly in India. They are mostly concentrated in urban areas making it difficult for nearly 70% of the population living in rural areas to access any such care. Interventions for rehabilitation include assessment, developing an individual programme based on residual deficits and instituting combined rehabilitation measures. Minor support services—wound management, relieving contractures—should be available at *taluka* hospitals and above. Low-cost, useful walking-aid equipment such as crutches, wheelchairs and walkers should be readily available for use in community centres and above. While medical specialists would be able to provide advanced care, simple and regular physiotherapy exercises, reassurance and psychological support should be available at all levels of care. The required skills and training should be promoted for personnel at all levels from a PHC and above. Facilities for physiotherapy, minor reconstructive surgery for contractures, prevention and management of pressure sores, etc. should be available in district hospitals. Several drugs such as analgesics, antibiotics, dressing materials, plaster casts and anaesthetics should be easily available for regular use. Appropriate use of these techniques along with social support systems for employment, education and home care would be of help to many disabled persons and improve their quality of life.

Policies and programmes: A systems approach

Systems-related issues

Injuries are a reflection of absent or poor safety systems or policies and programmes. As injuries are not considered a major public health problem, a systems approach has been lacking to understand their causation and develop interventions with regard to broader systems-related factors. India does not have:

- A national policy on injury prevention and control (including trauma care)
- A nodal agency within any ministry to coordinate the range of activities
- A specified budget for injury prevention
- National guidelines/protocols on emergency care, injury prevention and trauma care
- Injury surveillance to track the growing epidemic (to identify crucial and targeted areas for intervention)
- A definitive policy on curriculum for undergraduate, postgraduate medical education and allied medical subjects
- A prioritized plan for human resource development and capacity-building programmes
- A mechanism for co-coordinated activities (as several agencies are involved in prevention and control of injuries)
- A prioritized, targeted, time-bound activity schedule for injury prevention and control
- A policy on safety and standards of products, and mechanisms to check safety of products.

A public health approach to the problem of injury emphasizes identifying the burden, understanding the determinants, implementing interventions and evaluating them to see whether and how they work (WHO 2002d). To understand problems and determinants, good quality and representative information is needed. Injury surveillance is a comprehensive tool available to track changing trends and patterns, identify causes, and evaluate interventions by systematic data collection, analysis and interpretation (WHO 2002c). Implementation of interventions needs developing, promoting, strengthening of existing mechanisms, which can show measurable changes at the community level. India has a number of laws but there is poor implementation, as is evident by the increase in the number of deaths and injuries. With multisectoral involvement in implementation of directly or indirectly related activities for injury prevention, there is no systematic, coordinated effort with one individual agency or Ministry. There has been an absence of collective effort in this area compared with many other national health problems such as HIV/AIDS, malaria, tuberculosis, etc. Injury prevention and control also requires informed decision-making by the Government, industry, NGOs and society. For a demonstrable reduction in deaths, injuries and disabilities, major initiatives are required in terms of formulation and implementation of policies and programmes on a cost-effective and sustainable basis. There is an immediate need

- To establish a lead agency (independent of any ministry) to effectively develop, implement, evaluate, coordinate, monitor and guide activities with sufficient resources, staffing and independent powers.
- To develop a national policy and strategies for injury prevention and control with a major thrust on reduction of RTIs, suicides, burn injuries, work-related injuries (including agricultural injuries) and violence. The strategy should aim at developing a plan of action (with measurable targets), which would be integrated, coordinated, cost-effective and sustainable.
- To formulate individual programmes with clearly defined goals, objectives, approaches and mechanisms based on consensus to implement a National Road Safety Programme (at the time of completion of this report, the Ministry of

Road Transport and Highways had formulated a draft proposal on National Road Safety Policy [URL: www.morth.nic.in]), National Suicide and Violence Prevention Programme, National Work Safety Programme (including the unorganized labour sector), National Burns Prevention Programme, National Poisoning Prevention Programme and others.

- To allocate adequate financial and human resources for injury prevention and control and safety promotion at all levels of national and State systems to promote programmes.
- To prioritize and implement all known and proven counter-measures for prevention and control of RTIs, suicides, burn injury, occupational injuries, etc. by greater political commitment, and involvement of professionals and policy-makers.
- To focus on developing institutional mechanisms by establishing advanced centres, which could work independently to find solutions by providing adequate human and financial resources.
- To develop a comprehensive national policy on building an effective trauma care system including emergency care, acute hospital care and rehabilitative care along with strengthening the skills of the existing manpower, allocation of resources and promoting rational use of technology.
- To institute mechanisms to systematically collect, analyse, disseminate injury-related data from health and related sectors by establishing surveillance mechanisms and trauma registries to develop evidence-based understanding of problems and solutions with a focus on developing cost-effective and sustainable intervention policies and programmes.
- To facilitate urgent mechanisms for capacity building, strengthening the knowledge base and promoting research across all sectors (road engineering, vehicle manufacture, health, the police, legal professionals and others) connected with injury prevention and control. Within the health sector, strengthening the knowledge and skills of medical and allied students, and upgrading the skills of doctors at various levels of health care are essential.

The benefits of successful implementation of the above policies and programmes include: (i) a reduction in deaths caused by injuries; (ii) a reduction in the number and severity of disabilities caused by them; (iii) an increase in the number of productive working years through reduction of death and disability; (iv) a decrease in the costs associated with initial treatment and continued rehabilitation of trauma victims; (v) a reduced burden on local communities as well as the State and Central Governments in support of disabled trauma victims; and (vi) a decrease in the impact of the disease on 'second trauma' victims—their families.

Acknowledgements

I am highly thankful to the National Commission on Macroeconomics and Health for giving me an opportunity to undertake and complete this complex task. I am thankful to Dr D. Nagaraja, Director/Vice-Chancellor, National Institute of Mental Health and Neuro Sciences for granting permission to undertake this study.

My sincere thanks to Dr Dinesh Mohan, Coordinator, Transportation Research and Injury Prevention Programme, Indian Institute of Technology, New Delhi, and Dr Manjul Joshipura, Director, National Academy of Traumatology, Ahmedabad, for their valuable inputs and suggestions, and for reviewing the final reports.

I am indebted to Dr Ranjith Kumar M. and Dr Gayatri S. Pandit, Research Officers for their diligent and meticulous efforts in data collection. I am also thankful to the National Crime Records Bureau and Registrar General of India (Sample Registration System and Medical Certification of Causes of Death Divisions), Government of India, New Delhi and their respective State Government officers for timely provision of available reports.

References

Aaron R, Joseph A, Abraham S, Jayaprakash M, George K, Prasad J, *et al.* Suicides in young people in rural southern India. *The Lancet* 2004;**3:**1–4.

Adarsh K, Mohan D, Mahajan P. Studies on tractor related injuries in northern India. *Accident; Analysis and Prevention* 1998;**1:**53–60.

Adarsh K, Varghese M, Mohan D. Equipment-related injuries in agriculture: An international perspective. *Injury Control and Safety Promotion* 2000;**7:**1–12.

Aeron Thomas A, Jacobs GD, Sexton B, Gururaj G, Rahman F. The involvement and impact of road crashes on the poor: Bangladesh and India case studies. Transport research laboratory, published project report, PPR010; July 2004.

Ahuja RB, Bhattacharya S. An analysis of 11,196 burns admissions and evaluation of conservative management techniques. *Burns* 2002;**28:**555–61.

Ashok J, Gopinath N, Murthy N, Rao G. Domestic accidents in urban India. *Proceedings of the 7th world conference on injury prevention and control*. Vienna; 2004.

Baker SP. Where have you been and where are you going with injury control? In: Mohan D, Tiwari G (eds). *Injury prevention and control*. New York: Taylor and Francis Publishers; 2000.

Banerjee SR. Agricultural child labor in West Bengal. *Indian Pediatrics* 1993;**30:**425–9.

Bangdiwala SI. Methodological considerations in the analysis of injury data. In: Mohan D, Tiwari G (eds). *Injury prevention and control.* New York: Taylor & Francis Publication; 2000:27–34.

Barss P, Smith A, Baker S, Mohan D. *Injury prevention: An international perspective. Epidemiology, surveillance and policy.* New York: Oxford University Press; 1998.

Batra AK. Burn mortality: Recent trends and sociocultural determinants in rural India. *Burns* 2003;**29:**270–5.

Berger LR, Mohan D. *Injury control: A global view.* New Delhi: Oxford University Press; 1996.

Bhalla SB, Kale SR, Mohan D. Burn properties of fabrics and garments worn in India. *Accident Analysis and Prevention* 2000;**32:**407–20.

Bharti P, Nagar AM, Umesh T. Pattern of trauma in western Uttar Pradesh. *Neurology India* 1993;**41:**49–50.

Bose A, George K, Joseph A. Drowning in childhood: A population based study. *Indian Pediatrics* 2000;**1:**80–4.

Central Statistical Organization. *Select socioeconomic statistics of India, 2002*. New Delhi: Ministry of Statistics & Programme Implementation, Government of India; June 2004.

Chandrashekaran R, Gyanashelan J, Sahai A, Swaminathan RP, Perme B. Psychiatric and personality disorders in survivors following their first suicide attempt. *Indian Journal of Psychiatry* 2003;**2:**45–8.

Channabasavanna SM, Gururaj G. Head injuries and helmets: Implications for policies in developing countries. *Journal of Police Research and Development* 1994;Jan–March:19–24.

Christopher TGD, Gallagner SS. *Practical knowledge skills and strategies. Injury prevention and public health.* Maryland: Aspen Publication; 1999.

Colohan ART, Alves WM, Gross R, Torner JC, Mehta VS, Tandon PN, *et al.* Head injury mortality in two centres with different emergency medical services and intensive care. *Journal of Neurosurgery* 1989;**71:**202–7.

Datta B, Misra G. Advocacy for sexual and reproductive health: The challenge in India. *Reproductive Health Matters* 2000;**16:**24–34.

Diego De Leo. Struggling against suicide; the need for an integrative approach. *Crisis* 2002; 1:23–31.

Du L, Faludi G, Palkovits M, Bakish D, Hrdina PD. Serotonergic genes and suicidality. *Crisis* 2001;**2:**54–60.

Ghee C, Silcock D, Astrop A. Socio-economic aspects of road accidents in developing countries. *Transport Research Laboratory* 1997;**247:**1–29.

Ghuliani KK, Tyagi NK, Narang R, Nayar S. An epidemiological study of burn injury. *Indian Journal of Public Health* 1988;**1:**24–30.

Goel A, Kumar S, Bagga MK. Epidemiological and trauma injury and severity score. Analysis of trauma patients at a tertiary care centre in India. *The National Medical Journal of India* 2004;**17:**186–8

Gordon JE, Gulati PV, Wyon JB. Traumatic accidents in rural tropical region: An epidemiological study in Punjab, India. *American Journal of Medical Science* 1962;**243:**382–402.

Gururaj G, Aeron Thomas A, Reddi MN. *Underreporting of road traffic injuries in Bangalore. Implications for road safety policies and programmes. Proceedings of the 5th world conference on injury prevention and control.* New Delhi: Macmillan India Ltd; 2000b.

Gururaj G, Channabasavanna SM, Das BS, Kaliaperumal VG. Epidemiology of head injuries. National Institute of Mental Health and Neuro Sciences, PR/3/93.

Gururaj G. Behavior and road safety: A multi dimensional issue—duplication for road safety programs in developing countries. In: Holst HV, Nygren A, Anderson AE (eds). *Transportation, traffic safety and health.* New York: Springer; 1998:327–49.

Gururaj G, Girish N, Isaac MK, Subbakrishna DK. Final report of the project 'Health behavior survillence' submitted to the Ministry of Health and Family Welfare, Government of India; 2004.

Gururaj G, Isaac MK, Subbakrishna DK, Ranjani R. Risk factors for completed suicides: A case–control study. *Journal of Injury Prevention & Safety Promotion* 2005b;**11:**183–91.

Gururaj G, Isaac MK. Epidemiology of suicides in Bangalore. National Institute of Mental Health and Neuro Sciences, publication no. 33; 2001a.

Gururaj G, Isaac MK. Suicides-beyond numbers. National Institute of Mental Health and Neuro Sciences, publication no. 34; 2001b.

Gururaj G, Reddi MN, Aeron Thomas A. Epidemiology of road traffic injuries in Bangalore. *Proceedings of the 5th world conference on injury prevention and control.* New Delhi: Mc Millan Publishers 2000a.

Gururaj G, Shastry KVR, Chandramouli AB, Subbakrishna DK, Kraus JF. Traumatic brain injury. National Institute of Mental Health and Neuro Sciences, Publication no. 61, 2005a.

Gururaj G, Suryanarayana SP. Burden and impact of injuries: Results of population-based survey. Proceedings of the 7th world conference on injury prevention and control, Vienna; 2004:275–6.

Gururaj G. Epidemiology of traumatic brain injuries; Indian scenario. *Neurological Research* 2000;**24:**24–28.

Gururaj G. Injuries in South East Asia. Cause for concern and call for action. Report submitted to WHO South East Asia Regional Office, New Delhi, 2004a (unpublished document).

Gururaj G. The effect of alcohol on incidence, pattern, severity and outcome from traumatic brain injury. *Journal of the Indian Medical Association* 2004b;**102:**157–60.

Gururaj G. The First India Injury Report—problems to solutions. Report submitted to the National Commission on Macroeconomics and Health, Ministry of Health and Family Welfare, New Delhi, 2005.

Haddon Jr W. The changing approach to the epidemiology, prevention and amelioration of trauma: the transition to approaches etiologically rather than descriptively. *American Journal of Public Health 1968;* **58:**1431–8.

Haq MU. Human development in South Asia 2000. The gender question. New Delhi: Oxford University Press; 2000:10–12.

Heise LL, Raikes A, Watts CH, Zwi AB. Violence against women: A neglected public health issue in less developed countries. *Social Science & Medicine* 1994;**9:**1165–79.

Helen DP. Interpersonal violence aggression and antisocial behaviors in adolescents. *Indian Journal of Pediatrics* 1999;**66:**589–602.

India 2003—a reference manual. Ministry of Information and Broadcasting, Government of India; 2003.

International Center for Research on Women. *Domestic violence in India: A summary report of four case studies.* Washington, DC: 2000.

International Clinical Epidemiologists Network. India safe: Studies of abuse in the family environment in India: A summary report, 2000.

International Labour Organization. *Protecting and saving lives at work: The emerging challenge in Asia,* 1994.

Jejeebhoy SJ. Associations between wife-beating and fetal and infant deaths: Impressions from a survey in rural India. *Studies in Family Planning* 1998;**3:**300–8.

Jha N, Srinivasa DK, Goutam R, Jagadish S. Injury pattern among road traffic accident cases: A study from south India. *Indian Journal of Community Medicine* 2003;**2:**85–90.

Joshipura M, Hyder AA, Rehmani R. Emergency care in South East Asia: Challenges and opportunities, road traffic injuries. *Journal of College of Physicians and Surgeons of Pakistan* 2004;**14:**731–5.

Joshipura MK, Shah HS, Patel PR, Divata PA, Desai PM. Trauma care systems in India. *Injury* 2003;**34:**1–8.

Krishnamurthy K, Khan FA, Gowri DM, Anand B. Suicidal intent in schizophrenic patients—a serious risk. *Journal of Post Graduate Psychiatry* 2000:59–67.

Kumar P, Chirayil PT, Chittoria R. Ten years of epidemiological study of pediatric burns in Manipal, India. *Burns* 2000;**26:**261–4.

Maheswari J, Mohan D. Road traffic injuries in Delhi: A hospital based study. *Journal of Traffic Medicine* 1989;**17:**23–7.

Malhotra N, Sood M. Sexual assault—a neglected public health problem in the developing world. *International Journal of Gynecology and Obstetrics* 2000;**71:**257–58

Malhotra P, Dhar S, Dogra S, Kaul S, Raina RK. Pattern of injuries in hydro electric project. *Journal of the Indian Medical Association* 1995;**5:**171–2.

Martin SL, Moraco KE, Garro J, Tsui AO, Kupper LL, Chase JL, *et al.* Domestic violence across generations: Findings from northern India. *International Journal of Epidemiology* 2002;**31:**560–72.

Mathur N, Sharma KKR. Medicoeconomic implication of industrial hand injuries in India. *The Journal of Hand Surgery* 1988;**3:**325–7.

Ministry of Road Transport and Highways, Transport Research Wing. *Motor transport statistics of India,* New Delhi: Government of India; 2001–02.

Mittal VK. A study of magnitude, causes, and profile of victims of

injuries with selected farm machines in Punjab. Final report of ICAR ad-hoc research project. Ludhiana, India: Department of Farm Machinery and Power Engineering, Punjab Agricultural University; 1996 (unpublished).

Mock C, Lormand JD, Goosen J, Joshipura M, Peden M. *Guidelines for essential trauma care.* Geneva: World Health Organization; 2004.

Mock C. Improving pre-hospital trauma care in rural areas of low-income countries. *The Journal of Trauma* 2003;**54:**1197–8.

Mock CN, Adzotor KE, Conklin E, Denno DM , Jurkovich GJ. Trauma outcomes in the rural developing world: Comparison with the urban level trauma 1 center. *The Journal of Trauma* 1993;**35:**518–23.

Mock CN, Jurkovich GJ, David nii-Amon–Kotei, Arreola Rice C, Maier RV . Trauma mortality patterns in three nations at different levels: Implications for global trauma care systems development. *The Journal of Trauma: Injury, infection and critical care* 1998;**44:**804–14.

Mohan D, Anderson R. Injury prevention and control: International course on injury prevention and control. TRIPP, New Delhi; 2000.

Mohan D, Bawa PS. An analysis of road traffic fatalities in Delhi, India. *Accident; Analysis and Prevention* 1985;**1:**33–45.

Mohan D, Patel R. Design of safer agricultural equipment: Application of ergonomics and Epidemiology. *International Journal of Industrial Ergonomics* 1992;**10:**301–9

Mohan D, Varghese M. Fireworks cast a shadow on India's festival of lights. *World Health Forum* 1990;**11:**323–6.

Mohan D. Injuries in India: A survey. *ICSSR Research Abstracts* 1992;**21:**221–2.

Mohan D. Road safety in less motorised environment: Future concerns. *International Journal of Epidemiology* 2002;**31:**527–32.

Mohan D. The road ahead: Traffic injuries and fatalities in India. Transportation Research and Injury Prevention Programme. Indian Institute of Technology, Delhi, April 2004.

Mohan D, Tiwari G. Traffic safety in low income countries: Issues and concerns regarding technology transfer from high-income countries. In: *Reflections of the transfer of traffic safety knowledge to motorizing nations*. Melbourne: Global Traffic Safety Trust; 1998:27–56.

Mukerji G, Shobha C, Patidar GP, Gupta S. Epidemiology of pediatric burns in Indore, India. *Burns* 2001;**27:**33–8.

Murlidhar V, Nobhojit R. Measuring trauma outcomes in India: An analysis based on TRISS methodology in a Mumbai University hospital. *Injury* 2004;**35:**386–90.

Murray CJL, Lopez AD. *The global burden of diseases: A comprehensive assessment of mortality and disability from diseases, injuries and risk factors in 1990 and projected to 2020.* Volume 1. Boston: World Health Organization, Harvard School of Public Health and World Bank; 1996.

Nag PK, Patel VG. Work accidents among shift workers in industry. *International Journal of Industrial Ergonomics* 1998;**21:**275–81.

Narayana G. Family violence, sex and reproductive health behavior among men in Uttar Pradesh, India: Policy project. New Delhi: The Futures Group; 1996 (unpublished).

National Crime Records Bureau. *Accidental deaths and suicides in India.* New Delhi: Ministry of Home Affairs, Government of India; 2001b.

National Crime Records Bureau. *Crime in India.* New Delhi: Ministry of Home Affairs, Government of India; 2001a.

National Sample Survey Organization. Disability in India. NSSO 47th round (July–December 1991). Ministry of Statistics and Programme Implementation, Government of India; 1991.

National Sample Survey Organization. Disability in India. NSSO 52nd round. Ministry of Statistics and Programme Implementation, Government of India; 1994.

National Sample Survey Organization. Disability in India. NSSO 58th round. Ministry of Statistics and Programme Implementation, Government of India; 2003.

Patel SP, Gaw AC. Suicide among immigrants from the subcontinent: A review. *Psychiatry Services* 1996;**5:**517–20.

Planning Commission. Report of the Working Group on Accidents, Injury Prevention and Control, 2003.

Planning Commission. Study of the nature, extent, incidence and impact of domestic violence against women in the states of Andhra Pradesh, Chattisgarh, Gujarat, Madhya Pradesh and Maharashtra. Report by Yuganthar Education Society, Nagpur, 2004.

Rathod AM, Tripathi R, Arora R. Domestic violence against pregnant women interviewed at a hospital in New Delhi. *International Journal of Gynaecology and Obstetrics* 2002;**76:**83–5.

Rautji R, Dogra TD. Rail traffic accidents: A retrospective study. *Medicine, Science, and the Law* 2004;**1:**67–70.

Registrar General of India. *Census of India 1991.* Ministry of Home Affairs, Government of India, 1991.

Registrar General of India. *Census of India 2001.* Ministry of Home Affairs, Government of India, 2003.

Registrar General of India. Medical Certification of Causes of Death. New Delhi: Ministry of Home Affairs; 1998a.

Registrar General of India. Survey of Causes of Death (rural), vital statistics division. Series 3, no 31, 1998b.

Road Peace. World's first road death. Available from URL: http://www.roadpeace.org/articles/worldfirstroaddeath (accessed in 2003).

Robertson LS. *Injuries: Causes, control strategies and public policy.* Cambridge, MA: Lesington Broks; 1983.

Sahadev P, Lacqua MJ, Singh B, Dogra DT. Road traffic fatalities in Delhi: Causes, injury pattern, and incidence of preventable deaths. *Accident; Analysis and Prevention* 1994;**3:**377–84.

Sarin SM, Bajpai RK, Mittal N. A note on road traffic accidents in India. New Delhi: Central Road Research Institute; 2000 (unpublished).

Sathyasekaran BWC. Study of the injured and the injury pattern in road traffic accident. *Indian Journal of Forensic Sciences* 1991;**5:**63–8.

Sathyasekaran BWC. Population-based cohort study of injuries. *Injury* 1996;**27:**695–8.

Segal UA. Family violence a focus on India. *Aggression and Violent Behavior* 1999;**2:**213–31.

Sharma B, Harish D, Sharma V, Krishnan V. Kitchen accidents vis-a-vis dowry death. *Burns* 2002;**28:**250–3.

Sharma BR, Harish B, Sharma V, Krishnan V. Road traffic accidents: A demographic and topographic analysis. *Medicine, Science, and the Law* 2001;**3:**266–75.

Sharma BR, Harish D, Sharma V, Vij K. Poisoning in northern India: Changing trends, causes and prevention there of. *Medicine, Science and the Law* 2002;**42:**251–7.

Shneidman E. *Definition of suicide.* New York: John Wiley & Sons; 1985.

Sidhu DS, Sodi S, Banarjee G. Mortality profile in trauma victims. *Journal of Indian Medical Association* 1993;**91:**16–17

Singh D, Singh A, Sharma AK, Sodhi L. Burn mortality in Chandigarh zone: 25 years autopsy experience from a tertiary care hospital of India. *Burns* 1998;**24:**150–6.

Srivastava MK, Sahoo RN, Ghotekar LH, *Dutta S, Danabalan M, Dutta JK et al.* Risk factors associated with attempted suicides: A case–control study. *Indian Journal of Psychiatry* 2004;**46:**33–8.

Subramanyam M. Epidemiology of burns in a district hospital in western India. *Burns* 1996;**6:**439–42.

Sudarshan MK. Assessing burden of rabies in India. Association for Prevention and Control of Rabies in India, 2004.

Sudhir D. Occupational health services for agricultural workers. *Indian Journal of Occupation and Environmental Medicine* 1998;**2:**96–111.

Suresh K. Impact of child survival and maternal care interventions over the incidence and prevalence of disability. *Action Aid Disability News* 1997;**8:**12–15.

Taly AB, Gururaj G, Gourie-Devi M, Das BS, Rao S, Subbakrishna

DK. Assesment of neurological disabilities among hospitalized patients. *European Journal of Neurology* 1996;**3:**86.

Tiwari G. Traffic flow and safety: Need for new models in heterogeneous traffic. In: Mohan D, Tiwari G (eds). *Injury prevention and control.* London: Taylor and Francis; 2000: 71–88.

Tiwari G, Mohan D, Fazio J. Conflict analysis for prediction of fatal crash locations in mixed traffic streams. *Accidental Analysis and Prevention* 1998;**30:**207–15.

Tiwari SP, Gite PS, Dubey AK, Kot LS. Agricultural injuries in central India: Nature, magnitude, and economic impact. *Journal of Agricultural Safety and Health* 2002;**1:**95–111.

Tousignant M, Seshadri S, Raj A. Gender and suicide in India: A multi perspective approach. *Suicide and life-threatening behavior* 1998;**1:**50–62.

Trunkey DD. Trauma. *Scientific American* 1983;**28:**249.

Unisa S. Childlessness in Andhra Pradesh, India; treatment-seeking and consequences. *Reproductive Health Matters* 1999;**13:**54–63.

Vahia NV, Sonavane S, Gandhi A, Vahia I. Suicide and depression. *Journal of the Indian Medical Association* 2000;**5:**232–6.

Varghese M, Mohan D. *Transportation injuries in rural Haryana, North India.* Proceedings of the international conference on traffic safety. New Delhi: Macmillan India Ltd; 2003:326–29.

Varghese M. Occupational injuries among agricultural workers in rural Haryana, India. *Journal of Occupational Accidents* 1990;**12:** 237–44.

Venkoba Rao, Madhavan T. Depression and suicide behaviour in the aged. *Indian Journal of Psychiatry* 1983;**4:**251–9.

Verma PK. An epidemiological study of accidents among rural population. MD thesis submitted to the Delhi University, April 1998

Verma PK, Tiwari KN. Epidemiology of road traffic injuries in Delhi. Results of a survey. *Regional Health Forum* 2004;**8:**6–14.

Vijay Kumar L, Rajkumar S. Are risk factors for suicide universal? A case–control study in India. *Acta Psychiatrica Scandinavica* 1999;**99:**407–11.

Vijayendra R. Wife-beating in rural south India: A qualitative and econometric analysis. *Social Science & Medicine* 1997;**8:**1169–80.

World Health Organization. *Handbook for the documentation of interpersonal violence prevention programmes.* Geneva: WHO; 2004c.

WHO. In: Holder Y, Peden M, Gururaj G (eds). *Injury surveillance guidelines.* Geneva: WHO; 2002c.

WHO. In: Krug E, *et al.* (eds). *Injury: A leading cause of the global burden of disease.* Geneva: WHO; 1999.

WHO. In: Krug EG, Dahlberg LL, Mercy JA, *et al.* (eds). *World report on violence and health.* Geneva: WHO; 2002b.

WHO. In: Peden M, Scurfield R, Sleet D, Mohan D, Hyder AA, Jarawan E, *et al.* (eds). *World report on road traffic injury prevention.* Geneva: WHO; 2004a.

WHO. Injury prevention and control. An epidemiological survey of injuries in area of Municipal Corporation of Delhi. 2003b, New Delhi, SEA-injuries-5.

WHO. *Organization of emergency medical services.* Geneva: WHO; 1983.

WHO. *Shaping the future.* Geneva: WHO; 2003a.

WHO. Strategic plan for injury prevention and control in South East Asia, 2002d, New Delhi, SEA-accident-8.

Annexure

Annexure A: Causes of various diseases/health conditions

NATIONAL COMMISSION ON MACROECONOMICS AND HEALTH, NEW DELHI

Table A1 Causes of blindness

Disease/ health condition	Direct (clinical-point of care)	Direct (health system)	Indirect (socio-economic determinants)	Indirect (lifestyle RF)	Indirect (genetic, medical history, age, sex)
Cataract	• Intraocular inflammation • Injury to the eyes • Metabolic causes • Congenital (intra-uterine infections) • Corticosteroids (systemic and topical)		• Poor socioeconomic status • Illiteracy	• Exposure to sunlight • Poor nutrition • Smoking • Alcohol • Cheap cooking fuel	• Age • Acute diarrhoea in early life • Diabetes • Glaucoma • Myopia • Hypertension • Sex—more common in females
Refractive errors and low vision	• Non-utilization of spectacles • Diabetes • Cataract • Eye surgery • Trauma	• Non-screening of eyesight • Lack of school health services	• Rural areas • Illiteracy		• Anatomical aberrations of the eye

RF: risk factors

Table A2 Causes of chronic obstructive pulmonary disease (COPD) and asthma

Disease/ health condition	Direct (clinical-point of care)	Direct (health system)	Indirect (socioeconomic determinants)	Indirect (lifestyle RF, awareness)	Indirect (genetic, medical history, age, sex, ethnicity RF)
COPD	Delayed treatment-seeking	Lack of awareness of symptoms of COPD	Air pollution	• Type of cooking fuel used • Tobacco smoking	Sex—more common in males
Asthma	Viral upper respiratory tract infections	Lack of awareness on symptoms or causes of asthma	• Air pollution • More common in rural areas	• Poor housing conditions such as poor ventilation, less living space • Type of fuel and method used for cooking—chullah, stove • Occupational asthma —industry-related Smoking	• Family history • Slightly more common in males than females • Season—more common in winters

COPD: chronic obstructive pulmonary disease

Table A3 Causes of cancer

Disease/ health condition	Direct (clinical-point of care)	Direct (health system)	Indirect (socioeconomic determinants)	Indirect (lifestyle RF, awareness)	Indirect (genetic, medical history, age, sex, ethnicity RF)
Cancer	Some infections (HBV, HPV)	• Lack of awareness of signs and symptoms for early diagnosis of cancers • Lack of screening of high-risk groups	• Environmental pollution • Industrial toxins	• Consumption of tobacco • Excessive fat in the diet • Excessive alcohol consumption • Reproductive and sexual behaviour • Occupational hazards • Other lifestyle factors	• Genetic predisposition • Hormones

HBV: hepatitis B virus; HPV: human papillomavirus

Table A4 Causes of chronic otitis media

Disease/ health condition	Direct (clinical-point of care)	Direct (health system)	Indirect (socioeconomic determinants)	Indirect (lifestyle RF, awareness)	Indirect (genetic, medical history, age, sex, ethnicity RF)
Chronic otitis media	• Recurrent upper respiratory tract infections • Allergy • Enlarged adenoids • Seasonal viral infections	• Lack of adequate medical attention • Lack of awareness on basic ENT and dental care	• Poverty • Malnutrition • More common in rural areas • Poor environmental sanitation	Poor personal hygiene	

ENT: ear, nose, throat

Table A5 Causes of childhood diseases/health conditions

Disease/health condition	Direct (clinical-point of care)	Direct (health system)	Indirect (socioeconomic determinants)	Indirect (lifestyle RF, awareness)	Indirect (genetic, medical history, age, sex, ethnicity RF)
Birth asphyxia	• Low birth weight • Obstetric complications (APH, cord prolapse, PIH, etc.) • Foetal malformations	• Unskilled birth attendant • Inadequate maternal care • Poor/no emergency obstetric care • Absence of a credible referral system • Lack of community awareness on danger signs of a sick child	• Maternal illiteracy • Low socioeconomic status	Maternal age <18 or >35 years	
Neonatal sepsis	• Low birth weight • Acute maternal intrapartum infection (including STI) • Prolonged and preterm rupture of membranes	• Unhygienic delivery and postnatal conditions • Lack of community awareness on danger signs of a sick child • Unskilled birth attendant • Inappropriate community practices (e.g. cord care practices, branding, etc.)	• Maternal illiteracy • Low socioeconomic status	• Delayed and non-exclusive breastfeeding • Delayed recognition and care-seeking	Increased biological risk in males
Low birth weight	• Obstetric complications • Medical illness (e.g. malaria) • Multiple pregnancy	Lack of community awareness on dietary requirements of pregnant women	• Maternal illiteracy • Low socioeconomic status	• Maternal work and inadequate rest during pregnancy • Adolescent pregnancy • Maternal age <18 or >35 years	

(Cont.)

Table A5 (*cont.*) Causes of childhood diseases/health conditions

Disease/health condition	Direct (clinical-point of care)	Direct (health system)	Indirect (socioeconomic determinants)	Indirect (lifestyle RF, awareness)	Indirect (genetic, medical history, age, sex, ethnicity RF)
	• Foetal malformations • Inadequate maternal care • Poor maternal health			Interpregnancy interval <2 years	
Diarrhoea	• Vitamin A deficiency • Low birth weight	Lack of community awareness on prevention and management of diarrhoea	• Maternal illiteracy • Low socioeconomic status • Lack of sanitation • Contaminated water and food	• Bottle-feeding • Non-exclusive breastfeeding for 6 months	
Pneumonia	• Lower respiratory tract infection in family • Low birth weight/ severe malnutrition • Vitamin A deficiency	• Lack of community awareness • Inappropriate immunization for age	• Illiteracy • Low socioeconomic status	• Use of biomass fuel • Passive smoking • Lack of community awareness • Non-exclusive breast-feeding for 6 months	More common in males
Malnutrition	• Recurrent infections • Low birth weight	• Lack of awareness about dietary requirements of children • Delay in care-seeking	• Poverty • Natural disasters (drought, flood, etc.) • Non-energy-dense feeds • Inadequate quantity of weaning food	• Community feeding practices • Non-exclusive breast-feeding for 6 months	

APH: antepartum haemorrhage; PIH: pregnancy-induced hypertension; STI: sexually transmitted infection

Table A6 Causes of maternal diseases/health conditions

Disease/health condition	Direct (clinical-point of care)	Direct and indirect (health system)	Indirect (socioeconomic determinants)	Indirect (lifestyle RF, awareness)	Indirect (genetic, medical history, age, sex, ethnicity RF)
Puerperal sepsis	• Prolonged labour • Prolonged leakage of membranes • Multiple unclean per vaginal examinations • Manual removal of the placenta • Retained placental bits • Unclean/unsterile instruments • Inadequate antibiotic therapy when indicated	• Delay in referral • Appropriate health facility too distant from home • Delivery by untrained person	Low socioeconomic status		
Septic abortion	• Foreign body inserted into the genital tract • Injury to genital organs • Retained foetal tissue • Unclean/unsterile instruments	Abortion by untrained person	• Illegal abortion • Fear of loss of confidentiality • Prenatal sex determination	• Pregnancy in unmarried girls • Ignorance • Use of abortifacients	
Eclampsia	• Associated diabetes • Underlying renal disorder • SLE	Lack of antenatal care (especially recording of the blood pressure)	Illiteracy	• Insufficient calcium intake • Ignorance	• Elderly woman • Immunological causes • Genetic predisposition

(Cont.)

Table A6 (*cont.*) Causes of maternal diseases/health conditions

Disease/health condition	Direct (clinical-point of care)	Direct and indirect (health system)	Indirect (socioeconomic determinants)	Indirect (lifestyle RF, awareness)	Indirect (genetic, medical history, age, sex, ethnicity RF)
Postpartum haemorrhage					
a) Uterine inversion	• Adherent placenta • Strong traction on the umbilical cord	• Delivery by unskilled person • Specialist available at a distance	• Poverty • Delayed transportation	• Ignorance • Home delivery	
b) Retained placenta	• Placenta accreta • Tearing of the cord	• Delivery by unskilled person • Specialist available at a distance • Blood bank facility available at a distance	• Poverty • Delayed transportation	• Home delivery	
Premature rupture of membranes	• Genital tract infection • Occult amniotic fluid • Multiple foetuses • Abruptio placentae • Polyhydramnios • Cervical incompetence	Lack of antenatal care			
Anaemia	• Poor intake of iron during pregnancy • Intolerance to iron • Worm infestation • Malaria • Malabsorption of iron	• Lack of adequate antenatal care • Inadequate supply of iron–folic acid (IFA) tablets	• Low socioeconomic status • Poverty • Illiteracy	• Poor reserves of iron • Multiparity • Inadequate birth spacing • Teenage marriage and pregnancy • Ignorance	
Bleeding in pregnancy					
a) Bleeding in early pregnancy	• Threatened abortion • Inevitable abortion • Incomplete abortion • Ectopic pregnancy • Molar pregnancy • Complications due to medical termination of pregnancy • Injury to the genital tract				
b) Bleeding in late pregnancy (APH)	• Placenta praevia • Abruptio placentae • Undetermined APH • Chronic hypertension • Gestational hypertension				• Elderly woman • Multiparity
Prolonged/ obstructed labour	• Cephalopelvic disproportion • Malposition • Malpresentation • Cervical dystocia • Prolonged rupture of membranes • Uterine dysfunction	• Unbooked cases • Home delivery • Hospital at a distance		Ignorance	• Short-statured mother • Bad obstetric history • Multiparity • Foetal anomalies

RF: risk factors; SLE: systemic lupus erythematosus; APH: antepartum haemorrhage

Table A7 Causes of tuberculosis (TB)

Disease/ health condition	Direct (clinical-point of care)	Direct and indirect (health system)	Indirect (socioeconomic determinants)	Indirect (lifestyle RF, awareness)	Indirect (genetic, medical history, age, sex, ethnicity RF)
Tuberculosis (TB)	Contact with a smear-positive case of TB	• Ignorance and lack of awareness of TB • Unimmunized child	• Malnutrition • Poverty • Low socioeconomic status • Overcrowding • Illiteracy • Natural disaster	• Smoking • Alcoholism • Stigma associated with TB	• Male • Age >45 years • HIV infection • Patient on immunosuppressive therapy • Diabetes mellitus • Silicosis

HIV: human immunodeficiency virus

Table A8 Causes of cardiovascular diseases

Disease/health condition	Direct (clinical-point of care)	Direct and indirect (health system)	Indirect (socioeconomic determinants)	Indirect (lifestyle RF, awareness)	Indirect (genetic, medical history, age, sex, ethnicity RF)
Coronary heart disease/stroke/ transient ischaemic attack (TIA)	• High blood pressure • Diabetes • High total cholesterol • Low high-density liproprotein (HDL) cholesterol • Obesity • Renal/vascular disease		• Upper socioeconomic status • Migration • Literacy status • Acculturation	• Smoking • Physical inactivity • Diet • Stress • Psychological factors	• Age • Sex • Ethnicity
Congestive heart failure	• Underlying heart disease • Rheumatic heart disease • High blood pressure • Left ventricular hypertrophy • Diabetes • Obesity—general and central			Smoking	• Age • Sex • Ethnicity
Rheumatic fever	• Streptococcal infection • Underlying infection of valves • Poorly adapted autoimmune response to group A β-haemolytic streptococci		• Overcrowding • Low socioeconomic status • Low literacy status		Age (mostly in children)
Hypertension	• Obesity—central and generalized • Diabetes		Upper socioeconomic status	• Diet (low fruit and vegetable or excess sodium intake) • Physical inactivity • Psychological factors	• Age • Sex • Ethnicity
Diabetes	Obesity—general and central		• Upper socioeconomic status • Urbanization	• Diet • Physical inactivity • Smoking	• Age • Sex • Family history of diabetes • Ethnicity

Table A9 Causes of dental diseases/health conditions

Disease/health condition	Direct (clinical-point of care)	Direct and indirect (health system)	Indirect (socioeconomic determinants)	Indirect (lifestyle RF, awareness)	Indirect (genetic, medical history, age, sex, ethnicity RF)
Dental caries	• Loss of a few teeth and failure to replace them • Deep pits and fissures in teeth • Malalignment of teeth • Crowding of teeth • Gum recession • Trace elements—Zn, Pb, Fe • Viscous salivary secretion • Enzymes—lactoperoxidase, lysozyme, lactpferrius • Reduced secretion (xerostomia) • Immunoglobin A	• Lack of availability/poor access to health facility • Lack of health insurance	• Low socioeconomic status • Low literacy • Urban/rural location	• Poor dietary habits • Social and cultural practices • Excessive intake of refined carbohydrates • Oral clearance rate	• Age • Sex • Fluoride content of teeth
Periodontal diseases	• Dental plaque and occlusion • Traumatic occlusion • Food impaction	• Lack of health insurance • Poor knowledge of oral health	• Low socioeconomic status • Low literacy	• Chewing and smoking tobacco • Oral hygiene	• Idiopathic (gingival fibromatosis) • Deficiency of vitamins A and C • Endocrine disturbances (puberty, pregnancy, menopause, hyper-thyroidism, hyperpara-thyroidism, diabetes) • HIV infection • Immunosuppressant drugs • Anaemia, leukaemia
Dentofacial anomalies and malocclusion	• Trauma and accidents • Abnormalities in number, size and shape of teeth • Abnormal labial frenum and mucosal barriers • Premature tooth loss • Delayed eruption of permanent teeth • Abnormal eruptive path • Ankylosis	Untreated dental caries and improper dental restorations		• Deficiency of vitamin D, calcium and phosphates • Abnormal suckling • Mouth breathing • Thumb and finger sucking • Tongue thrusting and tongue sucking • Abnormal swallowing habits	• Maternal diet and metabolism • Maternal German measles • Position of foetus *in utero* • Intake of certain drugs during pregnancy • Birth injury • Cerebral palsy • Temporomandibular joint surgery • Psychogenic tics and bruxism • Posture • Endocrine imbalance • Muscular dystrophies • Infectious diseases such as poliomyelitis
Oral cancer	• Infectious diseases—HPV, HSP, AIDS, syphilis, candidiasis • Chronic irritation—faulty prosthesis, sharp teeth	• Poor access to oral health care facilities • Radiation	• Industrial pollution—asbestos, lead, leather, textile industries • Low socioeconomic and literacy level	• Tobacco smoking/chewing • Paan masala/gutka chewing	• Compromised immune status • Deficiency of vitamins A, B complex and zinc

(Cont.)

Table A9 *(cont.)* Causes for dental diseases/health conditions

Disease/health condition	Direct (clinical-point of care)	Direct and indirect (health system)	Indirect (socioeconomic determinants)	Indirect (lifestyle RF, awareness)	Indirect (genetic, medical history, age, sex, ethnicity RF)
Dental fluorosis			• Poor nutritional status • Industrial pollution • Exposure to high levels of fluorides in drinking water • Diet (sea food, poultry, grain and cereal products, tea, green leafy vegetables)	Diet (sea food, poultry, grain and cereal products, tea, green leafy vegetables)	• Tropical climate (excess ingestion of water) • Renal diseases • Thyroid and thyrotrophic hormones • Decreased bone phosphatase activity • Deficiency of vitamin D, calcium and phosphates

HPV: human papilloma virus

Table A10 Causes of mental illnesses/health conditions

Disease/health condition	Direct (clinical-point of care)	Direct and indirect (health system)	Indirect (socioeconomic determinants)	Indirect (lifestyle RF, awareness)	Indirect (genetic, medical history, age, sex, ethnicity RF)
General	Physiological factors	• Lack of mental care services • Non-availability of medicines	Absence of clearly defined mental health policies	• Poor level of awareness • Lack of care-seeking	
Schizophrenia	• Alcohol and drug abuse • Post-viral infections	Drugs beyond the reach of the poor	• Violence • Lack of income and employment • Poverty • Stigma	• Living alone • Lack of family support systems	• Genetic predisposition • Personality/nature of individuals • Immunological factors
Alcohol and drug abuse		Lack of early recognition and diagnosis	• Easy availability of alcohol and drugs • Extensive promotion by the media • Lack of clear policies on production, availability, distribution and promotion of alcohol and drugs	• 'Liberalized' values among people • Peer group influences • Poor life skills	• Genetic predisposition • Personality profile of individual • Family history of usage
Dementia	History of mental disorder	Deprivation of basic care		• Isolation and other social issues • Absence of supportive care	
Epilepsy	• Brain injury • Febrile convulsions • Pyogenic meningitis	Inaccessibility to drugs			Genetic factors

Table A11 Causes of malaria and other vector-borne diseases

Disease/health condition	Direct (clinical-point of care)	Direct and indirect (health system)	Indirect (socioeconomic determinants)	Indirect (lifestyle RF, awareness)	Indirect (genetic, medical history, age, sex, ethnicity RF)
Malaria	Lack of adequate and timely treatment		• Malnutrition • Low socioeconomic status • Occupation • Illiteracy • Migration of labourers	• Non-use of personal protection against mosquito bites • Outdoor sleeping habits • Lack of health care-seeking behaviour • Collection of water favouring breeding of mosquitoes	Immunosuppressive drugs
Kala-azar (additional factors)				Proximity to animal stables (favouring sandfly breeding)	Environment—high temperature, high humidity, alluvial soil
Japanese encephalitis (additional factors)		Poor intersectoral coordination with agriculture, animal husbandry and local government	• Poor community participation for control of mosquito breeding • Intensive rice cultivation • Proximity to piggery • Asylum for migratory birds	Mixed dwelling	
Dengue (additional factors)			• Poor socioeconomic status • Illiteracy	• Poor water storing practices • Not drying coolers once every week • Lack of personal protection against mosquito bite	

Table A12 Causes of leprosy

Disease/health condition	Direct (clinical-point of care)	Direct and indirect (health system)	Indirect (socioeconomic determinants)	Indirect (lifestyle RF, awareness)	Indirect (genetic, medical history, age, sex, ethnicity RF)
Leprosy	Exposure to a patient with leprosy	Lack of adequate health services	• Illiteracy • Malnutrition • Overcrowding • Lack of ventilation		

Table A13 Causes of HIV infection/AIDS

Route of HIV infection	Direct (clinical-point of care)	Direct and indirect (health system)	Indirect (socioeconomic determinants)	Indirect (lifestyle RF, awareness)	Indirect (genetic, medical history, age, sex, ethnicity RF)
Sexual transmission	Incompletely treated STI/RTI	• Ignorance about STIs • Low coverage with quality life skills education to adolecents and out-of-school children • Unregulated, unqualified practitioners traditionally accepted for treatment of STIs • Poor coordination between private and public sectors • Poor access to quality risk reduction counselling • Low acceptability/awareness about syndromic management	• Poverty • Natural calamities—famine, earthquake • Lower empowerment of women • Single male member migration	• Unprotected sex with casual partner or HIV-infected person • Practice of risky sexual behaviour • Social stigma associated with risky behaviour, STIs and 'at risk' sub-populations • Social taboo on discussions on sexuality • Alcohol or other substance abuse • Poor treatment-seeking behaviour	

(Cont.)

Table A13 *(cont.)* Causes of HIV infection/AIDS

Route of HIV infection	Direct (clinical-point of care)	Direct and indirect (health system)	Indirect (socioeconomic determinants)	Indirect (lifestyle RF, awareness)	Indirect (genetic, medical history, age, sex, ethnicity RF)
		guidelines among health care providers • Low coverage with quality, targeted interventions among 'at risk' subpopulations			
Transfusion-associated	• Practice to provide plasma • Non-adherence to rational use of blood	• Poor adherence to biosafety precautions • Lack of wide availability of HIV-tested blood products • Occupational—lack of protective equipment at workplace • Ignorance about post-exposure prophylaxis • Lack of access to post-exposure prophylaxis drugs	• Unemployment • Poverty • Social instability	• Injecting drug use • Peer pressure	
Mother-to-child transmission	Vaginal delivery	Lack of access to HIV testing and counselling		Ignorance about MTCT of HIV infection	

STI: sexually transmitted infection; RTI: reproductive tract infection; MTCT: mother-to-child transmission

Table A14 Causes of injuries

Type of injury	Direct (clinical-point of care)	Direct and indirect (health system)	Indirect (socioeconomic determinants)	Indirect (lifestyle RF, awareness)	Indirect (genetic, medical history, age, sex, ethnicity RF)
Road traffic injuries	Lack of standard treatment protocols	• Lack of advocacy by health personnel for injury prevention and control • Poor emergency care services • Lack of medical treatment facilities • Lack of skill-based training of health personnel • Absence of triage • Lack of communication facilities in health care settings	• Increasing motorization • Lack of road safety policy • Non-functioning road safety councils at all levels • Absence of mandatory laws and poorly implemented laws regarding wearing of helmets and use of seat belts • Poor design and visibility of vehicles • Poor design, maintenance and quality of roads • Accident black spots • Lack of integrated rehabilitation programmes for road traffic injury patients • Delay in transportation of patients, especially in rural and semi-urban areas	• Consumption of alcohol • High speed	
Fall-related injuries	Lack of standard treatment protocols	• High cost of care • Lack of trauma audits • Absence of triage • Improper referral • Lack of facilities to treat polytrauma	• Non-compliance of laws on work safety and home safety • Poor design of homes • Lack of safety equipment for construction workers	Influence of alcohol	

(Cont.)

Table A14 (*cont.*) Causes of injuries

Type of injury	Direct (clinical-point of care)	Direct and indirect (health system)	Indirect (socioeconomic determinants)	Indirect (lifestyle RF, awareness)	Indirect (genetic, medical history, age, sex, ethnicity RF)
			• Poor quality of climbing devices • Lack of insurance and compensation benefits		
Burns-related injuries		• Lack of health care facilities • High costs of treatment	• Absence of fire safety mechanisms in public places • Unsafe electrical appliances • Unsafe stoves • Easily flammable clothes • Lack of transportation facilities, especially in rural areas	Risk-taking behaviour of people	
Occupational injuries	Lack of knowledge of first aid	• Lack of health care facilities in small industries and rural areas • Lack of emergency care in the unorganized labour sector	• Poor work safety laws and poor implementation of laws • Hazardous workplaces • Use of unsafe technology • Greater use of toxic chemicals and materials • Lack of periodic inspection of workplaces • Child labour	Inadequate experience of workers	
Drowning	Lack of knowledge of first aid	• Poor emergency care services • Lack of medical treatment facilities • Lack of skill-based training of health personnel • Lack of communication facilities in health care settings • Lack of advocacy by health personnel for injury prevention and control	• Presence of unprotected wells, ponds and lakes • Natural disasters such as cyclones and floods		
Poisoning	Absence of specific antidotes in peripheral hospitals	• Poor emergency care services • Lack of medical treatment facilities • Lack of skill-based training of health personnel • Lack of communication facilities in healthcare settings • Lack of advocacy by health personnel for injury prevention and control	• Lack of laws and systems to regulate the availability and use of OPC and drugs • Increasing use of toxic chemicals • Unsafe workplaces	• Absence of parental supervision • Drugs within the easy reach of children	
Suicide	• Lack of skills to detect those with suicidal ideation • Lack of screening of high-risk groups	• Easy availability of OPC and drugs • Lack of supportive care facilities in schools and workplaces • Lack of transportation	• Impact of media portrayal • Medicolegal issues and complications • Growing disparity in society • Stigma in society	• Increasing domestic violence • Alcohol-related problems	

(Cont.)

Table A14 (*cont.*) Causes of injuries

Type of injury	Direct (clinical-point of care)	Direct and indirect (health system)	Indirect (socioeconomic determinants)	Indirect (lifestyle RF, awareness)	Indirect (genetic, medical history, age, sex, ethnicity RF)
		systems • Lack of first-aid facilities • Lack of health interventions for suicide attempters	• Lack of employment opportunities • Increasing stress in society		
Violence	Absence of screening for patients who have experienced violence	• Lack of counselling and other support systems within the health sector • Lack of emergency helplines • Absence of crisis prevention centres	• Changing society values • Easy availability of weapons • Lack of employment opportunities • Lack of educational opportunities • Stigma with regard to violence • Non-availability of help from concerned sectors • Growing disparity between people and society • Discriminatory laws in society • Growing depiction of violence in the media		

OPC: organophosphorus compounds

Annexure B: Interventions for the management of diseases/health conditions at different levels of care

NATIONAL COMMISSION ON MACROECONOMICS AND HEALTH, NEW DELHI

S. No.	Activity	Community	Subcentre	PHC	CHC	District level
	Maternal health					
1	Creating awareness on danger signs during pregnancy and childbirth		✓			
2	Preparing for the birth of a child (birth preparedness)	✓				
3	Early identification of a complicated case during pregnancy/delivery	✓				
4	Referral of complicated case to an appropriate health facility	✓				
5	Arranging for transportation of patients with complications to an appropriate health facility	✓				
6	Accompanying the patient to an appropriate health facility	✓				
7	Admitting the patient for treatment			✓		
8	Administering drugs (oxytocics/anticonvulsants/antibiotics)			✓		
9	Donating blood	✓				
10	Arranging blood in case a voluntary donor is not available				✓	
11	Performing a caesarean section if required				✓	
12	Follow-up of a case at home		✓			
13	Promoting contraception, birth spacing and terminal methods	✓				
14	Organizing sterilization operation camps			✓		
15	Performing sterilization operations in camps and at CHCs				✓	
16	Early detection and registration of pregnancy (in the first trimester)	✓				
17	Providing regular ANC check-ups		✓			
18	Creating awareness on the availability of safe abortion services in the district	✓				
19	Providing safe abortion services			✓		
20	Preventing child marriage	✓				
21	Providing TT immunization to adolescents		✓			
22	Screening for anaemia in adolescents	✓				
23	Providing IFA supplements to adolescents	✓				
24	Checking the nutritional status of adolescents	✓				
25	Conducting safe deliveries at home	✓				
26	Promoting institutional deliveries	✓				
27	Providing training to TBAs for early identification of complications		✓			
28	Creating awareness about the symptoms of RTIs/STIs	✓				
29	Screening for RTIs/STIs		✓			
30	Providing treatment for RTIs/STIs			✓		

(Cont.)

S. No.	Activity	Community	Subcentre	PHC	CHC	District level
	Child health					
1	Providing basic newborn care		✓			
2	Noting down the birth weight		✓			
3	Home visits for newborn care		✓			
4	Helping the mother to initiate breastfeeding	✓				
5	Promoting exclusive breastfeeding for six months	✓				
6	Discouraging bottle feeding	✓				
7	Encouraging mothers to feed colostrum to the newborn and not discard it	✓				
8	Early identification and initial management of neonates with asphyxia		✓			
9	Management of neonates with birth asphyxia				✓	
10	Early identification of cases of neonatal sepsis		✓			
11	Management of cases of neonatal sepsis				✓	
12	Giving immunization to children		✓			
13	Monitoring the growth of children by recording the weight periodically	✓				
14	Promoting personal hygiene—washing hands before feeding and after toilet	✓				
15	Recognition of signs and symptoms of dehydration	✓				
16	Promoting increased fluid intake/ORS in case of dehydration	✓				
17	Hospitalization and administration of IV fluids for severe dehydration			✓		
18	Recognition of signs and symptoms of pneumonia	✓				
19	Home management of ARI	✓				
20	Management of cases with pneumonia		✓			
21	Management of cases of severe pneumonia			✓		
22	Home-based management of children with malnutrition	✓				
23	Hospitalization for management of children with malnutrition				✓	
	Blindness					
1	Creating awareness (about symptoms and treatment)	✓				
2	Identification of suspect cases of cataract	✓				
3	Referral of suspect cases of cataract to a health facility	✓				
4	Screening for cataract and refractive errors by PMOA			✓		
5	Operation for cataract				✓	
6	Follow-up of operated cases of cataract		✓			
7	Routine screening for refractive errors in schoolgoing children		✓			
8	Referral of a child with refractive errors to the PHC		✓			
9	Creating awareness on the need for eye care	✓				
10	Establishing eye banks and making them operational					✓
11	Promoting eye donation in the community	✓				
	HIV infection/AIDS					
1	Creating awareness (about symptoms, spread, prevention, treatment)	✓				
2	Identification of persons with high-risk behaviour	✓				
3	Promoting the use of condoms/safer sex by people in the reproductive age group	✓				
4	Supply of condoms to sexually active people	✓				
5	Promotion of voluntary blood donation	✓				
6	Counselling of persons with HIV infection/AIDS		✓			
7	Testing for HIV infection in a person with high-risk behaviour				✓	

(Cont.)

S. No.	Activity	Community	Subcentre	PHC	CHC	District level
8	Screening for RTIs/STIs		✓			
9	Clinical diagnosis of RTIs/STIs		✓			
10	Laboratory diagnosis of RTIs/STIs					
11	Treatment of a patient with RTI/STI		✓			
12	Screening of donated blood for the presence of HIV infection					✓
13	Clinical diagnosis of AIDS					✓
14	Supply of drugs for the treatment of AIDS				✓	
15	Supervising the administration of drugs to a patient with HIV infection/AIDS	✓				
16	Targetted supervision	✓				
17	School AIDS programme		✓			
18	Care and support	✓				
	Tuberculosis					
1	Creating awareness (about symptoms, spread, treatment, BCG vaccination)	✓				
2	Identification of suspected cases of TB	✓				
3	Referral of suspected cases of TB to a health facility	✓				
4	Clinical diagnosis of TB			✓		
5	Taking a sputum sample of a suspected case			✓		
6	Laboratory diagnosis of TB and follow-up visits			✓		
7	Supply of drugs to a patient with TB			✓		
8	Supervising the intake of drugs	✓				
9	Follow-up for complete treatment of a patient with TB		✓			
10	Follow-up of contacts of sputum-positive cases of TB		✓			
11	Providing BCG vaccination		✓			
12	Community mobilization to remove the stigma associated with TB	✓				
13	Treating complicated cases of TB					✓
	Leprosy					
1	Health education	✓				
2	Early identification and referral of a suspected case of leprosy	✓				
3	Clinical diagnosis of a case of leprosy			✓		
4	Starting of treatment			✓		
5	Follow-up of treatment		✓			
6	Management of complications			✓		
7	Prevention and care of deformity			✓		
8	Purchase of MCR footwear					✓
9	Rehabilitation					✓
	Malaria and other vector-borne diseases					
1	Health education	✓				
2	Identification of suspected cases of malaria (any case of fever)	✓				
3	Taking the body temperature of a suspected case of malaria	✓				
4	Transporting patients with suspected malaria to a health facility	✓				
5	Clinical diagnosis of malaria		✓			
6	Preparing a blood slide of a suspected case	✓				
7	Laboratory diagnosis of malaria—blood slide examination			✓		
8	Supply of drugs to a patient with malaria	✓				

(Cont.)

S. No.	Activity	Community	Subcentre	PHC	CHC	District level
9	Follow-up of complete (radical) treatment of a patient with malaria		✓			
10	Treating patients with complicated malaria				✓	
11	Preventing accumulation of water in open places	✓				
12	Weekly draining out of water from coolers and cleaning them	✓				
13	Keeping overhead tanks covered with lids	✓				
14	Using bednets at night (optional)	✓				
15	Using mosquito repellants	✓	✓			
16	Taking precautions against mosquito bites while going to a jungle	✓				
17	Developing hatcheries for larvivorous fish	✓				
18	Spraying insecticides	✓				
19	Pouring mineral oil over small collections of water in the neighbourhood	✓				
20	Entomological surveillance for malaria and vector-borne diseases	✓				✓
	Cancers					
1	Health education		✓			
2	Motivation for cancer screening		✓			
3	Collection of Pap smear		✓			
4	Inspection of the cervix		✓			
5	Teaching self-inspection of the oral cavity and breast		✓			
6	FNAC and Pap smear					✓
7	Cerviscopy					✓
8	Staining of cytology smears					✓
9	Biopsy			✓		
10	Cancer-related physical examination			✓		
11	Management of pre-cancers			✓		
12	Management of early cancers					✓
13	Colposcopy					✓
	Mental health					
1	Early detection of problem/screening	✓				
2	Early detection of psychiatric emergencies			✓		
3	Providing psychiatric first aid			✓		
4	Referral			✓		
5	Diagnosis of cases				✓	
6	Short-term medical management				✓	
7	Long-term medical management					✓
8	Health promotion and behaviour modification					✓
9	Rehabilitation					
	a Community resource centre				✓	
	b Residential facility					✓
10	Training					
	a Health personnel					✓
	b Patient and family members			✓		
	c Community volunteers		✓			
11	Liaison with patients	✓				
12	Ensuring continuity of care		✓			

(Cont.)

S. No.	Activity	Community	Subcentre	PHC	CHC	District level
	COPD and asthma					
1	Health education	✓				
2	Early identification of symptomatics	✓				
3	Peak flow gauge measurement		✓			
4	Spirometry for assessing lung function					✓
5	Medical management as outpatient			✓		
6	Medical management of inpatients				✓	
	Dental health					
1	Dental check-up			✓		
2	Atraumatic restorative treatment (ART)			✓		
3	Silver filling				✓	
4	Aesthetic fillings					✓
5	Indirect restorations	To be done at tertiary level hospitals				
6	Root canal treatment					✓
7	Scaling and polishing of teeth				✓	
8	Surgical procedures (gingivectomy, flap operation, mucogingival surgeries, endodontic surgeries)			✓		
9	Orthodontics—removable					✓
10	Orthodontics—fixed appliances	To be done at tertiary level hospitals				
11	Complete dentures				✓	
12	Partial dentures (removable)				✓	
13	Partial dentures (fixed)	To be done at tertiary level hospitals				
14	Biopsy				✓	
15	Surgical extraction (impaction)					✓
16	Fracture reduction/cyst enucleation/benign growth excision					✓
	Diabetes					
1	Health education	✓				
2	Screening		✓			
3	Getting blood sugar test done			✓		
4	Starting treatment			✓		
5	Management on insulin				✓	
6	Screening and treatment of complications				✓	
7	Follow-up for compliance		✓			
8	Management of retinopathy, nephropathy and other complications					✓
	Hypertensive stroke					
1	Screening	✓				
2	Starting treatment			✓		
3	Management of difficult cases					✓
4	Health education	✓				
5	Follow-up for compliance		✓			
6	Rehabilitation			✓		

(Cont.)

S. No.	Activity	Community	Subcentre	PHC	CHC	District level
	Cardiovascular diseases					
1	Health education	✓				
2	Screening			✓		
3	Starting treatment			✓		
4	Management of difficult cases of hypertension				✓	
5	Management of other difficult cases					✓
6	Follow-up for compliance		✓			
7	Management of acute myocardial infarction	To be done at tertiary level hospitals				
	Oto–rhino–laryngyological diseases (ENT)					
1	Ear health education	✓				
2	Early detection of otitis media		✓			
3	Medical management of ENT diseases			✓		
4	Surgical management of ENT diseases					✓
	Injuries					
1	Assess consciousness, look for respiratory distress and call for help	✓				
2	Assessment of the airway and manual manoeuvre	✓				
3	Use of suction			✓		
4	Endotracheal intubation				✓	
5	Cricothyroidotomy and surgical intervention					✓
6	Application of external pressure and elevation of the affected part	✓				
7	Assessment of shock and medical management of injury			✓		
8	Providing first aid	✓				
9	Stabilization and immobilization of simple fractures	✓				
10	Stabilization and immobilization of advanced fractures			✓		
11	Referral			✓		
12	Orthopaedic and neurosurgical management					✓
13	Social rehabilitation	✓				
14	Medical rehabilitation					✓
15	Admission of cases of attempted suicide				✓	
16	After care for suicide attempters					✓
17	Flushing animal-bite wound with running water	✓				
18	Controlling bleeding with occlusive dressing		✓			
19	Immobilizing the limb below the level of the heart in the neutral position		✓			
20	Scraping the skin surface			✓		
21	Identification of the type of snake-bite and wound management			✓		
22	Prevention and control of anaphylactic reactions to antivenom				✓	
23	Post-exposure treatment in dog-bite with vaccine and administration of immunoglobulin			✓		

PHC: primary health centre; CHC: community health centre; TT: tetanus toxoid; IFA: iron–folic acid; ANC: antenatal care; TBA: traditional birth attendant; RTI: reproductive tract infection; STI: sexually transmitted infection; ORS: oral rehydration salt; IV: intravenous; ARI: acute respiratory infection; PMOA: paramedical opthalmic assistant; BCG: bacille Calmette-Guérin; TB: tuberculosis; MCR: microcellular rubber; FNAC: fine-needle aspiration cytology; Pap: Papanicolaou; COPD: chronic obstructive pulmonary disease

Background Papers of the National Commission on Macroeconomics and Health

MINISTRY OF HEALTH AND FAMILY WELFARE
GOVERNMENT OF INDIA, 2005