MANGESH SURYAKANT PEDNEKAR

# The Impact of Tobacco Use and/or Body Composition on Adult Mortality in Urban Developing Country Population

## Results from the Mumbai Cohort Study, Mumbai, India, 1991–2003

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ACADEMIC DISSERTATION
To be presented, with the permission of
the Faculty of Medicine of the University of Tampere,
for public discussion in the Auditorium of
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To my beloved mother:
Ms. Suman Suryakant Pednekar

# Contents

# List of Tables



# List of Figures

# List of Original Publications

This dissertation is based on the following original publications, referred to in the text by Roman numerals I–V:

I. Gupta PC, **Pednekar MS**, Parkin DM and Sankaranarayanan R (2005): Tobacco associated mortality in Mumbai (Bombay) India. Results of the Bombay Cohort Study. Int J Epidemiol 34:1395–1402.

II. **Pednekar MS** and Gupta PC (2007): Prospective study of smoking and tuberculosis in India. Prev Med 44:496–498.

III. **Pednekar MS**, Hakama M, Hebert JR and Gupta PC (2008): Association of body mass index with all-cause and cause-specific mortality: findings from a prospective cohort study in Mumbai (Bombay) India. Int J Epidemiol 37:524–535.

IV. **Pednekar MS**, Gupta PC, Shukla HC and Hebert JR (2006): Association between tobacco use and body mass index in urban Indian population: implications for public health in India. BMC Public Health 6:70.

V. **Pednekar MS**, Gupta PC, Hebert JR and Hakama M (2008): Joint Effects of Tobacco Use and Body Mass on All-Cause Mortality in Mumbai, India: Results from a Population-based Cohort Study. Am J Epidemiol 167:330–340.
Erratum. Am J Epidemiol. (in press).

# Abbreviations

| | |
|---|---|
| AD | Anno Domini |
| AIDS | Acquired Immuno Deficiency Syndrome |
| BC | Body Composition |
| BMC | Bombay Municipal Corporation |
| BMI | Body Mass Index |
| CED | Chronic Energy Deficiency |
| CI | Confidence Interval |
| cm | centimetre |
| COPD | Chronic Obstructive Pulmonary Disease |
| e.g. | for the sake of example |
| g | gram or gramme |
| GDP | Gross Domestic Product |
| HIV | Human Immunodeficiency Virus |
| IARC | International Agency for Research on Cancer |
| ICD | International Classification of Diseases |
| i.e. | that is |
| IHD | Ischemic Heart Disease |
| ILTD | Indian Leaf Tobacco Development Division |
| ITC | Indian Tobacco Company |
| kg | kilogramme |
| km | kilometre |
| m | metre |
| mg | milligram |
| MCS | Mumbai Cohort Study |
| NNMB | National Nutrition Monitoring Bureau |
| OR | Odds Ratio |
| PAF | Population Attributable Fraction |
| PBCR | Population Based Cancer Registry |
| RBD | Registration of Births and Deaths |
| RR | Relative Risk |
| $ | dollar |
| SES | Socio-economic Status |
| TB | Tuberculosis |
| TRC | Tobacco Related Cancer |
| vs. | Versus |
| WHO | World Health Organization |
| WHS | World Health Survey |
| & | and |

# Summary

Worldwide, there are two important risk factors underlying the major causes of death, tobacco use and nutritional status. Of the total 55.9 million global annual deaths, tobacco use and nutritional status, together, account for approximately 20%. Information about excess mortality from different forms of tobacco use other than cigarette smoking that are widely prevalent in India (such as *bidi* smoking and the various forms of smokeless tobacco use), is very limited. Using Mumbai Cohort Study (MCS) data, the present study reports on the association of various kinds of tobacco habits that are prevalent in India with all-causes of mortality and with major causes [such as cancers, tuberculosis (TB), etc.].

Nutrition research in India has focused primarily on the problem of undernutrition, particularly among vulnerable women and children. Currently, India is undergoing a rapid economic transition. At this stage in the associated epidemiologic transition, the country is facing the double burden of communicable and non-communicable diseases. In all such transitions, nutrition plays a central role.

The joint effect of tobacco use (mainly smoking) and body mass on mortality has not been well characterized, although a body of evidence is accumulating on the individual effect of smoking on the association of body mass and mortality. Most investigators have either adjusted for tobacco use or stratified by tobacco use in assessing the association of body mass index (BMI) with mortality. However, to the best of our knowledge, none of the large prospective studies have assessed the joint effect of tobacco use and BMI on mortality. Using MCS, this was the first such attempt, from a developing country population, where both under- and over- nutrition and tobacco use are major public health concerns.

Using the Mumbai electoral role list as the selection frame, a total of 148,173 individuals aged ≥ 35 years were recruited in two phases for a prospective follow-up study. During phase I (1991–1994), men and women aged ≥ 35 years were recruited, and during phase II (1994–1997) only men aged ≥ 45 years were recruited. The endpoint of the prospective cohort study was mortality; hence, the survey was confined to individuals aged ≥ 35 years.

An active house-to-house follow-up (to ascertain vital status) after an average 5.5 years was also conducted in two phases; individuals recruited during phase I were

followed up during the years 1997–1999 and individuals recruited during phase II were followed-up during the years 2000–2003. The information on cause of death was abstracted from the death registers of Bombay Municipal Corporation (BMC) and linked with cohort deaths to ascertain specific cause of death occurring in the Mumbai cohort. Causes of death were coded using the international classification of diseases 10$^{th}$ revision (ICD–10).

The baseline survey included the following 2 components: (1) anthropometric measurements; and (2) interviewer administration of structured questionnaire to obtain information on age, gender, education, religion, mother tongue, and information related to frequency and type of tobacco usage.

At active follow-up (1997–2003), 140,908 (95%) individuals were traced. Among these, 13,261 deaths were recorded, of which 85% occurred within the study area. It was possible to abstract cause of death information from BMC death registers for 9,259 deaths. These were coded using ICD–10.

In Mumbai, tobacco use prevalence was higher among men (≈70%) than women (≈60%), but women were primarily smokeless tobacco users (as smoking prevalence was <1%). For men, the adjusted death rate (per 1,000 person-years) among smokers (23.3) was higher than those among never-users of tobacco (13.6) and the adjusted (using Cox regression model) relative risk (RR) was 1.55 [95% confidence interval (CI): 1.42–1.69]. Adjusted RR was 1.37 (95% CI: 1.23–1.53) for cigarette smokers and 1.64 (95% CI: 1.47–1.81) for *bidi* smokers, with a statistically significant dose-response relationship for number of *bidis* or cigarettes smoked per day. Women were essentially smokeless tobacco users and the adjusted RR was 1.25 (95% CI: 1.15–1.35).

Smoking among men increased the risk of dying from respiratory diseases (RR: 2.12, 95% CI: 1.57–2.87), TB (RR: 2.30, 95% CI: 1.68–3.15), circulatory diseases (RR: 1.21, 95% CI: 1.04–1.40) and all neoplasms (RR: 2.60, 95% CI: 1.78–3.80). The adjusted RR of dying from TB among *bidi* smokers was 2.60 (95% CI: 2.02–3.33) times higher than never-smokers, with a statistically significant trend for daily frequency of *bidi* smoking. Also, the risk of self-reported TB among *bidi* smokers was 5.23 (95% CI: 4.01–6.82) times higher than among never-smokers.

The RRs for smokeless tobacco users, although higher than unity, did not reach statistical significance for several categories of diseases, except for all neoplasms (RR: 1.57, 95% CI: 1.16–2.13), circulatory diseases (RR: 1.19, 95% CI: 1.02 to 1.38) among women and respiratory diseases (RR: 1.50, 95% CI: 1.12 to 2.03) and TB (RR: 1.46, 95% CI: 1.07 to 2.00) among men.

In Mumbai, 19.1% of women and 17.4% of men were underweight (BMI<18.5), while 29.4% of women and 20.2% of men were overweight (BMI≥25). There were more obese (BMI≥30) women (6.9%) than men (2.7%) in Mumbai. The association of BMI with all-cause mortality was non-linear. The adjusted (using Cox regression model) RRs were estimated compared with individuals having normal BMI [18.5–<25.0 kilogram(kg)/metre$^2$ (m)$^2$]. An increased risk of dying at both extremes (very high and low BMI values) was observed. Despite adjusting for possible confounders including tobacco use, there were no substantial variations in the results regarding the main effect of relative weight. Restricting the analyses to never-users of tobacco also yielded similar results; i.e., increased risk of dying at both extremes of BMI.

Elevated risk of death was observed across all underweight categories among women (RRs: 1.94 for BMI<16.0 kg/m$^2$; 1.38 for BMI 16.0–<17.0 and 1.24 for BMI 17.0–<18.5) and men (the corresponding RRs were 2.24, 1.45, and 1.27 respectively). Extremely thin (BMI<16.0 kg/m$^2$) cohort members were at highest risk for death due to TB (RRs: 7.20 and 14.94 in women and men respectively), cancers (RRs: 1.87 and 2.44 respectively), and respiratory diseases (RRs: 3.46 and 4.35 respectively); and the risk remained high despite excluding deaths that had occurred during the first 2 years of follow-up.

Individuals with above normal BMI were at lower risk of dying than those having normal BMI. Overweight (BMI 25.0–<30.0) women (RR: 0.89, 95% CI: 0.81–0.98) and men (RR: 0.87, 95% CI: 0.82–0.93) were at lower risk; however, obese (BMI≥30.0) men <60 years of age had an increased risk of dying (RR: 1.22, 95% CI: 1.01–1.48). Overweight men and women were at ≈60% and ≈30% decreased risk of death due to TB and respiratory system diseases respectively; while obese women were at ≈30% increased risk of dying due to diseases of the circulatory system.

All forms of tobacco use were found to be associated with the prevalence of low BMI, and the prevalence of low BMI was highest among *bidi* smokers (32%). Further, there existed a dose-response gradient among men smokers and women smokeless tobacco users across various underweight categories.

MCS results also show that all forms of tobacco use and BMI had a joint effect on increased risk of death. Among men, obese smokers and obese never-users of tobacco were at 56% and 34% increased risk of dying respectively, compared with overweight never-users of tobacco. Similarly, at highest risk were extremely thin males who smoked *bidis* (RR: 3.45, 95% CI: 2.98–3.99) or cigarettes (RR: 3.32, 95% CI: 2.68–4.11).

Tobacco use and undernutrition are serious problems in India. The present study indicates that obesity may emerge as a serious public health problem.

All forms of tobacco smoking increased the risk of dying in Mumbai. In addition to smoking, different forms of smokeless tobacco use also resulted in excess mortality. Using MCS findings, a total of 24% male and 6% female deaths (aged 35–69 years) were found to be attributable to their tobacco usage. Also, 41.6% of men's and 20.7% of women's cancer deaths were found to be attributable to their tobacco usage. *Bidi* smoking was found to be as harmful as cigarette smoking and was found to be responsible for around 32% of TB deaths.

Therefore, MCS findings provide supportive evidence from the population of a developing country about the association of tobacco usage with increased risk of death; primarily for various cancers and TB.

MCS results showed that both chronic underweight and overweight are equally present in an urban population of India. However, the important public health implications for the burden of diseases are associated with only the upper extreme (obese) and all underweight body composition (BC). The results from MCS highlight the immediate need to identify and to address both underweight and obese portions of the distribution in identifying vulnerable targeting interventions.

Our study reported that tobacco use is a risk factor for low BMI. Further, tobacco use and low BMI had synergistic effect on mortality in men and antagonistic effect in women, independent of whether additive or multiplicative interaction was assumed.

The policy implications for prevention would be that improving the nutritional status of those underweight and preventing use of tobacco results in the immediate highest yield.

# 1. Introduction

## 1.1 Theme of the study

Worldwide, there are two important risk factors underlying the major causes of death, tobacco use and nutritional status (Beaglehole and Yach 2003; Ezzati et al. 2004; Boyle et al. 2006; Lopez and Mathers 2006; Mathers and Loncar 2006). Their effects are now increasing rapidly, with high prevalence rates of smoking and other forms of tobacco use in many parts of the world (Peto et al. 1994; Anderson 2006; Jha et al. 2006a), and a virtual epidemic of overweight and obesity and chronic undernutrition in other parts of the world (Sørensen 2000; de Onis et al. 2004; Uauy and Lock 2006). Of the total 55.9 million annual deaths worldwide, tobacco usage and nutritional status are together responsible for approximately 20% of deaths (Ezzati et al. 2004).

After China [where tobacco accounted for about 12% of male deaths at ages 35–69 years in 1990 (Niu et al. 1998)], India is the second most populous country, comprising around 17% of the world's population, and contributing 16% of the world's deaths (Jha et al. 2006b). India is the world's second largest producer and consumer (unmanufactured) of tobacco (Reddy and Gupta 2004; Crofton and Simpson 2006). India has a long history of tobacco use, including smokeless tobacco use and many forms of smoking, of which cigarette smoking is only a minor part (Bhonsle et al. 1992; Reddy and Gupta 2004). All forms of tobacco use may carry serious health consequences (Sanghvi and Notani 1989; Reddy and Gupta 2004); therefore, the burden of deaths and diseases from tobacco use in India may be greater than those included in global estimates, which are based primarily on cigarette smoking (Gupta 1991). A cohort study was undertaken in Mumbai with a view to obtaining all-cause and cause-specific tobacco attributable mortality in India (I, II).

Worldwide, obesity is recognized as a serious health problem. Studies conducted mainly in the West have shown that obesity is closely associated with increased risks of many diseases, disability, and reduced life expectancy (National Institutes of Health

1998; World Health Consultation on Obesity 1998). Although the problem is not as widespread as it is in the West, a recent report by World Health Organization (WHO) and others (The International Diabetes Institute et al. 2000) has shown an alarming increase in the disease risk due to obesity in the Asian countries. However, there are very few or no datasets to enable a longitudinal study of the association between obesity and mortality from India or other parts of the developing world, where underweight (BMI<18.5 kg/m$^2$) continues to be bigger concern. We report the results from a cohort study regarding the association of BC with all-cause and cause-specific mortality among men and women aged ≥ 35 years in Mumbai, India (III).

The association between low BMI and smoking is well documented (Molarius et al. 1997; Bovet et al. 2002). However, recent studies show that the nature of this relationship depends on educational level, gender, ethnicity, and frequency (e.g., number of cigarettes per day) of smoking (Molarius and Seidell 1997; Laaksonen et al. 1998). In India, tobacco is used in various forms (Bhonsle et al. 1992; Reddy and Gupta 2004). The high prevalence of both tobacco use as well as low BMI raises important questions about its impact on public health in India. Paper IV focuses on providing a detailed analysis of the relation between different forms of tobacco use and low BMI.

In recent years, many prospective epidemiological studies have evaluated the relationship between BMI and mortality (Singh et al. 1999a; Haapanen-Niemi et al. 2000; Song and Sung 2001; Meyer et al. 2002; Calle et al. 2003; Engeland et al. 2003; Flegal et al. 2005; McGee DL and Diverse Populations Collaboration 2005). The relationship between body weight and all-cause mortality is controversial and the observed relationship was from linear to J- or U-shaped (Manson et al. 1995; Troiano et al. 1996; Meyer et al. 2002; Ajani et al. 2004; Flegal et al. 2005; McGee DL and Diverse Populations Collaboration 2005; Gu et al. 2006). Inconsistency in findings may be due to the confounding effects of smoking behaviour and some serious illness that are associated with higher mortality and lower BMI (Manson et al. 1987; Willett et al. 1999). Most studies have either adjusted for, or stratified by tobacco use while assessing the BMI and mortality association. However, none of the large prospective studies (mainly from developing countries) have assessed the joint effect of tobacco use and BMI on mortality. Therefore, Paper V focuses on providing a detailed analysis of the joint effects of tobacco use and BC on all-cause mortality.

## 1.2 Background population

### *1.2.1 India*

The Republic of India, commonly known as India, is a sovereign country in South Asia. It is the seventh largest country by geographical area, the second most populous country, and the most populous liberal democracy in the world (Country profile: India 2007).

India is a union of twenty-eight states and seven federally governed union territories. All states, the union territory of Puducherry, and the National Capital Territory of Delhi have elected governments. The other five union territories have centrally appointed administrators.

With an estimated population of 1.1 billion (CIA Fact Book: India 2007), India is the world's second most populous country. Almost 70% of Indians reside in rural areas, although in recent decades migration to larger cities has led to a dramatic increase in the country's urban population. India's largest urban agglomerations are Mumbai (formerly Bombay), Delhi, Kolkata (formerly Calcutta), Chennai (formerly Madras), Bangalore, Hyderabad and Ahmedabad.

With a gross domestic product (GDP) growth rate of 9.4% in 2006-07, the Indian economy is among the fastest growing in the world (Government of India 2007). The Indian economy has grown steadily over the last two decades; however, its growth has been uneven when comparing different social groups, economic groups, geographic regions, and rural and urban areas (World Bank 2006). Although income inequality in India is relatively small (Gini coefficient: 0.33 in year 2000), it has been increasing recently. Despite significant economic progress, a quarter of the nation's population earns less than the government-specified poverty threshold of $0.40 per day. In addition, India has a higher rate of malnutrition among children under the age of three than any other country in the world (World Bank 2006).


**Figure 1.** Administrative divisions of India

India has a labour force of 509.3 million, 60% of which is employed in agriculture and related industries (CIA Fact Book: India 2007). Major agricultural crops include rice, wheat, oilseed, cotton, jute, tea, sugarcane, and potatoes. The agricultural sector accounts for 28% of GDP; the service and industrial sectors make up 54% and 18% respectively. At the time of India's independence in 1947, its literacy rate was 12.2%. Since then, it has increased to 64.8% (53.7% for females and 75.3% for males) in 2007. The state of Kerala has the highest literacy rate (91%); Bihar has the lowest (47%). There is considerable variation within states, within districts and even between urban and rural areas. The national gender ratio is 944 females per 1,000 males. In India the life expectancy at birth is 68.6 years and the median age is 24.8, and the population growth rate of 1.6% per annum; there are 22.7 births and 6.6 deaths per 1,000 people per year (CIA Fact Book: India 2007).

### *1.2.2 Mumbai*

Mumbai, formerly known as Bombay, is the capital of the state of Maharashtra, the most populous city of India, and by some measures the most populous city in the world with an estimated population (as of 2007) of about 13.1 million (World Gazetteer 2007). Mumbai is located on Salsette Island, off the west coast of Maharashtra. Along with its neighbouring suburbs, it forms the world's fourth most populous metropolitan area with a population of about 19.4 million and is projected to rise to 25.7 million by 2020 due to an annual growth rate of 2.2% (Demographia 2007). The city has a deep natural harbour and the port handles over half of India's passenger traffic and a significant amount of cargo.


**Figure 2.** Administrative divisions of Mumbai

The climate of the city, being in the tropical zone, and near the Arabian Sea, may be broadly classified into two main seasons — the humid season, and the dry season. Up until the 1980s, Mumbai owed its prosperity largely to textile mills and the seaport, but the local economy has since been diversified to include engineering, diamond-polishing, healthcare and information technology. Mumbai's status as the state capital means that state and federal government employees make up a large percentage of the city's workforce. Mumbai also has a large unskilled and semi-skilled labour population, who primarily earn their livelihood as hawkers, taxidrivers, mechanics and other such blue collar professions. The port and shipping industry too employs many residents, directly or indirectly.

Along with the rest of India, Mumbai, its commercial capital, has witnessed an economic boom since the liberalization of 1991, the finance boom in the mid nineties, and the IT, export, services, and BPO (business process outsourcing) boom in the current decade. The middle class in Mumbai is the segment most affected by this boom and is the driver behind the consequent consumer boom. Upward mobility among *Mumbaikars* (residents of Mumbai) has led to a direct increase in consumer spending.

The population of Mumbai (as of 2007) is about 13.1 million (World Gazetteer 2007). There are 809 females to every 1,000 males – which is lower than the national average. The overall literacy rate of the city is above 86%, which is higher than the national average (Census of India 2007). The religions represented in Mumbai include Hindus (68% of the population), Muslims (17% of the population), and Christians and Jains (4% each). The remainder are Parsis, Buddhists, Sikhs, Jews and atheists (Census of India 2007). According to the 1991 census, the ethnic groups' demographics are – Marathi people (42%), Gujarati people (18%), *North Indian* (21%), Kannadigas (5%), Tamil people (3%), Sindhis (3%) and others.

Like other large cities in the developing world, Mumbai suffers from the same major urbanization problems seen in many fast growing cities in developing countries – widespread poverty and poor public health, employment, civic and educational standards for a large section of the population. With available space at a premium, Mumbai residents often reside in cramped, relatively expensive housing, usually far from workplaces, and therefore requiring long commutes on crowded mass transit, or congested roadways.

## 1.3 Birth and death registration in India

The registration mechanism of birth and death in India is considered as permanent and compulsory, primarily for its value as a legal document and secondly for its usefulness as a source of statistics. The registration of births and deaths (RBD) has been enacted all over India through the enforcement of the RBD Act –1969 (The Registration of Births and Deaths Act 1969). Births and deaths are required to be reported to local registrars in the local urban or rural bodies (PRIA 2005). Registration is free (local bodies charge a small fee for copies); there is a late fee for late registration (over 30 days after the event) and a moderate fine for non-registration (National Commission on Population 2001).

Even though a 'birth certificate' is considered an assurance for a hurdle free future for our children, only 65% of the Indian population has gone through the registration process. That is, with about 26 million births taking place in our country in a year; only about 17 million births are registered.

The data available on death registration shows that an average of only 60% of deaths that occurred could be registered every year across the country. The concern over poor registration of deaths still prevails in the states of Assam, Bihar and Jharkhand. In these states, averages of only 30% of deaths are registered (PRIA 2005). Although, the extent of registration varies across different parts of the country, in Mumbai, almost all deaths are registered and certified medically.

# 2. Review of the Literature

## 2.1 Tobacco

### *2.1.1 Burden for public health*

Tobacco is packed with harmful and addictive substances. Scientific evidence has shown conclusively that all forms of tobacco cause health problems throughout life, frequently resulting in death or disability. Smokers have markedly increased risks of multiple cancers, particularly lung cancer, and are at far greater risk of heart disease, strokes, emphysema and many other fatal and non-fatal diseases. Those who chew tobacco, risk cancer of the lip, tongue and mouth.

Women suffer additional health risks. Smoking in pregnancy is dangerous to the mothers as well as to the foetus, especially in countries where health facilities are inadequate. Maternal smoking is not only harmful during pregnancy, but has long-term effects on the baby after birth. This is often compounded by exposure to passive smoking by the mother, father or other adult members.

Of everyone alive today, 650 million will eventually be killed by tobacco globally. No other consumer product is as dangerous, or kills as many people. Tobacco kills more than Acquired Immuno Deficiency Syndrome (AIDS), legal drugs, illegal drugs, road accidents, murder, and suicide combined. Tobacco already kills more men in developing countries than in industrialized countries, and it is likely that deaths among women will soon be the same. Annual deaths due to tobacco in industrialized countries were 1.3 million in 1975 and reached 2.1 million in 2001 and will increase further to 3 million by 2025–30, whereas in developing countries this was 0.2 million in 1975, 2.1 million in 2001, and 7 million in 2025–30, which is a very rapid increase. While 0.1 billion people died from tobacco use in the 20$^{th}$ century, ten times as many will die in the 21$^{st}$ century. The main diseases by which smoking kills people are substantially different in America (where vascular disease and lung cancer predominate) (Peto et al. 1994), in China [where chronic obstructive pulmonary disease (COPD) predominates, causing even more tobacco deaths than lung cancer] (Liu et al. 1998; Niu et al. 1998), and in India (where half of the world TB deaths take place, and the ability of smoking to increase the risk of dying from TB may well be of particular importance) (Gajalakshmi et al. 2003).

Around the world, it is estimated that there are currently 30 million who start to smoke every year. There are already a billion smokers, and by 2030 about another billion young adults will have started to smoke. On current smoking patterns, worldwide mortality from tobacco is likely to rise from about 4–5 million deaths a year in 2001 to about 10 million a year around 2030. A survey conducted in 1990 showed that in 44 industrialized countries, smoking caused an average of 24% of all male deaths – but 35% of these deaths are in middle age (35–69 years). It also caused 7% of all female deaths, overall. This proportion was about 12% in Chinese middle-aged men and was expected to rise to about 33% by 2030.

### *2.1.2 Smoking forms of tobacco use in India*

Tobacco was introduced into India by Portuguese traders around Anno Domini (AD) 1600. Its use and production proliferated to such a great extent that today India is the second largest producer of tobacco in the world. Soon after its introduction, tobacco became a valuable commodity of barter trade in India.

**Cigarette** smoking is the second most popular smoking form of tobacco used in India after *bidis*. The first cigarette factory, the Indian Tobacco Company of what is now known as ITC (formerly Imperial Tobacco Company) was established in Monghyr, Bihar, in 1906. In 1912, the first brand ‘Scissors’ was launched. The history of Indian Leaf Tobacco Development Division (ILTD), ITC’s research subsidiary, describes the history of the production of cigarette tobacco in India. By 1928, the ILTD had obtained definite results and markedly increased the area under cultivation of Virginia tobacco (Technology Information, Forecasting and Assessment Council 2007). Many national and multinational companies all together manufacture about 100 brands of cigarettes (Bhonsle et al. 1992).

***Bidis*** are the most popular form of smoking of tobacco in India. It was described as a product about the size of the little finger, containing a small quantity of tobacco wrapped in the leaf of a tree and sold in bundles of 20–30. This description of the finger-sized products corresponds to the *bidi* contemporarily available in India (Sanghvi 1992). Thirty-four percent of the tobacco produced in India is used for making *bidis*. *Bidis* are puffed more frequently than cigarettes to prevent them from going out. *Bidis* are made by rolling a dried, rectangular piece of tendu leaf with 0.15–0.25 gram (g) of sundried, flaked tobacco (Bhonsle et al. 1992).

The ***hookah*** is an Indian water pipe in which the tobacco smoke passes through water before inhalation. It was more common among women, the reason being that it was inconvenient for men to carry a *hookah*, whereas women remain at home most of the time.

A ***cheroot*** is a roll made from tobacco leaves. *Cheroots* were commonly smoked by both Indian men and women in south India.

***Chuttas*** are coarsely prepared *cheroots*. They are usually the products of cottage and small-scale industries, or are made at home. Nearly 9% of the tobacco produced in India is used for making *chuttas*. It is estimated that about 3000 million *chuttas* are made annually in India. *Chutta* smoking is widespread in the coastal areas of Andhra Pradesh, Tamil Nadu and Orissa.

The term 'reverse smoking' is used to describe smoking while keeping the glowing end of the tobacco product inside the mouth. Reverse *chutta* smoking is practised extensively by women in the rural areas of Visakhapatnam and the Srikakulam district of Andhra Pradesh (Pindborg et al. 1971).

Unlike *bidis* and *chuttas*, ***dhumtis*** are not available from vendors but are prepared by the smokers themselves. *Dhumti* is a kind of a conical cigar made by rolling tobacco leaf in the leaf of another plant. The overall prevalence is 4% in Goa (Bhonsle et al. 1976). *Dhumtis* may be occasionally smoked with the lighted end inside the mouth.

**Pipe** smoking is one of the oldest forms of tobacco use. The different kinds of pipes used for smoking range from the small-stemmed European types made of wood to long-stemmed pipes made from metal or other material.

***Hooklis*** are clay pipes commonly used in western India. Once the pipe is lit, it is smoked intermittently. On average, 15 g of tobacco is smoked daily. *Hookli* smoking was common among men in the Bhavnagar district of Gujarat (Mehta et al. 1969).

A ***Chillum*** is a straight conical pipe made of clay, 10–14 centimetre (cm) long, held vertically. It is exclusive and common among men and is limited to the northern states of India, predominantly rural areas. *Chillum* smoking requires a deep pulmonary effort. Often, one *chillum* is shared by a group. They are made locally, are inexpensive and easily available. The *Chillum* probably predates the introduction of tobacco to India and was used for smoking opium and other narcotics (Wahi 1968).

### *2.1.3 Smokeless forms of tobacco use in India*

The term 'smokeless tobacco' is generally used to describe tobacco that is consumed without heating or burning at the time of use. Smokeless tobacco can be used orally or nasally. For nasal use, a small quantity of very fine tobacco powder mixed with aromatic substances called dry snuff is inhaled. This form of smokeless tobacco use, although still in practice, is not very common in India.

The oral use of smokeless tobacco is very prevalent in India; the different methods of consumption include chewing, sucking and applying tobacco preparations to the teeth and gums. Smokeless tobacco products are often made at home but are also manufactured. Recently, varieties of smokeless tobacco products have been produced industrially on a large scale, commercially marketed and are available in small plastic and aluminium foil packets. Small pieces of raw or commercially available finely cut tobacco, are used for chewing of tobacco alone; however, they do not appear to be very common in India.

Several smokeless tobacco preparations such as *mishri*, *gudhaku*, *bajjar* and creamy snuff, are intended primarily for cleaning the teeth. Such use, however, soon becomes an addiction. In India, there is a widespread misconception that tobacco is good for the teeth. Many companies take advantage of this misconception by packaging and positioning their products as dental care products without explicitly stating so. The reason is that by law, oral care products may not contain tobacco. The law is not strictly enforced and some oral care products may still contain tobacco (Sinha et al. 2004b).

***Paan (betel quid) with tobacco.*** Tobacco, introduced as a product to be smoked, gradually began to be used in several other forms in India. It became an important additive to *paan* (betel quid). *Paan* chewing as a habit has existed in India and South-East Asia for over 2000 years. Stone inscriptions from the year AD 473 are historical evidence of its existence. In Hindu culture (the predominant religion in India), *paan* chewing is referred to as one of the eight *bhogas* (enjoyments) of life (Bhonsle et al. 1992). *Paan* chewing was adopted even by invading kings and settlers in India. It was also a part of Mughal culture. Several Mughal rulers were great connoisseurs of *paan* and employed specialists skilled in preparing *paans* to suit all occasions. The social acceptance and importance of *paan* increased further during the Mughal era.

The practice of chewing betel quid reached India by the first century or earlier. Some scholars believe that it was introduced from the South Sea Islands, Java and Sumatra, through contacts with the South Pacific Islands (Gode 1961). *Paan* chewing became a widely prevalent form of smokeless tobacco use after tobacco use took root in

India (Gupta and Ray 2004). Women ate *paan* for cosmetic reasons as chewing it produced a bright red juice that coloured their mouth and lips. The ancient scriptures have mentioned the use of *paan* being forbidden to people who adopt a religious mode of life or observe vows, widows, menstruating women and students. This popular practice became a convenient vehicle for chewed tobacco. The inclusion of tobacco as one of the ingredients of *paan* highlights the importance of this product and wide social acceptability of tobacco chewing in ancient India. Tobacco was chewed by itself, with areca nut or with lime in India in as early as 1708 (Bhonsle et al. 1992).

*Paan* chewing, or betel quid chewing, is often erroneously referred to as 'betel nut chewing'. *Paan* consists of four main ingredients – betel leaf (*Piper betle*), areca nut (*Areca catechu*), slaked lime [Ca(OH2)] and catechu (*Acacia catechu*). Betel leaves contain volatile oils such as eugenol and terpenes, nitrates and small quantities of sugar, starch, tannin and several other substances (Reddy and Gupta 2004; The Tobacco Timeline 2007). Condiments and sweetening agents may be added as per regional practices and individual preferences. Some time after its introduction, tobacco became an important constituent of *paan*, and currently most habitual *paan* chewers include tobacco.

Tobacco is the most important ingredient of *paan* for regular users. It is used in the raw state (as in Kerala) as well as after processing. Processing, additives and names differ from place to place. Tobacco is referred to as *kaddipudi* and *hogesoppu* in Karnataka, *kadapan* in Orissa and West Bengal, and *pattiwala* in Uttar Pradesh. *Zarda* and *kiwam* are commercially manufactured varieties often used as ingredients in *paan*.

***Paan masala*** is a commercial preparation containing areca nut, slaked lime, catechu and condiments, with or without powdered tobacco. *Paan masala* contains almost all the ingredients that go into the making of a *paan*, but are dehydrated so that the final product is not perishable. It comes in attractive sachets and tins, which can be stored and carried conveniently. *Paan masala* is very popular in urban areas and is fast becoming popular in rural areas. Although the actual prevalence of this practice is not known, its popularity can be gauged by the production figures: according to commercial estimates, the Indian market for *paan masala* is now worth several hundred million US dollars.

**Tobacco, areca nut and slaked lime preparations.** Combinations of tobacco, areca nut and slaked lime are chewed in several regions of north India, where they are known by different names.

**Mainpuri tobacco.** In the Mainpuri district of Uttar Pradesh and nearby areas this preparation is very popular. It contains mainly tobacco with slaked lime, finely cut areca nut, camphor and cloves (Wahi 1968).

***Mawa*** preparation contains thin shavings of areca nut with the addition of some tobacco and slaked lime. Its use is becoming popular in Gujarat, especially among youth. *Mawa* use is also prevalent in other regions of the country. The prevalence of *mawa* chewing has increased tremendously in recent years. Its magnitude can be assessed from the fact that the Bhavnagar city administration appealed to the people not to litter the streets with the cellophane wrappers of *mawa*, as they clogged the city drains!

**Tobacco and slaked lime (*khaini*).** Use of a mixture of sun-dried tobacco and slaked lime, known in some areas as *khaini*, is widespread in Maharashtra and several states of north India. A regular *khaini* user may carry a double-ended metal container, one side of which is filled with tobacco and the other with slightly moistened slaked lime. A small quantity of tobacco is taken in the palm and a little slaked lime is added. The ingredients are then mixed vigorously with the thumb and placed in the mouth. In Maharashtra and Gujarat, *khaini* is placed in the premolar region of the mandibular groove, whereas in Bihar and Uttar Pradesh, it is generally held in the lower labial groove.

***Mishri*** is a roasted, powdered preparation made by baking tobacco on a hot metal plate until it is uniformly black. Women, who use it to clean their teeth initially, soon apply *mishri* several times a day. This practice is common in Maharashtra (Mehta et al. 1972; Gupta 1996) and Goa (Pednekar and Gupta 2004).

***Gul*** is a pyrolysed tobacco product. It is marketed under different brand names in small tin cans and used as a dentifrice in the eastern part of India. Its use has also been reported among schoolchildren in Bihar, Arunachal Pradesh, Nagaland, Assam, UP and Uttaranchal (Mehta et al. 1969; Bhonsle et al. 1992; Sinha et al. 2003b; Sinha et al. 2004b). Among adults in several northeastern states of India, female school personnel reported significantly higher *gul* use than males (Sinha et al. 2003a).

***Bajjar*** is dry snuff (also known as *tapkeer*) applied commonly by women in Gujarat on the teeth and gums.

***Lal dantmanjan*** is a dentifrice; a red-coloured tooth powder. Traditionally, it contained tobacco but after the passing of a law banning the use of tobacco in dental care products, the listing of tobacco as an ingredient was stopped. A laboratory test of five samples of red tooth powder that did not declare tobacco as an ingredient found a tobacco content of 9.3-248 milligram (mg) per g of tooth powder (Sinha et al. 2004b). The Global Youth Tobacco Survey (GYTS), which focuses on schoolchildren's in the age group 13–

15 years, found the prevalence of its use to be 49% in Bihar, 29% each in UP and Uttaranchal, 25% in Orissa, 9% in Mizoram, 5% in Nagaland, 4% each in Arunachal, Assam and Meghalaya, 3% in Tripura, and 2% each in Goa, Maharashtra, Manipur and Sikkim (Sinha et al. 2003b; Sinha et al. 2003c; Pednekar and Gupta 2004; Sinha et al. 2004b).

***Gudhaku*** is a paste made of tobacco and molasses. It is available commercially and is carried in a metal container, but can be made by the users themselves. It is commonly used in Bihar, Orissa, Uttar Pradesh and Uttaranchal. *Gudhaku* is applied to the teeth and gums, predominantly by women.

**Creamy snuff.** Commercial preparations of tobacco paste are marketed in toothpaste-like tubes. They are advertised as possessing anti-bacterial properties and being good for the gums and teeth. These products are thus used like regular toothpaste, but users soon become addicted. This practice seems popular with children in Goa (Vaidya et al. 1992; Pednekar and Gupta 2004).

**Tobacco water** (known as *tuibur* in Mizoram and *hidakphu* in Manipur) is manufactured by passing tobacco smoke through water. Its use was reported in the Aizawl district (7.2%) of Mizoram and in the Churchandpur district (6.5%) of Manipur; use was similar among males and females. The frequency of tobacco water use varied from 1 to 30 times per day; in Aizawl and Churchandpur districts (Sinha et al. 2004a).

**Nicotine chewing gum** containing 2% nicotine (brand name good-kha) has been launched as a help in tobacco cessation. For chewers, it is available in *gutka* flavour and for smokers, in mint flavour.

**Areca nut preparations.** Some areca nut preparations are chewed without the inclusion of tobacco, but this practice may be present concurrently with the use of smokeless tobacco or tobacco smoking. Alkaloids present in areca nut are known to give rise to carcinogenic nitrosamines and areca nut has recently been evaluated as a human carcinogen by the WHO (IARC 2004b). The use of areca nut by itself appears to be mildly addictive but when used with tobacco, the effect multiplies manifold. Chewing of areca nut products is very common in India; therefore, a brief resume of these products is included here.

In addition to being an ingredient of *paan*, occasional chewing of areca nut (usually processed) alone is quite common in India, but habitual chewing is comparatively rare. Its use was reported in Maharashtra and in other rural areas of India (Mehta et al. 1972). In Assam, a fermented form of areca nut, known as *tamol* or *bura tamol*, is chewed extensively. This is prepared by preserving raw areca nuts together with areca leaves in an underground pit with an inner lining of straw for four months. *Bura tamol* is often infected with fungus. This product contains high levels of arecoline.

Areca nut is known as ***supari*** in several parts of north India. Some commercial *supari* preparations are made by cutting dried areca nuts into pieces and roasting them in fat to which flavouring, sweetening agents and condiments are added. *Supari* is marketed in attractive aluminium foil packs, in tins and in simple paper packets. Offering *supari* to guests, especially after meals, is a prevalent and well-accepted social custom.

***Meetha*** (sweet) ***mawa*** consists of thin shavings of areca nut, grated coconut, dried fruits and other sweetening agents. It is commonly used in Gujarat and similar preparations with different names are used widely in other regions.

***Paan* without tobacco.** Occasional *paan* chewers generally prefer *paan* without tobacco. Chewing *paan* without tobacco, known as *tambula* in Sanskrit, is an ancient practice in India. Areca nut is an indispensable ingredient of *paan*. In addition, a wide range of chewing products including a chewing gum that may not contain either areca nut or tobacco but contains strong betel quid flavours is available in the market.

## 2.2 Nutritional status

### *2.2.1 Burden for public health*

Under-and over-nutrition problems and diet related chronic diseases account for more than half of the world's diseases and hundreds of millions of dollars in public expenditure. Malnutrition is a major health problem, especially in developing countries. Water supply, sanitation and hygiene, given their direct impact on infectious diseases, especially diarrhoea, are important for preventing malnutrition. Malnutrition essentially means "bad nourishment". It is basically not enough as well as too much food. Clinically, malnutrition is characterized by inadequate or excess intake of protein, energy, and micronutrient such as vitamins, and the frequent infections and disorders that results. Malnutrition in all its forms increases the risk of disease and premature death.

A chronic food deficit affects about 792 million people in the world (FAO 2000; WHO 2000a; Water-related Diseases 2007), including 20% of the population in developing countries. Worldwide, malnutrition affects one in three people (FAO 2000; WHO 2000a; Water-related Diseases 2007). Malnutrition affects all ages, but it is especially common among the poor and those with inadequate access to health education and to clean water and good sanitation. More than 70% of children with protein-energy malnutrition live in Asia, 26% live in Africa, and 4% in Latin America and the Caribbean (FAO 2000; WHO 2000a; Water-related Diseases 2007).

Malnutrition, “the silent emergency,” contributes to at least half of the 10.4 million child deaths every year (WHO 2000a). About 15% of non-elderly adults are too thin because of malnutrition and disease, which decrease their productivity and double their rate of premature mortality. At the same time, there are 1 billion people who are overweight, of whom 350 million are obese. 2 out of 3 overweight and obese people now live in developing countries and by 2010 more obese people will live in developing countries than in the developed world (Nutrition 2008). Over all about 2.6 millions deaths are attributable to overweight/obesity worldwide (Ezzati et al. 2004). Data such as these on low birthweight, stunting, thinness, and overweight are obtained from simple measurement of height and weight. Anthropometric measurements assess body size and composition, and reflect inadequate or excess food intake, insufficient exercise, and disease. They demonstrate that deprivation and excess may coexist not only across, but also within, countries and even households.

### *2.2.2 Body composition, nutritional status and BMI*

In the present dissertation BMI is used as a measure of body composition (BC) that indicates nutritional status. BMI became popular during the early 1980s as obesity started to become a discernible issue in prosperous Western society. BMI provided a simple numerical measure of a person's "fatness" or "thinness", allowing health professionals to discuss over- and under-weight problems more objectively with their patients. However, BMI has become controversial because many people, including physicians, have come to rely on its apparent numerical "authority" for medical diagnosis – but that has never been the purpose of BMI. It is meant to be used as a simple means of classifying sedentary (physically inactive) individuals with an average BC (WHO 1995).

In the assessment of the nutritional status of individuals and communities, anthropometric measurements play a very important role for the following reasons: departure from normal can often be detected earlier by anthropometry than by clinical examination: and anthropometry figures are more objective than clinical assessment.

In tropical countries, anthropometric assessment has most often focused on children under 5 years of age because such children are more often the victims of clinical undernutrition than other age groups. Adult malnutrition has received much less attention than that of children. This focus appears at least in part unjustified, and many public health workers have reported that parents often sacrifice their own feeding in times of serious food shortage (acute or chronic) in favour of young children in the family.

**Table 1.** International classification of adult underweight, overweight and obesity according to BMI

| Classification | BMI(kg/m²) | |
|---|---|---|
| | Principal cut-off points | Additional cut-off points |
| **Underweight** | **<18.50** | **<18.50** |
| Severe thinness | <16.00 | <16.00 |
| Moderate thinness | 16.00 – 16.99 | 16.00 – 16.99 |
| Mild thinness | 17.00 – 18.49 | 17.00 – 18.49 |
| **Normal range** | **18.50 – 24.99** | **18.50 – 22.99**<br>**23.00 – 24.99** |
| **Overweight** | **≥25.00** | **≥25.00** |
| Pre-obese | 25.00 – 29.99 | 25.00 – 27.49<br>27.50 – 29.99 |
| **Obese** | **≥30.00** | **≥30.00** |
| Obese class I | 30.00 – 34-99 | 30.00 – 32.49<br>32.50 – 34.99 |
| Obese class II | 35.00 – 39.99 | 35.00 – 37.49<br>37.50 – 39.99 |
| Obese class III | ≥40.00 | ≥40.00 |

*Source: Adapted from WHO 1995; WHO 2000b.*

BMI has been used by the WHO as the standard for recording obesity statistics since the early 1980s. In the US, BMI is also used as a measure of underweight, owing to advocacy on behalf of those suffering from eating disorders, such as anorexia nervosa and bulimia nervosa.

In 1992, a task force of the International Dietary Energy Consultative Group suggested that BMI be used to define adult chronic dietary energy deficiency (Norgan 1994). BMI can be calculated quickly and without expensive equipment. However, BMI categories do not take into account many factors such as frame size and muscularity. The categories also fail to account for varying proportions of fat, bone, cartilage, water weight, and more. BMI is a statistical categorization and it would be very useful in many ways.

BMI ranks along the index from around 15 (near starvation) to over 40 (morbidly obese). This statistical spread is usually described in broad categories: underweight, normal weight, overweight, obese and morbidly obese. The particular BMI values used to demarcate these categories vary depending on the authority, but typically a BMI of less than 18.5 is considered underweight and may indicate malnutrition, an eating disorder, or other health problems, while a BMI greater than 25 is considered overweight and above 30 is considered obese.

An analysis led by Lopez-Jimenez (Romero-Corral et al. 2006) of 40 studies involving 250,000 people showed that low BMI is associated with significantly higher total mortality, while overweight and obese patients do not have a significantly higher risk unless stratified by obese and severely obese. The implications of this finding can be confounded by the fact that many chronic diseases, such as diabetes, cause weight loss before eventual death. These findings could be explained by the lack of discriminatory power of BMI to differentiate between body fat and lean mass.

Studies conducted mainly in the West have shown that obesity is closely associated with increased risks of many diseases, disability, and reduced life expectancy (National Institutes of Health 1998; World Health Consultation on Obesity 1998). Although the problem is not as widespread as it is in the West, a recent report by WHO and others (The International Diabetes Institute et al. 2000) has shown alarming increases in the disease risk due to obesity in the Asian countries. However, there are very few or no datasets, to enable a longitudinal study of the association between obesity and mortality in Asian populations. It has been hypothesized that there may be differences in the association of overweight and obesity with adverse health outcomes, because the frame sizes of Asians tend to be smaller than those of Europeans (Deurenberg et al. 2002; WHO Expert Consultation 2004). A report co-sponsored by the WHO and others (The International

Diabetes Institute et al. 2000) proposed the new definition for overweight and obesity with a focus on the Asia-Pacific region with BMI [weight (kg)/height$^2$ (m)$^2$] $\geq$ 23. However, evidence used to establish this classification was obtained from a limited numbers of prevalence studies and not from more conclusive incidence or mortality data from cohort studies.

The nutritional status of the Indian population varies significantly across regions (Vijayaraghavan and Rao 1998; Shetty 2002; Nutrition country profiles 2006). While certain regions are associated with extremely high rates of childhood undernutrition (ranging from 20-80%), some demonstrate a high prevalence of adult undernutrition (>50%), and some have both (Vijayaraghavan and Rao 1998; Shetty 2002; Nutrition country profiles 2006). Earlier, developing countries such as India largely focused on the high prevalence of undernutrition (Vijayaraghavan and Rao 1998). However, now there is evidence of a double burden of undernutrition as well as overnutrition (Vijayaraghavan and Rao 1998; Shetty 2002; Nutrition country profiles 2006).

# 3. Objective of the Study

Globally, two major determinants of death are tobacco use and nutritional status. The objective of this study is to assess the effect of tobacco use and BC on all-cause and cause-specific mortality in a developing country population.

The aims of this study were specifically to estimate with a population based follow-up study during 1991–2003, in Mumbai, India:

- The association between different forms of tobacco use and BC (BMI) (IV)
- The effect of tobacco use on all-cause and cause-specific mortality (I, II)
- The effect of BC (BMI) on all-cause and cause-specific mortality (III)
- The joint effect of or interaction between tobacco use and BC (BMI) on all-cause mortality (V)

# 4. Materials and Methods

## 4.1 Material

Mumbai (Bombay) is large, densely populated metropolitan city [density 16,461 inhabitants/kilometre$^2$ (km$^2$)] with a population of 9.93 million (1991 census) in an area of 603 km$^2$. It is divided into three parts: the main city, the suburbs, and the extended suburbs. The survey was confined to the main city, which is the most densely populated area (density 48,830 inhabitants/km$^2$) with a population of 3.42 million, covering an area of about 70 km$^2$. A total of 148,173 individuals aged ≥35 years were recruited in two phases. During phase I (1991–1994), men and women aged ≥35 years were recruited, and during phase II (1994–1997) only men aged ≥45 years were recruited. In order to increase the number of deaths that will occur in the cohort, younger age groups (aged < 35 years) were not considered for the study.

Electoral rolls were organized by area with a polling station of 1,000–1,500 individuals as the smallest geographical unit. The polling stations were the basis of a convenience sampling aiming at representativeness between larger geographical areas. Within a selected polling station, all eligible individuals were approached for participation. The process was continued until the preset number of 150,000 individuals was covered.

The electoral rolls provided name, age, sex, and address of all the individuals aged ≥18 years. The rolls were assumed to be fairly complete as almost everyone is entitled to vote and are generally updated before every major election through house-to-house visits. We excluded polling stations that served upper-middle-class and upper-class housing complexes. Such complexes were not accessible to us because of security issues (i.e., they were essentially ‘‘gated communities’’). The proportion of polling stations excluded varied from area to area. Some areas that were known to be affluent – for example, those containing only skyscraper apartment complexes – were excluded completely, whereas fewer than 10% of polling stations were excluded in others.

For a selected polling station, all eligible people (aged ≥35 years) listed on its electoral roll were approached by trained field supervisors and were interviewed, with the exception of very sick or bedridden individuals. Sometimes individuals not listed on the electoral roll were also interviewed and included in the sample if they insisted that they were permanent residents. Such people formed about 5% of the sample. Their resident status was confirmed through their listing in "ration cards" that are issued by the BMC. Every householder keeps the card because, apart from getting certain food items at subsidised prices, it proves residence, allowing the person access to all city and state government services.

The interviews were conducted by using handheld computers (electronic diaries). The electronic diary had a language compiler installed in its Read Only Memory (ROM). The questionnaire was programmed on a personnel computer and transferred to a storage device in the diary. The computer program provided appropriate sequencing of the questions, skipped irrelevant questions for an individual, accepted only valid codes for answers, and preformed range and consistency checks on the spot. These computers, along with their storage devices, were brought back to the project office once a week, and the data were electronically transferred first to a personal computer and then to a mainframe computer.

The interviews were conducted in local languages (Marathi, Hindi) but the information was recorded in English. Mostly, the responses were recorded as codes because almost all questions were close-ended. The study satisfies all the criteria regarding the ethical treatment of human subjects, especially those formulated by the Indian Council of Medical Research (Indian Council of Medical Research 1988b).

## 4.2 Methods

The baseline survey included the following 2 components: (1) anthropometric measurements by weight and height and (2) interviewer administration of a structured questionnaire to obtained information on age, gender, education, religion, mother tongue (language), and information related to frequency and type of tobacco usage.

Weight was measured using a bathroom scale accurate to 0.5 kg. The scale was kept on a flat surface and the subject was requested to step on it in bare feet without holding on to anything. Subjects were measured in normal apparel, which in Mumbai is light cotton because of the tropical weather year round. The weight was recorded to the nearest kg. Height was measured using a specially constructed instrument consisting of a steel platform to which was attached a steel measure tape. With the subject standing erect on the steel platform, the tape was pulled vertically above the head, and then brought down to touch the flat ruler placed horizontally on the crown of the head. Height was recorded to the nearest cm.

Respondents were interviewed and classified according to present and past tobacco use as (a) having never used tobacco, (b) ex-smoker, (c) ex-smokeless tobacco user, (d) ever smoker, (f) ever smokeless tobacco user, and (g) ever mixed user (smokeless and smoking). The prevalence of past users was small, 2.2% among women (almost all smokeless tobacco users) compared to 57.5% current users and 4.5% among men (2.8% smokers and 1.7% smokeless tobacco users) compared to 69.3% current users (Gupta 1996) and they were combined with current users and referred to as ever-users. The results for smokers and mixed users (smoker as well as smokeless tobacco user) were generally similar, therefore for the sake of simplicity were considered as a single category of smokers. Further these smokers were divided into those who smoked i) cigarettes and ii) *bidis* only or with other smoking type. The smokeless tobacco use was divided into –i) *mishri* alone ii) *mishri* plus other smokeless tobacco products iii) betel quid iv) other tobacco and v) areca nut only. Betel-quid contains almost invariably some tobacco in this study.

BMI is defined as the individual's body weight divided by the square of their height. The formulas universally used in medicine produce a unit of measure of ($kg/m^2$). BMI can also be determined using a BMI chart, which displays BMI as a function of weight (horizontal axis) and height (vertical axis) using “contour lines” for different values of BMI or colours for different BMI categories.

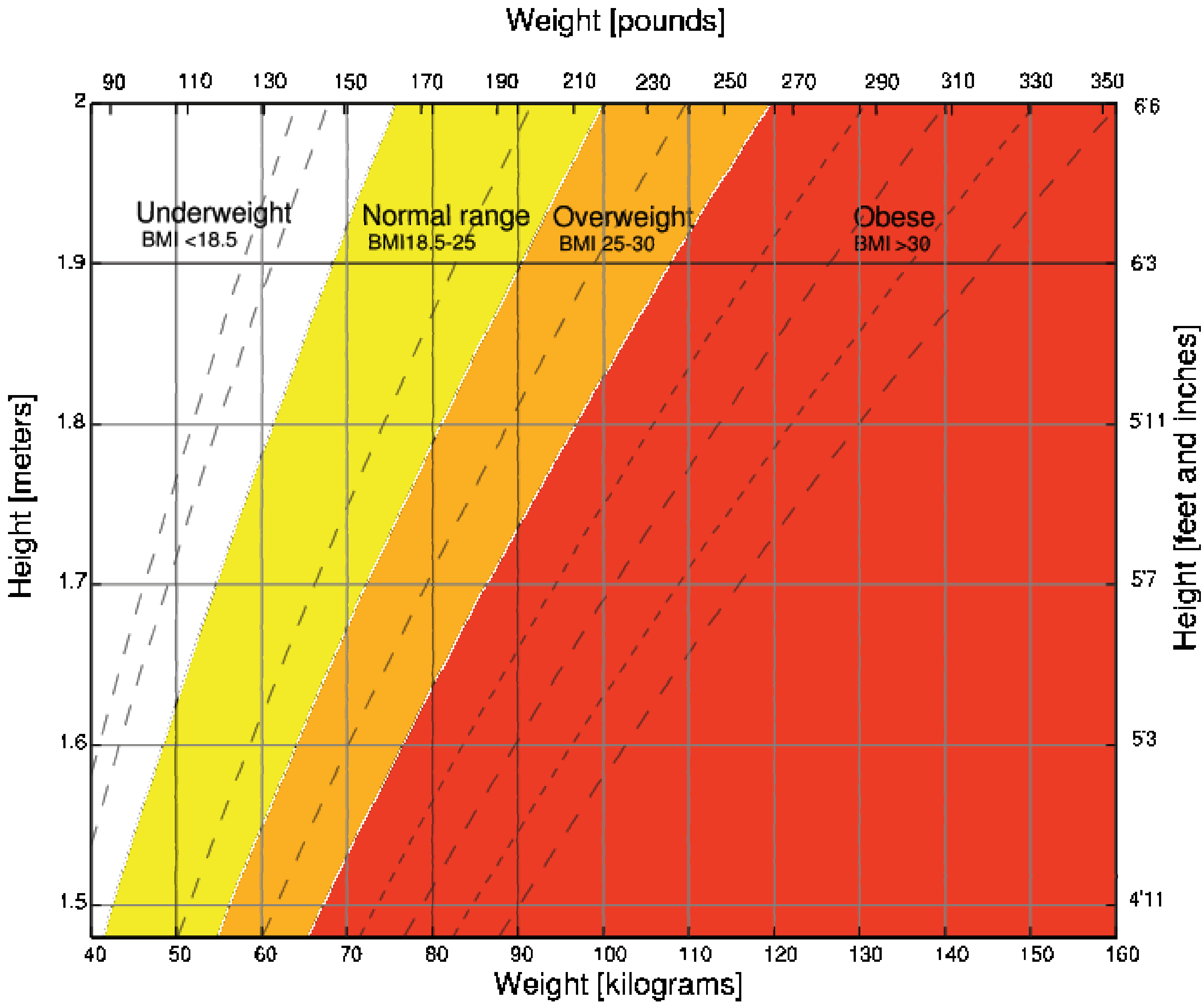


**Figure 3.** Graphical representation of BMI classification

BMI categories were defined by using the following 2 cut-off values: 18.5 kg/m$^2$ and 25.0 kg/m$^2$ for underweight and overweight respectively. The underweight BC was further subdivided into the following 3 groups: extremely thin (BMI <16.0 kg/m$^2$), very thin (BMI 16.0–<17.0 kg/m$^2$) and thin (BMI 17.0–<18.5 kg/m$^2$); similarly, the overweight BC was subdivided into the following 2 groups: overweight (BMI 25.0–<30.0 kg/m$^2$) and obese (BMI ≥30.0 kg/m$^2$).

## 4.3 Follow-up

An active house-to-house follow-up was conducted on average 5.5 years after the initial survey. The field investigators were provided with the list of names and addresses of cohort members and were instructed to revisit each person. If the person was alive and available, a face-to-face re-interview was conducted. If the person was reported to have died, the date and place of death were recorded with maximal accuracy. Permanent migration from the study area was considered as withdrawal from the study, and the date of migration was noted. The re-interviews for individuals recruited in phase I were conducted during the period 1997–1999 and for phase II during the period 2000–2003.

## 4.4 Cause of death

Under the uniform system of RBD, reporting and RBD is compulsory in India. Although the quality of registration varies across different parts of the country, in Mumbai, almost all deaths are registered and certified medically. The cause of death was sought from BMC death registers. In Mumbai, death is reported on a standard death certificate in accordance with the WHO guidelines. The medical section of the death certificate has four lines. The first line is for the major or underlying cause initiating the sequence of events leading to death. The second line is for immediate cause of death (state the disease, injury or complication which caused death, not the mode of dying such as heart failure, asthenia, etc). The third line is for antecedent cause (morbid condition if any, giving rise to the above cause, stating underlying condition last). The fourth line is for any other conditions that contributed to death, but are not related to the causes stated in the first three lines. A properly completed certificates shows only one condition for each line and, in accordance with WHO guidelines, the listed conditions form a causal sequence initiated by the underlying cause. The certificate is generally filled in by the family doctor or by the attending physician (when death occurs in hospital). If the circumstances of death are suspicious or violent, the certificate is submitted for completion to the medical examiner and the results are notified with a substantial delay.

Considering the diagnosis of ill-defined causes as a marker for the quality of causes of death certification, in the MCS the proportion of ill-defined causes among matched deaths was <5% (ICD codes R50–69, R95–99).

For all deaths (occurred within MCS area) aged ≥27 years (additional deaths of a persons aged 27 to 34 were collected to avoid occasional errors in the recorded age at death), additional details including demographic information of the decedent and the underlying cause(s) of death were recorded from BMC death registers by project personnel. This work amounted to record approximately over 20,000 deaths per year from BMC death registers. Then the deaths recorded during the follow-up of the cohort were linked with the dataset obtained from BMC death registers. Linking was performed using age, sex, name, address, and date of expiry. The most important variables for linking were found to be name and address of the deceased. Linking was performed manually in order to obviate any errors due to the differences in the spelling of names between 2 datasets. For matched deaths, the underlying cause of death was derived from the cause information copied from the corporation death registers and was coded according to the ICD-10 guidelines. Also for 1685 randomly selected matched deaths, an independent check was performed by sending trained field investigator to the household of the deceased. In all the 1685 revisited deaths, matching was found to be 100% accurate.

## 4.5 Statistical analyses

### *4.5.1 Cohort analyses*

Univariate analysis was performed for the total survey population separately for men and women. Person-years were calculated by using the date of recruitment and the date of endpoint ascertainment (defined as the date of expiry, re-interview, migration, or censor). In cases where the exact date of death (2.6% deaths) or migration (1.4% migrations) was not available, a midpoint between date of recruitment and date of ascertainment (date unknown) or $1^{st}$ July (year known) was used. Age-adjusted death rates for men and women were calculated by using overall five-year age-specific person-years as weights (i.e., the direct method). Multivariate analysis was performed by using Cox proportional hazard regression modelling (Cox 1972) and adjusted RRs with 95% CIs were estimated.

The response variable, death, was a dichotomous variable (yes or no) and time to event (or censor) was continuous.

To study the joint effect of tobacco use and BMI on mortality, expected RRs were calculated. Expected multiplicative RR was calculated by multiplying marginal RRs for different categories of tobacco users with different categories of BMIs. Expected additive RR was calculated as sum of the marginal RRs minus one. Observed RR less than the expected RR indicated antagonistic effect and higher than the expected RR indicated synergistic effect. A population attributable fraction (PAF) was calculated using a formula (Rockhill et al. 1998) $\boldsymbol{\sum pd_i[(RR_i-1)/RR_i]}$, where '$pd_i$' represents the proportion of the total deaths in the population arising from the $i^{th}$ exposure category and $RR_i$ is the (adjusted) RR for the $i^{th}$ exposure category (relative to the reference or unexposed stratum).

### *4.5.2 Cross-sectional analyses*

Descriptive statistics (mean, standard deviation, and proportion within categories) were calculated for the total survey population and by tobacco use for both men and women separately. Because the number of ex-smokers among women was small, no separate analyses were conducted.

The response variable, BMI, was converted into a dichotomous variable by using two cutpoints: 18.5 kg/$m^2$ and 25.0 kg/$m^2$. Multivariate analysis was performed using logistic regression modelling and adjusted odds ratios (OR) with 95% CIs were estimated.

# 5. Results

## 5.1 Baseline and follow-up (III, IV, V)

Of the total 148,173 cohort members, 60% were men. There were 39% of men and 59% of women smokeless tobacco users; while, the respective proportions of smokers were 31% and <1%.

**Table 2.** Percentage distribution of the population of baseline survey by age (in years), tobacco habits, BC and gender: MCS, Mumbai, India, 1991–1997

| Variable | Men (n=88,658) | Women (n=59,515) | Total (n=148,173) |
|---|---|---|---|
| **Age** | | | |
| 35–39 | 9.4 | 24.8 | 15.6 |
| 40–44 | 7.9 | 16.9 | 11.5 |
| 45–49 | 26.7 | 15.4 | 22.2 |
| 50–54 | 16.1 | 13.3 | 15.0 |
| 55–59 | 12.2 | 9.9 | 11.2 |
| 60–64 | 11.1 | 9.2 | 10.3 |
| 65–69 | 7.6 | 5.1 | 6.6 |
| 70 and up | 8.9 | 5.5 | 7.5 |
| **Tobacco users** | | | |
| Never | 30.1 | 40.3 | 34.2 |
| Smokeless | 38.5 | 59.3 | 46.8 |
| Smoker | 31.4 | 0.4 | 19.0 |
| **BC** | | | |
| Extremely thin | 4.3 | 5.7 | 4.9 |
| Very thin | 3.9 | 4.3 | 4.1 |
| Thin | 9.2 | 9.1 | 9.1 |
| Normal | 62.4 | 51.4 | 58.0 |
| Overweight | 17.5 | 22.5 | 19.5 |
| Obese | 2.7 | 6.9 | 4.4 |

In MCS, there were 62% of men and 51% of women had normal BMI. The prevalence of overweight (pre-obesity and obesity) was higher than the prevalence of thinness among both men (20.2% vs. 17.4%) as well as women (29.4% vs. 19.1%) (Table 2). The prevalence of severe thinness (BMI<16 kg/m$^2$) and obesity (BMI≥30 kg/m$^2$) was higher among women (5.7% and 6.9% respectively) than men (4.3% and 2.7% respectively).

Figure 4 presents the response rates for house-to-house follow-up. Of the total 148,173 cohort members, 7,265 could not be traced; the most common reason was the demolition of their residential building (6,452 persons) for redevelopment. Among the remaining 140,908 persons, 13,261 deaths were reported.

Of the 127,647 persons who were found to be alive, 25,777 subjects had migrated outside the study area, for 90,282 individuals a face-to-face re-interview was conducted, and the remaining 11,588 individuals were not available for re-interview even after multiple visits.

## 5.2 Tobacco (I, II)

Table 3 shows the number of person-years, deaths and the death rates by gender and tobacco use. A total of 533,445 person-years were observed. Of the total person-years contributed by men, 27.4% were by smokers, 46.1% by smokeless tobacco users, and 26.5% by never tobacco users. Similarly, these proportions were 0.4%, 59.3%, and 40.3% respectively for women.

Users of all forms of tobacco had higher death rates than corresponding never tobacco users. For men, the adjusted death rate (per 1,000 person-years) among smokers (23.3) was higher than those among never-users of tobacco (13.6), the adjusted RR was 1.55 (95% CI: 1.42–1.69). Although very few women smoked (RR: 1.40, 95% CI: 0.99–1.97), the RR appeared to be similar to that of men. The death rates among men with two major types of smoking habits prevalent in Mumbai, cigarette and *bidi*, were higher than the corresponding rate in never tobacco users. The adjusted RR was 1.37 (95% CI: 1.23–1.53) for cigarette smokers and 1.64 (95% CI: 1.47–1.81) for *bidi* smokers. A statistically significant dose-response relationship was observed for *bidi* (*p* value for trend <0.00001) as well as for cigarette (*p* value for trend <0.00001) smokers.

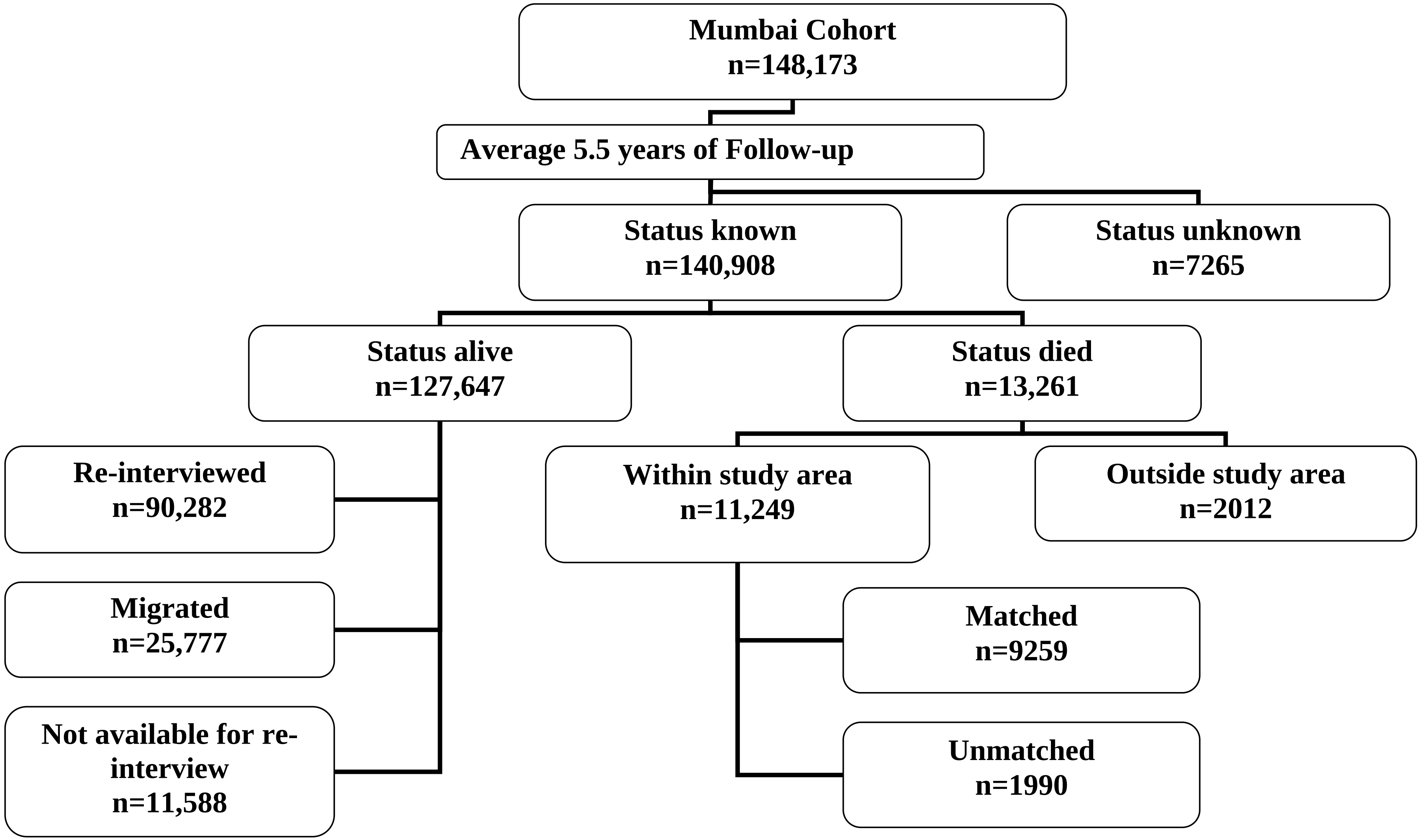


**Figure 4.** Response rates in house-to-house follow-up of the study subjects: MCS, Mumbai, India, 1991–2003

**Table 3.** Person-years of follow-up, number of deaths, age adjusted death rates (per 1,000 person years), relative risks (RR) and 95% confidence intervals (CI) for all-cause mortality in different types of tobacco using men and women: MCS, Mumbai, India, 1991–1999

| Gender and Tobacco habits | Person years | Deaths | Death rates | CI | RR* | CI |
|---|---|---|---|---|---|---|
| **Men** | | | | | | |
| Total | 210,129 | 4,119 | 17.9 | 17.3–18.5 | | |
| **Never tobacco use** | 55,717 | 854 | 13.6 | 12.6–14.6 | Reference | |
| **Smokeless tobacco** | 96,884 | 1,787 | 17.3 | 16.5–18.1 | 1.16 | 1.06–1.26 |
| *Mishri* alone | 14,658 | 226 | 16.1 | 14.1–18.1 | 1.14 | 0.97–1.33 |
| *Mishri* plus | 38,981 | 782 | 18.3 | 17.0–19.6 | 1.18 | 1.07–1.31 |
| Betel-quid | 24,368 | 436 | 16.2 | 14.6–17.8 | 1.10 | 0.98–1.24 |
| Other tobacco | 17,039 | 316 | 18.5 | 16.5–20.5 | 1.24 | 1.08–1.41 |
| Areca nut | 1,838 | 27 | 11.5 | 6.6–16.4 | 0.83 | 0.56–1.21 |
| **Smoking** | 57,528 | 1,478 | 23.3 | 22.1–24.5 | 1.55 | 1.42–1.69 |
| Number of *Bidis* (per day)† | | | | | | |
| 1–5 | 6,658 | 177 | 23.1 | 19.5–26.7 | 1.42 | 1.20–1.68 |
| 6–10 | 5,594 | 182 | 25.8 | 21.6–30.0 | 1.59 | 1.35–1.87 |
| 11–15 | 5,838 | 184 | 26.8 | 22.7–30.9 | 1.62 | 1.37–1.91 |
| ≥16 | 12,865 | 427 | 29.1 | 26.2–32.0 | 1.78 | 1.57–2.02 |
| ***Any*** | 31,376 | 982 | 26.5 | 24.7–28.3 | 1.64 | 1.47–1.81 |
| Number of cigarettes (per day)† | | | | | | |
| 1–5 | 11,709 | 177 | 17.5 | 15.1–19.9 | 1.20 | 1.02–1.42 |
| ≥ 6 | 14,444 | 319 | 20.5 | 18.2–22.8 | 1.50 | 1.32–1.71 |
| ***Any*** | 26,152 | 496 | 18.9 | 17.2–20.6 | 1.37 | 1.23–1.53 |
| **Women** | | | | | | |
| Total | 323,316 | 3,412 | 11.4 | 11.0–11.8 | | |
| **Never tobacco use** | 130,294 | 907 | 8.9 | 8.4–9.4 | Reference | |
| **Smokeless tobacco** | 191,625 | 2,470 | 12.7 | 12.2–13.2 | 1.25 | 1.15–1.35 |
| *Mishri* alone | 88,002 | 743 | 12.3 | 11.6–13.0 | 1.21 | 1.10–1.34 |
| *Mishri* plus | 71,817 | 1,323 | 14.1 | 13.2–15.0 | 1.36 | 1.24–1.48 |
| Betel-quid | 20,153 | 236 | 9.8 | 8.4–11.2 | 0.96 | 0.83–1.11 |
| Other tobacco | 5020 | 80 | 14.1 | 10.8–17.4 | 1.37 | 1.09–1.73 |
| Areca nut | 6,633 | 88 | 9.9 | 7.5–12.3 | 1.05 | 0.84–1.31 |
| **Smoking** | 1,398 | 35 | 17.1 | 10.3–23.9 | 1.40 | 0.99–1.97 |

* Adjusted for age and education.
† Chi-square for trend is significant, $p<0.00001$.

Smokeless tobacco use increased the risk of dying among both men (RR: 1.16, 95% CI: 1.06–1.26) and women (RR: 1.25, 95% CI: 1.15–1.35). Among women, the RRs were higher for those who used *mishri* alone, *mishri* plus other smokeless tobacco products, and chewing tobacco or tobacco containing product; these categories showed adjusted RRs of 1.21 (95% CI: 1.10–1.34), 1.36 (95% CI: 1.24–1.48) and 1.37 (95% CI: 1.09–1.73) respectively. Among men, the RRs were statistically significantly higher for the users of *mishri* plus other smokeless tobacco products (RR: 1.18, 95% CI: 1.07–1.31) and chewers of tobacco or product containing tobacco that mostly comprised tobacco and lime (RR: 1.24, 95% CI: 1.08–1.41). Interestingly, betel quid or areca nut (without the use of tobacco) did not show a statistically significant RR neither among men nor women.

Table 4 shows the RRs for major causes of death by gender. Smoking among men increased the risk of dying from respiratory diseases (RR: 2.12, 95% CI: 1.57–2.87), TB (RR: 2.30, 95% CI: 1.68–3.15), circulatory diseases (RR: 1.21, 95% CI: 1.04–1.40), and for all neoplasms (RR: 2.60, 95% CI: 1.78–3.80). Among respiratory diseases, smoking increased the risk of dying from pneumonia (RR: 2.46, 95 CI: 1.40–4.30) and COPD (RR: 2.13, 95% CI: 1.45–3.14); similarly among neoplasms, for oropharyngeal (RR: 19.69, 95% CI: 2.65–146.17) and respiratory (RR: 4.05, 95% CI: 1.51–10.85) neoplasms. Deaths from digestive diseases and all other coded causes (primarily senility) did not show any statistically significant association with smoking. Among circulatory diseases, cerebrovascular deaths were associated with smoking (RR: 1.54, 95% CI: 1.09–2.19), whereas deaths from ischemic heart diseases (IHD) failed to reach statistical significance (RR: 1.17, 95% CI: 0.99–1.39). In addition, smoking also increased the risk of dying from accidents (RR: 2.65, 95% CI: 1.43–4.90); however, the effect of confounding from alcohol was not taken into consideration. Female smokers were very few and the results are reported only into original paper (I).

**Table 4.** Number of deaths, relative risks (RR) and 95% confidence intervals (CI) for all-cause and cause-specific mortality in different types of tobacco using men and women: MCS, Mumbai, India, 1991–1999

| Gender and Causes of death | Never tobacco use | Smokeless | Smoker |
|---|---|---|---|
| **Men** | | | |
| **All** | | | |
| Deaths | 854 | 1,787 | 1,478 |
| RR* (95% CI) | Reference | 1.16 (1.06–1.26) | 1.55 (1.42–1.69) |
| **TB** | | | |
| Deaths | 58 | 160 | 152 |
| RR (95% CI) | Reference | 1.46 (1.07–2.00) | 2.30 (1.68–3.15) |
| **Neoplasms** | | | |
| Deaths | 40 | 91 | 103 |
| RR (95% CI) | Reference | 1.40 (0.95–2.06) | 2.60 (1.78–3.80) |
| **Circulatory Diseases** | | | |
| Deaths | 323 | 535 | 430 |
| RR (95% CI) | Reference | 0.94 (0.82–1.09) | 1.21 (1.04–1.40) |
| **Respiratory Diseases** | | | |
| Deaths | 63 | 169 | 148 |
| RR (95% CI) | Reference | 1.50 (1.12–2.03) | 2.12 (1.57–2.87) |
| **Digestive Diseases** | | | |
| Deaths | 35 | 61 | 53 |
| RR (95% CI) | Reference | 0.93 (0.61–1.42) | 1.35 (0.87–2.08) |
| **Accidents** | | | |
| Deaths | 14 | 45 | 42 |
| RR (95% CI) | Reference | 1.71 (0.93–3.14) | 2.65 (1.43–4.90) |
| **All other coded deaths** | | | |
| Deaths | 110 | 233 | 154 |
| RR (95% CI) | Reference | 1.18 (0.93–1.49) | 1.26 (0.98–1.61) |
| **Women** | | | |
| **All** | | | |
| Deaths | 907 | 2,470 | 35 |
| RR* (95% CI) | Reference | 1.25 (1.15–1.35) | 1.40 (0.99–1.97) |
| **TB** | | | |
| Deaths | 46 | 123 | 5 |
| RR (95% CI) | Reference | 1.40 (0.99–2.00) | 5.92 (2.31–15.17) |
| **Neoplasms** | | | |
| Deaths | 65 | 177 | 2 |
| RR (95% CI) | Reference | 1.57 (1.16–2.13) | 1.85 (0.45–7.60) |
| **Circulatory Diseases** | | | |
| Deaths | 283 | 735 | 7 |
| RR (95% CI) | Reference | 1.19 (1.02–1.38) | 0.84 (0.37–1.88) |
| **Respiratory Diseases** | | | |
| Deaths | 107 | 292 | 4 |
| RR (95% CI) | Reference | 1.04 (0.82–1.31) | 1.15 (0.42–3.15) |
| **Digestive Diseases** | | | |
| Deaths | 10 | 18 | |
| RR (95% CI) | Reference | 0.95 (0.42–2.14) | |
| **Accidents** | | | |
| Deaths | 20 | 31 | |
| RR (95% CI) | Reference | 0.76 (0.42–1.38) | |
| **All other coded deaths** | | | |
| Deaths | 140 | 372 | 6 |
| RR (95% CI) | Reference | 1.26 (1.02–1.55) | 1.57 (0.69–3.58) |

* Adjusted for age and education.

The RRs for smokeless tobacco users, although higher than unity, did not reach statistical significance for any specific category of diseases except for all neoplasms (RR: 1.57, 95% CI: 1.16–2.13), circulatory diseases (RR: 1.19, 95% CI: 1.02–1.38) [within circulatory diseases; IHD (RR: 1.25, 95% CI: 1.05–1.49)] among women and deaths from respiratory diseases (RR: 1.50, 95% CI: 1.12–2.03) and TB (RR: 1.46, 95% CI: 1.07–2.00) among men.

## 5.3 Body mass index (III)

Table 5 shows the number of person-years, deaths and the death rates by gender and BC. A total of 774,129 person-years were observed, of which 57% were contributed by men. Highest death rates were observed among extremely thin men (48.42 per 1,000 person-years) and women (24.32 per 1,000 person-years), while the lowest were observed among overweight men (15.06 per 1,000 person-years) and women (9.51 per 1,000 person-years).

Death rates were higher among men than women across different categories of BMI. Men and women with BMI below normal had higher death rates than those with normal BMI; by contrast, men and women with BMI above normal had lower death rates. The adjusted RR of dying increases with increasing severity of thinness; this was observed among both men (RRs: for thin=1.27, very thin=1.45, and extremely thin=2.24) and women (RRs: 1.24, 1.38, and 1.94 respectively). By contrast, overweight men (RR=0.87) and women (RR=0.89) were at a lower risk of death compared with corresponding normal weight men and women.

**Table 5.** Person-years of follow-up, number of deaths, age adjusted death rates (per 1,000 person years), relative risks (RR) and 95% confidence intervals (CI) according to BC in men and women: MCS, Mumbai, India, 1991–2003

| Gender and BC | Person-years | Number of deaths | Death rate | CI | RR* | CI |
|---|---|---|---|---|---|---|
| **Men** | | | | | | |
| Total | 443,892 | 9,589 | 19.43 | 19.02–19.84 | | |
| **Below normal** | 75,559 | 2,804 | 30.66 | 29.43–31.89 | 1.57 | 1.50–1.65 |
| Extremely thin | 17,764 | 1,079 | 48.42 | 45.26–51.58 | 2.24 | 2.09–2.39 |
| Very thin | 17,449 | 609 | 28.62 | 26.15–31.09 | 1.45 | 1.33–1.58 |
| Thin | 40,345 | 1,116 | 23.63 | 22.15–25.11 | 1.27 | 1.19–1.36 |
| **Normal** | 277,519 | 5,340 | 17.49 | 17.00–17.98 | Reference | |
| **Above normal** | 90,814 | 1,445 | 15.38 | 14.58–16.18 | 0.90 | 0.85–0.95 |
| Overweight | 78,939 | 1,226 | 15.06 | 14.21–15.91 | 0.87 | 0.82–0.93 |
| Obese | 11,875 | 219 | 17.41 | 15.06–19.76 | 1.05 | 0.92–1.21 |
| **Women** | | | | | | |
| Total | 330,237 | 3,412 | 12.61 | 12.23–12.99 | | |
| **Below normal** | 62,332 | 1,092 | 18.45 | 17.39–19.51 | 1.52 | 1.41–1.64 |
| Extremely thin | 18,164 | 502 | 24.32 | 22.08–26.56 | 1.94 | 1.75–2.15 |
| Very thin | 14,124 | 204 | 16.12 | 14.04–18.20 | 1.38 | 1.19–1.60 |
| Thin | 30,044 | 386 | 15.25 | 13.86–16.64 | 1.24 | 1.11–1.39 |
| **Normal** | 170,002 | 1,596 | 11.61 | 11.10–12.12 | Reference | |
| **Above normal** | 97,903 | 724 | 9.61 | 9.00–10.22 | 0.91 | 0.83–0.99 |
| Overweight | 75,068 | 552 | 9.51 | 8.82–10.20 | 0.89 | 0.81–0.98 |
| Obese | 22,835 | 172 | 10.04 | 8.75–11.33 | 0.97 | 0.83–1.14 |

* Adjusted for age, education, religion, mother tongue, and tobacco habits.

Table 6 presents the all-cause and cause-specific deaths, and adjusted RRs compared to a normal BMI (BMI 18.5–<25 kg/m$^2$). Increased risk of dying due to TB, cancers (men only), non-medical causes (men only), and respiratory system diseases was observed across different underweight categories, and the risk remained high despite excluding deaths occurred during first 2 years of follow-up. Among all different causes, the highest risk was observed for deaths due to TB among extremely thin men (RR: 14.94, 95% CI: 12.43–17.97) and women (RR: 7.20, 95% CI: 4.96–10.44) when compared with corresponding normal weight men and women. Non-medical causes, primarily includes accidental deaths (≈ 60%) and deaths due to injuries (≈ 30%); were also found to be associated with increased risk of dying across various categories of underweight men.

Overweight men and women were at ≈60% and ≈30% decreased risk of dying due to TB and respiratory diseases respectively; while obese women were at ≈30% increased risk of dying due to circulatory diseases. The numbers of obese women were few, however.

## 5.4 Tobacco use and BMI (IV)

All forms of tobacco use were found to be associated with the prevalence of low BMI, and the prevalence of low BMI was highest (32%) among *bidi* smokers.

Table 7 explores the dose-response relationship with low BMI divided into three categories: thin, very thin and extremely thin with frequency of smoking among men and smokeless tobacco use among women. There exists a clear gradient in ORs for almost every row and every column. The lowest OR was observed among thin men who smoke 1–5 times a day (OR: 1.21, 95% CI: 1.06–1.39) and the highest OR was among extremely thin men who smoke ≥11 times a day (OR: 2.54, 95% CI: 2.17–2.98). Similarly, the lowest OR was observed among thin women who use smokeless tobacco 1–2 times a day (OR: 1.32, 95% CI: 1.22–1.43) and the highest OR was among extremely thin women who use smokeless tobacco ≥11 times a day (OR: 2.19; 95% CI: 1.70–2.84).

**Table 6.** Number of deaths, relative risks (RR) with 95% confidence intervals (CI) for all-cause and cause-specific mortality according to BC in men and women: MCS, Mumbai, India, 1991–2003

| Gender and Causes of death | BC | | | | | |
|---|---|---|---|---|---|---|
| | Extremely thin | Very thin | Thin | Normal | Overweight | Obese |
| **Men** | | | | | | |
| **All** | | | | | | |
| Deaths | 1079 | 609 | 1116 | 5340 | 1226 | 219 |
| RR* (CI) | 2.24(2.09–2.39) | 1.45(1.33–1.58) | 1.27(1.19–1.36) | Reference | 0.87(0.82–0.93) | 1.05(0.92–1.21) |
| **TB** | | | | | | |
| Deaths | 245 | 87 | 121 | 226 | 26 | 5 |
| RR (CI) | 14.94(12.43–17.97) | 5.45(4.25–6.99) | 3.41(2.73–4.26) | Reference | 0.42(0.28–0.63) | 0.57(0.23–1.37) |
| **Neoplasms** | | | | | | |
| Deaths | 61 | 39 | 68 | 302 | 70 | 12 |
| RR (CI) | 2.44(1.85–3.23) | 1.69(1.21–2.36) | 1.41(1.09–1.84) | Reference | 0.84(0.64–1.09) | 0.95(0.53–1.70) |
| **Circulatory Diseases** | | | | | | |
| Deaths | 197 | 132 | 272 | 1813 | 482 | 87 |
| RR (CI) | 1.31(1.13–1.51) | 0.95(0.79–1.13) | 0.94(0.82–1.06) | Reference | 1.00(0.91–1.11) | 1.22(0.98–1.52) |
| **Respiratory Diseases** | | | | | | |
| Deaths | 171 | 76 | 129 | 412 | 66 | 6 |
| RR (CI) | 4.35(3.62–5.21) | 2.17(1.69–2.77) | 1.80 (1.48–2.20) | Reference | 0.63(0.48–0.81) | 0.39(0.18–0.88) |
| **Digestive Diseases** | | | | | | |
| Deaths | 29 | 20 | 43 | 177 | 43 | 9 |
| RR (CI) | 2.41(1.62–3.57) | 1.66(1.05–2.65) | 1.62(1.16–2.27) | Reference | 0.87(0.62–1.21) | 1.24(0.64–2.44) |
| **Others medical causes** | | | | | | |
| Deaths | 97 | 74 | 141 | 585 | 124 | 24 |
| RR (CI) | 1.92(1.54–2.38) | 1.61(1.26–2.06) | 1.46(1.22–1.76) | Reference | 0.82(0.68–0.99) | 1.12(0.75–1.69) |
| **Non-medical causes** | | | | | | |
| Deaths | 31 | 17 | 40 | 130 | 29 | 7 |
| RR (CI) | 3.14(2.11–4.67) | 1.75(1.06–2.92) | 1.89(1.32–2.69) | Reference | 0.85(0.56–1.27) | 1.44(0.67–3.08) |

* Adjusted for age, education, religion, mother tongue, and tobacco habits.

**Table 6. (continue.)**

| Gender and Causes of death | BC | | | | | |
|---|---|---|---|---|---|---|
| | Extremely thin | Very thin | Thin | Normal | Overweight | Obese |
| **Women** | | | | | | |
| **All** | | | | | | |
| Deaths | 502 | 204 | 386 | 1596 | 552 | 172 |
| RR* (CI) | 1.94(1.75–2.15) | 1.38(1.19–1.60) | 1.24(1.11–1.39) | Reference | 0.89(0.81–0.98) | 0.97(0.83–1.14) |
| **TB** | | | | | | |
| Deaths | 53 | 21 | 23 | 63 | 11 | 3 |
| RR (CI) | 7.20(4.96–10.44) | 4.09(2.49–6.72) | 2.04(1.26–3.29) | Reference | 0.41(0.21–0.77) | 0.36(0.11–1.16) |
| **Neoplasms** | | | | | | |
| Deaths | 29 | 12 | 26 | 123 | 40 | 14 |
| RR (CI) | 1.87(1.24–2.82) | 1.18(0.65–2.13) | 1.21(0.79–1.85) | Reference | 0.75(0.52–1.07) | 0.87(0.50–1.51) |
| **Circulatory Diseases** | | | | | | |
| Deaths | 89 | 53 | 102 | 498 | 211 | 72 |
| RR (CI) | 1.19(0.95–1.50) | 1.19(0.89–1.58) | 1.09(0.88–1.35) | Reference | 1.08(0.92–1.28) | 1.32(1.03–1.69) |
| **Respiratory Diseases** | | | | | | |
| Deaths | 97 | 27 | 55 | 168 | 49 | 7 |
| RR (CI) | 3.46(2.68–4.46) | 1.71(1.14–2.57) | 1.65(1.21–2.24) | Reference | 0.77(0.56–1.06) | 0.39(0.18–0.84) |
| **Others medical causes** | | | | | | |
| Deaths | 77 | 31 | 57 | 219 | 78 | 25 |
| RR (CI) | 2.29(1.76–2.98) | 1.52(1.04–2.22) | 1.33(0.99–1.78) | Reference | 0.92(0.71–1.19) | 1.07(0.70–1.62) |
| **Non-medical causes** | | | | | | |
| Deaths | 7 | 4 | 14 | 41 | 12 | 4 |
| RR (CI) | 1.17(0.52–2.64) | 1.15(0.41–3.22) | 1.86(1.01–3.43) | Reference | 0.71(0.37–1.36) | 0.80(0.29–2.26) |

* Adjusted for age, education, religion, mother tongue, and tobacco habits.

**Table 7.** Odds ratios (OR) and 95% confidence intervals (CI) of three categories of low BC by frequency of tobacco use and gender using corresponding normal weight never tobacco user as a reference category: MCS, Mumbai, India, 1991–1994

| Gender and Frequency of Tobacco use per day | Low BC | | | | | |
|---|---|---|---|---|---|---|
| | Thin | | Very thin | | Extremely thin | |
| | OR* | CI | OR* | CI | OR* | CI |
| **Men** | | | | | | |
| Smoking | | | | | | |
| 1–5 | 1.21 | 1.06–1.39 | 1.37 | 1.13–1.68 | 1.45 | 1.19–1.77 |
| 6 – 10 | 1.68 | 1.46–1.93 | 1.93 | 1.58–2.35 | 1.97 | 1.62–2.39 |
| ≥ 11 | 1.86 | 1.66–2.09 | 2.22 | 1.88–2.62 | 2.54 | 2.17–2.98 |
| **Women** | | | | | | |
| Smokeless tobacco use | | | | | | |
| 1–2 | 1.32 | 1.22–1.43 | 1.50 | 1.34–1.68 | 1.72 | 1.55–1.90 |
| 3–5 | 1.46 | 1.35–1.57 | 1.71 | 1.53–1.91 | 1.76 | 1.59–1.95 |
| 6–10 | 1.59 | 1.42–1.78 | 1.63 | 1.38–1.92 | 2.05 | 1.78–2.35 |
| ≥ 11 | 1.84 | 1.48–2.29 | 2.46 | 1.87–3.24 | 2.19 | 1.70–2.84 |

* Adjusted for age and education.

## 5.5 Joint effect of tobacco use and BMI on mortality (III, V)

Figure 5 suggests a pattern of increased risk of dying at both extremes (very high and very low) of BMI when adjustment was made for possible confounders, including tobacco use. A similar (increased risk of dying at both extremes) relationship was observed even after restricting the analyses only to never users of tobacco.

Table 8 presents observed and expected joint effect of tobacco use and BC on mortality. The observed joint effect of smoking and thinness in terms of RR was higher than the expected, indicating synergistic interaction. The observed joint effect of smokeless tobacco use and thinness in terms of RR was higher (lower) than the expected, indicating synergistic (antagonistic) interaction in men (women).

The observed joint effect of tobacco use and obesity in terms of RR was lower than the expected, indicating antagonistic interaction in men. The observed RR was identical with expected in women. However, there were only few deaths among obese persons.

In general, the results were independent of whether additive or multiplicative interaction was assumed.

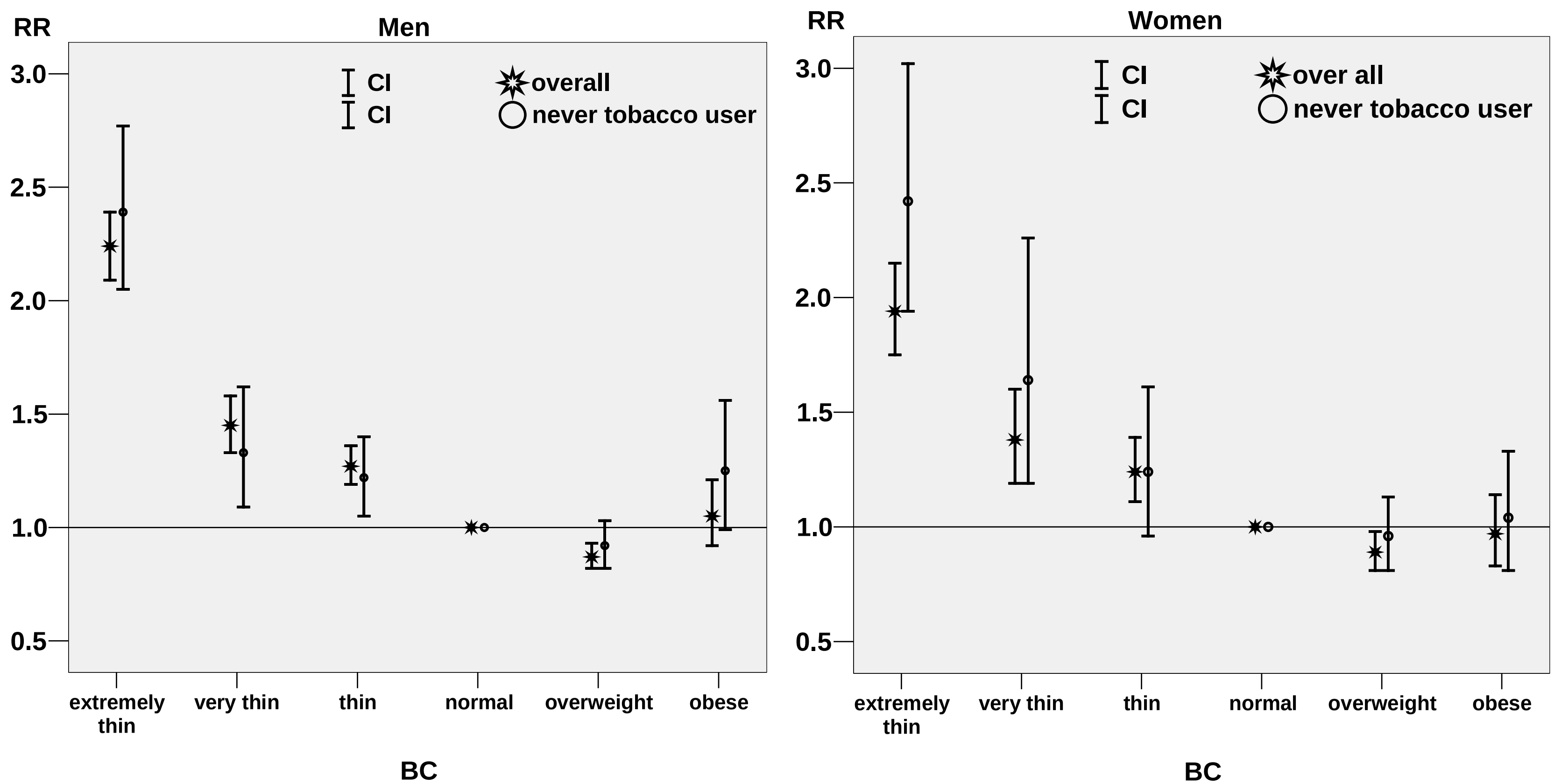


**Figure 5:** Relative risks (RR) with 95% confidence intervals (CI) for different BC, adjusted for age, education, religion, mother tongue, and tobacco habits in total material and in never tobacco users (adjusted for age, education, religion, mother tongue): MCS, Mumbai, India, 1991–2003

**Table 8:** Observed and expected relative risks (RR) with 95% confidence intervals (CI) of total mortality by gender, tobacco use and BC assuming multiplicative and additive joint effect: MCS, Mumbai, India, 1991–2003

| Gender and Tobacco habits | BC | | | | | |
|---|---|---|---|---|---|---|
| | Extremely thin | Very thin | Thin | Normal | Overweight | Obese |
| **Men** | | | | | | |
| **Never user** | | | | | | |
| Observed RR (CI) | 2.68 (2.26, 3.19) | 1.50 (1.21, 1.86) | 1.37 (1.16, 1.62) | 1.10 (0.98, 1.23) | Reference | 1.34 (1.05, 1.71) |
| **Smokeless tobacco user** | | | | | | |
| Observed RR (CI) | 2.75 (2.38, 3.19) | 1.71 (1.44, 2.03) | 1.53 (1.33, 1.77) | 1.18 (1.06, 1.32) | 1.02 (0.86, 1.17) | 1.17 (0.90, 1.51) |
| Expected RR | | | | | | |
| Multiplicative | 2.73 | 1.53 | 1.40 | 1.12 | 1.02 | 1.37 |
| Additive | 2.70 | 1.52 | 1.39 | 1.12 | 1.02 | 1.36 |
| **Smoker** | | | | | | |
| Observed RR (CI) | 3.36 (2.93, 3.85) | 2.38 (2.04, 2.78) | 2.02 (1.76, 2.31) | 1.61 (1.44, 1.80) | 1.38 (1.20, 1.59) | 1.56 (1.21, 2.01) |
| Expected RR | | | | | | |
| Multiplicative | 3.70 | 2.07 | 1.89 | 1.52 | 1.38 | 1.85 |
| Additive | 3.06 | 1.88 | 1.75 | 1.48 | 1.38 | 1.72 |
| **Women** | | | | | | |
| **Never user** | | | | | | |
| Observed RR (CI) | 2.70 (2.12, 3.45) | 1.80 (1.29, 2.53) | 1.37 (1.04, 1.80) | 1.07 (0.90, 1.26) | Reference | 1.09 (0.84, 1.42) |
| **Smokeless tobacco user** | | | | | | |
| Observed RR (CI) | 2.22 (1.86, 2.64) | 1.55 (1.25, 1.91) | 1.48 (1.23, 1.77) | 1.20 (1.03, 1.40) | 1.04 (0.87, 1.24) | 1.14 (0.89, 1.46) |
| Expected RR | | | | | | |
| Multiplicative | 2.81 | 1.87 | 1.42 | 1.11 | 1.04 | 1.13 |
| Additive | 2.74 | 1.84 | 1.41 | 1.11 | 1.04 | 1.13 |

## 5.6 Public health impact of tobacco use and BMI (V)

Table 9 shows the percent of deaths in terms of PAF that could be potentially prevented by pursuing the elimination of corresponding risk factor. For example 5% male and 11% female deaths could potentially prevented by pursuing the elimination of both smokeless tobacco use and underweight. Similarly, around 8% of male deaths could potentially prevented by pursuing the elimination of both smoking and underweight.

**Table 9.** Population Attributable Fractions (PAF) of all-cause mortality, by BC, tobacco use and gender: MCS, Mumbai, India, 1991–2003

| Gender and Tobacco habits | BC | | | |
|---|---|---|---|---|
| | Below normal | Normal | Overweight | Obese |
| **Men** | | | | |
| Never users | 2.3 | 1.3 | Reference | 0.2 |
| Smokeless tobacco users | 5.0 | 3.2 | 0.1 | 0.1 |
| Smoker | 7.9 | 7.7 | 1.2 | 0.3 |
| **Women** | | | | |
| Never users | 2.9 | 0.8 | Reference | 0.2 |
| Smokeless tobacco users | 11.1 | 5.7 | 0.4 | 0.3 |

# 6. Discussion

## 6.1 Tobacco use

There are an estimated 1.3 billion adult smokers (over 15 years old) among the world's six billion people (Guindon and Boisclair 2003). If the prevalence of tobacco use remains constant, the number of smokers will rise to 1.7 billion between 2020 and 2025 (Guindon and Boisclair 2003). Four-fifths of current smokers live in low-income or middle-income countries.

There are important gender differences in tobacco use, with global prevalence among males (48%) about four times higher than among females (10%) (Guindon and Boisclair 2003). There may be considerable female smoking that is underreported, or unreported, because of gender norms that stigmatize smoking among women. Male-female differences in use are highest in the Western Pacific Region and lowest in the Americas and Europe, where about one quarter of women smoke (Corrao et al. 2000). The most recent data for China show a dramatic gender gap (63% among men and 3.8% among women) (Yang et al. 1999).

Typically, the smoking epidemic starts in most populations among men and higher-income groups, and later affects women and low-income groups. However, global male rates have peaked and have stabilized or are in slow decline, while the prevalence of tobacco use among women is increasing (Mackay 2001; Molarius et al. 2001). In fact, the historical gender differences in uptake and prevalence are shrinking because of the increased prevalence of smoking among girls. Recent findings of the Global Youth Tobacco Survey, the largest global survey of adolescents aged 13 to 15 and tobacco use, show that almost as many young girls are smoking as young boys in many parts of the world (Global Youth Tobacco Survey Collaborating Group 2003). This is an indicator of the increasing global epidemic among women that will not peak until well into the 21st century. The prediction is that by 2025, 20% of the female population will be smokers, up from 12% in 2005. Even so, despite low prevalence in some countries, the large population base of countries like China and India means that tens of millions of women are already smokers. And, although the global prevalence of male tobacco use is not increasing, smoking rates among men and boys remain alarming, particularly in countries which are still in the early stages of the tobacco epidemic.

In India, there are currently about 195 million male and 45 million female tobacco users (Reddy and Gupta 2004). Around 45 surveys have been conducted since the 1960s in various urban and rural areas, covering different age groups, but only a handful were large enough to be representative of the area studied. Male tobacco use prevalence in 1995–1996 was 51.3% and in 1998–1999 it was 46.5% according to the National Sample Survey 52nd Round (National Sample Survey Organization 1996) and National Family Health Survey-2 (Rani et al. 2003). While the prevalence of tobacco use among females was 10.3% and 13.8% in those two successive surveys. Similarly, the National Household Survey of Drug and Alcohol Abuse (Srivastava et al. 2004) conducted in 25 states (excluding Jammu and Kashmir) in 2002, reported that 55.8% of males aged 12–60 years were current tobacco users. National trends seen in the data include higher tobacco use prevalence among males compared to females and among older age groups compared to younger. A more recent review article (Gupta and Ray 2004) reported the tobacco use prevalence from house-to-house surveys carried out in individual areas across India over a 40-year time span. In these studies, the tobacco use prevalence among men (age >15 years) ranged from 19 to 86% and among women from 7 to 77%. Overall, tobacco use prevalence is higher in rural areas. Gender-wise, chewing habits were practised about equally by men and women, while most smokers were men (Gupta and Ray 2004). The prevalence of chewing in men varied from 11 to 55% and in women from 10 to 39%. Smoking prevalence varied from 8 to 77% in men and 2 to 12% in women. MCS corroborates these national findings, in Mumbai, tobacco use prevalence was higher among men (≈70%) than women (≈60%), but women were primarily smokeless tobacco users (as smoking prevalence was <1%).

Tobacco use among women is prevalent in all regions and almost all sections of India; overall 2.4% of women smoke and 12% chew tobacco (Rani et al. 2003). The low prevalence of smoking among women in most areas is due to social unacceptability, but at the same time it is also common in parts of the north, east, north-east, and Andhra Pradesh (Reddy and Gupta 2004). In one study in rural Srikakulam, Andhra Pradesh, ‘reverse’ *chutta* smoking was practised by 59% of women and 35% of men. In rural Darbhanga, Bihar, smoking was practised by nearly 21.8% of women (mainly *hookah* smokers) and 11.4% of men. Also, in an area of Orissa 85% of women smoked *chuttas*, compared to 30% of men. Among school personnel in rural and urban Bihar, 31% of women and 47.4% of men reported smoking (Sinha et al. 2002).

However, smokeless tobacco use among women is high. In Mumbai, 59% of women used smokeless tobacco (Gupta 1996, Table 2). The prevalence was similar to that of other South-Asian female populations. Around 49% of UK-Bangladeshi females and 59% of rural Malaysian females use smokeless tobacco (Gan 1995; Croucher et al. 2002).

## 6.2 Tobacco use and mortality

Worldwide, an increased risk of death among cigarette smokers compared to non-smokers had been reported in several cohort studies (US Public Health Service 1964; US Department of Health and Human Services 2004). The present MCS, with a 37% increased risk of death for cigarette smokers (vs. never tobacco users), demonstrates that Indians are no different in this respect, although this increase may be on the lower side, probably due to lower frequency of number of cigarettes smoked per day (Gupta 1996). But there are differences in the nature of the effect of smoking seen in India and cigarette smoking in the West. For instance, in the USA, cigarette smoking leads primarily to lung cancer, accounting for over 70% of tobacco related cancer (TRC) deaths and a third of all-cancer deaths. In India, where, *bidi* smoking and use of smokeless tobacco are common (Reddy and Gupta 2004), the major effects of tobacco are seen in the oral cavity, pharynx, and oesophagus, which together account for almost 75% of tobacco-related cancers (IARC 2004a).

The excess annual mortality among middle-aged (35–69 year) male smokers in developed countries was reported as 701 per 100,000 (Peto et al. 1994). While in MCS, the corresponding value at the same age was 996 per 100,000, despite the fact that the number of *bidis* or cigarettes smoked per day was quite low [median 5 cigarettes or 12 *bidis* (Gupta 1996)]. The entire difference, however, was mainly due to *bidi* smoking (excess mortality 1,350 per 100,000 in *bidi* smokers vs. 503 per 100,000 in cigarette smokers). Our finding about higher excess risk of death among *bidi* smokers than cigarette smokers was similar to that reported elsewhere. (Rahman and Fukui 2000).

*Bidi* smoking is the dominant form of smoking in India, especially in rural areas. Being a large metropolitan city, in this cohort *bidis* and cigarettes were equally common but, in the country as a whole, 7 to 8 times more *bidis* are consumed than cigarettes. *Bidi* smoking is also practised in neighbouring countries (Rahman and Fukui 2000) and there are recent reports of its availability and popularity in the USA as well, especially among youth (Anonymous 1999b). The more recent global surveys (The GTSS Collaborative Group 2006a; The GTSS Collaborative Group 2006b) conducted among schoolchildren and school personnel, underlines the continuity of the popularity of *bidi* smoking not only among school personnel (Sinha et al. 2003a), but, also among schoolchildren aged 13–15 years, across India (Sinha et al. 2003b). The results for *bidi* smoking are therefore important not only for India but also for many other countries.

In Mumbai, *bidi* smoking caused 64% increased risk (compared to never tobacco users) of death among men. A similar increase (50–60%) was reported in previous cohort studies conducted in a neighbouring (Pune) district (Gupta et al. 1980) and in other parts of India as well in other countries (Gupta et al. 1984a; Rahman and Fukui 2000). Additionally, MCS reported higher RR at low exposure level, among *bidi* smokers (RR: 1.42 for 1 to 5 *bidi* per day) compared to the corresponding RR among cigarette smokers (RR: 1.20 for 1 to 5 cigarettes per day).

All forms of tobacco produce free radicals that deplete antioxidants like Vitamins C, E and carotenoids and cause oxidative damage to DNA, proteins and lipids (Nair et al. 1992; Bagchi et al. 1999; Mahimkar et al. 2001; Dietrich et al. 2003). Eventhough, a *bidi* contains a smaller amount of tobacco (about 0·2 g) than a cigarette (about 1 g), the concentrations of nicotine, tar and other toxic agents were found to be higher in *bidis* (Sanghvi and Notani 1989, Rahman and Fukui 2000). Also *bidi* smoking has a greater physiological and biochemical effect than cigarette smoking (Rahman and Fukui 2000, Gajalakshmi et al. 2003).

Besides the epidemiological observations, there is also experimental evidence showing that *bidi* smoke has an effect that is equally harmful as cigarette smoke to parameters such as pulse rate, blood pressure, platelet aggression time and serum-free fatty acid levels known to be related to the pathogenesis of coronary heart disease (IARC 2004a). Therefore, MCS not only highlights the burden of *bidi* smoking along with cigarette smoking, but highlights the need for a special attention towards *bidi* smoking in anti-smoking campaigns.

Another noteworthy tobacco form in India is the use of smokeless tobacco. In MCS, smokeless tobacco use increased the risk (vs. never tobacco use) of dying, among both men (RR=1.16) and women (RR=1.25). This increase was mainly observed due to the use of *mishri* (women only), *mishri* plus other forms of smokeless tobacco, and tobacco plus lime. Similar findings of increased risk of dying for smokeless tobacco user men (RR=1.2) and women (RR=1.3) were reported in a study conducted in Kerala (Gupta et al. 1984b). A systematic review carried out by Critchely et al. reported that chewing betel quid and tobacco is associated with a substantial risk of oral cancers in India. The most recent studies from the US and Scandinavia, did not reveal statistically significant findings, but a moderately positive association cannot be ruled out due to lack of power, although the types of smokeless tobacco were different (Critchely and Unan 2003).

Worldwide, it was estimated that, between 1950 and 2000, 10 million women died from smoking-related diseases (Crofton and Simpson 2006). Women tobacco users not only share the same health risks as men, but are also faced with health consequences that are unique to women, including those connected to pregnancy and cervical cancer.

In Mumbai, annual excess mortality among middle-aged (35–69 years) smokeless tobacco user women was 250 per 100,000, whereas in developed countries, similar excess mortality (312 per 100,000) was observed among women (aged 35–69 years) who smoke (Peto et al. 1994). Therefore, it is highly likely that, like smoking in developed countries, smokeless tobacco use in India may also result in increased all-cause mortality.

Considering the prevalence of smoking in India as 40% (30% *bidi*, RR: 1.82, 95% CI: 1.62–2.05; 10% cigarette, RR: 1.43, 95 % CI: 1.25–1.64) in men, and with an additional 15% use of smokeless tobacco (RR: 1.14, 95% CI: 1.03–1.26), a total of 23.7% of deaths among middle-aged (35–69 years) men could be attributed to their tobacco usage. This, translates into 527,500 deaths every year [taking all cause deaths in the 30–69 year age group 2,229,000 (Murray and Lopez 1996)]. Similar estimates were also reported in a case-control study in Chennai (Gajalakshmi et al. 2003) where about a quarter (552,000 deaths every year) of deaths in male *bidi* or cigarette smokers at ages 25–69 years were killed by their smoking. Similarly, taking the prevalence of smokeless tobacco use among women in India to be 15% (RR: 1.30, 95% CI: 1.18–1.43) and of smoking to be 3% (RR: 1.53, 95% CI: 0.97–2.42); about 5.7% of deaths among women (aged 35–69 years) could be attributed to their tobacco usage. This, translates into 83,000 deaths per year [taking all-cause deaths in 30–69 years age group 1,453,000 (Murray and Lopez 1996)].

According to the projections carried out by WHO in early 2006 (Mathers and Loncar 2006), the world will experience a substantial shift, in the distribution of causes of deaths, from younger age-groups to older age-groups, and from communicable diseases to non-communicable diseases, during the next 25 years. A considerable decline in mortality is projected to occur during these years for all of the principal communicable, maternal, perinatal, and nutrition causes, with the exception of Human Immunodeficiency Virus (HIV)/AIDS. Global deaths from HIV/AIDS are projected to rise from 2.8 million in 2002 to 6.5 million in 2030.

In India, the increase will be observed for deaths from cardiovascular diseases (29.0% in 2005 to 35.9% in 2030), cancers (8.0% to 11.9%), other chronic diseases (16.0% to 19.1%), and injuries (10.8% to 12.1%). In contrast, the decrease (36.2% to 21.0%) will be observed for deaths from communicable diseases (Ezzati et al. 2004).

The RRs of cancer deaths of the different sites of the upper alimentary and respiratory tracts ranged between 2.5 and 6.2 among chewers, 2.2–11.8 among smokers, and 6.2–31.7 among those who both chew and smoke (Gupta and Ray 2004). The RRs for smokers developing myocardial infarction and coronary artery disease ranged between 2 and 3 fold. However, a four-fold prevalence of COPD was observed among smokers (Notani et al. 1989). In MCS, smoking among men increased the risk of dying from respiratory diseases (RR: 2.12, 95% CI: 1.57–2.87) and circulatory diseases (RR: 1.21, 95% CI: 1.04–1.40). Among respiratory diseases, smoking increased the risk of dying from pneumonia (RR: 2.46, 95% CI: 1.40–4.30) and COPD (RR: 2.13, 95% CI: 1.45–3.14). Similarly, smokeless tobacco use increased the risk of dying due to circulatory diseases (RR: 1.19, 95% CI: 1.02–1.38) among women [within circulatory diseases; IHD (RR: 1.25, 95% CI: 1.05–1.49)] and respiratory diseases (RR: 1.50, 95% CI: 1.12–2.03) among men.

The TRC relative to all-sites of cancer, reported by the Population Based Cancer Registries (PBCRs) of Bangalore, Barshi (rural), Bhopal, Chennai, Delhi, Ahmedabad, Kolkata, and Mumbai for the years 2004–05; ranged from 34.7% in Bangalore to 51.0% in Ahmedabad (other than Ahmedabad urban) among males. Among females, the relative proportion of TRC ranged from 10.1% in Delhi to 16.8% in Chennai (National Cancer Registry Program 2007). The top five or six cancers in men are all tobacco-related cancers: of the lung, oral cavity, larynx, oesophagus and pharynx. In women, the leading cancer sites, besides breast and ovary, include those related to tobacco: cervix, oral cavity, oesophagus and lung (National Cancer Registry Program 2007).

In MCS, smokeless tobacco use increased the risk of dying from all cancers (ICD 10 codes; C00–97) among both women (RR:1.39, 95% CI: 1.01–1.91) and men (RR: 1.33, 95% CI: 1.03–1.72), after controlling for possible confounders such as age, education, religion, mother tongue, and BMI (results not shown). Similarly, smoking increased the risk of dying from cancers among men (RR: 2.95, 95%: 2.33–3.73), but failed to reach statistical significance (due to low prevalence of smoking) among women (RR: 1.97, 95% CI: 0.47–8.16). Using the above RRs for all-cancer deaths, a PAF was estimated by using a revised formula given by Rockhill et al. (Rockhill et al. 1998). We estimated that 41.6% of men's and 20.7% of women's cancer deaths could be attributable to their tobacco usage. These estimates were consistent for both men (40.6%) and women (15.0%) with that reported by PBCR Mumbai in terms of the relative proportion of TRCs.

Among men, not only smoking (RR: 6.08, 95% CI: 3.16–11.67) butalso smokeless tobacco use (RR: 2.11, 95% CI: 1.03–4.30) increased the risk of dying from respiratory neoplasms (ICD 10 codes; C30–39). Similar increased risks (i.e., for smoking: RR: 14.40, 95% CI: 4.44–46.68, and for smokeless tobacco use: RR: 2.83, 95% CI: 0.78–10.21) were observed for deaths due to cancer of the lip, oral cavity, and pharynx (ICD 10 codes; C00–14). Smoking also increased the risk of dying from ill-defined cancers (ICD 10 codes; C76–80) and cancers of the digestive organs (ICD 10 codes; C15–26) among men (RR: 2.58, 95% CI: 1.71–4.45 and RR: 1.98, 95% CI: 1.29–3.04 respectively) as well as women (RR: 10.68, 95% CI: 1.27–89.74 and RR: 3.83, 95% CI: 0.49–30.06 respectively).

Therefore, MCS findings provide corroborative evidence from developing country population about the association of tobacco usage with increased risk of dying from different cancers, in India, too.

Although tobacco use is causing excess mortality in India, the pattern of death seems to be different from that in industrialized countries. TB is a major cause of morbidity and mortality in developing countries, including India. The WHO estimates that the largest number of new TB cases and TB deaths in 2005 occurred in the South-East Asia Region, which accounted for 34% of incident cases (8.8 million) and 31% of deaths (1.6 million) globally. However, the estimated incidence rate in sub-Saharan Africa is nearly twice that of the South-East Asia Region, at nearly 350 cases per 100,000 individuals (Tuberculosis Fact Sheet Number 104 2007). By 2020, the global burden of TB is estimated to be 2.30 million, of which, 99% will be in developing countries (Anonymous 1997).

Smoking has been widespread for many decades among men in India (Gupta 1996; Kolappan and Gopi 2002; Gajalakshmi et al. 2003; Reddy and Gupta 2004). But the possible association between smoking and TB has not received much attention so far. Only either cross-sectional or case-control studies (Lam et al. 2001; Leung et al. 2003; Ariyothai et al. 2004; Altet-Gomez et al. 2005) have been reported on this association. One such case-control study reported four-fold increased odds of dying from TB among ever smokers (vs. never smokers) in urban (OR: 4.5, 95% CI: 4.0– 5.0) and rural (OR: 4.2, 95% CI: 3.7– 4.8) areas of Tamil Nadu (Gajalakshmi et al. 2003). Another case control study (Kolappan and Gopi 2002) from the same state, but from a different district (Tiruvallur), reported a similar increase (OR: 2.24, 95% CI: 1.27–3.94). Thus, two case-control studies from the same state (Tamil Nadu) reported a statistically significant association, even though there were some differences in reported ORs. This association was not visible in cohort studies, as most of the previously reported cohort studies were from industrialized countries, where TB is fairly rare. However, a recent cohort study, from Hong Kong (Leung et al. 2004), demonstrated an adjusted hazard ratio of 2.87 (95% CI: 2.00–4.11) among current cigarette smokers (vs. never smokers) for pulmonary TB, with a statistically significant dose response relationship.

The MCS with a large sample size corroborates the results reported in both the case-control studies (Kolappan and Gopi 2002; Gajalakshmi et al. 2003) from India and a cohort study from Hong Kong (Leung et al. 2004). In MCS, the risk was twice greater in ever smokers (RR: 2.12, 95% CI: 1.70, 2.66) than in never-smokers and it was nearly three times higher in *bidi* smokers (RR 2.60, 95% CI: 2.02–3.33).

Considering the RR (RR: 2.60, 95% CI: 2.02–3.33) for TB deaths from MCS and 30% *bidi* smoking prevalence in India, ≈32% of TB deaths in India can be attributed to *bidi* smoking. Similarly, for other (mainly cigarette) smoking (prevalence 10%), ≈6% of deaths can be attributed to other (mainly cigarette) smoking (this was 33% for smokers in Hong Kong (Leung et al. 2004). A more recent nationally representative case-control study (Jha et al. 2008) of smoking and death in India reported smoking associated excess deaths (38%) similar to those reported in MCS. Thus, smoking, particularly *bidi* smoking, seems to be a very important risk factor for increasing the burden of TB in India and MSC highlights the importance of *bidi* smoking in TB control programmes.

## 6.3 Body mass index

The recommended BMI cut-off levels may vary from time to time and country to country, making global, longitudinal surveys problematic. In 1998, the US National Institutes of Health brought US definitions into line with WHO guidelines, lowering the normal/overweight cut-off from BMI 27.8 to BMI 25. This had the effect of redefining approximately 30 million Americans, previously technically healthy to technically overweight. WHO and others recommends lowering the normal/overweight threshold for South-East Asian body types to around BMI 23 (The International Diabetes Institute et al. 2000), and expects further revisions to emerge from clinical studies of different body types.

In Singapore, the BMI cut-off figures were revised in 2005 with an emphasis on health risks instead of weight. Adults whose BMI was between 18.5 and 22.9 had a low risk of developing heart disease and other health problems such as diabetes. Those with a BMI between 23 and 27.4 were at moderate risk while those with a BMI of 27.5 and above were at high risk of heart disease and other health problems (Revision of Body Mass Index Cut-Offs in Singapore 2007).

Nutritional research in India has focused primarily on the problem of undernutrition, particularly among vulnerable women and children. Currently, India is undergoing a rapid economic transition. At this stage in the associated epidemiological transition, the country is facing the double burden of communicable and non-communicable diseases. As in all such transitions, nutrition plays a central role (Rosenkrantz 1972; Romieu et al. 1997; Puska 2002). Obesity, representing one extreme of the continuum, is a preventable risk factor for chronic degenerative diseases (World Health Consultation on Obesity 1998; WHO 2003), while chronic energy deficiency (CED), though less directly preventable, causes impaired physical capacity (Durnin and Passmore 1967, Durnin 1994), reduced economic productivity (Kennedy and Garcia 1994, Untoro et al. 1998), increased mortality (National Institute of Nutrition 1991; Campbell and Ulijasek 1994) and poorer reproductive outcomes (Kusin et al. 1994; Anonymous 1995, Schieve et al. 2000).

Routine monitoring of nutritional status through the collection of data on BC is a simple approach that is both economical and can be rapidly applied to large numbers of people. The BMI is a useful index of relative weight that can be applied to define obesity (Garrow 1988) and CED (Ferro-Luzzi et al. 1992; Shetty and James 1994) and can be used to assess individual and community nutritional status (Bailey and Ferro-Luzzi 1995). With its huge population and extremes of economic conditions, India is in a unique position in its epidemiological and nutritional transition that may presage the experience of many developing countries.

Although the BMI profile of a representative rural Indian population has been published by the National Nutrition-Monitoring Bureau (NNMB) (Naidu and Rao 1994), there is a lack of data on the BMI distribution of representative adult Indian urban populations – the group at highest risk of the extremes of the current transition.

Earlier published studies on BMI in India had reported either a high prevalence of thinness and a low prevalence of overweight or a low prevalence of thinness and a high prevalence of overweight. This contradictory reporting may be due to differences in locality (urban/rural) or population sample (high socio-economic/low socio-economic). A representative survey conducted in rural areas by NNMB found that over 40% of the women were thin, while the prevalence of overweight people was only 6.6% (Reddy et al. 1993). An earlier study from Mumbai reported a higher prevalence of overweight (44%) (Dhurandhar and Kulkarni 1992). However, sampling volunteers from members of clubs could have introduced a bias towards overweight in that study. Another five-city study that included Mumbai as one of its sites, reported a higher prevalence of overweight (37.5%) and a lower prevalence of thinness (5%) in women (Singh et al. 1999b). Published data from surveys in Delhi, another major city in India, reported 23–25% of men were overweight (Chadha et al. 1995; Gopalan 1998). However, the reported prevalence of overweight in females was highly divergent (35% and 60%) in those two studies. A prevalence of 21–27% for overweight has been reported from other urban areas in India (Rao et al. 1986; Sood et al. 1996). In general, earlier studies in urban areas have focused mainly on the prevalence of obesity, but the prevalence of thinness has not so far been reported except in one urban slum population, where 36.7% were underweight and 11.6% were overweight (National Institute of Nutrition 1996).

A more recent nationally representative World Health Survey (WHS) was conducted in six states of India; Assam, Karnataka, Maharashtra, Rajasthan, Uttar Pradesh, and West Bengal (WHS 2003). In WHS, the prevalence of underweight (BMI<18.5) among women ranged from 14.3% (Assam) to 38.3% (Uttar Pradesh), and among men it ranged from 14.3% (Assam) to 28.5% (Maharashtra). While the prevalence of overweight (BMI≥25) ranged from 7.5% (Uttar Pradesh) to 16.2% (West Bengal) and 6.7% (Rajasthan) to 11.6% (Karnataka) among women and men respectively. There were more obese (BMI≥30) women than men, in all the six states surveyed in India. Underweight was more prevalent in rural India than urban India, and this was true for both, women (30.8% vs. 21.0%) and men (24.9% vs. 18.8%). The reverse was true for overweight women (11.2% vs. 19.9%) and men (9.1% vs. 17.2%) in rural and urban India respectively. These results were more prominent for obese women (2.6% vs. 5.5%) and men (0.9% vs. 2.0%).

The BMI profile presented by MCS is from one of the largest urban survey done on adults in India and is consistent with results based on 500,000 individuals in the Chennai prospective study (Gajalakshmi et al. 2007). Besides being one of the largest cities in India, Mumbai is the country’s financial centre and is host to people from all states of India. The age distribution of the study sample was similar to that of urban India (Registrar General of India 1991; Shukla et al. 2002). Literacy in the study sample was 87.6% and 54.7% compared with 81.3% and 62.5% for urban India for men and women (15–59 age group) respectively (Registrar General of India 1991; Shukla et al. 2002). We excluded affluent housing residents and adults aged 20–34 years of age. While this might be seen as a limitation, it can be argued that middle to low income strata form a substantial segment of the population and are the groups in whom the epidemiological transition will have the most impact. Mumbai, and indeed most of urban India, is in the midst of a nutritional and epidemiological transition (Shetty 2002). Thus, the classic relationship that is observed between nutritional status and other socioeconomic variables in Western populations that are in the late stages of nutritional transition is not applicable to the Indian population (Griffiths and Bentley 2001; Popkin 2001; Popkin et al. 2001; Popkin 2002a; Popkin 2002b; Shetty 2002); indeed, they may be dramatically inverted. For example, affluent individuals in India are more likely to be overweight than their less affluent counterparts; by contrast, in the US more affluent individuals are more likely to be thin (Hebert 2005).

Although socio-economic status (SES) is important determinant in both contexts, its implications in each may be quite different and the correlates with health may differ similarly. Both sides of the energy balance equation, deficiency and excess of energy intake, drive differences in relative weight. Besides differences in the characteristics (and cost) of physical labour (Durnin and Passmore 1967, Durnin 1994), the cost of prepackaged convenience and other fast foods vary dramatically in relation to the more traditional foods (e.g., rice, beans, and pulses) between the pre- and post-transition countries. Such pre-packaged and fast food items are relatively expensive in relation to the minimum wages in India, but they are very inexpensive in the US (Drewnowski 2002). The ability to consume excess calories, with the concomitant result of increases in the prevalence of overweight and obesity, would depend on the cost per unit calorie relative to income and in comparison to the costs of other commodities. As food prices drop in relative terms, it ushers in a new phase of the nutrition transition (Popkin 2001) and with it, perhaps, a new epidemic of overweight with adverse health consequence in the developing world.

In Mumbai, around 19.1% women and 17.4% men were underweight ($BMI<18.5$), while 29.4% women and 20.2% men were overweight ($BMI\geq25$). There were more obese ($BMI\geq30$) women (6.9%) than men (2.7%) in Mumbai. The findings presented provide evidence that both overweight and thinness are equally prevalent in the urban population of Mumbai. Our results on the prevalence of underweight, overweight, and obesity are consistent with those reported in a recent nationally representative WHS (WHS 2003).

Thus, emerging problems related to overweight/obesity against a background of chronic underweight in large segments of the population raise important questions about policies aimed at health promotion. For example, in a country with a preponderance of overweight people a recommendation can be targeted at lowering BMI values without much concern for thinness (except among individuals with eating disorders). On the other hand, in a country with a preponderance of thinness, a recommendation can be targeted at increasing body weight without much concern about inducing obesity or overweight-related illness. The results from MCS highlight the immediate need to identify and to address both underweight and obese portions of the distribution in identifying vulnerable targeting interventions. Because age, education, and tobacco use were found to be independently associated with BMI, such targeting will need to take these factors into account.

The most vulnerable group at risk of thinness is the elderly. This segment of the population is growing worldwide and concern for the nutritional status of the elderly has been expressed in many countries. The BMI distribution observed in the elderly in this urban survey provides evidence that provisions for the care of this group needs to be considered carefully by health care policymakers.

Despite this, a high prevalence of thinness among the aged is of concern. This is in contrast to observations in elderly populations in affluent countries such as the UK, where 5% of men and 6% of women aged 75+ years were thin, and 64% men and 57% women were overweight (Prescott-Clarke and Primatesta 1996). There may be many reasons for the higher prevalence of thinness among elderly Indians. Because of cohort effects older individuals may have been thinner throughout their life span. Also, in the older age groups, morbidity may influence the BC with thinness. On the other hand, loss of income and independence may have caused a drop in BMI in the elderly group. It is most likely that a combination of all these factors influences BMI in the elderly.

In MCS the proportion of literate men, 87.6%, and women, 54.7%, was similar to that of the nationally reported proportion for urban men, 81.3%, and women, 62.5%, (Registrar General of India 1991; Shukla et al. 2002). From MCS, we observed that low BMI was associated with lower educational attainment, while high BMI (overweight/obese) was associated with higher educational attainment (Shukla et al. 2002). This pattern was observed for both men as well as women in Mumbai.

A global MONICA study (Molarius et al. 2000) conducted in 26 different countries reported no association between education and BMI in men in 18 countries, an inverse association in 6 countries, and a positive association in the remaining two countries (Russia and Poland). By contrast, in women, 22 out of 26 countries reported an inverse association between education and BMI. An inverse association is the norm in many affluent countries, most notably in the USA (Flegal et al. 1998).

The education-BMI association is not static, but changes over time and with the epidemiological transition. This was already observed in the UK. Although there exist an inverse relation between SES and BMI, a positive association between SES and overweight existed in the 1950s to early 70s (Sobal and Stunkard 1989). One possible explanation for this inverse in the association between education and BMI may be the relationship between education and occupation. In countries in transition (or mainly those facing a double burden of under- and over-nutrition), where less-educated people are in labour-intensive occupations and people with higher education are living a more sedentary lifestyle, education may be associated differently with low (<18.5) and high (≥25.0) BMI. This opposite association was observed in MCS; education was negatively associated with low BMI, while positively associated with high BMI (Shukla et al. 2002). By contrast, in economically advanced countries, lower education may be associated with higher unemployment or low-paid jobs that are not necessarily labour-intensive. Leisure time physical activity may be the major determinant of BMI. Increased health awareness and access to recreational facilities among the highly educated (affluent) groups compared to the less educated would result in an inverse relation between education and BMI. For example, in the USA (National Centre for Health Statistics 1997), obesity is twice as high in men with < a $9^{th}$-grade education (21.7%) compared to college graduates (11%) and two and half times higher in women with < a $9^{th}$-grade education (26.6%) compared to graduates (10.0%). Although in MCS, female college graduates in Mumbai had a similar prevalence of obesity (10.5%) as Americans; illiterate women had a lower prevalence of obesity (5.1%) and a higher prevalence of thinness (24.4%). Overall, Indian men have a very low prevalence of obesity; and a similar low prevalence was also observed in MCS. However, the prevalence was highest (5.2%) among graduate men in Mumbai.

## 6.4 BMI and mortality

Moderate to high prevalence of both under- and over-weight may exist within populations in different parts of the world (Doak et al. 2000). This was present in our population (Shukla et al. 2002) and was reported in other parts of India as well (Kapoor and Anand 2002; WHS 2003). Low BMI was associated with increased risk of mortality in this urban Indian population. These results underline the need for public health interventions aimed at reducing the burden of undernutrition. It should be noted that the observed relationships may change as the population makes the transition toward increased prevalence of overweight and obesity. Careful monitoring of this population will be necessary in order to alert the policymakers to future public health problems that are currently being observed in association with the obesity epidemic in the West.

Currently there are over 1 billion overweight (BMI≥25) people worldwide, of whom approximately 350 million are obese (BMI≥30). As the worldwide prevalence of obesity is increasing to epidemic proportions at an alarming rate (Flegal et al. 2002; Hedley et al. 2004), concomitant concerns regarding its effect on excess mortality have increased, although the association between body weight and all-cause mortality is more controversial—the relationships ranging from linear to J- or U-shaped (Manson et al. 1995; Troiano et al. 1996; Meyer et al. 2002; Ajani et al. 2004; Flegal et al. 2005; McGee DL and Diverse Populations Collaboration 2005; Gu et al. 2006). However, most of these studies were conducted in Western populations in which only a small proportion of the study participants had low BMI. Our findings were based on the first cohort study from India that has attempted to investigate the association of both, low and high BMI with mortality.

Despite the concerns that the epidemiological transition, in India and in other low-income countries, might result in a double burden of diseases related to both under- and over-nutrition, the findings from this unique and large prospective study suggest that undernutrition continues to be a major problem in India.

In MCS, BMI categories were based on baseline measurements; however, weight change appears to be an additional important risk factor independent of the initial BMI (World Health Consultation on Obesity 1998; Mikkelsen et al. 1999). However, in order to study the impact of weight changes on mortality, it is necessary to have repeated weight measurements (Byers 1999), data that were not available in MCS. In contrast to many other studies, height and weight were measured and not self reported. In the West, self-reported weights, despite their high correlation with measured weights, have been found to be lower (average 1.5 kg) than measured weights (Plankey et al. 1997; Kuczmarski et al. 2001). In addition to the underestimation of weight, height is often overestimated (Kuczmarski et al. 2001; Nawaz et al. 2001). While, the pattern of misreporting may indeed be very different in India, we obviated the problem entirely by actually measuring weight and height.

The categorization of BMI used in MCS was consistent with the recommendations of WHO (World Health Consultation on Obesity 1998). Recently, Stevens et al. (Stevens et al. 2000) validated the categorization in the WHO report by using a large American cohort of never-smokers. Compared with those predominantly Western populations, Asians have a lower average BMI value and a higher percentage of body fat for a given

BMI (Deurenberg et al. 2002; Chang et al. 2003). Therefore, there are problems with applying standard classification to Asians and to other ethnic groups uniformly (The International Diabetes Institute et al. 2000). For Asian populations, WHO defined overweight and obesity as BMI values of 23–<25 and ≥25 respectively. Using the Asian cut-off values, we observed (results not shown) a similar risk pattern (i.e., increased risk of dying at both extremes of BMI) that was observed using the conventional cut-off values. Although the RRs were apparently (but not statistically significant) lower for different BMI categories when we used the Asian cut-off values in place of the conventional cut-off values generally used in the West. Of course, this result was not surprising because the lower part of the Asian "overweight" category (i.e., >23 but <25) overlaps the normal weight category that serves as the referent for the other, more standard, comparison. Very few studies have reported findings based on the Asian cut-off values; further, these studies used different reference categories (Gu et al. 2006). Therefore, a direct comparison with those studies was not possible. We therefore conclude from our findings that the choice of cut-off should not be a major issue if the primary objective is to study the association of BMI with all-cause mortality.

### *6.4.1 Overweight and mortality*

While the debate regarding the specific BMI range that constitutes overweight and obesity is likely to continue, there is little doubt that morbid obesity is unhealthy and that it increases the risk of death. However, the results regarding overweight and mortality are inconsistent. Some studies have observed that overweight was associated with an increased risk of all-cause mortality (Meyer et al. 2002; Ajani et al. 2004; McGee DL and Diverse Populations Collaboration 2005); while, others have reported no excess mortality, particularly in the older age-groups (McGee DL and Diverse Populations Collaboration 2005). By contrast, few studies have reported a decreased risk of mortality (Flegal et al. 2005; McGee DL and Diverse Populations Collaboration 2005) compared to those in the normal weight category.

In MCS, decreased risk of death for overweight (BMI 25.0-<30.0 kg/m$^2$) men (RR=0.87) and women (primarily aged 60 years or more, RR=0.82) was observed compared to corresponding normal BMI men and women. They were at ≈60% and ≈30% decreased risk of dying from TB and respiratory diseases respectively. While obese men and women were at ≈20% increased risk of dying from circulatory diseases. Potential correlates of the relationship between BMI and all-cause mortality (age, education, religion, mother tongue, sex and tobacco use) were taken into account in MCS, whereas most of the other studies adjusted primarily for age. Obesity was rare therefore the results are subject to random variation.

In purely biological and physical terms, the aetiology of overweight is similar everywhere, i.e., it is simply due to a chronic imbalance between energy intake and expenditure. The epidemiological and nutritional transitions observed over many decades (or centuries in the developed countries) were associated with changes in the nature of work, which resulted in a decreased need for physical labour (and reduced energy expenditure) to earn income and drastic reductions in the cost of food (and a concomitant increase in energy intake). Currently, these transitions are being observed in many of the developing countries. The rate and extent of the transitions may vary but the various underlying factors that are responsible for the higher prevalence of obesity seems common. These include factors associated with urbanization and lifestyle factors including lower energy expenditure for physical labour due to mechanization and automation and increases in sedentary leisure-time activities such as watching television.

### *6.4.2 Underweight and mortality*

Our result demonstrates an inverse relationship between BMI (especially extreme thinness) and mortality; this may be due to inadequate energy intake. Previous findings of an elevated risk of death in the thinnest persons have been attributed to inadequate adjustment for smoking (World Health Consultation on Obesity 1998; Anonymous 1999a; Willett et al. 1999) and to those who lost weight as a result of underlying diseases (Willett et al. 1999; Stevens et al. 2001). Our study reported that tobacco use is a risk factor for low BMI, but, MCS results also demonstrated no impact of tobacco use on the relationship (i.e., main effect) between low BMI and mortality (Figure 5). The exclusion of individuals with pre-existing and sub-clinical diseases has not altered the observed association between BMI and mortality in other studies (Allison et al. 1999a; Allison et al. 1999b; Mikkelsen et al. 1999). Although we were not able to exclude individuals with chronic degenerative diseases, the association remained essentially unchanged even after excluding the deaths that had occurred during the first 2 years of follow-up (III). Therefore, it appears that inadequate energy intake, possibly associated with poor diet, accounts for the observed relationship between low BMI and mortality and that it is a consequence of poverty (with all that this entails).

The relation between cancer mortality and energy intake is known since the experiments of Tanaenbaum in 1940 (Tannenbaum 1940). The J- or U-shaped (Lew and Garfinkel 1979; Nomura et al. 1985) response curve that has been observed in several large datasets indicates that mortality from cancer can account for the largest part of the increased risk of death at both the extremes of relative weight (as estimated by the BMI). Nevertheless, some findings of null (Iribarren et al. 1995; Dorn et al. 1997; Tulinius et al. 1997) or even inverted relationships (Calle et al. 2003) with cancer mortality among those with a low BMI who died early during follow-up (Jarrett et al. 1982) supported the argument that increased risk may derive from the BMI-lowering effect of preexisting cancer. However, our findings were similar to those reported by Song et al. (Song and Sung 2001), i.e. cancer in individuals with low BMI was unchanged after excluding those with incidence disease early during follow-up; thereby suggesting that the effect of BMI on death from cancer is not an artifact of measurement timing.

However, we acknowledge that our results may be influenced by adiposity-related differences in the natural history or treatment of cancer or by a true biological effect of adiposity on survival. Additionally, it should also be noted that the increased risk of death at low BMI was observed for non-medical causes (primarily accidental deaths) as well. Further probing into the causes of death, the pattern follows expectation in that BMI is associated with chronic diseases of affluence (i.e., that related to circulatory and digestive system dysfunction) in a reverse J-shaped manner. In contrast, BMI is inversely associated with infectious diseases associated with poverty (respiratory system and TB deaths). Cancer deaths follow the poverty gradient; however, this may be related to the under-diagnosis of certain cancers, as recently noted (Hebert et al. 2006).

## 6.5 Tobacco use and BMI

In India (Ministry of Human Resource Development 1998), nearly the half of all rural adults and a quarter of urban adults have a low BMI (i.e., <18.5 kg/m$^2$). Although CED due to inadequate diet may be the main factor placing the population at risk of low BMI, factors other than diet may play a significant role in explaining the low BMI within this population. These factors may act directly (by affecting appetite or other aspects of physiology) or indirectly (by decreasing the purchasing power for food). From a public health perspective, unmasking these non-dietary determinants of low BMI in the population will help to understand the impact of such exposures. The MCS has found that all forms of tobacco use were associated with low BMI independent of (i.e., after accounting for) age, education, mother-tongue, and religion in this population. Further, there exists a dose-response gradient and the response at every dose was higher in women compared with men, a relationship observed in many tobacco-health disease analyses, including cancer related endpoints (Hebert and Kabat 1991; Kabat and Hebert 1991). The dose-response was statistically significant not only for smoker men, but also for smokeless tobacco using men and women (IV). These findings raise important questions about the magnitude of the impact of tobacco use on the health status of the population.

In MCS, both smoking as well as smokeless tobacco use was independently associated with prevalence of low BMI. Lower BMI in smokers compared to non-smokers has been reported worldwide (Rasky et al. 1996; Molarius et al. 1997). The smoking-BMI association has been attributed to the effect of smoking on physiological processes that lead to changes in appetite, food preferences and basal metabolic rate (Hofstetter 1986; Marti et al. 1989; Margetts and Jackson 1993; Perkins et al. 1996). There have been no previous reports on smokeless tobacco use and, as far as we are aware, this is the first to show smokeless tobacco use as a risk factor for low BMI. Also, the risk was greater in women than in men. Although our findings have implications for women of all ages, they demand special attention among women of reproductive age. This is because low BMI in women of reproductive age in the developing countries was found to be associated with poorer reproductive outcomes (Anonymous 1995). India has the highest prevalence and largest absolute share of low birthweight in the world (Balaji and Dustagheer 2000), while smokeless tobacco use was found to be associated with lower birthweight (Krishna 1978; Krisnamurthy 1991; Deshmukh et al. 1998; Gupta and Sreevidya 2004) and decreasing gestational age at birth (Gupta and Sreevidya 2004). A prospective study conducted in a neighbouring (Pune) district of Mumbai, on maternal determinants of low birthweight found that intake of micronutrient-rich foods was an important limiting factor for foetal growth, independent of maternal age, height, and weight (Rao et al. 2001). Hence, the depletion of antioxidant micronutrients by the toxic agents in smokeless tobacco may play a role in the biology of foetal growth restriction. This merits further investigation as tobacco use in India takes a variety of smokeless and smoking forms, of which cigarettes are only a minor part (Bhonsle et al. 1992).

Previously we have reported (Shukla et al. 2002) that illiteracy is a risk factor for low BMI in this population (OR = 6.52; 95% CI 5.38 to 7.89 for men and OR = 4.83; 95% CI 3.71 to 6.28 for women respectively). In Mumbai (Gupta 1996), the prevalence of tobacco use (especially *bidi* smoking and chewing) is inversely related to education (a good proxy for poverty in this population). *Bidi* smoking is more common than cigarette smoking among the illiterate in Mumbai. This is true for all of South Asia, where the prevalence of *bidi* smoking is reported to be 21–56% among men (Rahman and Fukui 2000). In the present study, we found *bidi* smoking was associated with the highest risk of low BMI.

Besides the direct physiological effect, tobacco use among the economically disadvantaged is known to reduce the resources available to purchase food, clothing, health care, and education, all of which contribute to poor nutritional status (Efroymson et al. 2001).

## 6.6 Joint impact of tobacco use and BMI on mortality

Worldwide, there are two important risk factors underlying the major causes of death: tobacco use and nutritional status (Murray and Lopez 1996; Murray and Lopez 1999; Beaglehole and Yach 2003; Ezzati et al. 2004; Boyle et al. 2006; Lopez and Mathers 2006; Mathers and Loncar 2006). Their effects are now increasing rapidly, with high prevalence rates of smoking and other forms of tobacco use in many parts of the world (Anderson 2006; Jha et al. 2006a) and a virtual epidemic of overweight/obesity and chronic undernutrition in other parts of the world (Sørensen 2000; de Onis et al. 2004; Uauy and Lock 2006). If the current patterns persist, there will be approximately 1 billion deaths from tobacco use in the 21st century, compared with only about 0.1 billion (100 million) during the entire 20th century. Of the 55.9 million deaths occurring annually worldwide, tobacco use and nutritional status together were responsible for approximately 20% (Ezzati et al. 2004).

In developed countries, smoking and excess bodyweight are two of the most important risk factors implicated in chronic diseases and premature death (The Surgeon General's Report 1990; Freedman et al. 2006). Smoking is associated with lower body mass, while quitting smoking is associated with significant weight gain (Williamson et al. 1991; Schoenborn et al. 2004). In fact, smoking cessation was estimated to be responsible for about one quarter of the increase in the prevalence of overweight among US men during the 1980s (Flegal et al. 1995).

Patterns of tobacco use in India (Reddy and Gupta 2004) are very different from those observed in other developed countries. Specifically, smoking is common among men, and it is mainly in the form of *bidis*, followed by cigarette smoking (Bhonsle et al. 1992; Reddy and Gupta 2004). *Bidi* smoking is as harmful as cigarette smoking.

MCS shows that all forms of tobacco use and BMI (a proxy for nutritional status) had joint effects on mortality. In this study, the increased risk of dying was observed among men and the risk increased from never users of tobacco with a BMI of 18.5–<25.0 to smokers with a BMI of <16.0. A similar increasing pattern of risk of dying was observed for overweight/obese men. Although there was a low prevalence of obesity among men in Mumbai (3 percent of never tobacco users, 2 percent of smokeless tobacco users, and 3 percent of smokers), never tobacco users as well as smokers who had a BMI of ≥30.0 were at 34 percent and 56 percent increased risks of dying respectively. Men and women using different types of smokeless tobacco and having a BMI of <16.0 had approximately twice the risk of death compared to the reference group. Similar increasing risks were observed for different types of smoking habits (mainly *bidi* and cigarette smoking). Tobacco use and undernutrition are known to be serious problems in India, and the current study indicates that their joint effect on mortality is synergistic in men, whereas the joint effect of tobacco usage and obesity was antagonistic. By contract, the joint effect of smokeless tobacco use and thinness was antagonistic in women. In general, the conclusion did not depend on whether the interaction was assumed to be additive or multiplicative.

## 6.7 Public health impact of tobacco use and BMI

Tobacco use and nutrition related problems are estimated to be responsible for around 20% of the global annual deaths (Ezzati et al. 2004). However, the estimate does not account for the interaction in the joint effect. A recent projection of global mortality and burden of disease (Mathers and Loncar 2006) projected total tobacco-attributable deaths are to rise from 5.4 million in 2005 to 6.4 million in 2015 to 8.3 million in 2030. Alarmingly, this increase will primarily be observed in low- and middle-income countries (3.4 million in 2002 to 6.8 million in 2030), while a decline of 9% will be seen in high income countries during the same period. Therefore, the finding from MCS about the joint effect of tobacco use and undernutrition on mortality raises serious public health concern in India. Using MCS results we estimated that around 8% of male deaths were attributable to their being underweight and smokers, while 5% of male and 11% of female deaths were attributable to their being underweight and smokeless tobacco users.

There should be no question that the major emphasis of any competent public health programmes should be on preventing youth from staring to use tobacco and in supporting tobacco cessation efforts among those already addicted. But at the same time, we also have to take into account the known fact that tobacco affects dietary requirements. For example, in the UK (Department of Health 1994), the recommendations for vitamin C intake for smokers is higher (80 mg/day) than for non-smokers (40 mg/day). So far, nutritional recommendations for the Indian population have not distinguished between tobacco non-users or users of tobacco in any form (Indian Council of Medical Research 1988a). Doing so would help to highlight both the scientific issues and socioeconomic implications of creating even more stringent requirements for more expensive food, such as that rich in antioxidants.

The findings from this study about the joint effect of tobacco use and undernutrition suggest a major need for further research as tobacco use may have even more far-reaching public health implications in India than previously thought. In addition, tobacco control research and intervention will promote other public health goals of improved nutritional status and corresponding health benefits.

# 7. Conclusions

Tobacco use and undernutrition are serious problems in India. The present study indicates that obesity may emerge as a serious public health problem.

All forms of tobacco smoking increased the risk of dying in Mumbai. In addition to smoking, different forms of smokeless tobacco use also resulted in excess mortality. Using MCS findings, a total of 24% of men's and 6% of women's deaths (aged 35–69 years) were found to be attributable to their tobacco usage. Also, 41.6% of men's and 20.7% of women's cancer deaths were found to be attributable to their tobacco usage. *Bidi* smoking was found to be as harmful as cigarette smoking and was responsible for around 32% of TB deaths.

Therefore, MCS findings provide supportive evidence from a developing country population about the association of tobacco usage with increased risk of dying; primarily for various cancers and TB.

MCS results showed that both chronic underweight and overweight are equally present in an urban population of India. However, the important public health implications for the burden of diseases are associated with only the upper extreme (obese) and all underweight BC. The results from MCS highlight the immediate need to identify and to address both underweight and obese portions of the distribution in identifying vulnerable targeting interventions. Despite concerns that the epidemiological transition, in India and in other low-income countries, may result in a double burden of diseases related to both under- and over-nutrition, the findings from this unique and large prospective study suggest that undernutrition continues to be a major problem in India. These results underline the need for public health interventions that are aimed at reducing the burden of undernutrition. It should be noted that the observed relationships may change as the population makes the transition toward increased prevalence of obesity. Careful monitoring of this population will be necessary in order to alert the policymakers to future public health problems that are currently being observed in association with the obesity epidemic in developed countries.

Our study reported that tobacco use is a risk factor for low BMI. Further, smoking and low BMI had synergistic effect on mortality in men. We found that around 8% of male deaths could potentially be prevented by pursuing the elimination of both smoking and underweight. Smokeless tobacco use and underweight had synergistic effect in men and antagonistic effect in women on mortality. We found that around 5% of male and 11% of female deaths could potentially be prevented by pursuing the elimination of both risk factors. The results were independent of whether additiveness or multiplicativeness was assumed.

Tobacco use and undernutrition are known to be serious problems in India, and the present study indicates that obesity may soon emerge as a serious public health problem. However, the effect of obesity on mortality is subject to a large random variation in this study.

The policy implications for prevention would be that improving the nutritional status of those underweight and preventing use of tobacco results in the immediate highest yield.

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# 10. Appendices

## Appendix 1. Content of the baseline health survey of Mumbai Cohort Study

**TATA INSTITUTE OF FUNDAMENTAL RESEARCH**
**HOMI BHABHA ROAD, MUMBAI 400 005**

**A HEALTH SURVEY IN MUMBAI CITY**

INCLUDED IN THE VOTERS' LIST (Y/N)? _____
If "N" then ask, WHETHER RATION CARD HOLDER (Y/N)? _____
If "N" then, STOP.

DATE (automatically inputted from system clock)
TIME (automatically inputted from system clock)

INTERVIEWER'S NAME : ______________________________________

CARD NO. : ____________________

WARD NO. : _________________

ELECTORAL NO. : _________________

SERIAL NO. (from electoral roll) : ______________

INTERVIEWEE'S NAME : __________________________________________

SEX : MALE ☐ Female ☐

AGE: ☐☐ yrs.

ADDRESS : ______________________________________________________

_________________________________________________________________

SECOND ADDRESS (IN BOMBAY) : _____________________________

_________________________________________________________________

HEIGHT (in cm.) : ______________

WEIGHT (in kg.) : ______________

BLOOD PRESSUE : LOW ________ HIGH ________

PEAK FLOW : ________

DATE OF BIRTH : (Day) ☐☐ (Month) ☐☐ (Year) ☐☐

PLACE OF BIRTH : ______________________________

RELIGION : (H)indu, ☐ (M)uslim, ☐ (C)hristian, ☐

(B)udhist, ☐ (S)ikh, ☐ (O)thers ☐

MOTHER TONGUE : (Urdu) ☐ (Gujarati) ☐ (Hindi) ☐ (Tamil) ☐

(Kannada) ☐ (Malayalam) ☐ (Telgu) ☐ (Marathi) ☐

(Others) ☐

LEVEL OF EDUCATION : (I)lliterate ☐ (S)econdary, ☐

(P)rimary, ☐ (M)idle, ☐ (C)ollege ☐

AGE WHEN FIRST STARTED WORKING : ☐☐ yrs.

WHAT WAS YOUR FIRST OCCUPATION? ______________________

WHERE DID YOU WORK? __________________________________

AGE WHEN CURRENT OCCUPATION WAS STARTED : ☐☐ yrs.

WHAT IS YOUR CURRENT OCCUPATION? ______________________

WHERE DO YOU WORK? ___________________

DO YOU USE TOBACCO AT PRESENT? YES ☐ NO ☐

If YES, AGE WHEN TOBACCO USAGE STARTED : ☐☐ yrs
And go step 1

DID YOU USE TOBACCO IN THE PAST? YES ☐ NO ☐
If No, to step 13.

DID YOU STOP USING > 6 MONTHS AGO? YES ☐ NO ☐ UNSURE ☐

AGE WHEN TOBACCO USAGE STARTED : ☐☐ yrs.
AGE WHEN TOBACCO USAGE STOPPED : ☐☐ yrs.

1. HOW ARE/ WERE MATERIAL USED? APPLIED ☐ CHEWED ☐
APPLIED & CHEWED ☐

2. IS/ WAS BETEL LEAF CHEWED? YES ☐ NO ☐
3. IS/ WAS ARECA NUT USED? YES ☐ NO ☐
4. IS/ WAS CATECHU USED? YES ☐ NO ☐
5. IS/ WAS LIME USED? YES ☐ NO ☐
6. IS/ WAS TOBACCO USED? YES ☐ NO ☐

7. DO/ DID YOU INCLUDE ANY OTHER MATERIAL? YES ☐ NO ☐
If "Y" then, GIVE DETAILS : ________________________________

8. FREQUENCY PER DAY : ☐☐ DURATION OF HABIT : ☐☐ yrs.

9. DO YOU CHEW ANY OTHE COMBINATION? YES ☐ NO ☐
If YES, GIVE DETAILS : ________________________________

10. DO YOU APPLY MISHRI? YES ☐ NO ☐
If YES, FREQUENCY PER DAY : ☐☐ DURATION OF HABIT ☐☐ yrs

11. DO YOU APPLY CREAMY SNUFF? YES ☐ NO ☐
IF YES, FREQUENCY PER DAY : ☐☐ DURATION OF HABIT : ☐☐ yrs.

12. DO YOU APPLY ANY OTHER MATERIAL? YES ☐ NO ☐
If YES, GIVE DETAILS : ________________________________
FREQUENCY PER DAY : ☐☐ DURATION OF HABIT : ☐☐ yrs.

13. DO YOU SMOKE AT PRESENT? YES ☐ NO ☐
If YES, AGE WHEN SMOKING WAS STARTED : ☐☐ yrs.
go to step 16

14. DID YOU SMOKE IN THE PAST? YES ☐ NO ☐
If NO, go to step 19.

15. DID YOU STOP SMOKING > 6 MONTHS AGO? YES ☐ NO ☐ UNSURE ☐

AGE WHEN SMOKING WAS STARTED : ☐☐ yrs.

AGE WHEN SMOKING WAS STOPPED : ☐☐ yrs.

16. DO/ DID YOU SMOKE BIDI? YES ☐ NO ☐

If YES, FREQUENCY PER DAY ☐☐ DURATION OF HABIT : ☐☐ yrs.

17. DO/ DID YOU SMOKE CIGARETTE? YES ☐ NO ☐

If YES, FREQUENCY PER DAY ☐☐ DURATION OF HABIT : ☐☐ yrs.

18. OTHER FORM OF SMOKING : HOOKAH ☐ CHUTTA ☐ CLAY PIPE ☐

OTHERS ☐ NONE ☐

If NONE, go to step 19;

If HOOKAH/CLAY PIPE/CHUTTA, FREQUENCY PER DAY : ☐☐

DURATION OF HABIT : ☐☐

Otherwise GIVE DETAILS ____________________

REMARKS: ____________________________________________

# Appendix 2. Content of the follow-up health survey of Mumbai Cohort Study

## MUMBAI PROSPECTIVE STUDY

### PART I (FOLLOWUP INFORMATION)

1. INTERVIEWER'S NAME

2. ICARD

3. NUMBER OF VISITS

4. HAS ADDRESS BEEN LOCATED (Y/N)? (IF 'Y' GOTO Q6)

5. NOT LOCATED DUE TO ((I)ncomplete address/(D)emolished/(N)on traceable) **:: STOP**

6. WHETHER THE PERSON CAN BE IDENTIFIED (Y/N)?
   IF Q6 ="N" **:: STOP**

7. NAME

8. HAS THE NAME CHANGED (Y/N)? (IF Q8 = 'N' GOTO Q11)

9. REASON ((A)lias/(M)arried/(O)ther)

10. NEW NAME

11. ANY GIVEN INFORMATION WRONG (Y/N)? (IF Q11 = 'N' GOTO Q16)

12. ((A)ddress/(R)eligion/(M)other tongue) IS/ARE WRONG
    If "A" Q13 & repeat Q11
    IF "R" Q14 & repeat Q11
    IF "M" Q15 & repeat Q11

13. NEW ADDRESS (ADD)

14. NEW RELIGION (REL)

15. NEW MOTHERTONGUE (MT)

16. WHETHER THE PERSON IS ALIVE (Yes/No/Unknown) ?
IF Q16 ="Y" goto Q19
IF Q16="N" goto Q24

17. LAST DATE OF KNOWN STAY IN STUDY AREA (DD/MM/YY)

18. DATE IS ACCURATE (1-4)
1. ALL CORRECT
2. ONLY MONTH & YEAR
3. ONLY YEAR
4. NOT SURE

**:: STOP**

19. WHETHER RESIDING AT MENTION ADDRESS (Y/N)?
(IF Q19 = 'Y' GOTO Q34)

20. DATE OF MIGRATION (DD/MM/YY)

21. DATE OF MIGRATION IS ACCURATE (1-4)
1. ALL CORRECT
2. ONLY MONTH & YEAR
3. ONLY YEAR
4. NOT SURE

22. PLACE WHERE MIGRATED (1-4)
1. WITHIN OUR STUDY AREA
2. IN MUMBAI
3. WITHIN MAHARASHTRA
4. OUT SIDE MAHARASHTRA

If Q22= 1 ask Q23 Else **:: STOP**

23. ADDRESS WHERE MIGRATED (IF Q22='1')

**:: STOP**

24. DATE OF DEATH (DD/MM/YY)

25. DATE OF DEATH IS ACCURATE (1-4)
1. ALL CORRECT
2. ONLY MONTH & YEAR
3. ONLY YEAR
4. NOT SURE

26. PLACE WHERE EXPIRED (1-4) [IF Q26<>'1' GOTO Q32]
1. WITHIN OUR STUDY AREA
2. IN MUMBAI
3. WITHIN MAHARASHTRA
4. OUT SIDE MAHARASHTRA

27. NAME AS ENTER IN DEATH CERTIFICATE

28. ADDRESS WHERE EXPIRED

29. CAUSE OF DEATH

30. PLACE OF BURIAL OR CREMATION

31. DEATH REGISTRATION NUMBER
**:: STOP**

32. DATE OF LEAVING THE STUDY AREA (DD/MM/YY)

33. DATE IS ACCURATE (1-4)
1. ALL CORRECT
2. ONLY MONTH & YEAR
3. ONLY YEAR
4. NOT SURE

**:: STOP**

34. ABLE TO INTERVIEW (Y/N)?
(IF Q34 = 'Y' GOTO Q38)

35. REASON FOR NOT INTERVIEWING (Lock/Native place/Work/Hospital/Refused/Temporary lock)
(IF Q35 <> 'H')

36. CAUSE OF HOSPITALIZATION

37. NAME OF THE HOSPITAL
**::STOP**

**PART II (MEASUREMENTS)**

38. AGE (IN YRS.)

39. DO YOU HAVE ANY PROBLEM TO TAKE MEASUREMENT (Y/N)?
(IF Q39="N" goto Q43)

40. SPECIFY REASON

41. HEIGHT (IN CM. UPTO ONE DECIMAL '999.9' FOR INVALID)

42. WEIGHT (IN KG. UPTO ONE DECIMAL '999.9' FOR INVALID)

43. LOW BLOOD PRESSURE ('999' FOR INVALID)

44. HIGH BLOOD PRESSURE ('999' FOR INVALID)

**PART III (HABITS)**

45. DO YOU USE TOBACCO AT PRESENT (Y/N)?
(IF Q45 = ‘Y’ GOTO Q48)

46. DID YOU USE TOBACCO IN THE PAST (Y/N)?
(Q46 = ’N’ GOTO Q51)

47. DID YOU STOP USING TOBACCO< 6 MONTHS AGO

48. FORM OF TOBACCO USE (1-3)
1. SMOKING
2. SMOKLESS
3. BOTH
(IF Q48 = ‘2’ goto Q50 )

49. DO YOU SHARE SMOKING PRODUCT (1-3)
1. FREQUENTLY
2. OCCASIONLLY
3. NEVER

50. ANY CHANGE IN YOUR TOBACCO HABIT DURING LAST FIVE YRS
1. NO CHANGE
2. STARTED
3. INCREASED
4. REDUCED
5. STOPPED

51. DO YOU USE ALCOHOL AT PRESENT (Y/N)?
(IF Q51=’Y’ GOTO Q53)

52. EVER USED ALCOHOL IN THE PAST (Y/N)?
(IF Q52=’N’ GOTO Q56)

53. HOW FREQUENTLY DO/DID YOU DRINK (1-5)
1. LESS THAN ONCE A MONTH
2. ≤ 5 TIMES A MONTH
3. ≤ 3 TIMES A WEEK
4. 4–5 TIMES A WEEK
5. ≥ 6 TIMES A WEEK

54. TYPE (1-7)
1. WHISKY, 2. COUNTRY/DESHI LIQUOR, 3. SPIRIT (LEGAL) 4. BEER
5. OTHER IMFL, 6. TODDY, 7. OTHERS

55. QUANTITY (IN ML.)

56. DO YOU HAVE ANY HEALTH PROBLEM?
1. BLOOD PRESSURE (Y/N)?
2. HEART DISEASE (Y/N)?
3. CANCER (Y/N)?
4. TB (Y/N)?
5. ASTHAMA (Y/N)?
6. DIABETIES (Y/N)?
7. OTHER (Y/N)?

57. SPECIFY DISEASE (IF Q56 = 'OTHER')

**::STOP**

# 11. Original Publications

BMC Public Health

BioMed Central

Research article

Open Access

# Association between tobacco use and body mass index in urban Indian population: implications for public health in India

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## Abstract

**Background:** Body mass index [BMI, weight (kg)/height ($m^2$)], a measure of relative weight, is a good overall indicator of nutritional status and predictor of overall health. As in many developing countries, the high prevalence of very low BMIs in India represents an important public health risk. Tobacco, smoked in the form of cigarettes or bidis (handmade by rolling a dried rectangular piece of temburni leaf with 0.15–0.25 g of tobacco) or chewed, is another important determinant of health. Tobacco use also may exert a strong influence on BMI.

**Methods:** The relationship between very low BMI (< 18.5 kg/$m^2$) and tobacco use was examined using data from a representative cross-sectional survey of 99,598 adults (40,071 men and 59,527 women) carried out in the city of Mumbai (formerly known as Bombay) in western India. Participants were men and women aged ≥ 35 years who were residents of the main city of Mumbai.

**Results:** All forms of tobacco use were associated with low BMI. The prevalence of low BMI was highest in bidi-smokers (32% compared to 13% in non-users). For smokers, the adjusted odds ratio (OR) and 95% confidence interval (CI) were OR = 1.80(1.65 to 1.96) for men and OR = 1.59(1.09 to 2.32) for women, respectively, relative to non-users. For smokeless tobacco and mixed habits (smoking and smokeless tobacco), OR = 1.28(1.19 to 1.38) and OR = 1.83(1.67 to 2.00) for men and OR = 1.50(1.43 to 1.59) and OR = 2.19(1.90 to 3.41) for women, respectively.

**Conclusion:** Tobacco use appears to be an independent risk factor for low BMI in this population. We conclude that in such populations tobacco control research and interventions will need to be conducted in concert with nutrition research and interventions in order to improve the overall health status of the population.

## Background

Body mass index [BMI, weight (kg)/height ($m^2$)], a measure of weight adjusted for height, is a simple and inexpensive index that is often used as a proxy for overall health of populations. [1] Body habitus, as described by BMI, is related to skeletal size, muscle mass, and adiposity. As such, it is influenced by diet, other aspects of lifestyle, and other environmental factors. The association between low BMI and smoking is well documented. [2,3] However, recent studies show that the nature of this relationship depends on educational level, gender, ethnicity, and frequency (e.g., number of cigarettes/day) of smoking. Often, the relationship changes with time. In Finland, for example, the inverse association between smoking and BMI weakened between 1982–1987 and became positive later. [4] Educational attainment is known to influence the relation between smoking and BMI in North European populations. [5,6] In these populations, smoking is inversely associated with BMI at lower levels of education and positively associated with BMI at higher levels of education. A U-shaped relation between frequency of smoking and BMI has been observed by some investigators. [7]

In India, tobacco is used in various forms. [8] In addition to smoking cigarettes, bidis are commonly smoked, as they are much cheaper. Bidis are handmade by rolling a dried rectangular piece of temburni leaf (*Diospyros melanoxylon*) with 0.15–0.25 g of sun-dried, flaxed tobacco. In addition, use of smokeless tobacco, in a variety of forms, is widespread among both men and women. The most common form of smokeless tobacco use is mishri, a black powder obtained by roasting and powdering tobacco, which is then applied to the gums using a finger. Another common form is chewing of betel-quid, a combination of betel-leaf, areca nut, slaked lime, tobacco, and condiments; combinations of ingredients are altered according to individual preferences. Use of all forms of tobacco is associated with higher all-cause mortality in the Indian population. [9-11] Previously, we reported that tobacco using (smoking and smokeless) is associated with low BMI in an Indian population. [12] The high prevalence of tobacco use and its association with low BMI raises important questions about its impact on public health in India, a country which has a high prevalence of low BMI among adults. The focus of this paper is to provide a more detailed analysis of the relation between different forms of tobacco use and BMI and discuss the public health implications of these associations.

## Methods

The data presented in this report were obtained from a baseline cross-sectional survey conducted between 1992–1994 for a cohort study on tobacco-attributable mortality. [13] The survey was carried out in the main city of Mumbai, formerly Bombay – the largest city in India. A sampling frame was constructed from the electoral rolls. The sampling unit was a 'polling station,' consisting of 1000 to 1500 eligible voters. Rolls were assumed complete, as they are updated before every major election through house-to-house visits. Electoral rolls were organised by geographic areas. The selection of polling stations was done in a non-random manner to exclude those with apartments having high security, as it became evident during the pilot study that it would not be possible to gain access to these apartments.

Investigators approached all individuals aged ≥ 35 years (cut off chosen because of the overall goal of studying tobacco-attributable mortality in the cohort) listed in the selected polling stations for interview and anthropometric measurements. Individuals not present on the voters' list also were interviewed and included in the sample if their residence status was confirmed by their having a 'ration card.' These cards, issued by the Bombay Municipal Corporation, act as a proxy for residence cards and permit access to all city and state government services (including receiving certain food items at subsidized prices). Such individuals comprised about 5% of the sample. Less than 1% of individuals approached refused for interview and/ or allow anthropometric measurements to be taken. A total of 99,958 adults, 40,071 males and 59,527 females, were recruited and surveyed. The study satisfied all criteria of ethical treatment of human subjects; especially those formulated by the Indian Council of Medical Research.

The survey included two components: 1. measurement of height, weight, blood pressure; and 2. interviewer-administration of a structured questionnaire to obtain information on age, occupational history, education, religion, language, and tobacco-related behaviour. Weight was measured using a bathroom scale accurate to 0.5 kg. The scale was kept on a flat surface and the subject was requested to step on it in bare feet without holding on to anything. Subjects were measured in normal apparel, which in Mumbai is light cotton because of the tropical weather year around. The weight was recorded to the nearest kg. Height was measured using a specially constructed instrument consisting of a steel platform to which was attached a steel measure tape. With the subject standing erect on the steel platform, the tape was pulled vertically above the head, and then brought down to touch the flat ruler placed horizontally on the crown of the head. Height was recorded to the nearest cm.

All data were entered directly in a handheld computer in the field and transferred to a PC once a week in the Project office. Respondents were classified according to present and past tobacco use as (a) having never used tobacco, (b) ex-smoker, (c) ex-smokeless tobacco use, (d) ever smoker, (f) ever smokeless tobacco user, and (g) ever mixed habit (smokeless and smoking). Ever smokers and ever mixed users clubbed together were further divided into those who smoked i) cigarettes ii) bidis and iii) cigarettes + bidis. The smokeless-tobacco use further divided into -i)

**Table 1: Anthropometric values (mean ± SE of mean) by gender and tobacco use**

| | Men | | | | | Women* | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | *n* | *Age (years)* | *Height (cm)* | *Weight (kg)* | *BMI (kg/m²)* | *n* | *Age (years)* | *Height (cm)* | *Weight (kg)* | *BMI (kg/m²)* |
| **Never users** | 10493 | 49.6 ± 0.12 | 161.5 ± 0.07 | 59.0 ± 0.11 | 22.6 ± 0.04 | 23965 | 46.3 ± 0.07 | 148.9 ± 0.04 | 52.5 ± 0.07 | 23.7 ± 0.03 |
| Ex-users | | | | | | | | | | |
| Ex-smoking | 693 | 59.7 ± 0.48 | 161.8 ± 0.26 | 57.9 ± 0.44 | 22.0 ± 0.15 | - | - | - | - | - |
| Ex-smokeless | 824 | 58.4 ± 0.43 | 160.3 ± 0.25 | 57.2 ± 0.38 | 22.2 ± 0.13 | 1284 | 53.7 ± 0.34 | 147.9 ± 0.17 | 49.5 ± 0.30 | 22.6 ± 0.13 |
| Ex-mixed | 270 | 61.3 ± 0.73 | 160.3 ± 0.42 | 57.3 ± 0.74 | 22.2 ± 0.25 | - | - | - | - | - |
| **Ever users** | | | | | | | | | | |
| Smoking | 6017 | 50.8 ± 0.15 | 161.3 ± 0.09 | 55.8 ± 0.15 | 21.4 ± 0.05 | 159 | 54.5 ± 1.03 | 150.5 ± 0.63 | 48.8 ± 0.85 | 21.5 ± 0.34 |
| Smokeless | 18365 | 50.1 ± 0.08 | 160.5 ± 0.05 | 56.1 ± 0.08 | 21.8 ± 0.03 | 35296 | 49.3 ± 0.06 | 147.3 ± 0.03 | 48.0 ± 0.06 | 22.1 ± 0.02 |
| Mixed | 5194 | 50.1 ± 0.16 | 161.6 ± 0.09 | 55.2 ± 0.15 | 21.1 ± 0.05 | 107 | 51.9 ± 1.19 | 150.0 ± 0.74 | 47.3 ± 1.09 | 21.0 ± 0.44 |
| **Types of smoking**** | | | | | | | | | | |
| Cigarette | 4980 | 49.0 ± 0.16 | 162.6 ± 0.09 | 59.1 ± 0.16 | 22.3 ± 0.05 | 17 | 51.5 ± 2.20 | 157.6 ± 1.50 | 60.2 ± 1.48 | 24.3 ± 0.72 |
| Bidi | 5290 | 51.9 ± 0.16 | 160.2 ± 0.09 | 52.5 ± 0.14 | 20.4 ± 0.05 | 217 | 54.2 ± 0.90 | 149.7 ± 0.52 | 46.8 ± 0.73 | 20.9 ± 0.30 |
| Cigarette + bidi | 739 | 49.9 ± 0.44 | 162.2 ± 0.25 | 54.5 ± 0.40 | 20.7 ± 0.14 | 9 | 57.7 ± 3.86 | 153.0 ± 3.56 | 50.6 ± 2.43 | 21.7 ± 1.05 |
| **Types of Smokeless** | | | | | | | | | | |
| Mishri | 2724 | 48.9 ± 0.21 | 159.9 ± 0.13 | 56.4 ± 0.20 | 22.0 ± 0.07 | 15899 | 46.0 ± 0.08 | 147.7 ± 0.05 | 48.8 ± 0.08 | 22.4 ± 0.04 |
| Mishri + other | 7426 | 50.8 ± 0.13 | 160.0 ± 0.08 | 55.0 ± 0.12 | 21.4 ± 0.04 | 13401 | 52.5 ± 0.10 | 146.6 ± 0.05 | 46.5 ± 0.09 | 21.6 ± 0.04 |
| Betel quid | 4639 | 50.3 ± 0.17 | 161.1 ± 0.10 | 57.5 ± 0.16 | 22.1 ± 0.06 | 3844 | 50.8 ± 0.18 | 148.0 ± 0.10 | 49.3 ± 0.18 | 22.5 ± 0.08 |
| Other tobacco | 3234 | 49.1 ± 0.20 | 160.9 ± 0.12 | 56.4 ± 0.19 | 21.7 ± 0.07 | 942 | 50.0 ± 0.40 | 147.8 ± 0.21 | 47.7 ± 0.36 | 21.8 ± 0.16 |
| Areca nut | 342 | 51.7 ± 0.68 | 161.5 ± 0.36 | 59.4 ± 0.60 | 22.7 ± 0.20 | 1210 | 51.5 ± 0.35 | 148.0 ± 0.18 | 50.5 ± 0.33 | 23.0 ± 0.14 |

*Due to very small numbers of female ex-smokers and with ex-mixed habit the data are not included
**Ever smokers and mixed users were clubbed together

mishri ii) mishri + other forms of tobacco iii) betel quid iv) other tobacco and v) areca nut. For analysis of educational background, respondents were classified as (a) illiterate - received no education (b) primary - up to five years of education (c) middle - 6 to 8 years of education (d) secondary - 9 to 12 years of education (e) college - those who have received education past secondary level. Religion and mother tongue was used for analysis of cultural background. For mother tongue, the categories used were-Marathi, Hindi, Gujarati, Tamil, Urdu and other. For religion, the respondents were classified as Hindu, Muslim, Christian, Jew, Buddhist, and other.

Descriptive statistics (mean, standard deviation, and proportion within categories) were calculated for the total survey population and by tobacco use for both men and women separately. Because the number of ex smokers and cigarette smokers among women was small, separate analyses were not conducted on these two categories.

Multivariable analysis was performed using logistic regression. The response variable, BMI, was converted into a dichotomous variable by using two cut points: 18.5 and 25.0 kg/m². These are the conventional cut points indicating underweight and overweight, respectively. Three possible models corresponding to these two cut-off points were fit. Age (in 5-year age groups), education, mother tongue, religion and tobacco use were fit as independent variables in the final model. For dose response, the number of times (per day) tobacco was smoked, chewed or applied was refereed as frequency of habit per day and grouped as 1 to 5/day, 6 to 10/day and ≥ 11/day.

## Results

In general, non-users of tobacco had higher BMI values in this population (Table 1). In women, the few cigarette smokers represented (n = 17) had slightly higher BMI values and did never users. In both genders bidi smoking, appeared to be strongly associated with lower average BMI in this population. The difference between bidi smokers and those with never users of tobacco was more pronounced in women (2.8 BMI units lower than for subjects who did not use tobacco in any form) compared with men (2.2 BMI units lower than those with no habits). Often concern is directed at individuals who are at the extremes of the relative weight distribution.

Table 2 shows that the prevalence of low BMI (<18.5 kg/m²) was higher amongst all forms of tobacco users. Amongst smokers, the prevalence of low BMI was 2.5 times higher in bidi-smokers. However, the prevalence of underweight in cigarette smokers was not very different compared to non-tobacco users. Smokeless tobacco users, of all types had high prevalence of low BMI.

Table 3 gives the odds ratios for low BMI associated with tobacco habits controlling for age, education, mother tongue, and religion. All forms of tobacco use (except areca nut) were associated with higher risk of low BMI. Bidi-smoking was associated with the highest risk amongst all forms of tobacco use. In men, the odds ratio

**Table 2: Prevalence (%) of low and normal BMI by tobacco use with 95% confidence intervals**

| Tobacco use | Low BMI (<18.5 kg/m²) | | Normal BMI (18.5–25.0 kg/m²) | |
|---|---|---|---|---|
| | **Men (95% CI)** | **Women* (95% CI)** | **Men (95% CI)** | **Women* (95% CI)** |
| **Never users** | 13.5 (12.8 to 14.2) | 13.2 (12.8 to 13.6) | 62.4 (61.5 to 63.3) | 50.1 (49.5 to 50.7) |
| **Ex-users** | | | | |
| Smoking | 18.9 (16.0 to 21.8) | - | 61.0 (57.4 to 64.6) | - |
| Smokeless | 15.7 (13.2 to 18.2) | 19.2 (17.0 to 21.4) | 63.5 (60.2 to 66.8) | 51.9 (49.2 to 54.6) |
| Mixed | 17.8 (13.2 to 22.4) | - | 56.3 (50.4 to 62.2) | - |
| **Ever users** | | | | |
| Smoking | 25.2 (24.1 to 26.3) | 28.1 (21.1 to 35.1) | 58.8 (57.6 to 60.0) | 54.1 (46.4 to 61.8) |
| Smokeless | 19.9 (19.3 to 20.5) | 23.3 (22.9 to 23.7) | 62.1 (61.4 to 62.8) | 52.2 (51.7 to 52.7) |
| Mixed | 26.4 (25.2 to 27.6) | 35.1 (26.1 to 44.1) | 59.5 (58.2 to 60.8) | 47.9 (38.4 to 57.4) |
| **Types of smoking**** | | | | |
| Cigarette | 16.3 (15.3 to 17.3) | - | 60.8 (59.4 to 62.2) | - |
| Bidi | 32.1 (30.8 to 33.4) | 32.7 (26.5 to 38.9) | 57.9 (56.6 to 59.2) | 50.7 (44.0 to 57.4) |
| Cigarette + bidi | 32.6 (29.2 to 36.0) | - | 55.1 (51.5 to 58.7) | - |
| **Types of Smokeless** | | | | |
| Mishri | 17.1 (15.7 to 18.5) | 20.9 (20.3 to 21.5) | 64.1 (62.3 to 65.9) | 52.6 (51.8 to 53.4) |
| Mishri + other | 21.5 (20.6 to 22.4) | 26.6 (25.9 to 27.3) | 63.1 (62.0 to 64.2) | 52.3 (51.5 to 53.1) |
| Betel quid | 17.7 (16.6 to 18.8) | 21.4 (20.1 to 22.7) | 61.0 (59.6 to 62.4) | 50.9 (49.3 to 52.5) |
| Other tobacco | 20.0 (18.6 to 21.4) | 27.0 (24.2 to 29.8) | 61.2 (59.5 to 62.9) | 50.4 (47.2 to 53.6) |
| Areca nut | 12.3 (8.8 to 15.8) | 17.2 (15.1 to 19.3) | 63.7 (58.6 to 68.8) | 52.1 (49.3 to 54.9) |

*Data for female ex-smokers and cigarette not analysed because of small numbers
**Ever smokers and mixed users were clubbed together

for low BMI associated with bidi smoking was about twice as large compared with cigarette smoking. For smokeless tobacco use, the risk was greater in women compared with men.

**Table 3: Adjusted odds ratio for low BMI (< 18.5) by tobacco use* (Referent category was non-tobacco users with normal BMI, 18.5–25.0)**

| Tobacco use | OR (95% CI) † *Men* | *Women* |
|---|---|---|
| **Never users** | 1.0 | 1.0 |
| **Ever users** | | |
| Smoking | 1.80 (1.65, 1.96) | 1.59 (1.09, 2.32) |
| Smokeless | 1.28 (1.19, 1.38) | 1.50 (1.43, 1.59) |
| Mixed | 1.83 (1.67, 2.00) | 2.19 (1.40, 3.41) |
| **Types of smoking**** | | |
| Cigarette | 1.22 (1.11, 1.35) | - |
| Bidi | 2.36 (2.16, 2.58) | 2.01 (1.48, 2.74) |
| Cigarette + bidi | 2.64 (2.22, 3.13) | - |
| **Types of Smokeless** | | |
| Mishri | 1.18 (1.05, 1.33) | 1.41 (1.32, 1.50) |
| Mishri + other | 1.38 (1.26, 1.51) | 1.72 (1.61, 1.83) |
| Betel quid | 1.23 (1.12, 1.36) | 1.46 (1.33, 1.60) |
| Other tobacco | 1.37 (1.23, 1.53) | 1.80 (1.53, 2.11) |
| Areca nut | 0.89 (0.64, 1.25)$^{NS}$ | 1.18 (1.00, 1.40)$^{NS}$ |

*Results are controlled for age, educational status, mother tongue and religion
†All ORs were significant at $p < 0.0001$ except the one marked $^{NS}$
**Those who smoked and used smokeless tobacco were counted as smokers

Table 4 shows a dose-response gradient in the tobacco use-BMI relationship. The dose-response was significant for smoking as well as smokeless tobacco use among men as well as women.

Table 5 explores the dose-response relationship further with low BMI divided into three categories: < 16.0; 16.0–17.0; 17.0–18.5 with frequency of smoking among men and smokeless tobacco use among women. A clear gradient in odds ratios is seen for almost every row and every column.

On comparing adjusted odds ratios with unadjusted odds ratios (data not shown), the adjusted odds ratios were always smaller indicating that controlled variables were confounders. The differences however, were not large; the highest reduction in odds ratio was from 2.79 to 2.19 for women with BMI < 16.0 and frequency of smokeless tobacco use > 10 per day; suggesting that there was little possibility of residual confounding affecting the results.

## Discussion

In India, nearly half of all rural adults and a quarter of urban adults have a low BMI (i.e., <18.5 kg/m²). [14]. Although, chronic energy deficiency due to inadequate diet may be the main factor placing the population at risk of low BMI, factors other than diet may play a significant role in explaining the low BMI within this population. These factors may act directly (by affecting appetite or other aspects of physiology) or indirectly (by decreasing the purchasing power for food). From a public health perspective, unmasking these non-dietary determinants of low BMI in the population will help in understanding the impact of such exposures. The present study has found that all forms of tobacco use are associated with low BMI independent of (i.e., after accounting for) age, education, mother tongue, and religion in this population. Further, there exists a dose-response gradient and the response at every dose was higher in women compared with men, a relationship observed in many tobacco-health disease analysis, including cancer related end points [15,16]. This finding raises important questions about the magnitude of the adverse impact of tobacco use on the health status of the population.

### *Tobacco use among the socio-economically disadvantaged communities*

Previously we have reported that illiteracy is an independent risk for low BMI in this population (OR = 6.52; 95% CI 5.38 to 7.89 for men and OR = 4.83; 95% CI 3.71 to 6.28 for women, respectively). [12] In Mumbai, the prevalence of tobacco use (especially bidi smoking and chewing) is inversely related to education (a good proxy for poverty in this population). [13] Bidi smoking is more common than cigarette smoking among the illiterate in Mumbai. This is true for all of the South Asia where the prevalence of bidi-smoking is reported to be 21–56% among men. [17]

In the present study, we found bidi- smoking was associated with the highest risk of low BMI (adjusted for education). All forms of tobacco produce free radicals that deplete antioxidants like Vitamin C, E and carotenoids and cause oxidative damage to DNA, proteins and lipids. [18-21] Concentrations of nicotine, tar and other toxic agents are higher in bidis than cigarettes and bidi smoking has a greater physiological and biochemical effect than cigarette smoking. [9,17]

Antioxidant-rich foods such as green-leafy vegetables and fruits that may help reduce the oxidative stress caused by tobacco [22] are usually lacking in the diet of the poor [23]. This makes them more vulnerable to tobacco-induced oxidative stress with more damaging effects than in a well-nourished population. [24] Our studies, conducted in three different parts of rural India, indicate relatively low intakes of antioxidant nutrient intake among smokers [25-27], calling attention to this as a widespread public health concern. In this population, infectious agents and pollution are the other environment factors that may play a role in this interaction. Tobacco use [28-30] and poor nutrition [31] impair the immune system. Hence, tobacco users are more susceptible to infectious agents. This has been demonstrated for the relationship between pulmonary tuberculosis (TB) and smoking. [32,11] Smoking has been associated with higher relative risk of TB mortality and prevalence of active TB. This was true in both rural (RR 4.2, 95% CI 3.7 to 4.8) and urban (RR 4.5, 95% CI 4.0 to 5.0) India. [11] The risk was higher for bidi-smoking, the predominant smoking habit in this population. Thus, there is strong evidence that tobacco use in this population contributes to an increased burden of infectious disease. On the other hand, infections will further increase oxidative stress in tobacco-users. Hence, the interactions between malnutrition, tobacco use and infections make this group more vulnerable to smoking-related mortality and morbidity.

Besides the direct physiological effect, tobacco use among the economically disadvantaged is known to reduce the resources available to purchase food, clothing, health, and education, all factors that contribute to poor nutritional status. [33] This explains why changes in the relationship between BMI and smoking change with the secular trend toward affluence [4].

### *Tobacco use among women of reproductive age*

Smoking is not yet very common among Indian women. However, smokeless tobacco use among women is high. In our survey population 59% of women used smokeless tobacco. This is similar to that reported in other South Asian female populations. Some 49% of the UK- Bangladeshi female population and 59% of rural Malaysian females use smokeless tobacco. [34,35] In addition to causing oral cancer, smokeless tobacco use may be associated with increased risk of osteoporosis [36] and breast cancer. [37] In this study, we found that smokeless tobacco use is associated with a greater risk of low BMI in women compared to men. A similar increased risk for cancer from smokeless tobacco use was reported in women compared with men. [38] This may be a result of gender differences in the biology and/or nutritional status. Although the findings of this paper have implications for women of all ages, low BMI in women of reproductive age in the developing countries is associated with poorer reproductive outcomes. [39]. Smokeless tobacco use is associated with lower birthweight [40-43] and decreases gestational age at birth [43] in India. A prospective study on maternal determinants of low birth weight in India found that intake of micronutrient-rich foods was an important limiting factor for fetal growth independent of maternal age, height, and weight. [44] Hence, depletion of antioxidant micronutrients by the toxic agents in smokeless tobacco may be playing a role the biology of fetal growth restriction by smokeless tobacco. India has the highest prevalence and largest absolute share of low birth weight in the world. [45] Therefore, it would be important to estimate the contribution of the singular and combined effect of tobacco use, low intake of micronutri-

**Table 4: Adjusted odds ratio* for low BMI (<18.5)by frequency of tobacco use (Non tobacco users with normal BMI 18.5–25.0 were referent category)**

| Tobacco use | OR (95% CI)† Men | Women |
|---|---|---|
| Never users | 1.0 | 1.0 |
| **Frequency of use per day** | | |
| Smoking | | |
| 1–5 | 1.31 (1.17 to 1.45) | 1.50 (1.01 to 2.21) |
| 6–10 | 1.81 (1.62 to 2.01) | 2.02 (1.04 to 3.90) |
| ≥ 11 | 2.11 (1.93 to 2.30) | 2.20 (1.28 to 3.79) |
| Smokeless | | |
| 1–2 | 1.25 (1.13 to 1.38) | 1.46 (1.38 to 1.55) |
| 3–5 | 1.39 (1.28 to 1.52) | 1.59 (1.50 to 1.69) |
| 6–10 | 1.25 (1.14 to 1.38) | 1.72 (1.58 to 1.88) |
| ≥ 11 | 1.40 (1.20 to 1.62) | 2.08 (1.77 to 2.45) |

*Results are controlled for age and level of education
† All ORs and trends were highly significant (p < 0.0001)

ent- rich foods, and low maternal BMI to the burden of low birth weight in India.

### *Policy implications*

There should be no question that a major emphasis of any competent public health programs should be on preventing youth from starting to use tobacco and in supporting tobacco cessation efforts among those already addicted. Also, it is known that tobacco effects dietary requirements. For example, in the UK, the recommendations for vitamin C intake for smokers is higher (80 mg/day) compared to non-smokers (40 mg/day). [46] Thus far, nutritional recommendations for the Indian population have not distinguished between tobacco nonusers, or users of tobacco in any form. [47] Doing so would help to highlight both the scientific issues and be socioeconomic implications of creating even more stringent requirements for more expensive food, such as thoses rich in antioxidants.

### *Limitations*

The results in this paper are from a cross-sectional study so all limitations of cross-sectional data apply to them. In cross-sectional studies, exposure and outcome are assessed at the same time point, and one may not be sure which one came before the other? In the present data set there does not appear any specific reason to suggest that those with low BMI were more prone to start using tobacco. Cross sectional study results may also be affected by differential mortality rates in different subgroups of exposure and outcome. In our study the highest mortality would be most probably in low BMI – tobacco user group and lowest in normal BMI – non tobacco user group. Both these groups contribute to the numerator of odd ratio so the bias if any, is not unidirectional. Another limitation may seem to be the fact that we have not focused on overweight (BMI >25). This is however deliberate; the inverse association between overweight and tobacco use is well established and the same was reported in current data set. [12] The overweight category (BMI > 25.0) was excluded from the referent category (18.5 – 25) to guard against artificial inflation of odds ratios. '

## Conclusion

The effects of tobacco use on the incidence of certain diseases, particularly cancers of the aerodigestive tract and urinary bladder are well documented. The findings of this study that all forms of tobacco use are associated with low BMI (a proxy for nutritional status) suggest a strong need for further research on as tobacco use may have even more far-reaching public health implications in India than previously thought. If the association is evaluated as causal then tobacco control research and intervention also will benefit other public health goals on improved nutritional status and consequential health benefits,. These results have potential to affect the population living in the developing world.

**Table 5: Adjusted odds ratio* for three categories of low BMI and frequency of tobacco use(Non-tobacco users with normal BMI 18.5–25.0 were referent category)**

| Frequency of tobacco use/day | | | |
|---|---|---|---|
| **Smoking** (Men) | Mild thinness (BMI 17.0–18.5) | Moderate thinness (BMI 16.0–17.0) | Severe thinness (BMI<16.0) |
| 1–5 | 1.21 (1.06 to 1.39) | 1.37 (1.13 to 1.68) | 1.45 (1.19 to 1.77) |
| 6–10 | 1.68 (1.46 to 1.93) | 1.93 (1.58 to 2.35) | 1.97 (1.62 to 2.39) |
| > = 11 | 1.86 (1.66 to 2.09) | 2.22 (1.88 to 2.62) | 2.54 (2.17 to 2.98) |
| **Smokeless** (Women) | | | |
| 1–2 | 1.32 (1.22 to 1.43) | 1.50 (1.34 to 1.68) | 1.72 (1.55 to 1.90) |
| 3–5 | 1.46 (1.35 to 1.57) | 1.71 (1.53 to 1.91) | 1.76 (1.59 to 1.95) |
| 6–10 | 1.59 (1.42 to 1.78) | 1.63 (1.38 to 1.92) | 2.05 (1.78 to 2.35) |
| > = 11 | 1.84 (1.48 to 2.29) | 2.46 (1.87 to 3.24) | 2.19 (1.70 to 2.84) |

* Results are controlled for age and level of education

## Competing interests

The author(s) declare that they have no competing interests.

## Authors' contributions

MS Pednekar conceptualized and conducted all data analysis presented here and participated in preparation and finalisation of the article. PC Gupta, HC Shukla, and JR Hebert participated in all aspects of the conceptualization and preparation of the article. All authors read and approve the final manuscript.

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