Chapter 8

Overweight and obesity (high body mass index)

W. Philip T. James, Rachel Jackson-Leach, Cliona Ni Mhurchu, Eleni Kalamara, Maryam Shayeghi, Neville J. Rigby, Chizuru Nishida and Anthony Rodgers

Summary

It is widely acknowledged that being overweight is associated with an amplified risk of disease, particularly if body fat is deposited within the abdomen, as suggested by a high waist-circumference measurement. This chapter aims to estimate the burden of disease attributable to overweight and obesity as indicated by a high body mass index (BMI), by age, sex and subregion.[1]

BMI, which is calculated as weight (kg) divided by height squared (m^2), was chosen as a simple measurement of body weight in relation to height. While increases in both body fat and lean tissue cause increments in BMI, relationships between body weight and health are conventionally expressed in terms of BMI rather than body fat. Data on population weight and height, often collected as part of general medical or economic surveys, were obtained, typically from specially-commissioned analyses from ministries of health. Where these data sets or published representative information were lacking, earlier data published for each country were used. All information based on studies of select groups within a population were excluded. In addition, only data obtained by actual measurement of heights and weights by trained observers were included. As data were not available for some countries, it was necessary to extrapolate from data for other countries or subregions when deriving estimates of BMIs for the different age groups in each subregion.

Analyses of the relationship between BMI and both mortality and morbidity suggested that the theoretical optimum mean population BMI was approximately 21 kg/m^2. This value is far removed from those now found in many parts of the world. The analyses based on this continuous relationship therefore replaced the usual categorical analyses based on rates of overweight and obesity in the different subregions.

The disease outcomes assessed in relation to excess weight were type II diabetes (diabetes mellitus), ischaemic heart disease, stroke, hypertensive heart disease, osteoarthritis, and cancers of the postmenopausal breast, colon, endometrium and kidney. As it was evident that adult BMIs of >21 kg/m^2 were associated with the development of disease, the burden of disease attributable to high BMI was calculated from this baseline. New analyses based on 33 cohort studies carried out within the Asia-Pacific region were used to estimate the incremental risk of cardiovascular disease associated with each unit increase in BMI above 21 kg/m^2. The relationship between BMI and the risk of type II diabetes was derived from both unpublished and published data comprising measured anthropometry and fasting blood sugar measurements, extracted from nationally representative studies. Equivalent increments in the risks of co-morbidities associated with body-weight gain were assumed for all parts of the world.

High mean BMIs and elevated rates of overweight and obesity were found in the Americas, Europe, the Middle East and in the Western Pacific. It is estimated that rates of obesity vary geographically from 2–3% in some Asian countries to 75% in several Pacific Island nations. Currently, there are more than 300 million obese and more than 750 million overweight individuals in the world.

The proportions of the global burden of disease attributable to increases in BMI were 58% for type II diabetes, 21% for ischaemic heart disease, 39% for hypertensive disease, 23% for ischaemic stroke, 12% for colon cancer, 8% for postmenopausal breast cancer and 32% for endometrial cancer in women, and 13% for osteoarthritis. This means that the global burden of disease attributable to excess BMI in adults amounted to more than 30 million disability-adjusted life years (DALYs) in 2000, mostly incurred from ischaemic heart disease and type II diabetes. There were two and a half million deaths associated with this exposure. These are average global figures and there are remarkable variations by subregion and by disease. Thus EUR-C has the greatest burden of DALYs, this being dominated by the impact of high BMI on ischaemic heart disease, whereas the two African subregions have the lowest burden of DALYs. The burden of diabetes attributable to high BMI is greatest in WPR-B and AMR-B, with AMR-A also having a substantial burden. DALYs attributable to stroke were also dominated by the impact of high BMIs in both EUR-C and WPR-B, while the burden of DALYs caused by cancer was substantial in the European subregions, AMR-A, AMR-B and WPR-B.

Current trends were used to predict the increases in BMI and disease burden that are likely to occur by 2030, assuming that no new measures are taken to counteract the rapid recent increases in body weight in all parts of the world. On this basis, it is predicted that the burden of disease will increase substantially in most parts of the world, but there will probably be remarkable variations by subregion.

1. Introduction

Although the measurement and analysis of body weights and heights have been recognized as general indices of health for many years, it is only comparatively recently that the World Health Organization (WHO) has set out criteria for assessing underweight and overweight in both children and adults (WHO 1995). These new analyses of the impact of excess body weight came from insurance data generated in the first half of the 20th century which were used to identify optimum weights-for-height above which life expectancy was reduced, for both men and women. In the second half of the 20th century, it became clear that abnormalities in blood lipids relating to the risks of ischaemic heart disease were amplified by excessive body-weight gain, as was the risk of high blood pressure, type II diabetes, gallbladder disease and some cancers. It also became clear that the mechanical impact of excess body weight induced breathlessness and promoted arthritis in the weight-bearing joints. In developed countries, overweight women were stigmatized, with marked consequences on their sense of well-being, social interactions and even their employment and marriage prospects.

The traditional concerns of governments and policy-makers have focused on undernutrition, with greater emphasis being placed on the continuing problem of childhood protein–energy malnutrition, which is found especially in children aged 0–4 years. This condition is still prevalent in many countries despite economic progress (James et al. 2000), as described in chapter 2. Many nations now have reasonable systems for monitoring children's growth and can provide estimates of the prevalence of stunting, wasting and overweight in children aged <5 years (de Onis and Blössner 2000). Unfortunately, the value of monitoring the weights and heights of older children and adults has not been appreciated until fairly recently. Since the 1997 WHO Expert Consultation on Obesity (WHO 2000), there has been a substantial increase in the number of publications presenting newly-analysed data from past studies in different parts of the world. Thus, the regular national NHANES surveys in the United States of America (Stevens et al. 1999) allowed the magnitude of the problem of overweight to be recognized, and many cardiovascular surveys, for example the WHO MONICA surveys (Dobson et al. 1998), also documented high prevalences of overweight and obesity in Europe and Australasia. Other surveys such as the INTERSALT study (Dyer and Elliott 1999) revealed high prevalences of excess weight in some developing countries, including Brazil (James and Francois 1988). The data presented by many of these studies are not representative and do not include validation of the measurements of height and body weight. Nevertheless, it is apparent that many new national surveys are now being undertaken and a much more extensive database is expected to become available within the next few years.

This chapter was based on an extensive search of the literature to identify appropriate data sets and also specifically-commissioned analyses provided by a number of individuals, organizations and governments.

2. Choice of exposure variable

2.1 Definitions of body weight and of risk factors

The use of the BMI

The present analysis is based exclusively on the use of the BMI, which is calculated as weight (kg) divided by height squared (m^2). The height and weight of both children and adults are crude indices of the impact of many environmental factors, (including diet and infections) on the genetic growth potential of the individual over short and long periods of time, and affect many health outcomes.

BMI is the most appropriate simple indicator by which weight-for-height can be related to health outcome. WHO (1995) therefore proposed the use of BMI to monitor both undernutrition and overweight. The power of height is taken as 2.0 although it has been shown in many analyses that 1.5 might be more appropriate for women on the basis that this index in population studies proves to be approximately height-independent (Micozzi et al. 1986). Nevertheless, international convention, as represented by two major WHO Technical Consultations (WHO 1995, 2000), endorsed the use of a common BMI scheme for adults irrespective of sex or age.

Preliminary analyses of the global burden of disease associated with higher BMI, based on the current data sets, suggested that the population distribution of BMI values for men and women in each age group provided more valuable information than simply the proportions of the population who are classified as overweight and obese. These categories of overweight and obesity are used extensively by clinicians for patient management decisions, by the public and by policy-makers. Therefore, the proportions of overweight and obese people in the population are included in this chapter despite the fact that this information was not used in the calculation of the contribution of different values of BMI to the disease burden.

In these subsidiary analyses, the standard WHO BMI categories were used, except that the term "overweight" was taken as referring to BMI values of 25.0–29.9 kg/m^2 only and did not include the "obese" category, i.e. BMI of $\geq$30 kg/m^2, since these two groups, overweight and obesity, are often referred to independently. More extreme categories of obesity have been specified (WHO 2000), but were not included in the current global analyses.

Recently, it has been proposed that a lower BMI range of "healthy", "normal" or "acceptable" weights should be applied to groups of Asian

people (see below), but in the current analysis an assessment was made of the impact of increments in BMI on disease risk in different parts of the world, which was therefore not dependent on different schemes for categorizing overweight and obesity.

Body weight in children

It has become increasingly common for epidemiologists to express heights and weights of children in terms of the same BMI as used in adults, despite detailed analyses showing that BMI varies by age and sex during growth. Criteria have therefore now been developed for specifying the normal weight-for-height of children in terms of BMI for each age group, by sex, until adult height is achieved at approximately 18 years of age. There are three approaches to the categorical analysis of BMI in children: the traditional approach whereby an "abnormal" group is taken to be more than two standard deviations from the mean, the new International Obesity Task Force (IOTF) approach that relates BMI categories in childhood to the accepted classification in adults (Cole et al. 2000), and a new Centers for Disease Control and Prevention (CDC) set of standards whereby obesity is specified as >95th BMI percentile of carefully selected representative data from the United States (Ogden et al. 2002).

It is recognized that children of similar body proportions but of different heights at the same age will have different BMI values and that to obtain height-independent indices would require a sequential adjustment in the power value of height from about age 5 years upwards (Franklin 1999). Nevertheless, given that population comparisons are being made here, rather than the monitoring of the growth of individuals, weights and heights for children have been expressed in terms of BMI and these calculations have been applied only to children aged 5–18 years. The large body of nationally representative data for children aged <5 years collated by WHO and presented in chapter 2 provides the relevant information for this age group. However, the burden of disease estimates presented here are only for adults aged ≥30 years. This age limit was chosen because there are as yet insufficient prospective studies of an appropriate magnitude in children and young adults to allow quantitative analyses of the impact of excess weight gain on the incidence of noncommunicable diseases in individuals aged <30 years.

2.2 Other exposure determinants

Body fat

It is often assumed that health-related data would ideally be related to good measures of body fatness, and that the combination of weight and height in the form of BMI provides a crude index of body fatness. In practice, however, too few studies have measured body fatness and health outcomes at different ages and in different societies to allow an analysis

of whether a more specific measure of body fat than BMI would give greater predictive power for health outcomes. Given the many prospective studies that use BMI, the current convention has been maintained while recognizing that recent data show that different ethnic groups have substantially different proportions of body fat at the same value of BMI. For example, the ratio of fat : lean tissue is highest in Indian people, while values for Chinese people are intermediate between those for Indians and Caucasian peoples (Deurenberg et al. 2002), and Polynesians are increasingly recognized as having a relatively high proportion of lean tissue (Swinburn et al. 1999). On this basis, Deurenberg and colleagues have suggested that different BMI values should be chosen if the intention is to standardize international comparisons on the basis of body fat (Deurenberg et al. 1998, 2002). There are, however, no international studies as yet which would allow all population groups to be set a particular BMI value based on their fat : lean tissue ratios and the relationship of these indices to health outcomes.

Therefore, this chapter maintains the current convention of using BMI as an indicator of body fatness in adults. It is recognized that not only do women have substantially more fat tissue than men at equivalent BMIs (Shetty and James 1994), but also that both men and women lose lean tissue during the course of their adult lives such that, at an equivalent BMI, a 75-year-old man or woman has substantially greater proportion of fat than a 25 year old (James et al. 1988). This is not a cohort effect since the same changes have been shown, at least in men, in the Baltimore study of ageing, which evaluated the changing body composition of the same men over a 50-year period (James et al. 1989).

Corrections for unusual body proportions

The proportions of the major body parts which contribute to height may be different in different ethnic groups. For example, some African tribes are considered to have exceptionally long legs, whereas the indigenous populations of Central America are often cited as being small with very short legs (Norgan 1994a). It can readily be shown that even if the proportions of both the trunk and legs are equal in very short and tall peoples, the actual BMIs of these peoples will be very different. This has led Norgan (1994b) to develop a simple correction for BMI measurement based on the ratio of sitting height to total height. Although this is well-recognized (WHO 1995) and is valuable when looking at particular groups, the limited availability of good data on sitting height meant that it was not possible to incorporate this correction for BMI into the current analyses.

Waist circumference

Originally it was hoped that sufficient data would become available on waist circumference to allow an assessment of the usefulness of this measure in predicting the health of different communities. There are

many analyses that demonstrate that waist circumference provides a reasonable indicator of the quantity of abdominal fat, which correlates with the amount of intra-abdominal or visceral fat (Despres et al. 2001). This fat is considered to be metabolically rather different from subcutaneous fat in its responsiveness to dietary change and in its array of metabolite and hormonal outputs. An excess of abdominal fat has been associated with a range of metabolic abnormalities and diseases (Despres et al. 2001). The measurement of waist circumference is often found to be more valuable than BMI itself, for example, in predicting the likelihood of ischaemic heart disease (Lapidus et al. 1984; Larsson et al. 1984) or diabetes (Chan et al. 1994). The National Institutes of Health (NIH) report from the United States (NIH 1998) used waist circumference measurements as a suitable indicator of additional risk within a given range of BMI. In some studies, there seems to be additional predictive power when the waist:hip ratio rather than just waist circumference is used. The hip measurement indicates the degree of fat accumulation around the hips and this deposition may help in some way to limit the health impact of abdominal fat accumulation (Seidell et al. 2001b). Nevertheless, there seems to be increasing acceptance that, for general use, a single measure of waist circumference provides a simple index of fat distribution and additional risk (Seidell et al. 2001a).

Proposals have been also made for lower cut-off points for measurements of waist circumference for use in Asian communities (WHO/IASO/IOTF 2000) and new Chinese analyses have also proposed different values (Zhou 2002). There is also now increasing evidence that many communities, e.g. African and Hispanic Americans in the United States, Indians in India and elsewhere, the Chinese and Latin Americans have a greater propensity as adults to accumulate excess adipose tissue in the abdominal area than Caucasians in Europe or the United States (Ford et al. 2002; Sánchez-Castillo et al. 2003; Sargeant et al. 2002; Singh et al. 1995; Zhou et al. 2002). Although the selective accumulation of abdominal fat is indicative of a much greater risk of diabetes, hypertension, ischaemic heart disease, strokes and gall bladder disease, cross-sectional studies of the African diaspora in West Africa, the Caribbean and the United States show that the relationship of waist circumference to disease seemed to vary by region, perhaps because of concomitant regional dietary differences (Okosun et al. 1998, 2000). Nationally representative data and long-term cohort studies of the health impact of different indices of abdominal obesity in different communities are also currently insufficient to allow the use of some measures of waist circumference to estimate the BMI–disease relationship in different parts of the world.

The propensity to abdominal obesity within a community seems to be markedly influenced by stunting or a small size in childhood (Schroeder et al. 1999) and also by size at birth (Barker 1998). Changes in the hypothalamic–pituitary–adrenal axis controlling pituitary hormone and

corticosteroid metabolism in response to fetal nutritional deprivation and early postnatal events are also evident experimentally (Seckl et al. 2001) and abdominal obesity is associated with abnormal control of corticosteroid metabolism (Björntorp and Rosmond 1999). Evidence from India shows that children aged 4 and 8 years who were born small and later showed accelerated growth had a propensity to abdominal obesity with greater insulin resistance and higher blood pressure (Yajnik 2000). The current data available on a global basis do not, however, allow a systematic adjustment of health risk based on birth weights in different parts of the world, or the prediction of childhood BMIs from infant birth weights. A substantial proportion of the world's population that has been existing on marginal diets for centuries may have been sensitized to excess body-weight gain, this being reflected in the greater propensity to accumulate abdominal fat and in the higher prevalence of the metabolic syndrome of multiple risk factors for chronic diseases of adults such as diabetes, hypertension and ischaemic heart disease in Hispanic and non-Caucasian ethnic groups (Ford et al. 2002).

3. Methods of identifying sources and studies

3.1 Studies of interest

Studies of interest were identified using the following methods:

- Searches of the Medline and Embase databases were conducted systematically for all 191 countries of the world. Medline searches were performed with the keywords "BMI" and "obesity", each paired with "cardiovascular disease", "hyperlipidaemia", "cholesterol", "stroke", "ischaemic heart disease", "osteoarthritis", "diabetes mellitus type II", "cerebrovascular disease", and in combination with each country name, i.e. 2 x 8 x 191 searches. *Example*: BMI AND cardiovascular disease AND country X. Both United Kingdom and American English spellings were used in the searches. Embase searches were performed with the keywords "BMI", "obesity", "body mass", "body height", "weight", "children", and "adults". Countries were not specified in these searches.
- IOTF contacted each WHO Regional Nutrition Officer to request help with the analyses. The precise format for these was specified and each region was asked to help identify appropriate contacts from whom reliable national data on both BMI and diet could be obtained.
- Numerous direct contacts were made with governments and individuals to determine whether unpublished data were available. It is significant that with obesity now becoming a high profile issue throughout the world, many investigators, on learning of this

project, stated that they now wished to publish in their own right information on prevalence rates that had remained unpublished for several years.

- Relevant data sets were retrieved from online databases, or purchased and re-analysed. These included a United States Agency for International Development (USAID)-sponsored series of Demographic and Health Surveys (DHS) conducted by Macro International, the United States National Health and Nutrition Examination Survey (NHANES) III, and the 1998 Health Survey for England.
- Analyses of 33 cardiovascular cohort studies being conducted in the Asia-Pacific region and participating in the Asia-Pacific Cohort Studies Collaboration (APCSC) were also used to derive the relative risks of cardiovascular disease associated with increases in BMI.

For data from the identified studies, the following inclusion criteria were used.

- Nationally representative data were preferred.
- Clinical data were excluded whenever possible because they reflect a subgroup of the population with particular medical problems and could not be considered nationally representative.
- Only measured anthropometric data were used to assess the national information on BMI. A good correlation between measured and reported weights and heights can be found (Flegal and Troiano 2000), but many international analyses of reported vs measured heights and weights, including those from the United States, reveal discrepancies which underestimate weight and overestimate height, these discrepancies being particularly apparent in the groups of overweight people (see Niedhammer et al. 2000). Australian analyses have also shown that there can be very substantial differences in the prevalences of overweight and obesity as judged by the two approaches (Anonymous 1999). Nevertheless, for prospective analyses, the overall associations between reported weights and heights and health outcomes are unlikely to be seriously affected, even if the magnitude of the association becomes more uncertain. Therefore, some results of major studies employing self-reported weights and heights were used to illustrate disease relationships (although the quantitative associations used in estimates of impact on the health of the population are all derived from studies employing actual measurement of BMI). Given the levels of inaccuracy and bias associated with self-reported BMI, the use of such data was considered inappropriate for the purpose of estimating exposure. A small bias could have a substantial impact on the estimated prevalences of overweight and obesity in the tail of the BMI distribution. This criterion excludes many high profile publications that rely on self-reported weights and heights, particularly from

European Union surveys, and a large number of studies from the United States.

- A sufficiently large sample size was required, with preference being given to studies investigating ≥1000 individuals. For countries with no data, studies with smaller samples were not excluded.
- The earliest cut-off date for data collection was 1990, whenever possible. Where no suitable studies were available, studies dating from 1980 onwards were considered.
- For diabetes, only representative population measures of fasting plasma glucose were used. Ideally, representative data from children and adults tested with a standard glucose load are desirable for a full assessment of the prevalence of diabetes in relation to BMI, but there are very few studies with nationally representative data available from developing countries. No studies which involved the self-reporting of the presence of diabetes were considered appropriate for estimating national prevalences of diabetes, since surveys have repeatedly shown that representative assessments of population groups find a substantial proportion of unrecognized cases of diabetes within the community. In general, as age and BMI increase, the number of individuals with unrecognized diabetes also increases and there can therefore be substantial systematic biases. On this basis, only nationally representative data on fasting blood glucose levels, in combination with measured weights and heights, were used to assess the risk of diabetes associated with body-weight gain. The reason for relying on prevalence rather than incidence data for diabetes to estimate risk in relation to excess body weight is set out in a later section.

3.2 Measurements in children

The quantitative assessments of the disease burden caused by high BMI reported here only apply to adults aged ≥30 years. However estimates were also made of BMI values among children, by age, sex and subregion. This was for two reasons. First, these estimates are relevant to estimates of avoidable burden, since BMI levels track over time and so they will guide projections of the distribution of adult BMI in the next few decades. Second, high BMI is responsible for a disease burden in children and there is increasing evidence that high BMI in childhood markedly enhance the risks of disease once these children become adults; these relationships need to be included in hazard size estimates in the future. Therefore, these analyses attempted to estimate the distribution of BMIs for each 1-year age group for ages 5–18 years (i.e. until the typical end of child and adolescent normal growth [see the WHO Expert Technical Consultation on Anthropometry, WHO 1995]) and for ages 18–29 years combined.

In analysing the basis of overweight in children (Dietz and Bellizzi 1999), a concept was developed by IOTF which allowed a coherent set of nationally representative data on BMI percentiles at age 18 years to be obtained from both developed countries (prior to the recent emergence of many children with clear clinical obesity) and developing countries. The percentile values corresponding to BMIs of 25 and 30 kg/m^2 at age 18 years were used to derive sex- and age-specific cut-off points for the categorical analysis of overweight (see Cole et al. 2000). In the United States, CDC have also produced reference curves, but these are based arbitrarily on the 85th and 95th percentiles of carefully selected nationally representative data (Ogden et al. 2002). The data for this chapter are presented according to the IOTF system, Roberts and Dallal (2001) having concluded that the IOTF reference levels were more suitable for international comparisons.

4. Estimating mean BMI and prevalences of overweight and obesity

Given that representative data were not available for all 191 countries, it proved necessary to undertake a number of extrapolations. Some of the principal approaches are set out below.

4.1 Description of subregional availability of data

AFR-D

Childhood mean BMI data were from Mali and Senegal. In adults, subregional estimates were based on data from Cameroon, the Gambia, Ghana, Nigeria, Mali and Senegal. Data were also obtained for the Seychelles but were not included in the estimate. Mean BMI data are outlined in Table 8.1.

AFR-E

In children, subregional mean BMI data were from Ethiopia, South Africa and Zimbabwe. In adults, estimates were based on data from Ethiopia, Kenya, Malawi, South Africa, the United Republic of Tanzania and Zimbabwe. Data available from Kenya and the United Republic of Tanzania were limited to females only. Mean BMI data are outlined in Table 8.2.

AMR-A

For children, subregional mean BMI data were derived from both Canada and the United States. Although the Canadian data were not nationally representative, it was felt that these data should be used until nationally representative data become available in the required format. In adults, data were available from all countries in the subregion. Data from Cuba were provided in different age categories and were adjusted

Table 8.1 Mean BMI in AFR-D

		Mean BMI (kg/m²) Age group (years)						
Country (reference)	*Sex*	*5–14*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
Cameroon[a] (Rotimi et al. 1995)	Male	—	23.7	24.4	24.0	—	—	—
	Female	—	24.6	24.8	25.0	—	—	—
Gambia (Van der Sande et al. 1997)	Male	—	19.6	20.5	20.9	21.0	20.0	—
	Female	—	21.0	21.9	21.8	21.3	20.9	—
Ghana (DHS data provided by Macro International 1998)	Male	—	—	—	—	—	—	—
	Female	—	21.8	22.4	21.4	—	—	—
Mali (Re-analysed by Ferro-Luzzi, personal communication)	Male	14.8	18.9	20.5	20.8	20.3	19.6	20.2
	Female	14.9	19.9	21.1	20.6	20	19.5	20.8
Nigeria (Okesina et al. 1999)	Male	—	19.8	20.9	21.5	—	—	—
	Female	—	21.0	21.8	20.3	—	—	—
Senegal (Re-analysed by Ferro-Luzzi, personal communication)	Male	14.2	18.2	19.9	21.0	20.7	19.8	19.2
	Female	14.3	19.6	21.4	22.1	22.2	21.3	20.7
Seychelles (Bovet et al. 1991)	Male	—	22.9	23.5	23.1	23.2	—	—
	Female	—	23.2	25.7	27.2	27.5	—	—

— No data.

[a] Data provided for Cameroon were estimated from graphs as actual figures were not available.

according to the methodology outlined in section 4.4. Mean BMI data are outlined in Table 8.3.

AMR-B

Data on mean BMI in childhood were derived using findings from Argentina, Brazil and Mexico. The standard deviation for mean BMI in children was not available. To estimate standard deviation, the methodology outlined in section 4.6 was applied to data from EMR-B. For adults, subregional data were taken from Argentina, Barbados, Brazil, Mexico and Paraguay. Data were also obtained for Saint Lucia but were not included in the subregional analysis. Mean BMI data are outlined in Table 8.4.

AMR-D

Limited data were available for this subregion and neither population sample presented for children was considered to be nationally representative. However, it was considered inappropriate to exclude these data and to extrapolate from other subregions. For Guatemala, only data from children living in high altitude areas were considered in the calculations. Data for adults were only available for females. Subregional estimates for females were used to derive estimates for males, using

Table 8.2 Mean BMI in AFR-E

		Mean BMI (kg/m^2) Age group (years)						
Country (reference)	*Sex*	*5–14*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
Ethiopia (Re-analysed by Ferro-Luzzi, personal communication)	Male	14.2	17.5	18.3	18.0	18.0	17.9	19.8
	Female	14.5	18.9	18.6	17.3	16.7	17.6	18.6
Kenya (DHS data provided by Macro International, 1998)	Male	—	—	—	—	—	—	—
	Female	—	21.7	22.3	22.0	—	—	—
Malawi (Chilima and Ismail 1998)	Male	—	—	—	19.8	19.8	19.7	—
	Female	—	—	—	20.5	20.5	19.6	—
South Africa (T. Puoane et al. 1998, unpublished document)[a]	Male	13.8	21.5	24.2	25.3	24.8	24.4	—
	Female	14.0	24.4	28.5	29.9	28.8	27.7	—
United Republic of Tanzania (DHS data provided by Macro International, 1996)	Male	—	—	—	—	—	—	—
	Female	—	21.8	22.3	21.6	—	—	—
Zimbabwe (Re-analysed by Ferro-Luzzi, personal communication)	Male	15.3	19.5	20.8	21.0	21.0	20.1	20.0
	Female	15.4	21.3	23.0	23.5	21.8	20.5	20.3

— No data.

[a] Anthropometric patterns in South Africa: results from the National Demographic and Adult Health Survey 1998.

Table 8.3 Mean BMI in AMR-A

		Mean BMI (kg/m^2) Age group (years)						
Country (reference)	*Sex*	*5–14*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
Canada (Hanley et al. 2000;[a] Macdonald et al. 1997[b])	Male	19.1	23.7	25.6	26.8	26.6	26.3	—
	Female	20.2	23.2	24.1	26.3	26.7	26.4	—
Cuba (Provided by C. Nishida, personal communication, 1992)[b]	Male	—	22.2	23.5	23.5	—	—	—
	Female	—	22.4	24.3	25.4	—	—	—
USA (NHANES)[b]	Male	18.5	24.2	26.6	27.8	27.5	26.8	25.1
	Female	18.6	24.0	26.4	28.0	27.6	27.0	25.0

— No data.

[a] Childhood data.

[b] Adult data.

Table 8.4 Mean BMI in AMR-B

		Mean BMI (kg/m^2) Age group (years)						
Country (reference)	*Sex*	*5–14*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
Argentina (Hernandez et al. 1987)	Male	17.1	23.6	25.4	27.4	27.8	26.6	—
	Female	18.0	22.3	24.0	26.3	26.7	26.5	—
Barbados[a] (Rotimi et al. 1995)	Male	—	25.5	25.5	26.4	—	—	—
	Female	—	28.0	28.3	29.2	—	—	—
Brazil (Monteiro and Conde 1999)	Male	16.9	22.1	23.8	24.2	24.1	—	—
	Female	17.4	23.0	25.4	26.3	26.3	—	—
Mexico (Arroyo et al. 2000; C.P. Sánchez Castillo, personal communication, 2002[b])	Male	19.2	24.6	27.2	27.6	27.0	25.7	24.7
	Female	19.8	25.3	28.3	29.6	28.8	27.3	25.5
Paraguay[a] (Jimenez et al. 1998)	Male	—	22.3	25.2	25.1	23.1	—	—
	Female	—	21.9	27.0	29.4	27.6	—	—
Saint Kitts and Nevis, and Saint Lucia[a,b] (Rotimi et al. 1995)	Male	—	23.5	23.8	24.2	—	—	—
	Female	—	26.0	26.6	27.2	—	—	—

— No data.

[a] Data for Barbados, Paraguay, Saint Kitts and Nevis, and Saint Lucia were estimated from graphs as actual figures were not available.

[b] Childhood data.

Table 8.5 Mean BMI in AMR-D

		Mean BMI (kg/m^2) Age group (years)						
Country (reference)	*Sex*	*5–14*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
Guatemala (DHS 1998;[a] Martorell et al. 1995[b])	Male	14.6	—	—	—	—	—	—
	Female	14.9	24.4	25.8	26.8	—	—	—
Peru (DHS data provided by Macro International, 1996;[a] Gonzales et al. 1994[b])	Male	15.2	—	—	—	—	—	—
	Female	15.3	24.4	25.8	26.5	—	—	—

— No data.

[a] Adult data.

[b] Childhood data.

methodology outlined in section 4.4. The methodology outlined in section 4.4 was also used to obtain estimates for upper age categories. The original mean BMI data for females are outlined in Table 8.5.

EMR-B

In children, subregional data were from Bahrain, Lebanon and Saudi Arabia. In adults, regional estimates were derived from Bahrain, Cyprus,

Table 8.6 Mean BMI in EMR-B

		Mean BMI (kg/m^2) Age group (years)						
Country (reference)	*Sex*	*5–14*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
Bahrain (al-Mannai et al. 1996;[a,b] Musaiger and al-Mannai 2001;[a,c] Musaiger and Gregory 2000[d])	Male	16.0	22.9	27.2	26.7	25.1	—	—
	Female	16.9	24.6	30.4	30.0	29.9	—	—
Iran (Islamic Republic of) (Pishad 1996)[a]	Male	—	21.0	23.8	24.3	23.1	22.7	—
	Female	—	21.8	24.7	25.0	24.1	22.5	—
Jordan (Ajlouni et al. 1998)[a]	Male	—	24.9	27.0	28.5	28.0	26.2	—
	Female	—	26.3	30.8	32.5	31.9	30.1	—
Kuwait (al-Isa 1995)[a]	Male	—	26.7	28.3	28.6	25.0	—	—
	Female	—	26.7	30.3	31.5	29.8	—	—
Lebanon (Data re-analysed by N. Hwalla and N. Adra, 1996)[a,b]	Male	17.8	23.5	25.8	26.7	26.1	25.5	23.5
	Female	17.8	22.3	25.4	28.1	29.2	27.2	26.0
Saudi Arabia (al-Nuaim et al. 1996)[a]	Male	—	23.5	26.1	27.0	26.0	—	—
	Female	21.0	24.5	27.6	28.7	27.0	—	—
United Arab Emirates (el Mugamer et al. 1995)[a]	Male	—	24.7	25.6	26.5	24.6	—	—
	Female	—	26.8	27.8	28.8	25.4	—	—

— No data.

[a] Adult data.

[b] 18–29 years.

[c] >30 years.

[d] Childhood data.

the Islamic Republic of Iran, Jordan, Kuwait, Lebanon, Saudi Arabia and the United Arab Emirates. Only data for Lebanon were provided in the appropriate age categories; all other data were subject to the methodology outlined in section 4.4. Mean BMI data are outlined in Table 8.6.

EMR-D

Childhood data were not available in this subregion in the required format. Using the methodology outlined in section 4.5, it was concluded that it would be most appropriate to use data from the AFR-E subregion in order to determine the estimates for children. Mean BMI data for adults were limited to Pakistan and Egypt (females only). No data were available for the ≥80 years age category, therefore the methodology outlined in section 4.4 was applied. Mean BMI data are outlined in Table 8.7.

EUR-A

Availability of mean BMI data is described in Table 8.8.

Table 8.7 Mean BMI for adults in EMR-D

		Mean BMI (kg/m^2) Age group (years)					
Country (reference)	*Sex*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
Egypt (DHS, data provided by Macro International, 1992–1995)	Male	—	—	—	—	—	—
	Female	25.3	27.2	27.1	—	—	—
Pakistan (Data provided by Dr Habibullah, 1998)	Male	20.7	21.8	21.9	21.6	21.0	—
	Female	21.1	22.5	22.7	22.3	21.3	—

— No data.

EUR-B

In children, data on mean BMI were available from Bulgaria, Poland, Slovakia and Turkey. In adults, data on mean BMI were available from Romania, Slovakia, Tajikistan, Turkey and Uzbekistan, and are outlined in Table 8.9.

EUR-C

Limited data were available for children in EUR-C, from the Russian Federation only. In adults, data were available for Hungary, Latvia, Lithuania and the Russian Federation and are outlined in Table 8.10.

SEAR-B

There were no data available for children in this subregion, thus data from AFR-E were used, as specified in section 4.5. As no data for adults were available for Indonesia or Sri Lanka, subregional figures were based on data from Thailand, as outlined in Table 8.11. Normally these data would have been excluded as they came from attendees at a dental clinic. However, as no other data were available in the required format at the time, it was decided that they should be included in the absence of any more appropriate data.

SEAR-D

Data for children were available from Nepal. Data for adults were available for Bangladesh, India (females only) and Nepal. Mean BMI values available are shown in Table 8.12.

WPR-A

Data were available for both children and adults for Australia and Japan, as outlined in Table 8.13.

WPR-B

As no data were available for children in WPR-B, data from AFR-E were used as specified in section 4.5. Data for adults were available for China,

Table 8.8 Mean BMI in EUR-A

		Mean BMI (kg/m²) Age group (years)						
Country (reference)	*Sex*	*5–14*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
Belgium (Stam Moraga 1999)[a]	Male	—	24.8	25.3	26.5	26.3	26.1	—
	Female	—	23.1	24.1	27.2	27.8	27.9	—
Croatia (Data re-analysed by A. Kaic-Rak, 1992)[b]	Male	18.2	—	—	—	—	—	—
	Female	18.1	—	—	—	—	—	—
Czech Republic (V. Hainer, personal communication, 1997–1998)[b]	Male	—	25.5	26.9	28.2	28.6	—	—
	Female	—	23.6	25.7	28.3	29.8	—	—
Denmark (A. Robertson, personal communication 1995);[a] (Data re-analysed by A. Nielsen 1986–1997)[b]	Male	17.3	22.3	24.8	25.8	26	—	—
	Female	17.5	21.0	23.0	24.0	24.4	—	—
Finland (Lahti-Koski et al. 2000)[a]	Male	—	25.3	26.1	27.9	28.4	—	—
	Female	—	24.0	24.8	27.7	28.4	—	—
Germany (Bergmann and Mensink 1999, provided by G. Mensink;[a] Kromeyer-Hauschild et al. 1999[b])	Male	17.1	24.7	26.7	27.9	28.1	27.8	—
	Female	17.3	23.6	25.0	27.4	28.9	28.1	—
Greece (N. Katsilambros, unpublished data, 2000;[a,c] Trichopoulou et al. 2000[a,d])	Male	—	27.5	27.7	28.5	28.3	28.1	—
	Female	—	25.4	26.7	30.1	30.5	30.4	—
Iceland (V. Gudnason, unpublished data, 1991–1996)[a]	Male	—	25.1	26.2	27.1	27.7	26.3	25.5
	Female	—	28.5	24.9	26.4	27.8	26.4	26.2
Malta (A. Robertson, personal communication, 1994)[a]	Male	—	26.0	26.7	27.4	27.6	—	—
	Female	—	25.2	26.2	30.4	30.7	—	—
Netherlands (Data from the National Institute of Public Health and the Environment, and data from the MORGEN study, provided by T.L.S. Visscher, 1993–1997)[a]	Male	—	23.6	25.2	26.4	26.5	—	—
	Female	—	23.1	24.3	25.8	27.1	—	—
Portugal (Do Carmo et al. unpublished data presented at ECO 2000)[a]	Male	—	—	24.7	27.2	27.4	—	—
	Female	—	22.4	25.9	27.0	28.2	—	—
Spain (M. Fox, personal communication, 1990–1994;[a] (Moreno et al. 2000[b])	Male	18.9	24.2	25.8	26.8	—	—	—
	Female	19.0	22.6	24.7	27.4	—	—	—
Switzerland (M. Fox, personal communication, 1992/1993;[a] Zimmerman et al. 2000[b])	Male	17.2	23.0	24.4	25.7	25.8	25.5	—
	Female	17.3	21.2	22.1	23.5	24.3	24.3	—
United Kingdom (Health Survey for England, data re-analysed by IOTF 1998)[a,b]	Male	18.1	24.8	26.5	27.5	27.6	27.2	25.8
	Female	18.5	24.7	26.2	27.1	28.1	27.2	25.6

— No data.

[a] Adult data

[b] Childhood data.

[c] 18–29 years.

[d] >30 years.

Table 8.9 Mean BMI in EUR-B

		Mean BMI (kg/m^2) Age group (years)						
Country (reference)	*Sex*	*5–14*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
Bulgaria (Data re-analysed by S. Petrova, 1998)[b]	Male	18.0	—	—	—	—	—	—
	Female	17.9	—	—	—	—	—	—
Poland (Data re-analysed by I. Palczewska, 1999)[b]	Male	17.6	—	—	—	—	—	—
	Female	17.3	—	—	—	—	—	—
Romania (N. Hâncu, personal communication, 1999)[a]	Male	—	23.9	26.4	27.1	—	—	—
	Female	—	22.5	26.9	28.0	—	—	—
Slovakia (K. Babinska, personal communication, 1995–1999);[a] (Data collated by A. Bederova and re-analysed by K. Babinska, 1995–1999)[b]	Male	18.0	21.7	26.7	27.7	28.4	26.8	—
	Female	18.2	20.8	24.5	27.0	28.9	28.2	—
Tajikistan (A. Robertson, personal communication, 1998)[a]	Male	—	17.8	20.8	25.4	26.8	—	—
	Female	—	18.0	20.4	25.5	27.3	—	—
Turkey (Data re-analysed by G. Pekcan and N. Rak, 1993–1999)	Male	16.7	21.9	25.6	26.3	26.3	25.6	24.8
	Female	16.8	24.0	27.7	29.6	29.4	29.2	27.5
Uzbekistan (A. Robertson, personal communication, 1999)[a]	Male	—	20.7	21.2	22.5	—	—	—
	Female	—	20.2	20.1	22.0	—	—	—

— No data.

[a] Adult data.

[b] Childhood data.

Table 8.10 Mean BMI in EUR-C

		Mean BMI (kg/m^2) Age group (years)						
Country (reference)	*Sex*	*5–14*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
Hungary (Zajkas and Biro 1998)	Male	—	24.0	26.8	28.1	28.4	25.4	25.0
	Female	—	22.4	25.4	28.2	29.8	27.0	27.7
Latvia (A. Robertson, personal communication, 1997)	Male	—	25.6	27.0	29.0	29.2	—	—
	Female	—	22.1	24.2	27.6	28.8	—	—
Lithuania (A. Robertson, personal communication, 1999)	Male	—	24.8	25.7	26.5	26.8	—	—
	Female	—	23.2	24.9	27.6	28.7	—	—
Russian Federation (Data re-analysed by AD Deev, Russian Longitudinal Monitoring Survey—RLMS 1992)	Male	18.1	22.8	25.1	25.9	25.6	25.2	24.8
	Female	17.7	22.8	26.6	28.5	28.6	27.2	25.2

— No data.

Table 8.11 Mean BMI for adults in SEAR-B

		Mean BMI (kg/m^2) Age group (years)					
Country (reference)	*Sex*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
Thailand (Chaichareon et al. 1992)	Male	20.8	22.6	23.4	23.0	22.6	22.6
	Female	20.8	22.7	23.9	24.3	22.5	22.5

Table 8.12 Mean BMI in SEAR-D

		Mean BMI (kg/m^2) Age group (years)						
Country (reference)	*Sex*	*5–14*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
Bangladesh (M. Q-K and K. Talukder, personal communication, 2000)	Male	—	19.0	19.5	18.9	19.1	18.0	—
	Female	—	19.7	19.9	19.2	18.7	19.7	—
India (DHS data provided by Macro International, 1998)	Male	—	—	—	—	—	—	—
	Female	—	19.5	20.9	21.6	—	—	—
Nepal (Data re-analysed by A. Ferro-Luzzi personal communication, 1997)[a,b]	Male	14.4	19.0	20.1	19.8	19.6	18.2	19.4
	Female	18.5	20.4	20.7	20.2	19.6	19.1	16.0

— No data.

[a] Adult data.

[b] Childhood data.

Table 8.13 Mean BMI in WPR-A

		Mean BMI (kg/m^2) Age group (years)						
Country (reference)	*Sex*	*5–14*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
Australia (National Nutrition Survey 1995, data re-analysed by T. Gill, 2000)	Male	18.1	24.6	26.8	26.8	27.6	27.1	24.7
	Female	18.6	22.5	24.6	27.1	27.2	26.4	25.4
Japan (Yoshiike et al. 1998)	Male	17.6	21.8	23.0	23.4	22.7	22.3	21.5
	Female	16.9	20.5	22.1	23.3	23.5	23.0	22.3

Table 8.14 Mean BMI in WPR-B

Country (reference)	Sex	Mean BMI (kg/m^2) Age group (years) 15–29	30–44	45–59	60–69	70–79	≥80
China	Male	21.6	22.9	23.2	23.0	22.3	—
	Female	22.7	23.0	23.7	23.9	22.6	—
Malaysia (Khor et al. 1999)	Male	21.6	23.6	23.0	21.4	—	—
	Female	22.3	24.8	24.5	22.6	—	—
Republic of Korea (Jones et al. 1994)	Male	22.5	22.8	23.2	21.8	21.6	20.8
	Female	21.1	21.8	23.0	22.4	22.2	21.1
Samoa (McGarvey 1991)	Male	24.9	25.8	28.0	27.7	26.5	—
	Female	26.0	27.9	30.3	29.8	28.6	—
Solomon Islands (Eason et al. 1987)	Male	22.9	23.5	23.6	—	—	—
	Female	24.4	24.9	24.2	—	—	—
Viet Nam (Giay and Khoi 1994)	Male	19.3	19.5	19.0	18.2	—	—
	Female	19.8	19.4	18.6	17.8	—	—

— No data.

Malaysia, the Republic of Korea, Samoa, the Solomon Islands and Viet Nam, as outlined in Table 8.14.

4.2 Obtaining subregional estimates from country-specific estimates

In order to obtain subregional estimates of mean BMI, prevalences of overweight and obesity and their associated standard errors for sex- and age-specific categories, a meta-analysis was initially considered. In this approach, estimates from different studies would be combined into a single weighted estimate, using the variance of the estimate as the weight. The combined estimate of the variances would then be the inverse of the sum of the study-specific variances. There were two major drawbacks which made this approach unsuitable.

- Countries with unknown variances would be excluded from the subregional analysis for the estimate of mean BMI or prevalences of overweight and obesity.
- The method of weighting by the variance of the study (which is highly dependent on the sample size and the design of the study, with "better", larger studies having the smallest variances) assumes an equal population for each sample. The approach does not take into account differences in population sizes.

A second approach was to obtain a single estimate of mean BMI and the prevalences of overweight and obesity by using a population-weighted average. Standard deviations were estimated using standard statistical relationships.[2]

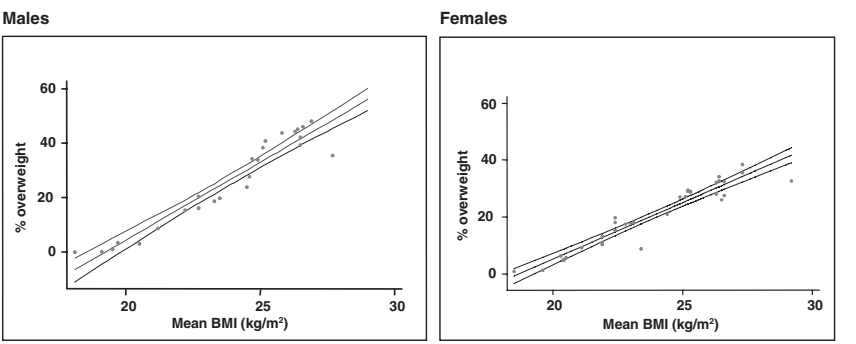
For simplicity, a simple average of the standard errors of the prevalences of overweight or obesity was used to obtain subregional estimates. In cases where no country data were available and the prevalences of overweight or obesity were based on predictions, the subregional standard error was that of the prediction.

4.3 Converting mean BMI to proportions of overweight and obesity and vice versa

BMI distributions are skewed in almost all age and sex categories throughout the world. Thus mean BMI with standard deviation does not accurately describe the whole BMI distribution and usually no further details of the distribution are available. However, Rose and Shipley (1990) showed that the prevalence of obesity (BMI ≥30 kg/m^2) was highly correlated with mean BMI in the selected group of adults, the prevalence of obesity increasing by 4.22% per unit (1 kg/m^2) increase in mean BMI. This approach was repeated in the present analysis, which was based on adult data (all ages ≥18 years combined) from 36 countries with continuous and categorical data for females and 26 countries with continuous and categorical data for males (Table 8.15). The mean BMI values for countries used in this estimation varied widely from 18.1 kg/m^2 to 29.2 kg/m^2. Mali was excluded from the current analyses in males because it was an outlier. Mean sex-specific BMI vs percentages of overweight and obesity are shown in Figures 8.1 and 8.2.

There is a clear positive linear association for both sexes. The regression equations that describe the graphs are:

Figure 8.1 Mean BMI and percentage of the adult population that is overweight



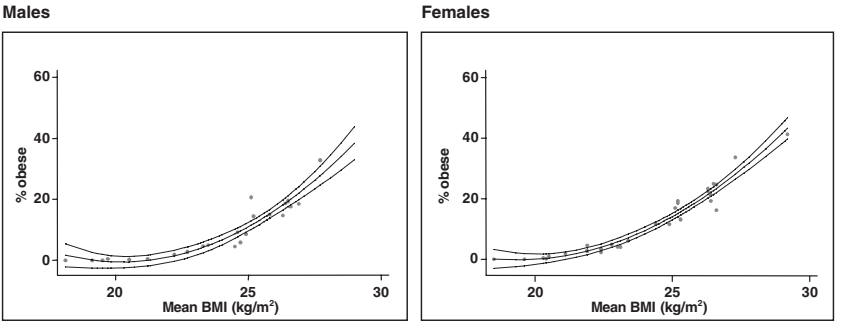
Note: These data points relate to population surveys from the countries listed in Table 8.15. The linear regression is shown together with the 95% confidence intervals of the prediction.

Table 8.15 Countries from which mean BMI data were used to derive equations for calculating the percentages of overweight and obese adults in the population

For female overweight and obesity	*For male overweight and obesity*
Australia	Australia
Bangladesh	Bangladesh
Brazil	Brazil
China	China
Denmark	Denmark
Egypt	Ethiopia
Ethiopia	Germany
Germany	Hungary
Ghana	Iceland
Guatemala	Japan
Hungary	Kuwait
Iceland	Lebanon
Japan	Nepal
Kenya	Norway
Kuwait	Republic of Korea
Lebanon	Russian Federation
Malawi[a]	Senegal
Mali	Slovakia
Nepal	Switzerland
Norway	Thailand
Peru	Turkey
Republic of Korea	United Kingdom
Russian Federation	USA
Senegal	Uzbekistan
Slovakia	Zimbabwe
South Africa[a]	
Switzerland	
Thailand	
Turkey	
United Kingdom	
United Republic of Tanzania	
USA	
Uzbekistan	
Zimbabwe	

[a] Overweight only.

Figure 8.2 Mean BMI and percentage of the adult population that is obese



Note: The predicted relationship is shown, together with the 95% confidence intervals of the prediction. The quadratic equations are given below.

$$\text{Males } (\%\ overweight) = -110.4 + 5.7 \times (mean\ BMI) \quad (1)$$

$$\text{Females } (\%\ overweight) = -74.2 + 3.97 \times (mean\ BMI) \quad (2)$$

In both cases, the β coefficient was highly statistically significant and the models accounted for >90% of the total variation. There was also a strong correlation between mean BMI and percentages of obese adults in the population, as seen in Figure 8.2.

$$\text{Males } (\%\ obese) = 205.1 - 20.4 \times (mean\ BMI) + 0.5 \times (mean\ BMI)^2 \quad (3)$$

$$\text{Females } (\%\ obese) = 168.5 - 17.4 \times (mean\ BMI) + 0.4 \times (mean\ BMI)^2 \quad (4)$$

The following conditions applied:

- the predictions had to be positive (since they are percentages);
- the predictions must be <100; and
- the sum of the predicted percentages of people in the overweight and obese categories must be ≤100 (the sum is equal to 100 in the extreme case whereby no individuals belong to the underweight or normal BMI categories).

These conditions hold simultaneously for mean BMIs of 21.3–29.7 kg/m^2 for males and 20.1–33.9 kg/m^2 for females. Predictions which fell outside these ranges were therefore not considered. These four models (Equations 1–4) for predicting the prevalences of overweight and obesity from mean BMIs were then applied to other populations when necessary.

Finally, the assumption was made that the equations held true for each age group and every country, so that the estimates could be

derived uniformly. This assumption seemed justified because wherever data were available the patterns of mean BMI and percentages of overweight and obesity were similar across age groups in most of the countries assessed.

4.4 Age and sex extrapolation

Obtaining estimates for the required age groups

Apart from data personally donated to or re-analysed by IOTF, all other data were reported in different age categories from those required for this work. To obtain data in these age categories, it was assumed that:

- the numbers of persons in each year within an age group were the same and equal to the total number of people in the age group divided by the number of years in the age group.
- the mean BMI and the standard deviation for each year within an age group were the same and equal to the mean BMI and standard deviation in the age group as a whole.

Single years or convenient groups of years were treated as different strata which were then combined to obtain the desired estimates in any age categorization.

Extrapolation to adult age groups for which no data were available

It was not always possible to obtain data for all age groups, particularly for the oldest (70–79 and ≥80 years) at a subregional level. WPR-B, where this problem was initially encountered for both sexes in people aged >70 years, is used as an example, although data subsequently became available for this subregion.

In the majority of the available data worldwide, the mean BMI increased with age and then started falling with rising age. Figure 8.3 shows the relationship between mean BMI and age in WPR-B. It was assumed that the mean BMI remained constant within each age group and changed only when moving from one age group to another.

The following regression equations describe the graphs:

$$\text{Females } (\textit{Mean BMI}) = 16.71 + 0.27 \times (\textit{age}) - 0.0024 \times (\textit{age}^2)$$

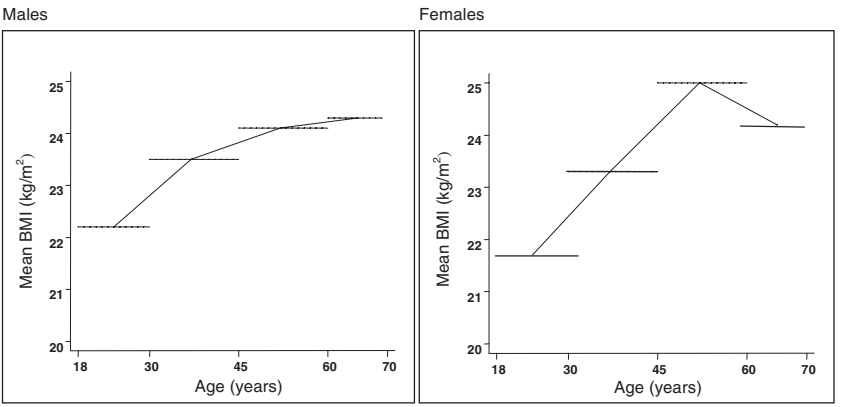
$$\text{Males } (\textit{Mean BMI}) = 16.62 + 0.14 \times (\textit{age}) - 0.0011 \times (\textit{age}^2)$$

Using these equations, the sex-specific mean BMI for each single year from age 70 years and above could be predicted. The overall mean BMI in the age group 70–79 years was set equal to the average of the mean BMIs for the individual years. The same procedure was used to estimate the mean BMI in the age group ≥80 years. The results are shown in Table 8.16. The illustrated approach was used for EMR-B.

Table 8.16 Predicted mean BMI values in the oldest age groups in WPR-B

	Mean BMI (kg/m²)	
Age group (years)	*Females*	*Males*
70–79	23.9	24.2
≥80	22.8	23.8

Figure 8.3 The relationship between mean BMI and age in WPR-B



Extrapolating data from one sex to the other

Many countries reported results either for both sexes combined or for one sex only. For example, on a subregional basis, no suitable data were available for males in AMR-D. The crude mean BMI for females in all subregions (calculated as the mean of the subregion-specific estimates for all ages combined) was 23.7 kg/m² and was 0.4 units greater than the crude mean BMI for men in all subregions ($P=0.7$). To determine whether this was the case in each age group, the age-specific differences in mean BMI between females and males were estimated for all subregions. For all age groups apart from the oldest (i.e. ≥80 years), the mean BMI for females was greater than that for males, as shown in Table 8.17.

These values served as correction factors when using data from males to estimate mean BMI for females and vice versa (without considering whether the differences between mean BMI for males and for females were significant). Thus, for each age group the respective correction factor was subtracted from mean BMI values for females to obtain the values for males. In most studies, it is found that women tend to have

Table 8.17 Age-dependent differences between mean BMI for females and for males

	Age group (years)					
	18–29	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
Differences in mean BMI (females – males)	0.3	0.5	0.7	0.8	0.3	–0.1

higher BMIs than men and this seems to be related to the deposition of fat rather than metabolically active lean tissue with body-weight gain. Women lay down a lower proportion of lean tissue and thus have to put on more weight before the slower increase in the mass of lean tissue raises the basal metabolic rate sufficiently to add to the exercise costs of their greater weight and achieve an energy output which finally matches their energy intake (James and Reeds 1997).

4.5 Estimating childhood data

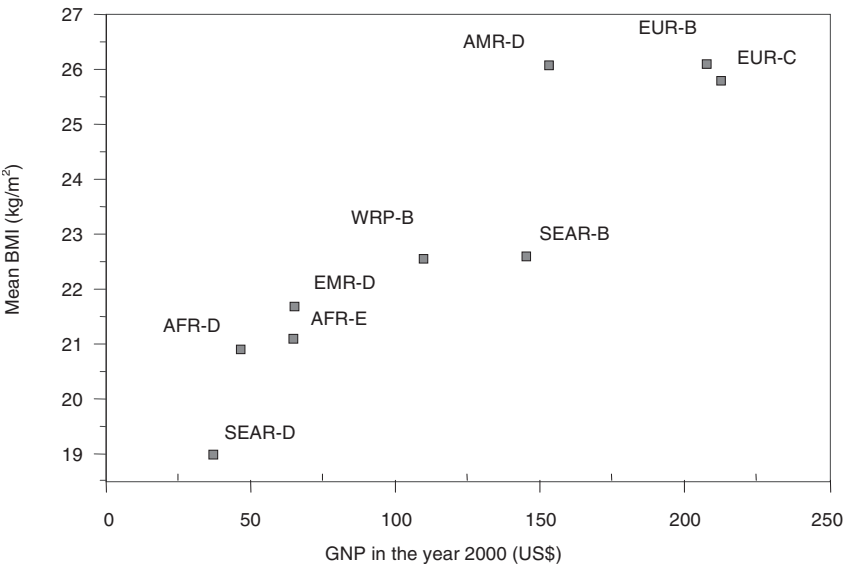
When no data were available for children for any of the countries within a subregion, an estimate was made of the distribution of BMI in children for that subregion by extrapolating from subregions with data and having equivalent economies, as judged by their gross national products (GNP). In the absence of any other data, WPR-A was extrapolated for AMR-A.

In general, plotting the mean age-standardized BMI for adults for each subregion (calculated as a simple average of population-weighted means for both sexes and for all ages ≥18 years) vs GNP (on a subregional basis) shows a broad relationship between increasing GNP and mean BMI in subregions with GNPs of <US$ 10 000 per year (Figure 8.4). For countries with the lowest GNPs, the assumption was made that BMIs for children would be similar in subregions with low GNP and low mean BMI for adults. This is subject to large uncertainty, given the complex underlying factors that determine body weight and height.

At the time of writing, no data were available for children in EMR-D and WPR-B. The GNPs of countries in EMR-D were similar to those in AFR-E; the average GNP in EMR-D was US$ 652 (with a range of US$ 110–1290), whereas the average GNP in AFR-E was US$ 647, (with a range of US$ 80–3520). Mean BMI values were also similar in both populations, with values being slightly higher in EMR-D. The distribution of BMIs in children in AFR-E was therefore applied to EMR-D. The distributions of GNP and BMI for adults in SEAR-B and WPR-B were also similar, but there were no data for children, so the AFR-E values were used for these subregions, AFR-E being the closest to these subregions economically and also in terms of BMI.

Further problems arose when the mean BMIs for children were available, but not the standard deviations, as in AMR-B. The standard

Figure 8.4 The relationship between mean BMI for adults and GNP in nine subregions



deviations of BMIs for children in AMR-B were therefore also assumed to have the same variability as in EMR-B.

Very little in the way of categorical data was available for children and it was therefore necessary to extrapolate extensively, pending appropriate data becoming available.

4.6 The estimated mean BMI and standard deviation for each subregion, by age and sex

These data are presented in the standard format proposed for the CRA project. The mean BMIs for each year for ages 5–17 years inclusive for males and females separately were obtained and are given in Tables 8.18 and 8.19.

The initial BMI analyses were made as previously, considering children in 1-year age groups, firstly for the countries with data. From these values, the mean BMIs of the 1-year age groups within the subregion were estimated. Assuming equivalent numbers of people in each year of each specified age group, it was possible to provide mean BMIs, standard deviations and confidence intervals for the age groups 5–14 years and 15–29 years used in the CRA analyses. It was recognized that these values could not be used in the usual way to predict different categories of excess weight because of the normal changes in body weight

Table 8.18 The mean BMIs for children and adults in all subregions, by sex and age[a]

		Mean BMI (kg/m²) Age group (years)																		
Subregion	*Sex*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*	*13*	*14*	*15*	*16*	*17*	*18–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
AFR-D	Male	14.2	14.0	14.1	14.1	14.5	14.6	14.7	14.9	15.1	15.7	16.1	16.3	16.9	20.1	21.2	21.7	20.5	20.5	20.0
	Female	13.9	13.6	13.8	14.0	14.0	14.5	15.1	15.1	15.9	17.0	17.2	19.0	18.9	21.3	22.1	21.0	21.0	20.2	20.8
AFR-E	Male	14.1	13.8	13.8	13.9	13.9	14.1	14.5	16.2	16.1	16.7	17.5	17.8	18.1	19.6	21.0	21.2	20.8	22.0	19.8
	Female	13.9	13.6	13.6	13.8	14.1	14.1	14.6	16.9	17.0	17.8	18.7	19.4	20.2	21.7	22.9	22.8	22.3	20.4	18.9
AMR-A	Male	15.9	16.4	16.9	17.2	18.2	18.5	19.4	20.0	20.5	22.5	22.2	22.5	23.4	24.5	26.4	27.6	27.4	26.7	25.1
	Female	16.0	16.0	17.3	17.2	18.3	18.6	19.8	20.1	22.1	22.3	22.2	22.9	23.0	24.1	26.1	27.7	27.5	26.9	25.0
AMR-B	Male	15.4	15.6	15.7	16.0	16.3	16.7	17.0	17.5	18.2	18.8	19.4	20.1	20.4	23.7	25.0	25.6	25.5	26.1	24.7
	Female	15.2	15.6	16.0	16.2	16.6	17.0	17.5	18.6	19.8	20.4	20.8	21.5	21.7	24.1	26.2	27.3	27.1	26.9	25.5
AMR-D	Male	15.8	15.5	14.9	15.3	15.2	15.1	15.9	16.0	17.1	17.6	18.8	19.4	20.1	24.1	25.3	25.9	26.0	26.3	26.3
	Female	15.6	15.3	15.2	15.1	14.9	14.7	16.0	16.7	18.9	19.2	20.5	21.7	22.1	24.4	25.8	26.6	26.8	26.6	26.2
EMR-B	Male	15.4	15.7	16.9	16.7	17.9	17.4	17.6	19.7	19.5	21.3	20.8	22.3	23.8	21.9	24.6	25.3	24.3	23.1	23.5
	Female	15.2	15.4	16.4	17.2	16.3	18.0	18.9	19.1	20.5	21.7	22.0	21.6	21.4	22.8	25.8	26.5	25.5	23.3	26.0
EMR-D	Male	14.1	13.8	13.8	13.9	13.9	14.1	14.5	16.2	16.1	16.7	17.5	17.8	18.1	20.7	21.8	21.9	21.6	21.0	20.1
	Female	13.9	13.6	13.6	13.8	14.1	14.1	14.6	16.9	17.0	17.8	18.7	19.4	20.2	22.3	23.8	22.8	22.3	21.3	18.9

EUR-A	Male	16.2	16.6	16.5	16.7	16.9	17.3	18.1	18.6	19.6	19.8	21.0	22.2	22.5	24.7	26.3	27.2	27.8	27.5	26.1
	Female	16.3	16.2	16.5	16.4	17.2	17.6	18.7	18.9	20.1	20.5	22.1	22.3	22.9	23.5	25.1	27.5	28.4	27.8	25.8
EUR-B	Male	15.1	15.5	15.5	16.0	16.7	17.2	18.6	18.4	18.6	19.3	19.7	20.4	20.6	22.1	25.0	26.5	27.3	25.8	24.8
	Female	15.0	15.2	15.5	15.8	16.4	16.9	17.6	18.2	18.9	19.9	20.0	20.5	21.4	23.2	25.6	27.9	28.8	29.0	27.5
EUR-C	Male	16.4	15.7	17.0	17.4	17.1	17.8	18.0	18.8	19.6	20.5	20.6	21.4	21.6	23.3	25.2	26.1	25.9	25.2	24.8
	Female	15.7	15.6	16.4	16.9	17.1	17.1	17.9	18.2	19.4	20.2	21.3	21.3	21.3	23.1	26.5	28.4	28.7	27.2	25.4
SEAR-B	Male	14.1	13.8	13.8	13.9	13.9	14.1	14.5	16.2	16.1	16.7	17.5	17.8	18.1	20.8	22.6	23.4	23.0	22.6	22.6
	Female	13.9	13.6	13.6	13.8	14.1	14.1	14.6	16.9	17.0	17.8	18.7	19.4	20.2	20.8	22.7	23.9	24.3	22.5	22.5
SEAR-D	Male	13.8	14.3	13.9	13.6	14.1	14.2	14.4	15.2	14.8	15.8	16.3	17.2	17.7	19.0	19.6	19.0	19.2	18.0	19.4
	Female	14.1	14.2	13.8	14.2	14.5	14.2	14.8	15.2	17.1	18.0	18.5	19.1	20.3	19.5	20.8	21.4	18.9	19.6	16.0
WPR-A	Male	16.3	15.6	15.9	16.5	16.8	17.6	18.1	19.2	19.9	20.4	20.9	21.4	21.8	22.4	23.7	23.9	23.3	22.8	21.9
	Female	16.2	15.3	15.6	16.0	16.4	17.0	17.5	18.4	19.5	19.8	20.1	20.5	20.5	20.9	22.5	23.8	23.9	23.3	22.6
WPR-B	Male	14.1	13.8	13.8	13.9	13.9	14.1	14.5	16.2	16.1	16.7	17.5	17.8	18.1	21.5	22.8	23.1	22.8	22.3	20.8
	Female	13.9	13.6	13.6	13.8	14.1	14.1	14.6	16.9	17.0	17.8	18.7	19.4	20.2	22.5	22.8	23.5	23.6	22.6	21.1

[a] Data analysed by 1-year age groups in childhood.

Note: Figures in shaded cells were estimated as outlined in the methodology section.

Table 8.19 The standard deviations of the BMIs for children and adults in all subregions, by sex and age[a]

		Age group (years)																		
Subregion	*Sex*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*	*13*	*14*	*15*	*16*	*17*	*18–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
AFR-D	Male	1.5	1.3	1.9	1.5	1.1	1.2	1.7	1.3	1.5	1.6	1.7	2.0	2.0	2.0	3.4	5.4	2.9	2.9	2.7
	Female	1.3	1.3	1.3	1.2	1.3	1.6	1.6	1.9	2.1	3.0	2.4	2.6	2.7	3.8	3.9	4.6	3.8	3.6	2.3
AFR-E	Male	1.4	1.2	1.2	1.2	1.1	1.4	1.0	3.0	2.1	2.3	2.4	2.4	2.4	3.1	5.2	4.6	4.2	3.6	1.8
	Female	1.3	1.3	1.3	1.3	1.2	1.8	1.4	2.6	2.5	3.0	2.9	2.9	2.9	4.2	4.8	5.2	5.1	5.1	2.4
AMR-A	Male	1.7	2.5	2.9	4.2	4.3	4.3	4.4	4.5	4.5	4.7	4.7	4.7	4.8	4.8	5.0	5.0	4.4	4.3	4.1
	Female	4.0	4.0	4.1	4.1	4.3	4.3	4.4	4.5	4.7	4.7	4.7	4.8	4.8	5.9	7.1	6.5	6.1	5.8	4.9
AMR-B	Male	1.5	1.4	2.3	1.9	2.8	4.1	3.8	3.7	3.7	3.9	3.6	3.5	3.2	4.1	4.1	4.3	4.0	3.9	3.8
	Female	1.4	2.1	2.8	3.8	3.3	3.6	3.9	4.1	4.1	4.7	4.0	4.2	5.1	4.8	5.1	5.3	5.2	5.2	4.3
AMR-D	Male	1.1	1.0	1.0	1.3	1.3	1.3	1.4	1.3	1.2	1.4	1.5	1.5	1.6	2.8	3.8	4.0	3.5	3.5	3.5
	Female	1.2	1.0	1.0	1.8	1.8	1.8	1.8	1.8	2.3	2.5	1.7	2.4	2.6	3.4	4.4	4.6	4.1	4.1	4.1
EMR-B	Male	1.5	2.0	2.9	2.1	3.0	2.4	2.9	4.5	4.0	3.4	3.8	3.2	3.7	3.8	4.1	3.9	4.0	3.7	3.6
	Female	1.4	1.9	2.2	5.2	2.6	2.9	3.4	4.1	4.3	4.8	5.3	5.7	4.9	4.1	5.1	5.3	4.8	3.7	5.8
EMR-D	Male	1.4	1.2	1.2	1.2	1.1	1.4	1.0	3.0	2.1	2.3	2.4	2.4	2.4	5.6	6.9	4.8	5.8	5.8	5.8
	Female	1.3	1.3	1.3	1.3	1.2	1.8	1.4	2.6	2.5	3.0	2.9	2.9	2.9	7.3	8.5	6.5	7.4	7.4	7.4

EUR-A	Male	1.2	3.9	2.1	2.3	2.4	2.4	2.9	2.9	3.2	3.0	2.9	3.6	3.6	3.8	3.7	3.9	3.8	3.8	3.7
	Female	1.7	2.2	2.0	2.1	2.6	2.6	3.2	3.1	3.1	3.2	4.2	3.9	3.4	4.1	4.7	4.7	5.2	4.9	4.3
EUR-B	Male	1.6	2.1	2.2	2.8	2.7	2.6	3.0	2.9	2.9	3.1	2.7	2.6	2.4	3.2	3.9	3.8	3.7	3.6	4.8
	Female	2.0	1.8	2.2	2.3	2.4	2.7	2.9	3.0	2.7	2.9	2.6	2.6	3.1	4.2	5.2	6	5.1	4.7	4.8
EUR-C	Male	2.5	2.6	2.9	3.2	2.6	3.0	2.6	3.1	3.1	3.3	2.3	2.3	2.6	2.9	3.5	3.9	3.8	3.9	3.9
	Female	2.5	2.6	2.8	3.1	3.3	2.9	2.9	2.8	2.5	3.1	3.1	3.0	2.4	4.2	5.0	5.1	5.2	5.1	5.0
SEAR-B	Male	1.4	1.2	1.2	1.2	1.1	1.4	1.0	3.0	2.1	2.3	2.4	2.4	2.4	2.2	2.8	2.3	3.9	2.5	2.5
	Female	1.3	1.3	1.3	1.3	1.2	1.8	1.4	2.6	2.5	3.0	2.9	2.9	2.9	3.6	2.3	2.1	4.1	4.5	4.5
SEAR-D	Male	1.4	0.8	0.9	1.2	1.1	0.9	1.0	1.4	1.4	1.3	1.6	1.7	1.7	2.1	3.2	2.8	2.9	2.7	3.1
	Female	0.9	1.2	1.3	1.9	1.5	1.2	1.4	1.6	2.7	2.1	2.2	2.6	2.2	3.2	4.0	4.6	3.7	5.9	2.0
WPR-A	Male	2.4	1.3	1.6	1.8	2.0	2.3	2.5	2.6	2.9	2.9	3.0	2.9	3.0	3.5	3.4	3.2	3.2	3.2	3.6
	Female	1.8	1.1	1.2	1.3	1.5	1.6	1.8	2.0	2.1	2.1	2.2	2.2	2.2	3.8	4.1	3.7	3.7	3.8	3.7
WPR-B	Male	1.4	1.2	1.2	1.2	1.1	1.4	1.0	3.0	2.1	2.3	2.4	2.4	2.4	2.7	3.3	3.1	3.3	3.6	1.8
	Female	1.3	1.3	1.3	1.3	1.2	1.8	1.4	2.6	2.5	3.0	2.9	2.9	2.9	5.2	4.1	3.6	5.0	3.7	1.9

[a] Data analysed by 1-year age groups in childhood.

Note: Figures in shaded cells were estimated as outlined in the methodology section.

Table 8.20 Mean BMIs by subregion, sex and age

		Mean BMI (kg/m²) Age group (years)						
Subregion	*Sex*	*5–14*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
AFR-D	Male	14.6	19.2	21.2	21.7	20.5	20.5	20.0
	Female	14.6	20.6	22.1	21.0	21.0	20.2	20.8
AFR-E	Male	14.7	19.2	21.0	21.2	20.8	22.0	19.8
	Female	14.9	21.2	22.9	22.8	22.3	20.4	18.9
AMR-A	Male	18.5	24.1	26.4	27.6	27.4	26.7	25.1
	Female	18.8	23.8	26.1	27.7	27.5	26.9	25.0
AMR-B	Male	16.7	22.9	25.0	25.6	25.5	26.1	24.7
	Female	17.3	23.5	26.2	27.3	27.1	26.9	25.5
AMR-D	Male	15.8	23.0	25.3	25.9	26.0	26.3	26.3
	Female	16.1	23.7	25.8	26.6	26.8	26.6	26.2
EMR-B	Male	17.8	22.0	24.6	25.3	24.3	23.1	23.5
	Female	17.9	22.5	25.8	26.5	25.5	23.3	26.0
EMR-D	Male	14.7	20.0	21.8	21.9	21.6	21.0	20.1
	Female	14.9	21.6	23.8	22.8	22.3	21.3	18.9
EUR-A	Male	17.6	24.2	26.3	27.2	27.8	27.5	26.1
	Female	17.9	23.3	25.1	27.5	28.4	27.8	25.8
EUR-B	Male	17.1	21.7	25.0	26.5	27.3	25.8	24.8
	Female	17.0	22.7	25.6	27.9	28.8	29.0	27.5
EUR-C	Male	18.0	22.9	25.2	26.1	25.9	25.2	24.8
	Female	17.6	22.7	26.5	28.4	28.7	27.2	25.4
SEAR-B	Male	14.7	20.2	22.6	23.4	23.0	22.6	22.6
	Female	15.0	20.5	22.7	23.9	24.3	22.5	22.5
SEAR-D	Male	14.4	18.6	19.6	19.0	19.2	18.0	19.4
	Female	15.0	19.5	20.8	21.4	18.9	19.6	16.0
WPR-A	Male	17.7	22.2	23.7	23.9	23.3	22.8	21.9
	Female	17.2	20.8	22.5	23.8	23.9	23.3	22.6
WPR-B	Male	14.7	20.8	22.8	23.1	22.8	22.3	20.8
	Female	15.0	21.9	22.8	23.5	23.6	22.6	21.1

Note: Figures in shaded cells were estimated as outlined in the methodology section.

and BMI during childhood development. The data on mean BMIs are presented in Table 8.20. Table 8.21 contains the standard deviations for these estimates, while Table 8.22 lists the number of subjects measured and used in this analysis, in order to provide a preliminary perspective on the validity of these estimates.

4.7 Final estimates of the prevalence of overweight and obesity, by age, sex and subregion

Table 8.23 shows the subregional prevalences of overweight, as defined by a BMI of between 25.0 and 29.9 kg/m². For the age groups 5–14 years

Table 8.21 The standard deviations of the mean BMI estimates by subregion, sex and age

		Age group (years)						
Subregion	*Sex*	*5–14*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
AFR-D	Male	1.5	2.0	3.4	5.4	2.9	2.9	2.7
	Female	1.6	3.5	3.9	4.6	3.8	3.6	2.3
AFR-E	Male	1.6	2.9	5.2	4.6	4.2	3.6	1.8
	Female	1.7	3.9	4.8	5.2	5.1	5.1	2.4
AMR-A	Male	3.8	4.8	5.0	5.0	4.4	4.3	4.1
	Female	4.3	5.7	7.1	6.5	6.1	5.8	4.9
AMR-B	Male	2.9	4.0	4.1	4.3	4.0	3.9	3.8
	Female	3.4	4.7	5.1	5.3	5.2	5.2	4.3
AMR-D	Male	1.2	2.5	3.8	4.0	3.5	3.5	3.5
	Female	1.7	3.1	4.4	4.6	4.1	4.1	4.1
EMR-B	Male	2.9	3.7	4.1	3.9	4.0	3.7	3.6
	Female	3.3	4.4	5.1	5.3	4.8	3.7	5.8
EMR-D	Male	1.6	4.9	6.9	4.8	5.8	5.8	5.8
	Female	1.8	6.3	8.5	6.5	7.4	7.4	7.4
EUR-A	Male	2.6	3.7	3.7	3.9	3.8	3.8	3.7
	Female	2.6	4.1	4.7	4.7	5.2	4.9	4.3
EUR-B	Male	2.6	3.1	3.9	3.8	3.7	3.6	4.8
	Female	2.5	3.9	5.2	6.0	5.1	4.7	4.8
EUR-C	Male	2.9	2.8	3.5	3.9	3.8	3.9	3.9
	Female	2.8	3.9	5.0	5.1	5.2	5.1	5.0
SEAR-B	Male	1.6	2.2	2.8	2.3	3.9	2.5	2.5
	Female	1.8	3.5	2.3	2.1	4.1	4.5	4.5
SEAR-D	Male	1.1	2.0	3.2	2.8	2.9	2.7	3.1
	Female	1.6	3.0	4.0	4.6	3.7	5.9	2.0
WPR-A	Male	2.2	3.4	3.4	3.2	3.2	3.2	3.6
	Female	1.7	3.5	4.1	3.7	3.7	3.8	3.7
WPR-B	Male	1.6	2.6	3.3	3.1	3.3	3.6	1.8
	Female	1.8	4.2	4.1	3.6	5.0	3.7	1.9

Note: Figures in shaded cells were estimated as outlined in the methodology section.

and 15–29 years, these prevalence figures can be used in their condensed form because allowances have already been made for the age- and sex-specific cut-off points in the BMI percentiles corresponding to a BMI of 25.0 kg/m^2 at age 18 years. The rather crude nature of the calculation of BMI does not take into account the different ages at which pubertal changes occur in different subregions of the world; but the figures for prevalence are more robust and usable than the data on mean BMI described earlier, given the implications of definitions and cut-offs for overweight and obesity among adolescents.

Table 8.22 The number of subjects used when estimating the mean and standard deviation in each age category for Tables 8.20 and 8.21

		Age group (years)						
Subregion	*Sex*	*5–14*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
AFR-D	Male	1254	2394	2008	1625	474	236	32
	Female	1180	4333	3695	1893	450	196	24
AFR-E	Male	3120	3899	1969	1295	879	333	18
	Female	3340	9099	5687	2265	1514	519	4
AMR-A	Male	2822	8585	11133	5286	2530	1773	695
	Female	2944	13641	14664	6405	2455	1825	781
AMR-B	Male	59127	39400	5590	3829	798	13	—
	Female	57726	38066	7290	5603	865	17	—
AMR-D	Male	1378	204	—	—	—	—	—
	Female	1325	7120	5652	337	—	—	—
EMR-B	Male	1000	4761	3717	2060	1170	227	102
	Female	1271	5541	3617	2193	1099	267	70
EMR-D	Male	—	8535	7626	4641	2482	1870	—
	Female	—	12129	11256	5103	2354	1418	—
EUR-A	Male	8549	8935	13341	14339	4471	2581	359
	Female	9215	10132	14966	16798	5615	3413	566
EUR-B	Male	4063	3472	1402	2381	499	298	52
	Female	4240	6138	3184	3411	1012	353	63
EUR-C	Male	1202	1966	2776	1949	860	210	79
	Female	1150	2171	3349	2415	1442	561	233
SEAR-B	Male	—	503	395	215	93	27	13
	Female	—	1273	988	423	105	20	12
SEAR-D	Male	392	919	765	412	159	36	5
	Female	392	2430	2224	790	134	38	3
WPR-A	Male	949	5074	7784	7676	4303	2191	482
	Female	895	5557	9390	9418	5179	3023	767
WPR-B	Male	—	3014	2636	2406	1397	—	—
	Female	—	4307	4026	3499	1882	—	—

— No data.

The prevalences of obesity corresponding to the subregional groupings by sex and age are listed in Table 8.24. Again, the accepted WHO criterion is taken, i.e. BMI ≥30 kg/m^2, and children have been assessed in relation to the cut-off percentile for BMI proposed by IOTF. This percentile varies by age and sex during childhood as growth and pubertal changes occur, but these collated values provide an overall estimate of the prevalences of substantial excess weight in both children and adults.

The estimated standard errors for the prevalences of overweight and obesity are given in Tables 8.25 and 8.26.

Table 8.23 The prevalence of overweight in children and adults, by subregion, sex and age

		Prevalence of overweight (%) Age group (years)						
Subregion	*Sex*	*5–14*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
AFR-D	Male	0.0	1.3	3.7	11.0	4.9	6.7	0.7
	Female	0.2	5.0	11.7	8.6	13.7	7.4	0.8
AFR-E	Male	0.4	0.1	0.5	1.3	1.9	0.7	0.0
	Female	0.7	5.9	8.4	7.1	1.2	1.8	0.0
AMR-A	Male	17.0	26.9	41.8	43.9	45.9	43.6	42.7
	Female	19.0	19.3	22.6	28.5	34.6	33.4	35.0
AMR-B	Male	19.5	30.1	45.3	47.2	43.9	42.3	42.8
	Female	19.8	28.0	39.0	39.5	40.2	31.1	29.9
AMR-D	Male	16.8	24.0	33.8	37.2	38.8	40.6	40.6
	Female	15.0	26.3	38.0	41.2	32.3	31.5	30.0
EMR-B	Male	15.1	18.1	33.1	37.2	23.8	25.5	36.4
	Female	17.7	17.9	39.5	37.8	25.1	24.0	25.0
EMR-D	Male	0.3	4.8	11.4	11.9	10.4	7.9	5.0
	Female	0.7	12.2	22.6	23.9	13.9	9.1	0.0
EUR-A	Male	15.3	29.7	47.4	53.2	52.8	57.2	46.9
	Female	18.0	17.3	28.1	39.4	41.9	50.0	37.7
EUR-B	Male	16.9	14.3	37.8	42.7	40.8	43.9	28.8
	Female	15.0	17.6	30.8	32.1	32.8	42.9	36.5
EUR-C	Male	20.0	20.5	37.1	41.6	39.3	42.2	35.8
	Female	16.9	17.1	33.6	37.8	38.1	36.3	31.0
SEAR-B	Male	0.3	2.7	19.0	27.0	24.7	17.5	17.5
	Female	0.7	5.5	20.3	34.6	37.1	21.9	21.9
SEAR-D	Male	0.0	0.0	6.6	9.4	5.4	4.9	0.0
	Female	0.0	3.9	12.9	18.8	8.7	6.8	0.0
WPR-A	Male	17.2	17.2	27.3	28.8	24.0	20.9	14.4
	Female	19.3	9.4	15.8	25.0	28.5	26.3	22.3
WPR-B	Male	1.5	13.8	24.3	27.0	28.3	13.6	9.0
	Female	1.4	10.5	18.7	21.8	21.6	14.0	9.6

Note: Figures in shaded cells were estimated as outlined in the methodology section.

5. Risk factor–disease relationships

This section reviews evidence for causality relating BMI to different disease and injury outcomes; provides a rationale for choosing 21.0±1.0 kg/m^2 (mean±SD) as the BMI value of theoretical population minimum-risk of adverse health effects; and summarizes the sources of data for the hazard estimates required for estimates of the attributable fraction for the population. To date, there have been no systematic reviews of cohort studies that present age- and sex-specific associations of adverse health outcomes with BMI as a continuous variable (rather than for

Table 8.24 The estimated prevalences of obesity, by subregion, sex and age

		Prevalence of obesity (%) Age group (years)						
Subregion	*Sex*	*5–14*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
AFR-D	Male	0.2	0.0	0.0	0.3	1.2	0.3	0.0
	Female	0.1	0.9	2.7	0.9	2.8	0.4	0.0
AFR-E	Male	0.2	0.0	0.1	0.0	0.0	0.4	0.0
	Female	0.1	0.5	2.5	2.1	0.9	0.4	0.0
AMR-A	Male	8.4	11.5	19.0	24.2	23.9	19.5	8.0
	Female	9.4	12.7	23.7	32.2	28.9	24.3	15.0
AMR-B	Male	7.1	9.2	18.5	22.2	19.7	20.0	20.7
	Female	5.7	11.8	28.5	38.5	32.8	22.6	20.8
AMR-D	Male	3.2	3.3	9.0	12.1	16.3	18.1	18.1
	Female	2.6	5.6	14.6	19.4	24.6	23.3	20.8
EMR-B	Male	6.1	3.7	6.8	8.9	6.2	3.0	9.1
	Female	3.3	6.4	14.5	17.1	18.0	15.8	25.0
EMR-D	Male	0.2	0.9	1.7	1.7	2.0	2.5	0.0
	Female	0.1	4.4	12.4	13.9	7.3	4.6	0.0
EUR-A	Male	9.4	6.9	14.4	18.7	22.7	19.2	11.6
	Female	13.2	9.5	13.5	22.5	31.6	34.3	15.7
EUR-B	Male	3.2	2.4	11.0	15.1	15.0	12.6	13.5
	Female	2.6	7.2	22.8	31.6	32.3	39.5	33.3
EUR-C	Male	5.7	1.9	9.2	14.7	14.7	8.9	9.0
	Female	3.7	5.7	22.2	35.4	36.4	26.8	18.2
SEAR-B	Male	0.2	0.5	2.3	2.3	2.2	7.5	7.5
	Female	0.1	0.6	3.1	5.5	5.7	9.4	9.4
SEAR-D	Male	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	Female	0.0	0.8	2.9	4.9	0.8	0.2	0.0
WPR-A	Male	8.4	7.2	4.7	4.5	3.7	3.3	1.9
	Female	9.4	3.5	4.5	5.7	6.5	5.3	3.5
WPR-B	Male	0.4	1.2	4.8	6.5	7.6	0.4	0.0
	Female	0.3	1.7	6.7	9.7	10.0	3.4	1.1

Note: Figures in shaded cells were estimated as outlined in the methodology section. The obesity rates in children were particularly variable in some subregions, e.g. EUR-B, because they were based on relatively small samples and the obesity cut-off point reflects an extreme percentile distribution of BMIs.

cut-off points, so that the full impact can be captured), and all affected outcomes and for different subregions. Therefore some trade-offs had to be made to obtain the best estimates of hazard size for this project. The following analysis is based on a series of systematic reviews, the two principal ones being of BMI and vascular disease, and BMI and cancer.

Table 8.25 The estimated standard errors of the measured or predicted prevalence of overweight, by subregion, sex and age

		Standard errors of the prevalence of overweight *Age group (years)*						
Subregion	*Sex*	*5–14*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
AFR-D	Male	0.05	1.74	2.24	2.47	2.37	3.65	2.05
	Female	0.21	0.84	0.99	1.62	2.43	3.26	2.70
AFR-E	Male	0.27	0.75	1.11	2.22	2.54	2.27	—
	Female	0.16	0.71	1.19	2.21	1.28	1.52	—
AMR-A	Male	2.29	2.32	2.00	2.31	2.59	2.57	2.40
	Female	2.39	1.34	0.80	1.06	1.33	1.11	1.50
AMR-B	Male	5.30	3.42	2.26	2.60	2.80	2.89	—
	Female	5.50	1.54	0.82	0.98	0.84	0.84	—
AMR-D	Male	3.15	0.61	—	—	—	—	—
	Female	2.87	1.50	1.60	6.45	—	—	—
EMR-B	Male	5.31	3.92	8.02	3.50	3.72	4.14	14.50
	Female	5.51	2.52	2.42	2.86	2.98	3.36	21.60
EMR-D	Male	0.26	0.00	—	—	—	—	—
	Female	0.16	1.46	1.10	5.00	—	—	—
EUR-A	Male	2.85	2.32	1.66	1.64	2.59	2.04	2.90
	Female	2.75	1.92	1.01	1.23	2.23	1.65	2.60
EUR-B	Male	3.15	2.15	3.03	3.79	5.05	7.50	6.30
	Female	2.84	1.09	1.24	2.27	4.30	5.75	6.10
EUR-C	Male	3.74	2.28	1.70	1.95	4.50	12.65	11.65
	Female	3.49	2.05	1.35	1.75	4.30	6.55	7.85
SEAR-B	Male	0.25	0.64	2.00	3.00	4.50	7.30	10.50
	Female	0.15	0.56	1.30	2.30	4.70	9.20	11.90
SEAR-D	Male	0.00	3.32	2.52	3.20	4.21	—	—
	Female	0.00	0.85	1.58	1.80	2.00	4.90	—
WPR-A	Male	2.32	2.23	1.72	1.78	2.11	2.43	3.59
	Female	2.42	1.36	0.85	0.95	1.19	1.36	2.19
WPR-B	Male	0.24	2.25	2.60	2.90	2.71	2.80	3.18
	Female	0.14	0.65	0.82	0.84	0.89	0.96	0.87

— No data.

Note: Figures in shaded cells were estimated as outlined in the methodology section.

5.1 Evidence for causality

Traditionally, excess body weight and obesity have been considered as risk factors in epidemiological terms, despite the fact that the International Statistical Classification of Diseases (ICD) has specified obesity as a disease in its own right since 1948. The effects of excess weight as a risk factor are at least partly mediated through changes in other risk

Table 8.26 The estimated standard errors of the measured or predicted prevalences of obesity, by subregion, sex and age

		Standard errors of the prevalence of obesity *Age group (years)*						
Subregion	*Sex*	*5–14*	*15–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
AFR-D	Male	0.11	0.53	0.67	0.89	0.85	1.10	—
	Female	0.11	0.43	0.73	1.21	1.00	1.03	—
AFR-E	Male	0.27	0.35	0.66	0.35	0.38	1.51	—
	Female	0.16	0.34	0.90	1.89	2.06	1.48	—
AMR-A	Male	1.80	1.19	0.90	1.14	1.57	1.62	1.20
	Female	1.90	1.09	0.99	0.92	1.08	1.05	1.40
AMR-B	Male	3.40	1.52	1.09	1.61	1.32	0.95	—
	Female	2.10	0.92	0.74	0.94	0.79	0.63	—
AMR-D	Male	1.01	0.11	—	—	—	—	—
	Female	1.31	0.73	1.45	5.50	—	—	—
EMR-B	Male	3.40	1.99	2.35	2.40	2.35	2.68	8.70
	Female	2.10	1.58	2.31	3.14	3.42	3.46	25.0
EMR-D	Male	0.26	0.00	—	—	—	—	—
	Female	0.16	0.77	1.20	4.10	—	—	—
EUR-A	Male	1.71	1.86	1.46	2.29	2.36	1.42	2.25
	Female	1.98	1.84	0.99	1.21	2.21	1.39	2.25
EUR-B	Male	1.00	0.78	2.76	2.40	4.40	4.30	4.70
	Female	1.29	0.49	1.04	2.37	4.15	5.55	5.90
EUR-C	Male	1.94	0.86	1.25	1.65	4.10	2.10	7.50
	Female	1.37	0.97	1.15	1.75	4.25	6.05	9.00
SEAR-B	Male	0.25	0.24	0.70	1.00	1.50	5.10	7.30
	Female	0.15	0.16	0.60	1.10	2.30	6.50	8.40
SEAR-D	Male	0.00	0.00	—	—	—	—	—
	Female	0.00	0.23	0.50	0.80	—	—	—
WPR-A	Male	1.80	1.15	0.91	1.04	1.26	1.46	2.05
	Female	1.90	1.02	0.74	0.89	1.09	1.24	1.74
WPR-B	Male	0.24	0.70	0.85	1.17	1.11	0.90	—
	Female	0.14	0.46	0.70	1.08	1.14	1.00	0.63

— No data.

Note: Figures in shaded cells were estimated as outlined in the methodology section.

factors, such as blood pressure and abnormal blood lipids. More recently, it has become clear that excess weight is not only of value in predicting the risk of suffering from particular diseases, but that intentional weight loss reduces these intermediate risk factors (such as high blood pressure); and experimental evidence is also emerging for reduced disease outcomes (Sjöström et al. 1999). This experimental evidence provides the strongest evidence of causality and is summarized in the following sections. The observational evidence from cohort studies linking

Table 8.27 Health outcomes considered in relation to excess body-weight gain

	ICD revision				
Disease	*ICD-6/7*[a]	*ICD-8*[b]	*ICD-9 BTL*	*ICD-9*	*ICD-10*
Ischaemic heart disease	A081	A083	B27	410–414, Proportion of: 427.1, 427.4, 427.5, 428, 429.0–429.2, 429.9, 440.9	I20–I25
Cerebrovascular disease	A070	A085	B29	430–438	I60–I69
Hypertensive disease	A083, A084	A082	B26	401–405	I10–I13
Type II diabetes	…	…	…	…	E11
Osteoarthritis	…	…	…	715	M15–M19
Colon and rectum cancers	A047, A048	A048, A049	B093, B094	153, 154	C18–C21
Breast cancer (females only)	A051	A054	B113	174	C50
Endometrial cancer	A053	A056	B122	179, 182	C54–C55

BTL Basic tabulation list.

… Not available.

[a] Intermediate list of 150 causes.

[b] List A: list of 150 causes.

BMI to adverse health outcomes is also described in more detail in the subsequent sections, as these studies provided estimates of exposure–disease hazard size. Together, these sources of data show that the associations found between BMI and many disease outcomes satisfy the widely-accepted criteria for causal relationships: they are strong, consistent, have a dose–response relationship and are biologically plausible (Hill 1965).

5.2 Health outcomes caused by excess body weight

The health outcomes that were considered are presented in Table 8.27; the causal relationship between excess body weight and these conditions is dealt with later.

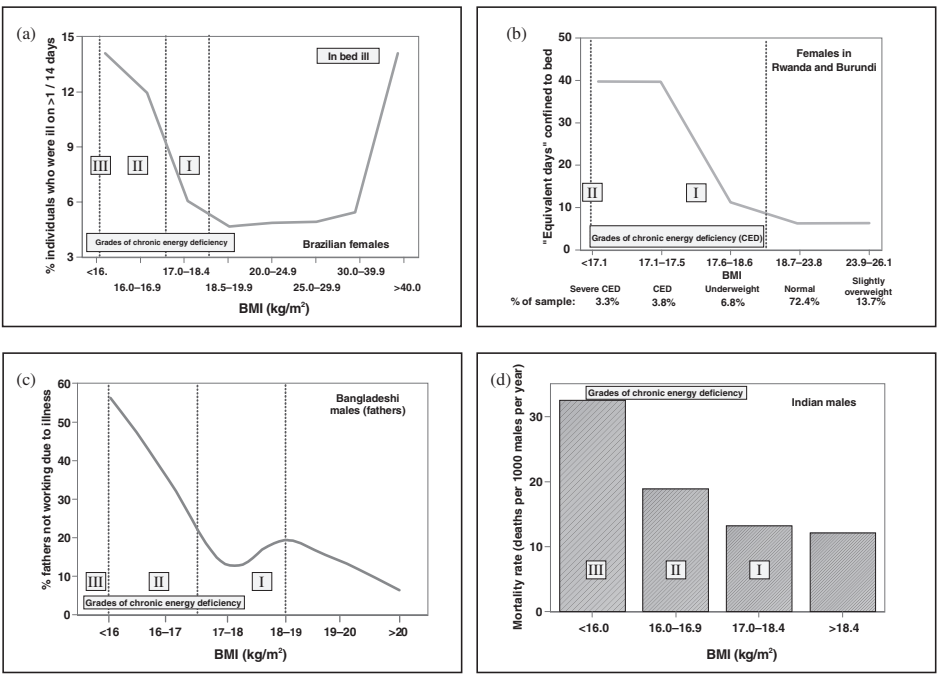
5.3 The optimum population BMI (theoretical-minimum-risk)

The theoretical-minimum-risk distribution of BMI in the population is that which is associated with the lowest health risks related to BMI. This choice of the theoretical minimum needs to take into account the fact that there are hazards associated with low as well as high BMIs (Shetty

and James 1994). The optimum trade-off is based on a balance between the level down to which the risk of developing diseases associated with high BMI persists (described in subsequent sections, but generally 20 kg/m^2, once the confounding effects of smoking and co-morbid prevalent diseases are accounted for) and the health hazards and reduced physical capacity found at lower BMIs. The handicaps related to underweight stem from a chronic dietary energy deficit and other phenomena related to undernutrition. These are summarized in Figures 8.5 and 8.6.

Analyses have been made (James and Francois 1994) of the relationship between the proportion of adults in the population who are underweight (i.e. BMI of <18.5 kg/m^2) and the median BMI of the population. This definition of underweight was accepted by WHO in both the 1995

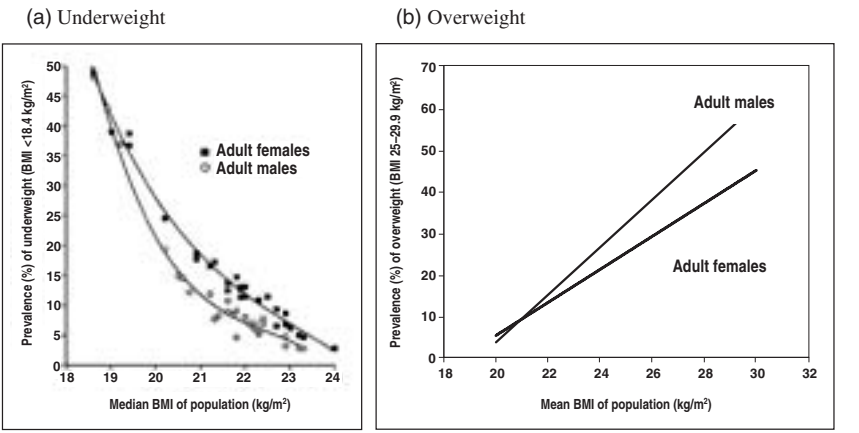
Figure 8.5 Morbidity and mortality at lower BMIs



Note: Data for Figure 8.5 were collated by Shetty and James (1994).

Source: (a) de Vasconcellos, based on data presented at a meeting "Functional Significance of Low Body Mass Index (BMI)", Rome 4–6 November, 1992. Fig. 6.3: BMI and the probability of illness among Brazilian women (PNSN Survey 1989, de Vasconcellos, personal communication); (b) P. Francois, unpublished data, 1990. BMI and "equivalent days" of illness from collated times of impaired activity among women in Rwanda and Burundi, 1982; (c) Adapted from J. Pryer, 1990. BMI and loss of labour days due to illness in Bangladesh; (d) Satyanarayana et al. 1991. Mortality rates for men according to BMI categories (Hyderabad).

Figure 8.6 Median or mean BMI and the prevalence of (a) underweight and (b) overweight in different populations



Source: (a) James and Francois (1994); (b) Regression lines taken from Figure 8.1.

report on the uses of anthropometry (WHO 1995) and in the more recent report on obesity (WHO 2000). Figure 8.6(a) presents unpublished representative data obtained from 27 large national surveys of developing countries. By specifying underweight adults as those with a BMI of <18.5 kg/m^2 it becomes clear that an optimum median BMI needs to be about 22.0 or more to ensure that fewer than about 10% of women are underweight (see below).

In order to compare the proportions of people with low and high BMIs in the population at different mean BMIs, data relating underweight (i.e. BMIs of <18.5 kg/m^2), to overweight, (i.e. BMIs of ≥25.0 kg/m^2), are required. To simplify this comparison, the two figures are presented side by side in Figure 8.6, but it should be noted that they are drawn from different data sets and that the data relating to underweight are presented in relation to the median rather than the mean BMI. However, at lower BMIs the mean and median values are very similar. It can be seen that when Figures 8.6(a) and (b) are compared, a BMI of 21.0–22.0 kg/m^2 emerges as an optimum BMI at which the chances of there being substantial proportions of either underweight or overweight people in the population are minimized. At a BMI of about 21 kg/m^2, the minimum proportion of underweight and overweight people in the population is about 10% for males, according to Figure 8.6(a), and for both sexes, according to Figure 8.6(b). To achieve a proportion of only 10% of women being underweight requires that the mean BMI of the population be about 22.5 kg/m^2. However, at this mean BMI already

about 15% of the female population is overweight, according to Figure 8.6(a). Given the clear evidence for health hazards in women, even at BMIs of 23 kg/m^2 and above (Willett et al. 1995), and the modest handicaps currently evident for women within the first grade of underweight, i.e. BMIs of 17.0–18.4 kg/m^2 (James et al. 1988), we concluded that at present a universal mean BMI of 21.0 kg/m^2 should be chosen as the optimum for both sexes in populations throughout the world.

Overall, the theoretical-minimum-risk was estimated to occur at a BMI of 21.0 ± 1.0 kg/m^2 (mean ± SD). It should be noted that this is below the level which some cohort studies have estimated to be the nadir of associations with mortality. This is because of the corrections for effects of disease on BMI that have been employed here and the focus on non-fatal as well as fatal events. This optimum BMI is similar to the lower limit of the range proposed by the WHO Technical Consultation on Obesity (WHO 2000), i.e. 21.0–23.0 kg/m^2. The inclusion of the upper limit of 23.0 kg/m^2 in the original WHO Technical Consultation stemmed from a concern that in some developing countries there might be a need for higher reserves of body energy to cope with potential natural disasters and crop failures leading to food deprivation. In practice, as will become evident, the increase in the rates of diseases associated with increases in BMI is evident from BMIs of about 20 kg/m^2.

5.4 Body weight and cardiovascular disease

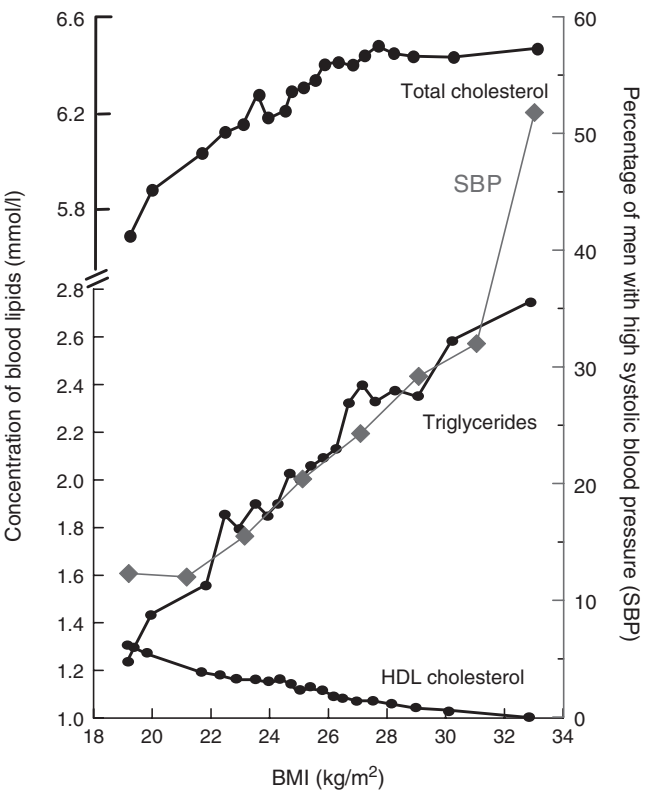
Causal relationships between excess weight and cardiovascular disease

Non-optimum levels of blood pressure, cholesterol and glucose are leading causes of cardiovascular disease. There is strong evidence that excess body weight is associated with adverse levels of these risk factors. A recent Cochrane systematic review focusing on randomized controlled trials (Mulrow et al. 2001) identified 18 trials totalling 2611 hypertensive participants with an average body weight of 84 kg. The data suggested that weight loss in the range of 4–8% of body weight produced an average reduction in systolic blood pressure of 3.0 mmHg, consistent with earlier reviews (Goldstein 1992; MacMahon et al. 1987). It has also been clearly shown in meta-analyses of intervention trials that losing excess weight improves blood lipid profiles (Dattilo and Kris-Etherton 1992), with a fall in total serum cholesterol and triglyceride levels, and an increase in high density lipoprotein (HDL) concentrations. It is reasonable to expect that such reductions in major intermediate risk factors would translate into reduced incidence of cardiovascular disease. Direct evidence from randomized trials on clinical outcomes is limited, owing to the challenges in the modern environment to achieve and maintain weight loss. Nonetheless, evidence is emerging for substantial reductions in diabetes incidence in trials which have weight loss as a major feature of the intervention in individuals at high risk. A detailed discussion can

be found in, for example, Williamson and Pamuk (1993) and in the latest publications from Sweden, where very large numbers of individuals are currently in the middle of long-term trials of the health impact of weight loss induced by surgical reconstruction of the intestine (Sjöström et al. 1999, 2000).

There is also evidence from prospective observational cohort studies for positive associations between BMI and a range of cardiovascular disease outcomes. These data provide the main source of estimates of hazard size in this analysis and are summarized in the relevant following sections. Some further insight into causality from selected cross-sectional and prospective studies is summarized in Figure 8.7, which

Figure 8.7 The relationship between BMI, high blood pressure (systolic pressure ≥160 mmHg) and concentrations of blood lipid in men aged 40–59 years in the United Kingdom



Source: Adapted from Shaper et al. (1997).

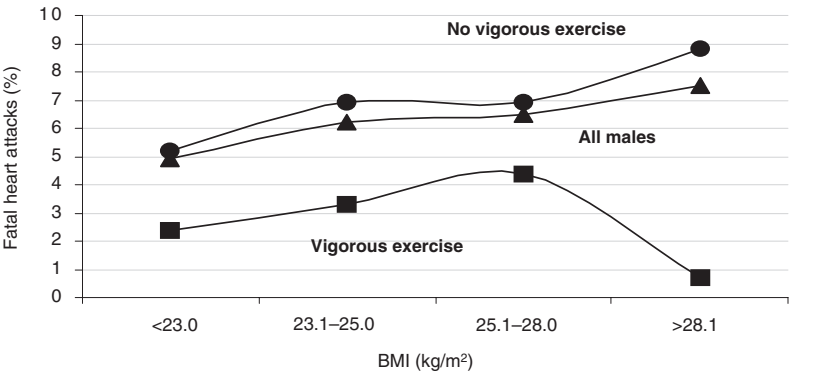
shows a progressive rise in total cholesterol with increasing BMI, from a BMI of 20 kg/m^2, and a sustained fall in HDL from BMI 20–30 kg/m^2. In addition, there is a clear association between high BMI and increases in serum triacylglyceride (triglyceride) concentration. Many studies have shown that there is a potential independent additional risk of ischaemic heart disease with increases in triglyceride concentrations.

The mechanisms by which increased body weight leads to the induction of cardiovascular diseases and excess mortality are not always clear. The effect is partly related to the frequent concomitant lack of physical fitness and physical activity in the overweight, but it is generally accepted that body-weight gain *per se* enhances insulin resistance, and thus physical inactivity is not the sole explanation. The development of insulin resistance is a powerful predictor of excess levels of triglycerides in the blood and of the propensity to develop type II diabetes.

In the INTERSALT study (Dyer et al. 1989), a significant association was found between BMI and systolic and diastolic blood pressures, which was independent of age, alcohol intake, smoking habits and urinary sodium and potassium excretion (this excretion rate being taken as an index of intake). Other cross-sectional analyses with measurements of BMI, blood pressure and lipids in West Africa, the Caribbean and the United States have also clearly shown increases in BMI associated with rising blood pressure (Wilks et al. 1996). Several mechanisms may explain why changes in body weight lead to alterations in blood pressure. Physical activity is one contributor, with inactivity tending to promote both weight gain and blood pressure increases. Weight change induced by diet without altering physical activity, however, also leads to changes in blood pressure. The mechanisms by which weight gain promotes a rise in blood pressure may involve the accentuation of insulin resistance, increases in the tone of the sympathetic nervous system control of the arterioles and the production by the adipose tissue itself of a variety of vasoactive cytokines and hormones, such as angiotensinogen, which increase blood pressure (Gorzelniak et al. 2002). These vasoactive compounds act in part by reducing sodium excretion by the kidney, thereby increasing the blood volume and therefore blood pressure.

It is well recognized that physical inactivity is an important contributor to body-weight gain and also increases the risk of diabetes, cardiovascular disease and some cancers. Data on the risks of excess BMI stratified by levels of physical inactivity (e.g. Figure 8.8 and also Paffenbarger et al. 1970) show that the hazards of high BMI are present at all levels of physical activity. This indicates that physical inactivity does not account for the full relationship between BMI and disease.

Figure 8.8 BMI, vigorous exercise and incidence of fatal heart attacks among male British civil servants



Note: This study was based on observations of 17 944 male British civil servants aged 40–65 years who self-reported at survey between 1968 and 1970. BMI values were taken at age 40–59 years. Average follow-up was 8.5 years. Death certificates were supplied by the National Health Service Central Register. Rates were standardized for age. Note that the group with the highest BMI and reporting vigorous exercise was "fewer than five people".

Source: Adapted from Morris et al. (1980) (Table III p. 1209). Morris defined vigorous exercise as >6 METS (metabolic equivalents, ml oxygen/kg min^{-1}) or 7.5 kcal min^{-1}, e.g. swimming, hill climbing, gardening, brisk walking, for longer than 30 minutes per day.

Systematic review of BMI and cardiovascular disease outcomes: the Asia-Pacific Cohort Studies Collaboration (APCSC)

To date, the only available systematic review providing age-, sex- and outcome-specific hazard size as a continuous variable is the APCSC, which included data from 33 cohorts (12 studies from Japan, 11 from mainland China, two from Singapore, two from Taiwan [China], one from Hong Kong Special Administrative Region of China [Hong Kong SAR], one from the Republic of Korea, one from New Zealand and three from Australia) (Table 8.28). The heights and body weights of individual participants were measured in all studies and BMI was calculated. Data from participants recorded as having a BMI of <12 kg/m^2 or >60 kg/m^2 were excluded from the analysis.

There was evidence of confounding due to disease at baseline and therefore health events that occurred within the first 3 years of follow-up were excluded from all analyses. Smokers were not excluded, but smoking was included as a covariate in all analyses. Participants were categorized as "smokers" if they classed themselves as current smokers, former smokers or ever-smokers (people who have smoked at any time), and as "non-smokers" if they indicated that they had never smoked.

Some cohorts included a smoking category entitled "not current"; participants in this category were excluded from the analyses since it was not possible to determine if they were former smokers or never-smokers.

In total, 310 283 participants contributed 2 148 354 person-years of follow-up with a mean duration of 6.9 years. Data on baseline BMI, smoking habits and >3 years of follow-up were available for these participants. The mean age of the participants at baseline was 47 years and 41% were female. Ten per cent of participants were from Japan, 15% were from mainland China, 55% were from other parts of Asia (Singapore, Taiwan [China], Hong Kong SAR and the Republic of Korea), and 20% were from Australia and New Zealand (ANZ). The contributing data sets are set out in Table 8.28.

The overall mean baseline BMI was 23.6 kg/m^2. The mean BMI for the Asian populations was 22.9 kg/m^2 while that for the ANZ populations was 26.4 kg/m^2. Table 8.29 presents the calculated increments in risk of cardiovascular disease associated with a one unit (1 kg/m^2) decrease in BMI and Figure 8.9 summarizes the relationship of BMI with all ischaemic heart disease events (adjusted for age, sex, cohort and smoking).

Figure 8.9 The relationship between BMI and all ischaemic heart disease events in the Asia-Pacific Cohort Studies Collaboration

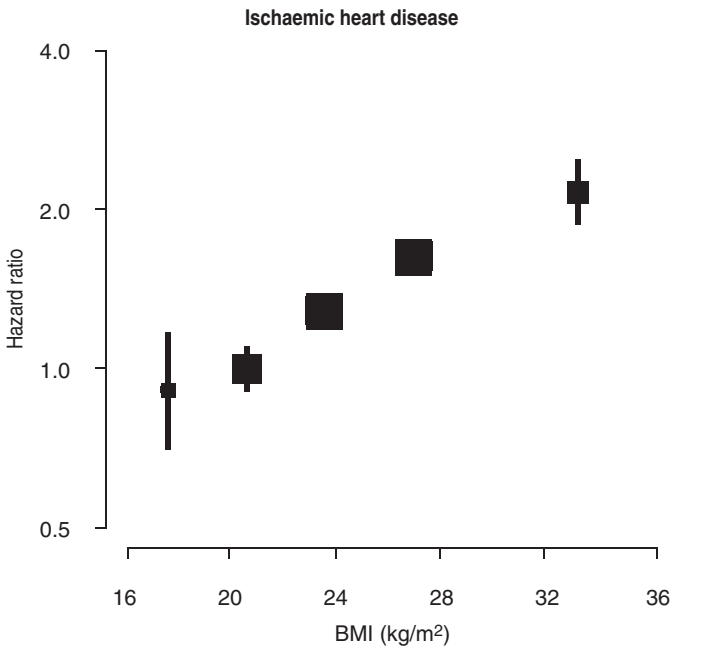


Table 8.28 Asia Pacific Cohort Studies Collaboration: cohorts contributing to the analyses of BMI

Country or area	Study name	n	Start year	Mean follow-up (years)	% female	Mean age (years)	BMI (kg/m^2)		No. of stroke events	No. of haemorrhagic stroke events	No. of ischaemic stroke events	No. of IHD events
							Mean	SD				
Australia	Busselton	7166	1966–1981	21.2	52	44	24.7	3.8	619	57	118	915
	Melbourne	41051	1990–1994	5.7	59	55	26.9	4.4	21	8	5	84
	Perth	10118	1979–1994	12.7	48	45	25.2	3.9	55	9	4	164
Mainland China	Anzhen	8165	1991	4.3	55	53	23.9	3.7	130	32	91	24
	Anzhen 02	735	1992	3.0	1	46	24.1	3.1	3	0	3	0
	Capital Iron & Steel Company	3664	1974	13.3	0	45	23.0	2.7	116	37	75	52
	CISCH	2132	1992	3.3	51	44	24.7	3.5	9	0	0	13
	East Beijing	321	1979	14.8	20	41	23.5	3.5	8	3	4	3
	Fangshan	1407	1991	3.7	67	47	24.0	3.4	15	2	9	3
	Huashan	44	1992	3.1	27	55	22.9	2.9	1	1	0	0
	Seven Cities Cohorts	10282	1987	9.8	53	52	22.1	3.2	164	112	52	37
	Six Cohorts	8824	1982	8.3	12	45	21.1	2.4	109	33	71	43
	Tianjin	4487	1984	6.0	39	53	22.9	3.8	116	43	10	36
	Yunnan	6238	1992	4.5	3	55	21.6	2.9	59	46	12	3
Hong Kong SAR	Hong Kong	842	1985	3.4	67	77	22.0	3.9	12	1	0	23
Taiwan, China	CVDFACTS	5387	1989	6.6	56	47	23.5	3.4	22	6	4	7
	Kinmen	178	1993	3.5	51	67	23.1	3.8	4	0	0	3

continued

Table 8.28 Asia Pacific Cohort Studies Collaboration: cohorts contributing to the analyses of BMI (*continued*)

Country or area	*Study name*	*n*	*Start year*	*Mean follow-up (years)*	*% female*	*Mean age (years)*	*BMI (kg/m²)*		*No. of stroke events*	*No. of haemorrhagic stroke events*	*No. of ischaemic stroke events*	*No. of IHD events*
							Mean	*SD*				
Japan	Aito Town	1124	1980	15.4	59	51	22.9	3.1	20	4	0	8
	Akabane	1804	1985	11.3	56	54	22.5	3.0	35	5	14	27
	Civil Service Workers	9020	1991	6.6	33	47	22.5	2.7	1	0	0	1
	Hisayama	1427	1961	19.9	56	55	21.6	2.7	285	55	209	78
	Konan	1033	1987	7.0	56	51	21.9	3.0	10	2	6	3
	Miyama	986	1988	6.5	56	60	22.2	3.0	4	0	2	1
	Ohasama	2155	1992	4.2	64	59	23.3	3.1	47	9	33	2
	Saitama	3534	1986	9.7	62	54	22.4	2.9	47	11	24	20
	Shibata	2207	1977	16.8	58	56	22.5	3.0	173	30	67	62
	Shigaraki Town	2457	1991	4.8	59	57	22.5	3.0	3	1	2	1
	Shirakawa	4590	1974	16.8	54	48	21.5	2.8	74	24	36	60
	Tanno/Soubetsu	737	1977	15.3	14	51	22.9	2.9	42	12	15	31
New Zealand	Fletcher Challenge	2383	1992	4.8	22	40	26.5	4.3	21	2	5	29
Republic of Korea	KMIC	160159	1990	5.5	33	44	23.0	2.5	999	295	429	256
Singapore	Singapore Heart	2326	1982	12.5	49	40	23.4	4.3	69	8	21	63
	Singapore NHS92	3300	1992	6.2	52	39	23.2	4.2	39	3	13	21
Total or average[a]		310283		6.9	41	47	23.0	3.1	3332	851	1334	2073

IHD Ischaemic heart disease.

[a] Weighted by person years of follow-up. Total person-years of follow-up is 2 148 354.

Table 8.29 Asia Pacific Cohort Studies Collaboration: summary of associations of cardiovascular disease with a one-unit (1 kg/m^2) decrease in BMI, by age

	No. of studies	*No. of participants*	*No. of events*	*Hazard ratio*[a]	*95% CI*
All ischaemic heart disease events					
30–44 years	31	137 916	73	0.89	(0.84–0.95)
45–59 years	32	205 109	504	0.91	(0.88–0.93)
60–69 years	33	76 301	511	0.95	(0.93–0.97)
70–79 years	28	28 366	576	0.96	(0.94–0.98)
≥80 years	26	5 869	414	0.97	(0.95–1.00)
Deaths from hypertensive disease					
30–44 years	31	136 265	0	1.00	(1.00–1.00)
45–59 years	32	205 007	5	0.92	(0.74–1.13)
60–69 years	33	76 272	16	0.86	(0.76–0.96)
70–79 years	28	28 446	29	0.89	(0.82–0.98)
≥80 years	26	5 979	39	0.94	(0.86–1.03)
All ischaemic stroke events					
30–44 years	31	137 917	41	0.85	(0.77–0.94)
45–59 years	32	205 109	411	0.92	(0.88–0.95)
60–69 years	33	76 345	345	0.94	(0.91–0.97)
70–79 years	28	28 449	316	0.94	(0.91–0.98)
≥80 years	26	5 967	222	0.98	(0.94–1.02)

Note: The first 3 years of follow-up were excluded and results were adjusted for age, sex, cohort and smoking habits.

[a] The age pattern of the hazard ratios presented was smoothed and the resulting age-specific estimates were used to derive the estimates of global burden of disease attributable to BMI:

Ischaemic heart disease: 0.88, 0.92, 0.93, 0.95, 0.98.

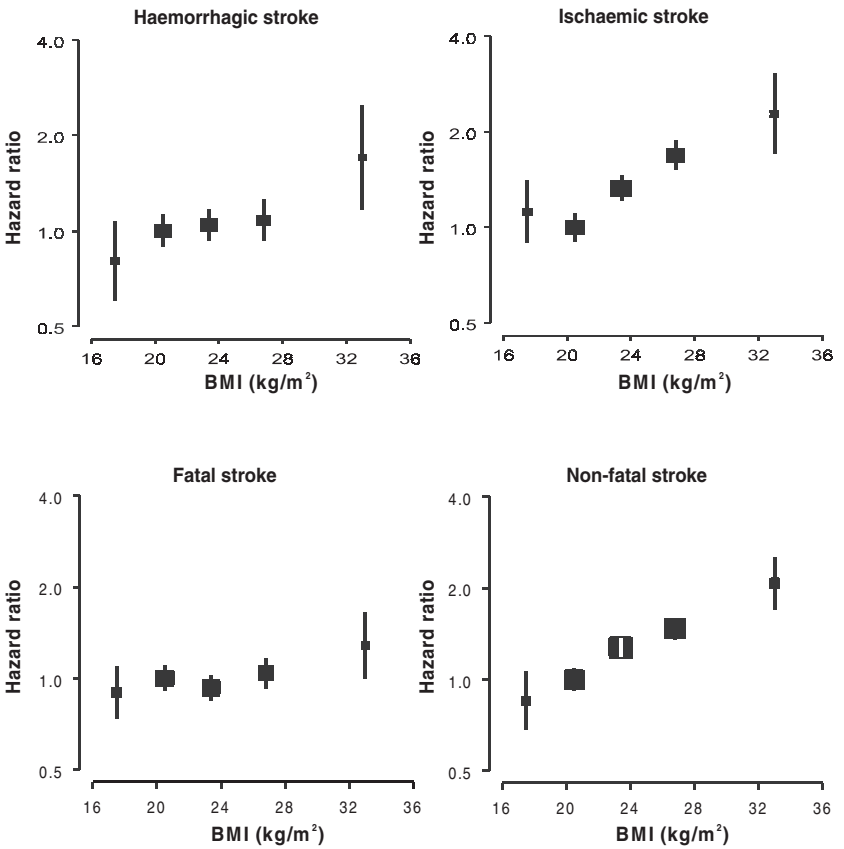
Hypertensive disease: 0.88, 0.91, 0.93, 0.95, 0.97.

Ischaemic stroke: 0.82, 0.85, 0.88, 0.90, 0.93.

The overall reduction in risk of ischaemic heart disease associated with a reduction in BMI of one unit in the age group 45–59 years in the APCSC data amounted to 9%, a very similar figure to that provided by the large North American and European prospective studies (reviewed in the next section).

The findings for stroke from the APCSC meta-analysis are displayed in Figure 8.10. A continuous relationship between increasing BMI and risk of non-fatal stroke is evident, but little association was seen between BMI and the risk of fatal stroke. In examining stroke subtypes, a continuous relationship between increasing BMI and risk of ischaemic stroke was apparent, but a weaker association was seen between BMI and the

Figure 8.10 The relationship between stroke events and BMI in adults in the cohort studies of the Asia-Pacific Cohort Study Collaboration



risk of haemorrhagic stroke. This suggests that the lack of association seen between BMI and fatal stroke may reflect in part the higher proportion of fatal strokes that are of the haemorrhagic subtype. Therefore estimates of global burden of disease attributable to stroke in this work were based solely on ischaemic stroke for which a continuous association with BMI is evident.

Other systematic reviews of BMI and cardiovascular outcomes

A systematic review of large cohort studies investigating ischaemic heart disease has recently been completed (Whitlock et al. 2002). These studies were almost all from North America and Europe and are described in Table 8.30. Most of the studies included measured BMI and dealt with

Table 8.30 The relationship between risk of ischaemic heart disease and BMI: large North American and European prospective cohort studies[a]

Study (reference)	*Maximum no. of cases analysed*	*Type of IHD outcome*	*BMI*	*Age at recruitment (years)*	*Shape of association*	*Risk reduction per kg/m² BMI reduction (%)*[b]
Nurses' Health study (Willett et al. 1995)	1292	Fatal and non-fatal	Self-reported	30–55	Positive	11
Adventist Mortality study (Lindsted et al. 1991)	1004	Fatal	Self-reported	30–89	Positive	8
British civil servants' study (Morris et al. 1980)	977	Fatal and non-fatal	Not reported	40–59	Positive? (no significance tests)	4
British Regional Heart study (Shaper et al. 1997)	974	Fatal and non-fatal	Measured	40–59	Positive	7
Manitoba Follow-Up study (Tate et al. 1998)	915	Fatal and non-fatal	Measured	18–62	Positive or J-shaped	4
Social Insurance Institution study (Rissanen et al. 1990)	868	Non-fatal	Measured	25–64	Positive	4
Whitehall study (Jarrett et al. 1982)	727	Fatal	Measured	40–64	Positive	6
NHANES I Epidemiologic Follow-up study (Cooper and Ford 1992)	(Male) 563 (Female) 696	Fatal and non-fatal	Measured	25–75 25–75	Not significant Positive or J-shaped	(Male) 6 (Female) 2
Gothenburg men's study (Rosengren 1999)	686	Fatal	Measured	47–55	J-shaped	8
Framingham study (Higgins et al. 1988)	(Male) 659 (Female) 540	Fatal and non-fatal	Measured	35–69 35–69	Positive or J-shaped Positive or J-shaped	(Male) 5 (Female) 4
Seven Countries study (Keys et al. 1972)	632	Fatal and non-fatal	Measured	40–59	Positive or J-shaped	7
Physicians' Health study (Rexrode 2001)	548	Fatal and non-fatal	Self-reported	40–84	Positive	7
Eastern and Southwestern Finland study (Jousilahti et al. 1999)	520	Fatal and non-fatal	Measured	25–64	Positive or J-shaped	4
Honolulu Heart Program (Rhoads and Kargan 1983)	511	Non-fatal	Measured	45–68	Positive	10

IHD Ischaemic heart disease.

[a] Studies recording 500 or more cases of IHD.

[b] Adapted from Whitlock et al. (2002). The large American Cancer Society (1960–1972) cohort (Calle et al. 1999; Stevens et al. 1998) recorded self-reported height and weight. However, cause-specific associations have not been published. A positive association between risk of all cardiovascular death, steeper in younger age groups, has been shown.

non-fatal as well as fatal outcomes. All but one showed a positive or J-shaped relationship between BMI and the risk of either fatal or non-fatal ischaemic heart disease. When the potential reduction in risk was calculated for each unit decrease in BMI, results were found to be reasonably similar, with most studies indicating a 5–10% reduction in rates of ischaemic heart disease. The overall unweighted average reduction in risk per unit BMI difference was 6%, with studies using self-reported BMI giving a weighted average of 9%, whereas the weighted average for the studies using measured BMI amounted to a 5% reduction. The mean age at death for these cohorts was estimated to be age 50–60 years, indicating that these data are highly consistent with those of the APCSC outlined earlier.

The conclusions of other systematic reviews are also consistent with the APCSC results, although comparisons are limited by the use of BMI categories; the main results are summarized in Table 8.31.

5.5 Type II diabetes

Causal relationships between excess weight and type II diabetes

The relationship between excess body-weight gain and type II diabetes is now considered so strong that there is increasing use of the term "diabesity" as a unifying concept. Not only is there a close association between higher BMIs and the risk of developing type II diabetes, but weight gain itself has also been identified as a particularly important risk factor. The impact of weight gain is markedly enhanced if it occurs in young adults who were already overweight or obese when they entered adult life (Colditz et al. 1995). More direct evidence for the importance of increases in weight in the development of diabetes comes from intervention studies. Over 80% of very obese diabetic adults treated by gastric bypass surgery to induce marked weight loss, for example 30 kg, become non-diabetic and over an 8-year post-surgery follow-up period, the incidence of new cases of diabetes in these patients is minimal (Sjöström et al. 2000). Four prospective studies, three of which were randomly controlled intervention studies, have also shown that changes in diet and exercise that induce a modest loss of weight in overweight or obese subjects with glucose intolerance can markedly reduce the subsequent development of type II diabetes over periods of 3–6 years (Diabetes Prevention Program Research Group 2002; Eriksson and Lindgarde 1991; Tuomilehto et al. 2001; Xiao-Ren Pan 1997). Weight-loss trials among obese patients with type II diabetes have also shown marked improvements in diabetic states or even a return to normal glucose tolerance. Lean et al. (1990) have also shown that the degree of weight loss achieved in newly-diagnosed patients with type II diabetes predicts their future life span. Williamson and Pamuk (1993) also observed that in the United States overweight and obese women with co-morbidities have not only a reduced overall mortality but also a selective reduction in death from

Table 8.31 The relationship between BMI and the development of cardiovascular disease: analyses from two systematic reviews

Table 8.31(a) Australia

	Relative risks associated with overweight and obesity							
	Overweight (BMI 25–29.9 kg/m²)				*Obese (BMI ≥30 kg/m²)*			
	Males		*Females*		*Males*		*Females*	
	<65	*≥65*	*<65*	*≥65*	*<65*	*≥65*	*<65*	*≥65*
Ischaemic heart disease based on Harris et al. (1993, 1997); Manson et al. (1990); Rimm et al. (1995)	1.35	1.00	1.40	1.00	1.80	1.20	2.00	1.25
Ischaemic stroke based on Rexrode et al. (1997)	1.35	1.00	1.25	1.00	1.50	1.15	1.60	1.20
Hypertension based on Ascherio et al. (1992); Sjöström et al. (1992); Witteman et al. (1989)	1.40	1.40	1.40	1.40	2.35	2.35	2.35	2.35

Source: Mathers et al. (1999).

Table 8.31(b) United Kingdom

	Relative risks associated with obesity (BMI ≥30 kg/m²)	
	Males	*Females*
Myocardial infarction	3.2	1.5
Stroke	1.3	1.3
Hypertension	2.6	4.2
Angina	1.8	1.8

Note: Relative risks specified only in relation to obesity (BMI ≥30 kg/m²). Derived from 48 unspecified studies after a systematic review of 3537 studies.

Source: National Audit Office (2001).

diabetes-related conditions if they intentionally lose modest amounts of weight of up to 9 kg (Williamson and Pamuk 1993).

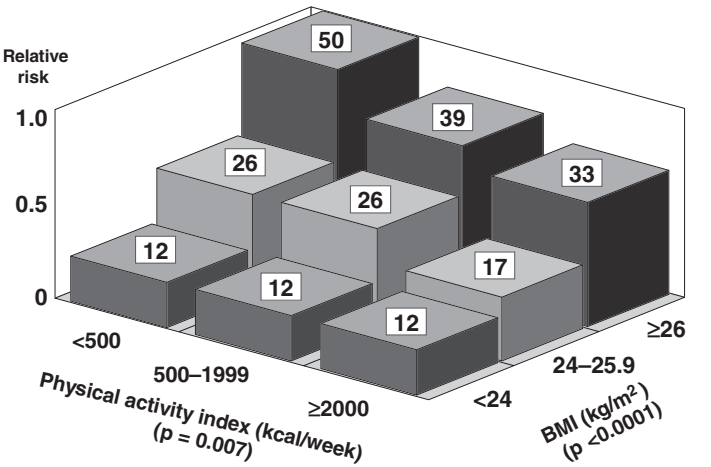
The mechanisms whereby weight gain leads to the development of type II diabetes are the subject of intense investigation, with the development of insulin resistance seen as dominant. Type II diabetes develops when the pancreatic capacity to generate insulin cannot maintain the markedly increased demand induced by insulin resistance. Insulin resis-

tance itself is affected not only by increases in weight, particularly if the extra energy is stored in abdominal, i.e. visceral, fat, but also by dietary composition. Dietary fat induces insulin resistance (Marshall et al. 1994; Sarkkinen et al. 1996; Vessby et al. 2001) and there is increasing interest in the possibility that rapidly absorbed carbohydrates, which cause sudden increases in concentrations of blood glucose, place extra demands on the pancreas (Willett et al. 2002). Adipose tissue itself, particularly visceral adipose tissue, secretes cytokines such as interleukin-6 (IL-6) and tumour necrosis factor (TNFα) which are recognized to be important inducers of insulin resistance. Circulating adiponectin, an adipocyte-derived hormone which markedly improves insulin sensitivity is reduced as the fat cells expand with body-weight gain, and is also modulated by sex hormones (Nishizawa et al. 2002). Physical inactivity also contributes to insulin resistance, with vigorous exercise leading to a rapid restoration of insulin sensitivity.

The quantitative contributions of each of these components are still uncertain, but the carefully controlled Chinese Prevention study on preventing type II diabetes (Xiao-Ren Pan et al. 1997) provides some information. In this study, three groups of overweight adults with glucose intolerance were assigned to either a low fat, low saturated fat and high vegetable and fruit diet, or to a modest increase in physical activity equivalent to 30 minutes of brisk walking daily, or were advised on both dietary and physical activity interventions. There was a marked and equivalent reduction in the incidence of type II diabetes in all three groups over the 6 years of follow-up. This implies that there is little interaction between diet and physical activity but that both can contribute to the development of insulin sensitivity.

Physical activity is potentially a major confounding effect. Physical activity is recognized as beneficial in enhancing the sensitivity of tissues to insulin, thereby enhancing the body's capacity to handle glucose. As noted above, increasing physical activity alone without dietary change reduced the incidence of diabetes in modestly overweight Chinese adults with glucose intolerance (Xiao-Ren Pan et al. 1997). This finding was consistent with those from a further two major controlled trials from Finland and the United States, which suggested that the combination of changes in diet and activity together with only modest weight losses reduced the incidence of type II diabetes in susceptible adults by 56%, this benefit increasing to 76% in those aged >60 years (Diabetes Prevention Program 2002; Tuomilehto et al. 2001). In these trials, the modest weight loss, for example 5%, was induced by a low-fat diet combined with physical activity. Given the important effect of physical activity, the quantification of any interaction between BMI and physical activity when determining the risk of onset of type II diabetes is necessary. As with coronary disease, the increase in risk of diabetes with increasing BMI is seen at all levels of physical activity (Figure 8.11).

Figure 8.11 The interaction of higher BMIs with physical activity in determining the age-adjusted incidence of type II diabetes per 10 000 person-years of follow-up, in males



Note: "Incidence" relates to the age-adjusted incidence of type II diabetes per 10 000 person-years of follow-up in male graduates of the University of Pennsylvania.

Source: Adapted from Helmrich et al. (1991).

Sources of hazard size estimates for BMI and type II diabetes

Incidence studies

The conclusions of systematic reviews from Australia (Anonymous 1999; Mathers et al. 1999) and the United Kingdom of Great Britain and Northern Ireland (National Audit Office 2001) are given in Table 8.32. In one of the Australian reports (Mathers et al. 1999), the risks of developing diabetes were arbitrarily halved in an attempt to take into account the impact of physical activity on diseases relating to obesity (see below). The first three of the four studies (Carey et al. 1997; Colditz et al. 1990, 1995; Njolstad et al. 1998) noted by this report involved very large groups of professionals in the United States (e.g. >100 000 subjects with individual prospective follow-ups of 12–18 years). Unfortunately, these data are based on self-reported heights, weights and disease. Although weights and heights reported in these study populations are found to have a good correlation with observed measurements, they are still likely to systematically underestimate the actual prevalences of overweight and obesity. The Victoria report (Anonymous 1999) looked at this issue and

Table 8.32 The relative risks of developing type II diabetes associated with overweight and obesity, as assessed by two governmental reviews

		Overweight (BMI 25.0–29.9 kg/m²)		*Obese (BMI ≥30 kg/m²)*	
Study	*Age (years)*	*Males*	*Females*	*Males*	*Females*
Australia[a] (Mathers et al. 1999)	<65	1.8	1.8	3.2	3.2
	≥65	1.8	1.8	3.2	3.2
United Kingdom (National Audit Office 2001)	All ages	—	—	5.2	12.7

— No data.

[a] Relative risk values arbitrarily halved to help account for any independent impact of physical activity or residual confounding.

found from its own analysis that "people who are obese selectively underestimated their weight and/or overestimated their height more than others. As a result, the proportion of people who are obese by measurement is 60% higher than estimates based on self-reported height and weight. The greatest discrepancies are found in adolescent men and older people". How this systematic underestimation of obesity impacts on the estimates of relative risk per BMI unit is unclear, but it may well result in overestimation, i.e. a bias away from the null. This is because the distribution of self-reported BMI is narrower than the distribution of actual BMI, and so the slope of associations between BMI and risk of disease is artificially steep. The use of self-reported disease status is another source of uncertainty in hazard estimates because in many such studies diabetes has been found to be underreported or under-diagnosed (Harris et al. 1998). If undrrecording of diabetes is associated with BMI level, this would also result in bias in hazard estimates (away from the null, if relatively more cases of diabetes failed to be identified among people with low BMI).

The prospective studies on male and female health professionals in the United States (Chan et al. 1994; Colditz et al. 1995) suggest age-adjusted relative risks of self-reported diabetes of between 4 and 14 in men aged 40–75 years and reporting a BMI of about 30 vs <23 kg/m²; whereas the relative risk for self-reported diabetes in the nurses of similar age and with reported BMIs of about 30 was about 28 compared with the rates for nurses with reported BMIs of <22 kg/m². A more robust estimate of the relative risk of developing type II diabetes comes from a detailed Norwegian study based on a sampling system which is representative of their most northern county and in which objective measurements of weight and height as well as measurements of fasting glucose concentrations were made (Table 8.33).

Table 8.33 Relative risk of developing type II diabetes in Norwegian men and women aged 35–52 years, by sex-specific quartiles of baseline BMIs

Quartiles of baseline BMI (kg/m²)		*Relative risk of type II diabetes over 12 years*	
Males	*Females*	*Males*	*Females*
<23.2	<22.0	1.0	1.0
23.2–25.0	22.0–24.1	1.8	1.8
25.1–27.0	24.2–27.1	2.5	2.1
≥27.1	≥27.2	13.0	30.0

Note: The above relative risk of developing diabetes for both males and females are interpolated from a graph in Njolstad et al. (1998).

By chance, the quartiles of BMI for men again allow an approximate distinction between those with BMIs of <23.0 kg/m² and <25.0 kg/m², i.e. their second quartile, the upper limit of which coincides with the WHO distinction between normal and overweight. The values for women need to be adjusted. These data relate to men and women aged 35–64 years, but were age-standardized. Nevertheless, it is clear that the relative risks of diabetes as derived from these data in general agree with data from the United States prospective studies, and that the values given for the upper quartile will be low estimates of the true risk of diabetes in those Norwegian men and women who have a BMI of ≥30.0 kg/m².

Prevalence studies

The ideal source of estimates of the hazard size would be large long-term trials or, in their absence, large, long-term cohort studies with direct measurements of BMI and blood glucose. The principal cause of type II diabetes appears to be excess weight gain in childhood and in adult life, and so relatively short-term prospective studies of the incidence of diabetes may not capture the full impact of persistent excess BMI. Large, nationally representative cross-sectional studies are available that use objective measurements of BMI and diabetes (in contrast to the few large, long-term prospective studies outlined earlier). Although the diagnosis of diabetes may itself lead to a loss in weight, this is likely to result in only a moderate bias to the null, since current BMI correlates so closely with BMI levels over many previous decades. The estimates of hazard size for this analysis of the burden of diabetes attributable to BMI were therefore derived from the age- and sex-specific associations of type II diabetes (based on fasting glucose values) with BMI from the Japanese National survey (Yoshike, personal communication; see Figure 8.12). The incremental risks for the required age categories were estimated on a linear basis (Table 8.34).

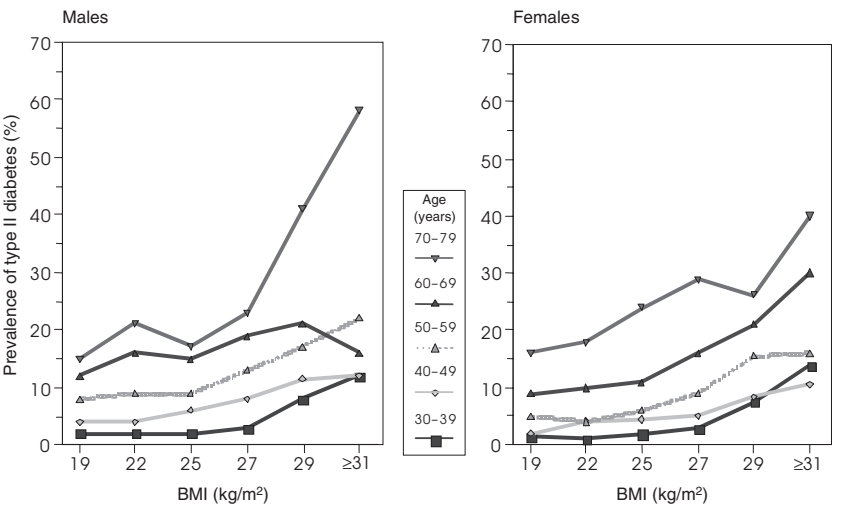
Table 8.34 The relative risk of developing type II diabetes, per unit (1 kg/m^2) increase in BMI

			Age group (years)						
Disease	*Sex*	*Country source*	*15–17*	*18–29*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
Type II diabetes	Males	Japan	—	—	1.36	1.24	1.18	1.27	1.27
	Females	Japan	—	—	1.47	1.34	1.21	1.20	1.20

— No data.

Source: These estimates were taken from a diagram of results (from the Japanese National survey) and are likely to be subject to some inaccuracy.

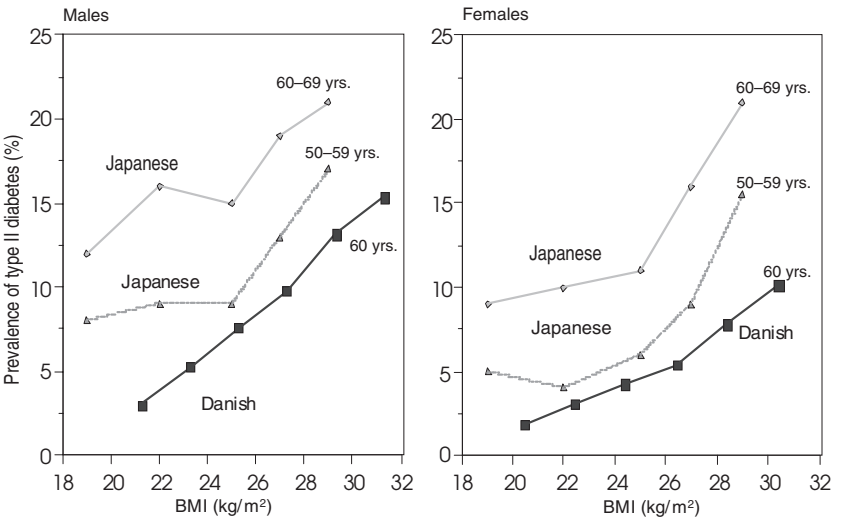
Figure 8.12 The prevalence of type II diabetes (based on fasting blood glucose concentrations) by age, sex, BMI and decade of adult life, in a representative sample of the Japanese population



Note: Based on rates of type II diabetes determined from measurements of fasting blood glucose ≥126 mg/dl or where treatment is already being given for type II diabetes (Yoshike, personal communication).

The proportional increases in the prevalence of diabetes associated with a given increase in BMI were consistent in the Japanese national survey with associations observed in a Danish representative survey (Drivsholm et al. 2001; see Figure 8.13). They are also broadly consistent with the results of prospective studies that measured weight and height, that is, the Norwegian study that assessed the risk of diabetes,

Figure 8.13 A comparison of the prevalence of type II diabetes (based on fasting blood glucose concentrations and measured BMI) in Danish and Japanese adults of comparable age



with measurements of blood glucose (Table 8.33) and the United States NHANES III analyses of prevalence rates of diagnosed diabetes (Table 8.35).

A perspective on the overall burden of ill-health associated with higher BMI can be gauged from nationally representative studies of measured BMI and the prevalence of type II diabetes. Thus Must et al. (1999) used the NHANES III study and highlighted the marked 20-fold increase in prevalence rates of reported type II diabetes in men and women aged >55 years compared with those aged <55 years, even in people with a measured "normal weight", that is, with BMIs of <25.0 kg/m^2. Superimposed on this age-related increase was the impact of high BMIs: the prevalence ratios for people aged <55 years with high BMI compared with those with a normal weight varied from 3.3 in men and 3.8 in women with BMIs of 25–29.9 kg/m^2, up to 8–11 in men and women with BMIs of 35–39.9 kg/m^2 (but with very broad confidence limits). Prevalence ratios were lower in the older age groups (1.8 in the overweight men and women, and 4.2 in men and 3.2 in women with BMIs of 35–39.9 kg/m^2).

Recently, there has been an interest in the issue of ethnic differences in the incidence of co-morbidities that result from increases in weight, with reports of an increased propensity to diabetes in Pima Indians, in those of Hispanic origin and in Asians, compared to the American Cau-

Table 8.35 Estimated prevalence ratios of type II diabetes by weight status category in adults in the United States-representative NHANES III study

	Prevalence ratio of type II diabetes				
	Weight status category				
Age group (years)	*Adjusted prevalence in individuals of normal weight*[a]	*Overweight (BMI 25.0–29.9 kg/m²)*	*Obesity class 1 (BMI 30.0–34.9 kg/m²)*	*Obesity class 2 (BMI 35.0–39.9 kg/m²)*	*Obesity class 3 (BMI ≥40.0 kg/m²)*
Males					
<55	0.2	3.27 (1.17–9.05)	10.14 (4.03–25.08)	7.95 (2.44–25.23)	18.08 (6.71–46.84)
≥55	5.3	1.77 (1.26–2.47)	2.56 (1.71–3.74)	4.23 (2.09–7.59)	3.44 (1.11–8.32)
Females					
<55	0.4	3.82 (1.75–8.21)	2.49 (1.01–6.12)	10.67 (4.02–27.11)	12.87 (5.69–28.05)
≥55	7.9	1.81 (1.41–2.31)	2.19 (1.56–3.01)	3.24 (2.13–4.67)	5.76 (4.17–7.42)

[a] For people with BMI of 18.5–24.9 kg/m, prevalence data were specified only for white adults, with current smokers included if aged <55 years, but also former smokers in the age group ≥55 years. Data for both age groups are adjusted for age, and prevalence ratios are adjusted for race and ethnicity, as well as smoking status. The ratios are set out in relation to the group of individuals of normal weight. The 95% confidence limits are shown in parentheses.

Note: The prevalence of type II diabetes depended on self-reporting because fasting glucose levels were available for only 44% of the sample.

Source: Must et al. (1999).

casian population (Edelstein et al. 1997; Seidell et al. 2001a). Figure 8.13 shows that the prevalence of diabetes in the Japanese population markedly exceeds that in the Danish population, for both sexes and for each BMI category, but the gradient of risk appears to be roughly the same in the two communities. Similar enhanced risks of diabetes are evident in Mexicans assessed in the latest national survey of the prevalence of diabetes and other diseases in relation to excess weight in Mexico (Sánchez-Castillo et al. 2003). These data were compared with the recalculated data relating only to those with fasting blood glucose measurements, from the NHANES III study in the United States. Prevalence rates for type II diabetes are 2–3-fold greater in Mexicans than in non-Hispanic Caucasians in the United States, on an age-standardized basis and, more importantly, prevalences in Mexicans exceed those in people of the United States, when BMI is taken into account. The differences were somewhat smaller, but still statistically significant when the age-dependent prevalences of diabetes were related to the waist circumferences of the two national groups. In the year 2000, the prevalence of abdominal obesity was greater in Mexicans than in non-Hispanic Caucasians in the United States, as measured in the NHANES III study (Sánchez-Castillo et al. 2003). Similar data are now emerging from mainland China Hong Kong SAR and India. In the near future, these comparisons may be extended so that the increment in risk for each pop-

ulation at each BMI level, taking age into account, can be obtained with some assurance on the basis of measured body weights, heights and fasting glucose levels. It will also be necessary to take into account the relationship between BMI and diabetes in Pacific Islanders, which may well prove to be different from that in other peoples, given their greater lean tissue : fat ratios (Bell et al. 2001). Nevertheless, it seems reasonable on present evidence to conclude that although in Asians and some other populations the absolute risk of type II diabetes is amplified, the incremental gradient of risk of diabetes in relation to increasing BMI is approximately the same as that found in Caucasians. On this basis, similar values for relative risk can be applied to all subregional groups.

5.6 Osteoarthritis

There is a well-documented association between high BMI and the development of osteoarthritis in both men and women (Cicuttini and Spector 1998). Osteoarthritis is an abnormality which involves damage to and eventually the destruction of the articular cartilage of the joint. New bone is formed at the joint surfaces, probably in response to the cartilaginous damage. The relationship between excess weight and the development of osteoarthritis has been studied in a number of population-based prospective, cross-sectional and retrospective trials, showing excess weight as the most important preventable risk factor for osteoarthritis. Cross-sectional studies have consistently reported increased risks of osteoarthritis in association with body-weight gain, with early studies suggesting a 2–7-fold greater risk in those individuals in the top compared with the bottom tertile of BMI (Table 8.36) and, in some studies, a greater risk in women than men (Cicuttini and Spector 1998). As the data were adjusted for race, ethnicity and smoking as well as age, no age-related data were available, but these are the best nation-

Table 8.36 Prevalence of osteoarthritis by weight status category, in adults

	Prevalence of osteoarthritis (%)					
	Weight status category					
Sex	*Underweight BMI <18.5*	*Normal weight BMI 18.5–24.9*	*Overweight BMI 25.0–29.9*	*Obesity class 1 BMI 30.0–34.9*	*Obesity class 2 BMI 35.0–39.9*	*Obesity class 3 BMI ≥40.0*
Males ($n = 6\,987$)	0.39	2.59	4.55	4.66	5.46	10.04
Females ($n = 7\,689$)	7.79	5.22	8.51	9.94	10.39	17.19

Source: Table 3 in Must et al. (1999).

ally representative data available for assessing the quantitative impact of BMI increases on the prevalence of osteoarthritis.

A number of mechanisms have been proposed to explain the association of adult body-weight gain with osteoarthritis. The physical burden associated with an increased load on the joints seems straightforward, but changes in movement and gravitational stresses as weight gain occurs are also a factor. Other mechanisms have, however, been invoked, including systemic changes in metabolism associated with hypertension, raised blood glucose and cholesterol concentrations, insulin resistance and elevated concentrations of blood uric acid, as well as hormonal changes induced by the metabolic effects of additional adipose tissue (Cicuttini and Spector 1998). Several of these factors could be acting on the metabolic integrity of the articular cartilage, as could other dietary factors, such as high fat intake, which have also been linked to this disease. The associations with hypertension tend to disappear once concomitant increased body weight is taken into account, and the link with hypercholesterolaemia is not sufficiently robust to warrant special consideration. Abnormal glucose metabolism is a more plausible mechanism, with the possible involvement of growth hormone (Cicuttini and Spector 1998), but epidemiological studies have not shown a consistent link between type II diabetes and osteoarthritis. Raised uric acid concentrations have been associated with osteoarthritis, but again data supporting the relationships are inconsistent.

It is clear that excess weight gain precedes the development of osteoarthritis rather than the reverse. This was initially reported in retrospective recall studies of reported former weights (Anderson and Felson 1998; Hart and Spector 1993), but three studies (Anderson and Felson 1998; Cicuttini et al. 1996; Hart and Spector 1993) have shown a strong association of excess weight gain with asymptomatic radiological evidence of osteoarthritis, such radiological evidence having been clearly shown to be a predictor of future disability (Acheson et al. 1974; Hochberg et al. 1989). Twin studies have shown that the heavier twin has a greater risk of developing osteoarthritis (Cicuttini et al. 1996), and the incidence of osteoarthritis is markedly enhanced in overweight women (Schouten et al. 1992). In population studies, the incidence of disability once osteoarthritis has developed is also particularly marked if the subjects are obese (Verbrugge et al. 1991). In addition to predicting the greater risk of developing osteoarthritis, weight gain has also been shown to enhance the progression of the disease. Among middle-aged women with early stage unilateral knee damage, those who were overweight had a 34% chance of developing osteoarthritis in the contralateral knee within 2 years and 22% also showed radiological progression of the disease in the initially affected joint (Cicuttini and Spector 1998).

Detailed population studies regarding the incremental risk of developing osteoarthritis of any joint over a wide range of BMIs are rare,

however, with Must et al. (1999) being the principal source of data. Must et al. used the NHANES III study with measurements of BMI and with reported rates of disability from osteoarthritis to derive risk values. Given the need to consider the linear relationship between BMI and disabilities rather than the type of categorical analyses shown in Table 8.36, the Must et al. (1999) study was used here. This study, as well as several others, shows that the absolute risk of osteoarthritis is greater in women than in men, but that the incremental risk of osteoarthritis per unit increase in BMI is approximately the same in both sexes. On the basis of this survey in the United States, the relative risks of osteoarthritis were derived without regard to adult ages or sex, as shown in Table 8.37. These analyses were not adjusted either for age or smoking status, but Must et al. (1999) found that there were no interactions between weight class and race or ethnicity for either sex.

Gout

Gout is a metabolic disease of uric acid metabolism, which has a genetic component and which involves acute episodes of arthritis leading to chronic arthritis and disability. Although much less common than osteoarthritis, gout has also been linked with excess weight gain in both cross-sectional and longitudinal studies (Cicuttini and Spector 1998). The abdominal distribution of body fat seems to be a particularly important risk factor, in keeping with mechanistic studies which suggest that hormonal or other agents which alter uric acid metabolism, such as diet and alcohol, are involved in precipitating an attack of gout. Although multivariate analyses have shown the importance of BMI from as early as age 35 years and excess weight gain as distinct risk factors (Roubenoff et al. 1991), with other large studies suggesting that excess weight as early as in adolescence is predictive of gout, there is no clear picture of the prevalence of or associated disability linked to gout on an international basis. Thus, although weight reduction is clearly recognized as potentially important in avoiding and limiting the risk of gout and its associated handicaps, it was decided not to include estimates of the

Table 8.37 The relative risk of developing osteoarthritis per unit (1 kg/m^2) increase in BMI

			Age group (years)						
Disease	*Sex*	*Country source*	*15–17*	*18–29*	*30–44*	*45–69*	*60–69*	*70–79*	*≥80*
Osteoarthritis	Males	USA	—	—	1.04	1.04	1.04	1.04	1.04
	Females	USA	—	—	1.04	1.04	1.04	1.04	1.04

— No data.

effects of excess BMI on gout and only to use data on weight gain in association with osteoarthritis.

5.7 Cancer

There have recently been two major re-analyses, incorporating systematic reviews, of the associations between excess weight gain and the development of different forms of cancer (Bergström et al. 2001; IARC 2002), which supersede those of Australia (Mathers et al. 1999) and the United Kingdom (National Audit office 2001). The latest review by the International Agency for Research on Cancer (IARC) lists the risk of different cancers in relation to both body weight and physical activity (IARC 2002). These analyses concluded that cancers of the colon, breast (postmenopausal), endometrium and kidney were statistically related to weight gain, each analysis being usually based on a systematic review of a large number of both case–control and prospective studies. The present analysis also drew heavily on the recent series of meta-analyses conducted by IARC staff and colleagues, which provided estimates of the coefficient of risk per unit BMI increase (Bergström et al. 2001). Since this chapter used continuous rather than categorical data, the conclusions of the main IARC report on statistically significant findings are used to select the cancers to be considered, but the coefficients of risk are taken from Bergström et al. (2001). Table 8.38 sets out these estimates. In view of the much lower incidence of and the uncertainty of the global statistics for kidney cancer, the estimates of the burden of cancer were confined to cancers of the breast in postmenopausal women, colon and endometrium.

Breast cancer

A distinction needs to be made between the risks of developing breast cancer before and after the menopause. Premenopausal breast cancer is less likely to develop in women with high BMIs, but this effect is only seen at BMIs of about >28 kg/m^2 and rates of mortality are not lower among women with higher BMIs. For the present analyses, the role of excess weight gain in the development and the burden of disease from breast cancer was confined to cases arising in postmenopausal women. Over 100 studies conducted in many populations have found that women with higher BMIs are at greater risk of postmenopausal breast cancer (IARC 2002). Bergström et al. (2001) used 27 of these studies to quantitatively evaluate the impact of excess weight. Age, age at menarche, parity, alcohol intake and diet are recognized confounders, but when all of these were taken into account the coefficient of risk per unit BMI was still statistically significant (Table 8.38). Furthermore, there are studies which overall suggest that those women who have limited their weight gain or have lost weight in early adult life tend to have a reduced risk of postmenopausal breast cancer (IARC 2002). Over 50 studies (e.g. Huang et al. 1997; Tretli 1989; Törnberg and Carstensen 1994) also

Table 8.38 The relative risks of developing cancer associated with increases in BMI, as calculated for European populations

Cancer	*Coefficient (±95% CI)*[a] *(Increase in incidence rates per unit [1 kg/m²] increase in BMI)*	*Relative risk of developing cancer*		*No. of studies*
		Overweight[b]	*Obesity*[c]	
Postmenopausal breast	1.03 (1.02–1.04)	1.12	1.25	13
Colon	1.03 (1.01–1.05)	1.15	1.33	6
Endometrial	1.10 (1.07–1.14)	1.59	2.52	4
Kidney	1.06 (1.03–1.08)	1.36	1.84	2

[a] Coefficient describes the increase in incidence rates per unit increase in BMI irrespective of age (and includes the 95% confidence intervals) based on covariance analysis.

[b] Defined as BMI 25.0–29.0 kg/m²; the relative risk is calculated from each study's provision of the distribution of BMIs within the studied population.

[c] Defined as BMI ≥30.0 kg/m²; the relative risk is calculated from each study's provision of the distribution of BMIs within the studied population.

Notes: 27 eligible studies but analysis restricted to cohort studies with incident cases only; adjustment for age, reproductive factors, alcohol and diet did not materially affect the relationship.

No effect was seen when restricting the data to incident cases; all studies accounted for age, diet (where data were available), alcohol and/or physical activity.

Data provided for men but the data were almost identical for women (1.07, CI 1.05–1.09). Studies were restricted to incident cases, with age adjustment and allowance for smoking but these restrictions did little to influence the results from the seven studies considered.

Source: Bergström et al. (2001).

suggest that women with a higher BMI at the time of diagnosis have poorer survival and an increased likelihood of recurrent breast cancer, irrespective of their menopausal status and after adjusting for the stage of cancer development and the type of treatment used.

Mechanistically, it seems clear that the risk of developing postmenopausal breast cancer is increased in women with raised plasma and tissue concentrations of estrogens. The activity of these hormones is greater when there are lower circulating concentrations of the sex hormone-binding globulin (SHBG). Obesity, with its associated insulin resistance, lowers SHBG levels; overweight women are also found to have higher circulating concentrations of total and bioavailable androgens and estrogens. Confirmation of the importance of these hormonal changes comes from the observation that women exposed to combined estrogens and progesterones as part of postmenopausal hormone replacement therapy subsequently have increased rates of breast cancer, the risk being greater in those on combined compared with estrogen-alone treatment (Weiderpass et al. 1999). The reduced risk associated with a late menarche and in women with anovulatory cycles is considered to relate to the lower exposure of the breast to bioactive estrogens (IARC 2002).

Colon cancer

Bergström et al. (2001) considered 19 studies, 12 of which were prospective, which generally tended to show a stronger relationship between excess weight and incidence of cancer in men than in women. The studies also allowed an assessment to be made of the confounding effects of physical activity, age, family history of colon cancer, ethnicity, social class and diet. For the full quantitative analyses, only six studies could be included (Gerhardsson et al. 1990; Giovannucci et al. 1995; Kune et al. 1990; Lee and Paffenbarger 1992; Martinez et al. 1997; Thun et al. 1992); and in these studies no sex-specific differences could be found, nor did restricting the analysis to incident cases alter the estimate. The same general relationships of weight to the development of large colonic adenomas were found in the IARC analyses (IARC 2002), the development of adenoma being seen as part of the progression of cellular changes leading to the development of colon cancer. Although fewer studies have considered rectal cancers separately, no relationship was found between BMI and rectal cancer.

The mechanisms by which weight gain might accentuate the risk of developing large adenomas and colon cancer are unclear, but the stronger association of high BMIs with large rather than small adenomas suggests that excess weight operates at a relatively late stage in the promotion of tumour formation. Excess weight is associated with a wide range of hormonal and metabolic effects that may be involved in the promotion of cancer. Dietary factors could, in theory, be confounders with high meat intake, especially processed meat, and a low intake of fibre-rich vegetables and fruit being particularly linked to colon cancer and also being part of a weight-gain-inducing, energy-dense diet. However, several of the studies also assessed diet and the impact of higher BMIs seemed to be independent of the direct dietary effects.

Endometrial cancer

Both case–control and cohort studies have shown a relationship between higher BMI and increased risk of developing endometrial cancer, even after adjusting for other risk factors relating to the reproductive system, such as age at birth of first child and parity (Le Marchand et al. 1991). There was a remarkably consistent relationship between high BMI and risk of endometrial cancer in 22 of the 25 studies assessed by IARC (2002), studies which considered only cancer incidence and adjusted for all suggested confounders showing similar relationships. Bergström et al. (2001) used four of the seven cohort and 17 case–control studies reviewed by IARC (2002) to calculate by meta-analysis the incremental risk shown in Table 8.38. The overall risks of endometrial cancer appeared to be equivalent at different ages, but adult weight gain seems to be particularly important whatever the early adult weight status. Upper body fatness may be particularly conducive to the process of car-

cinogenesis, but the standard measures of abdominal fatness have given inconsistent results.

The dominant mechanistic theory relates to the unopposed estrogen hypothesis, according to which estrogenic contraceptives or hormone replacement therapy enhance the risk of endometrial cancer, whereas progesterone-containing preparations confer protection. Estrogens are known to induce endometrial proliferation via local production of insulin growth factor (IGF-1), whereas progesterone induces the production of an endometrial IGF-1-binding protein. Women with low levels of plasma SHBG, high levels of androgens and, after the menopause, elevated levels of total and bioavailable estrogens have an increased risk of endometrial cancer, as have younger women with the polycystic ovarian disease, which is associated with chronic anovulation and therefore low rates of production of progesterone. All these findings, therefore, fit the concept of excess available bioactive estrogen, which induces endometrial cell proliferation. Insulin resistance and higher concentrations of circulating IGF-1 induced by the lower concentrations of IGF-binding proteins in women who gain weight may also be involved. The IARC report notes that, given the substantial changes in insulin resistance, IGF-1 and estrogen status which accompany weight loss, it is possible that weight reduction quite late in life could reduce the risk of the estrogen-promoted cancers of the postmenopausal breast and endometrium.

5.8 Body weight and total mortality

Clearly, the net associations of BMI with total mortality will depend crucially on the component causes of death, which vary substantially by age, sex and population. Effects on total mortality were not estimated directly in these analyses for this reason, only cause-specific estimates being made. However, for completeness, data relating BMI to total mortality (usually derived from middle-aged North American populations) are reviewed here.

An extensive review of the relationship between BMI and total all-cause mortality, based on a detailed systematic review, was published by Troiano et al. (1996). About 1000 citations dating from 1861 to 1991 were identified; of these, 22 suitable studies were selected, with 56 substudy groups (e.g. in relation to age, ethnicity, sex and smoking status) and 354 BMI groups. Most of the studies dealt with Caucasian men, but 14 substudies assessed Caucasian women. Only two substudies in Asian men (not women) were available and one substudy in Samoan men and women combined was assessed. The authors distinguished data derived from insurance companies from data derived from other populations because they demonstrated that insurance data, particularly those from the United States, were associated with lower mortality in relation to BMI than that found in the general population. It was inferred that these data, which formed the basis of many earlier official reviews on the

impact of obesity for governments, for example, in the United States and the United Kingdom, related to groups in society who were relatively affluent and therefore, for a variety of reasons, able to sustain better health.

Troiano et al. (1996) demonstrated evidence of a U-shaped curve of mortality in relation to BMI, but this curve varied depending on whether the data were derived from the United States or elsewhere (predominantly northern Europe and Scandinavia). When all data were included, as shown in Table 8.39, there was clear evidence of a minimum mortality at BMIs of 24–27 kg/m^2, the lower value being that obtained with longer, i.e. 30-year follow-ups of adults. Most of these data relate to individuals who were aged about 50 years and were followed for 30 years. The table dealing with all the studies shows a statistically significant increase of *z*-scores when the *z*-scores exceed 1.65. Clearly, even BMIs of ≤23 and ≥28 kg/m^2 are associated with higher mortality in these overall groups. It is noteworthy, however, that these analyses included

Table 8.39 The probability of death associated with BMI level, for either 10 or 30 years of follow-up (smokers and non-smokers included)

	Odds ratio		*z-score differences*	
BMI (kg/m^2)	*10 years*	*30 years*	*10 years*	*30 years*
19	2.08	2.89	1.16	2.29
20	1.86	1.97	1.64	2.43
21	1.65	1.48	2.00	2.50
22	1.46	1.21	1.74	2.32
23	1.30	1.06	1.25	1.68
24	1.16	—	0.84	—
25	1.07	1.00	0.50	0.11
26	1.01	1.07	0.18	0.82
27	—	1.20	—	1.45
28	1.04	1.39	0.63	2.10
29	1.15	1.68	1.27	2.87
30	1.36	2.12	2.18	3.84
31	1.73	2.69	3.04	4.84
32	2.23	3.49	3.13	5.03

— No data.

Notes: The odds ratios relate to the probability of death, with the lowest mortality for any BMI group taken as 1.0, the odds of the other groups then being calculated as the log of the increased mortality ratio. A z-score of ≥1.65 is evidence for a statistically significant increase ($P < 0.05$, one-tailed). Note that the minimum mortality occurred at a BMI of 27 kg/m^2 when there was a 10-year follow-up, but at 24 kg/m^2 with a 30-year follow-up. Men and women, smokers and non-smokers, USA and non-USA studies included.

Source: Data are from Table 4 of Troiano et al. (1996).

both smokers and non-smokers. Non-smokers, considered over the 30-year period, had systematically lower risks at any BMI than smokers and Troiano et al. (1996) used these data for their baseline mortality curves.

There has been extensive discussion over the last 30 years regarding the repeated finding of higher mortality rates associated with lower BMIs. It was recognized that the original inclusion of data from smokers in such calculations had a marked effect because smokers are at greater risk of mortality, but tend to be thinner because of their reduction in appetite and their increased metabolic rate, that is, increased total energy expenditure, which leads to lower body weights when these effects are in energy balance. Thus the excess of smokers in the group of "thin" adults imposes higher mortality rates on the group overall, despite the lower BMIs.

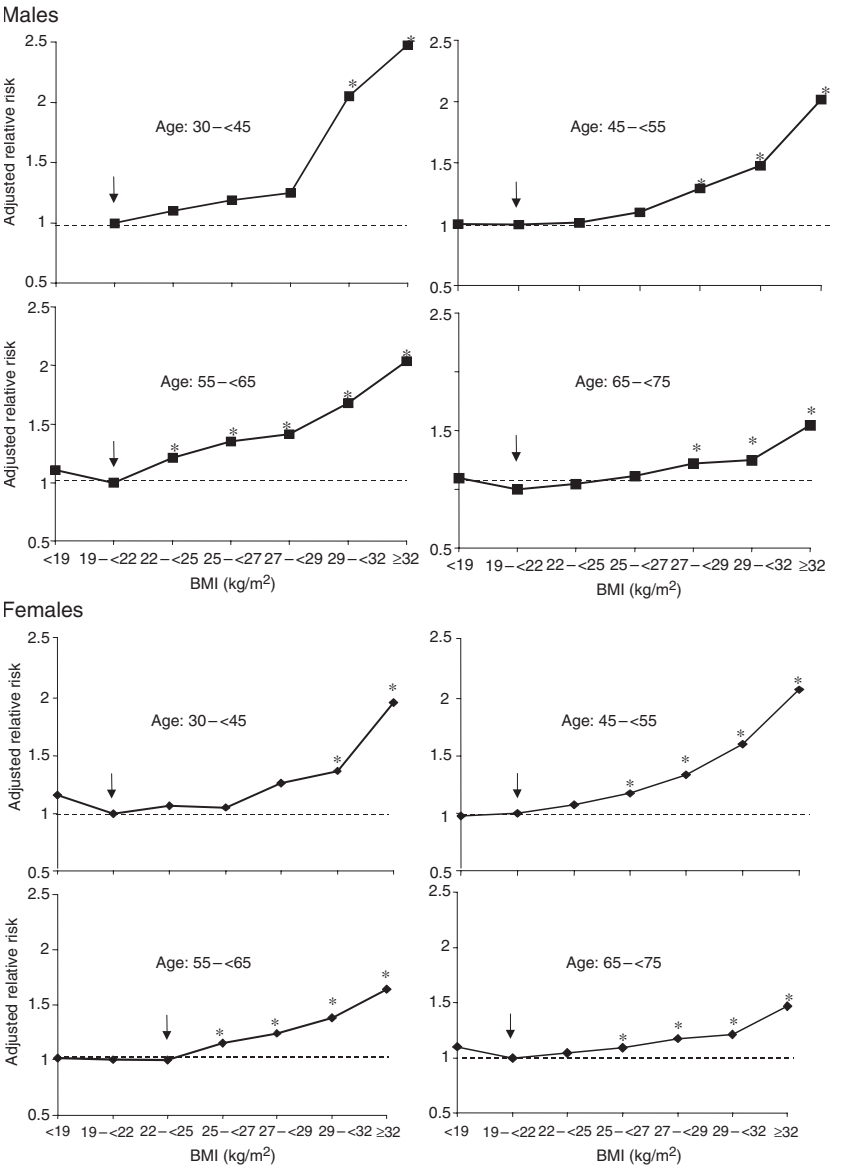
Further, the mortality rates of the groups with low BMIs may be enhanced by the presence of individuals with as yet undiagnosed diseases, for example, cancer, who may have lost weight before symptoms emerged or a diagnosis was made. A convention has therefore developed whereby the early deaths are excluded and only those deaths that occur 2–5 years after the initiation of any study are considered. By doing this, it is frequently found that the U-shaped curve converts to a J-shaped curve or log-linear relationship.

The data previously considered were standardized to men and women aged about 50 years. Stevens et al. (1998) have recently presented a detailed analysis based on about 325 000 men and women taking part in the American Cancer Society's Cancer Prevention Study. As already noted, this analysis in the United States relates to a relatively affluent fraction of the population and unfortunately used reported, not measured, weights and heights. Nevertheless, the sample is valuable in indicating the likely effect of age when assessing total mortality rates over a 12-year follow-up period. For this purpose, data were expressly chosen which related only to life-long non-smokers. Figure 8.14 shows the age-related risk of death from all causes. Note that men and women with a BMI of 19.0–21.9 kg/m^2 had the lowest total mortality. Small increases in risk were apparent at BMIs of 22.0–27.0 kg/m^2, but the increase in relative risk was more obvious in men than in women and in those aged <75 years. The risk of death from cardiovascular disease related to BMI was more clear-cut than the relative risk for all causes of death, as found in many studies (see below).

Figure 8.15 shows the decline in relative risk per unit increase in BMI with age. A very clear trend is seen, with the incremental risks being statistically significantly above those for the reference group up to the age of 75 years.

From Figure 8.14, it seems reasonable to conclude that the ideal BMI should be between 19.0 and 21.9 kg/m^2. These values relate to the individual risk of death in groups within a single population (in this

Figure 8.14 The relative risk of death from all causes, according to age and BMI: American Cancer Society's studies using self-reported weights and heights for BMI calculations

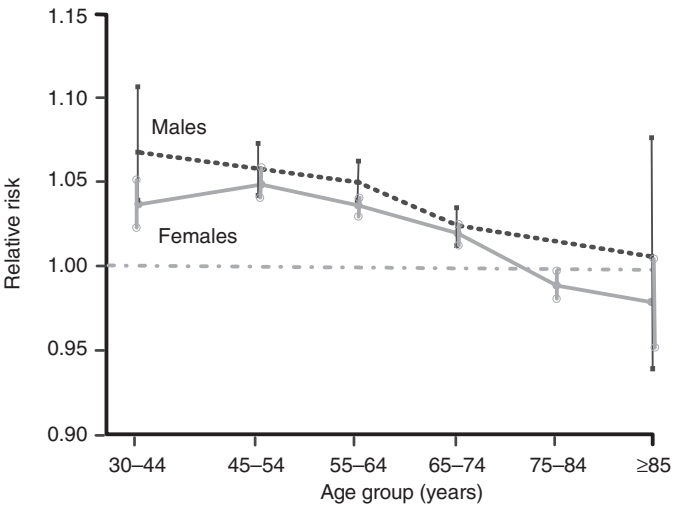


Key: Arrow denotes the BMI for minimum risk.

* Signifies a statistically different risk from that at BMIs of 19–22 kg/m^2.

Source: Data reformatted by Stevens from Stevens et al. (1998).

Figure 8.15 The change with age in the relative risk of death from all causes associated with a one-unit (1 kg/m^2) increase in BMI, in never-smokers



Note: The bars represent 95% confidence intervals and the trends are significant in all cases.

Source: Taken from Stevens et al. (1998) with the reference group having a BMI of 19.0–21.9 kg/m^2 in each age group.

case, the United States) and seem to apply to all age groups considered in the CRA analysis, from age 30–79 years. These findings are in keeping with the earlier analyses of the relationship between mean BMI and the minimizing of the prevalence of overweight, but would allow a higher proportion of underweight, which in a developing country would be a disadvantage. Given that the circumstances encountered by thin adults in the United States are probably different to those in developing countries, it seems reasonable to conclude that the data reinforce the choice of a low value for the minimum population mean BMI of 21.0–22.0 kg/m^2.

5.9 Excluded health outcomes

A number of other conditions have been widely quoted as concomitants of body-weight gain in affluent societies, including breathlessness, sleep apnoea, back pain, dermatitis, reactive depression and social isolation, menstrual disorders, infertility and gall bladder disease. The reasons for excluding these conditions are as follows.

Breathlessness: this is hard to quantify on an international basis and comparable data to those obtained in affluent societies in relation to excess weight are difficult to find in many parts of the world. It is also not clear to what extent different degrees of physical fitness in different parts of the world might affect an assessment of perceived breathlessness in those with excess weight.

Sleep apnoea is a well-described clinical complication of excess weight, which affects very obese children and adults. There are few data on its prevalence however, and it is generally considered to occur in those with more extreme forms of obesity.

Back pain: there are many causes of back pain, which is a very prevalent condition with substantial economic implications. Few studies however provide reliable estimates of hazard due to any specific risk.

Dermatitis: skin problems occur commonly in people who are obese, but few data are available other than from the developed world. Again, the description of skin problems associated with obesity is largely clinical and therefore this condition is not included.

Reactive depression and social isolation: it is well recognized that the societal response to obese individuals varies widely across the world. In some developing countries, the overweight and obese have traditionally been seen as successful individuals who are sufficiently wealthy or resourceful to have acquired enough food. In these circumstances, there is little indication of any social isolation or ensuing depression resulting from the overweight and obese being excluded from social interactions. This is quite different from accounts widely reported in North America and Europe where being obese, particularly for children and young women, is a social stigma which has clearly been related to poorer access to employment opportunities, lower earning power, a tendency to marry less affluent partners and a tendency to become personally distressed and socially isolated. Given the diversity in cultural perceptions of the benefits or handicaps of being overweight or obese, no attempt has been made at this stage to use representative data on body weight in adults in relation to mental health outcome.

Menstrual disorders and infertility: although severe underweight and anorexia nervosa have classically been associated with amenorrhoea and infertility, obesity is now increasingly recognized as a major feature of, for example, polycystic ovarian disease, which is associated with menstrual abnormalities, hirsutism and infertility. Weight loss markedly improves the condition of patients with this disorder and restores fertility. Little is known about the prevalence of this disease in different societies. The issue of infertility does not seem as yet to be of great societal concern if considered simply in terms of the probability of maintaining the population size. There is also no suggestion that the marked decline

in fertility seen, for example, in Europe (France, Italy and Spain), relates to the increasing prevalence of adiposity. Societal and social issues appear to be of far greater importance, so this outcome is also excluded from the current analysis.

Gallbladder disease: it has been recognized for several decades that excess weight gain is associated with a greater propensity to the development of gallstones and gall bladder disease. This relationship is clear-cut in developed societies, but as data on gall bladder disease are not collected systematically in many countries it is not possible to undertake an appropriate international analysis of the risk associated with weight gain.

5.10 Risk reversibility

The evidence on the reversibility of health hazards after reducing excessive BMIs has been given in each section setting out the relationship between increases in BMI and the development of individual diseases such as type II diabetes, ischaemic heart disease, hypertension and stroke, as well as cancers. The speed of reversibility depends on the condition. Thus an elevated blood pressure associated with weight gain can start to reverse within days of the beginning of weight loss; in association with other dietary measures and increased physical activity, lower body weights can also then limit the incidence of hypertension for 10 years (Stamler et al. 1980). Changes in blood lipids also begin within days of weight loss, although the restitution of low concentrations of HDL cholesterol to normal requires a period of several weeks at a stable lower body weight (Dattilo and Kris-Etherton 1992). The impact of weight loss on the development of ischaemic heart disease is more difficult to distinguish from other dietary changes accompanying the intended weight loss. Thus when individuals at a high risk of suffering a myocardial infarction are trained to markedly reduce their intake of fat, together with their intake of salt and sugars, as well as undertaking exercise training, they lose substantial amounts of weight and show a marked decrease not only in the principal risk factors for ischaemic heart disease (i.e. hypertension, dyslipidaemias and glucose intolerance) but also show a reduction within weeks in the incidence of angina and then in rates of myocardial infarction (Ornish et al. 1990); this is clearly evident within a 5-year period. The time needed to alter insulin resistance is also short (i.e. within a few days to weeks) and the resulting reduced incidence of type II diabetes becomes evident within 1 or 2 years, although to obtain statistically robust data in most studies about 3 years is needed (Tuomilehto et al. 2001).

6. Results

Tables 8.40–8.42 show the proportion of the included diseases, the number of deaths and disease burden attributable to increases in BMI

above 21.0 kg/m^2 in the different subregions of the world in 2000. Figure 8.16 summarizes the contributions of the diseases considered in this analysis to the total global burden of ill-health attributable to the effect of high BMI in 2000.

No lives are lost because of arthritis, but the global total mortality for cancers is appreciable, amounting to 74 000 for colon cancer, 47 000 for breast cancer and 32 000 for endometrial cancer, i.e. a total of 153 000 cancer deaths in 2000. However, these figures are dwarfed by the 491 000 deaths from diabetes, 489 000 from ischaemic stroke, 1 168 000 from ischaemic heart disease and 290 000 from hypertensive disease. Thus by considering only the major diseases affected by high BMI, a total of 2 592 000 deaths in 2000 were attributable to this risk factor. There are marked regional differences in this mortality burden, some of which reflect major differences in population sizes.

It is evident from the impact of weight gain on risk of arthritis that those subregions with a large proportion of adults with high BMIs, for example, AMR-A, AMR-B, EUR-A and EUR-B, are likely to have more of a burden linked directly to the physical demands of weight bearing, but much depends on the quality of the documentation of degrees of handicap associated with arthritis globally. The proportions of diabetes attributable to BMI increases range from 38–88% according to subregion. For diabetes and cardiovascular diseases, while the gradient of

Figure 8.16 The contribution of high BMI to the global burden of ill-health in the year 2000

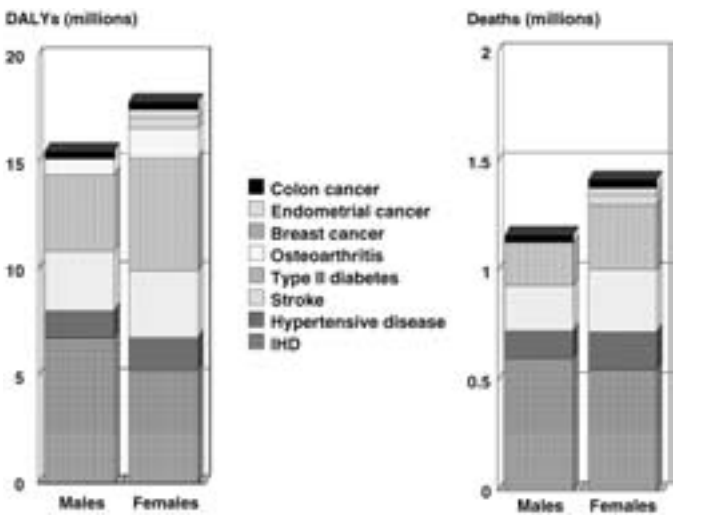


Table 8.40 Percentage of different diseases which is attributable to high BMI in adults aged ≥30 years, by subregion

	Osteoarthritis		*Colon cancer*		*Postmenopausal breast cancer*		*Endometrial cancer*		*Type II diabetes*		*Stroke*[a]		*Ischaemic heart disease*		*Hypertensive disease*	
Subregion	*Male*	*Female*	*Male*	*Female*	*Male*	*Female*	*Male*	*Female*	*Male*	*Female*	*Male*	*Female*	*Male*	*Female*	*Male*	*Female*
AFR-D	6	6	4	5	NA	3	NA	15	38	46	14	13	13	12	24	22
AFR-E	7	9	5	7	NA	4	NA	25	44	58	17	20	15	19	29	32
AMR-A	22	24	17	18	NA	12	NA	52	83	88	40	37	37	32	63	58
AMR-B	17	23	13	16	NA	10	NA	46	71	83	34	40	32	37	52	60
AMR-D	18	21	13	14	NA	8	NA	41	69	77	31	34	31	32	52	54
EMR-B	15	15	10	13	NA	7	NA	43	62	72	26	32	30	32	45	51
EMR-D	10	15	7	11	NA	5	NA	34	55	72	18	25	21	26	36	44
EUR-A	22	24	18	18	NA	13	NA	50	78	84	35	31	35	29	56	50
EUR-B	15	22	15	19	NA	12	NA	48	71	84	35	41	34	38	56	65
EUR-C	17	25	14	19	NA	13	NA	52	68	83	33	37	33	33	56	64
SEAR-B	9	10	7	8	NA	4	NA	24	44	56	18	21	17	19	32	36
SEAR-D	2	5	1	4	NA	2	NA	14	13	43	3	10	4	10	7	21
WPR-A	11	12	8	9	NA	5	NA	28	55	65	20	18	19	15	31	27
WPR-B	9	10	7	8	NA	5	NA	26	47	62	18	19	17	18	30	34
World	11	14	11	13	NA	8	NA	42	50	66	21	25	21	22	36	41

NA Not applicable.

[a] Percentage of stroke attributable to high BMI was based upon estimates for ischaemic stroke. The fractions for total stroke are given in the annex tables for the chapter (see the CD-ROM accompanying this book).

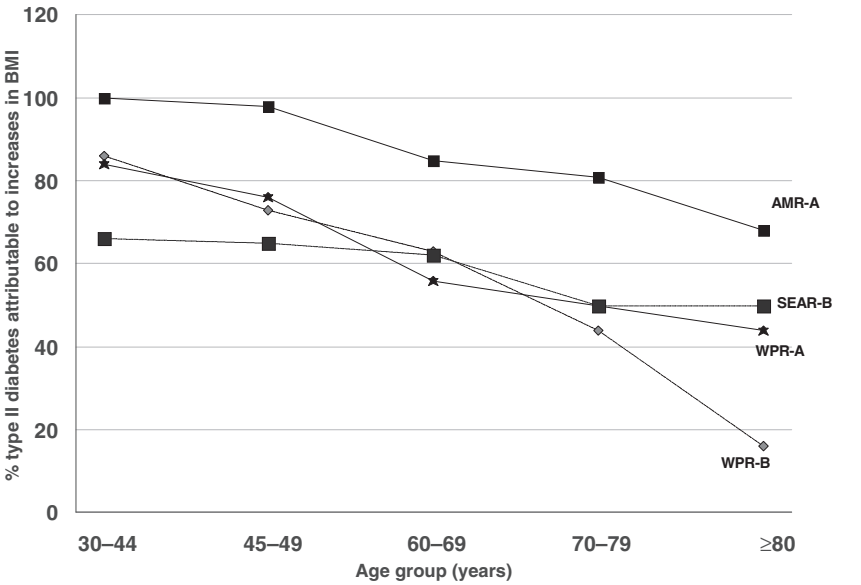
Table 8.41 Mortality (000s) from diseases caused by high BMI in adults aged ≥30 years, by subregion

Subregion	*Osteoarthritis*	*Colon cancer*	*Postmenopausal breast cancer*	*Endometrial cancer*	*Type II diabetes*	*Stroke*	*Ischaemic heart disease*	*Hypertensive disease*	*Total*
AFR-D	0	0	0	0	7	7	14	4	32
AFR-E	0	1	1	0	16	11	20	7	56
AMR-A	0	12	8	4	62	28	135	22	271
AMR-B	0	4	4	5	103	35	78	33	262
AMR-D	0	1	0	2	11	4	9	6	33
EMR-B	0	1	0	0	10	5	33	14	63
EMR-D	0	1	2	0	22	16	65	19	125
EUR-A	0	25	14	8	70	66	167	29	379
EUR-B	0	5	3	3	23	51	137	36	258
EUR-C	0	10	6	6	16	124	284	21	467
SEAR-B	0	2	1	1	32	13	34	19	102
SEAR-D	0	1	2	0	35	24	82	9	153
WPR-A	0	3	1	1	9	10	15	2	41
WPR-B	0	9	4	2	74	95	95	68	347
World	0	74	47	32	491	489	1168	290	2592

Table 8.42 Burden of disease in DALYs (000s) from diseases caused by high BMI in adults aged ≥30 years, by subregion

Subregion	*Osteoarthritis*	*Colon cancer*	*Postmenopausal breast cancer*	*Endometrial cancer*	*Type II diabetes*	*Stroke*	*Ischaemic heart disease*	*Hypertensive disease*	*Total*
AFR-D	37	6	5	2	148	110	199	57	564
AFR-E	55	9	14	5	240	188	282	94	887
AMR-A	243	104	77	46	1 171	400	1 243	195	3 479
AMR-B	193	41	40	77	1 367	504	892	309	3 423
AMR-D	22	5	5	22	164	51	91	62	422
EMR-B	33	7	6	4	273	84	447	136	990
EMR-D	70	12	17	6	515	217	865	208	1 910
EUR-A	348	185	127	66	876	619	1 271	166	3 658
EUR-B	176	46	35	37	401	559	1 304	306	2 864
EUR-C	266	95	66	69	526	1 291	2 741	208	5 262
SEAR-B	85	21	18	7	555	175	406	201	1 468
SEAR-D	95	8	20	5	916	309	1 156	117	2 626
WPR-A	74	31	11	9	224	135	134	11	629
WPR-B	421	104	45	33	1 503	1 280	1 139	709	5 234
World	2 118	676	486	386	8 877	5 921	12 170	2780	33 414

Figure 8.17 Selected subregional differences in the age-related proportion of type II diabetes attributable to increases in BMI in women



relative risk was considered the same in the different subregions, the absolute risk varied substantially because of other determinants of these diseases such as dietary variation, which causes additional changes in both serum cholesterol levels and blood pressure.

Figure 8.17 presents subregional differences in the age-related proportion of type II diabetes attributable to high BMI in women. The picture of differences is magnified and the clear downward gradient in the attributable fraction with increasing age is evident. Similar relationships are observed for all three cardiovascular end-points, but not for the cancers or osteoarthritis, where relative risks were independent of age.

7. Discussion

These analyses highlight the very substantial burden of ill-health incurred by increases in adult BMIs with the greatest impact in disease specific terms being the burden associated with the development of type II diabetes. The attributable fraction for high BMIs does vary markedly by subregion, which implies that other factors contribute to or interact with increases in BMI. The overall burden by subregion, however, depends on

the prevalences of both high BMIs and of the particular disease (e.g. ischaemic heart disease) as well as the overall size of the population. The current analyses do not take account of the interactions with other risk factors, for example, physical inactivity, which could confound the estimated burden attributable to high BMIs *per se*. However, increases in body weight also amplify other major risk factors such as blood pressure and blood cholesterol levels, and so discriminating a selective effect of high BMIs from dietary factors and physical activity is not straightforward. Nevertheless, on the basis of the current data, the impact of increases in BMI on the development of type II diabetes and on cardiovascular diseases and cancers in most parts of the world is substantial.

This is the first global analysis of the risks attributable to high BMIs and the results reinforce the recent recognition by WHO (2000) that this is one of the largest unrecognized public health problems that now need to be addressed. With the seemingly inexorable rise in mean BMIs in various populations, the projected impact on the global health burden will be very substantial by 2030 unless effective public health measures can be introduced soon.

8. Future exposure

8.1 Regional time trends in adult BMIs

There are as yet only a modest number of studies dealing with BMI changes in different populations, the most comprehensive being that conducted by Pelletier and Rahn (1998). In this study, a series of small data sets were found for several countries within different regions of the world. The data sets differed by sex, age and setting, i.e. whether they were studied in a clinical context, were related to urban or rural groups or differed in terms of their economic status. By specifying these variables, Pelletier and Rahn (1998) were able to identify the magnitude and significance of these variables and still develop generic equations for the overall BMI changes with time in different regions. These estimates of time trends per decade are presented in Table 8.43.

Given the fact that these data relate to the 1980s and that the equations are often based on data from clinics or other small groups, more recent evidence from larger and more representative samples was sought, again with the specification that the BMIs should be measured and that mean BMI values were available.

Popkin et al. (2002) have recently presented valuable new data on annual increments in the prevalence of overweight and obesity. Unfortunately this approach does not use the continuous approach to BMI–risk relationships and extrapolating from the equations for the curvilinear relationship between the mean BMI and the prevalence of

Table 8.43 Predicted regional rates of change in BMI per decade for adults

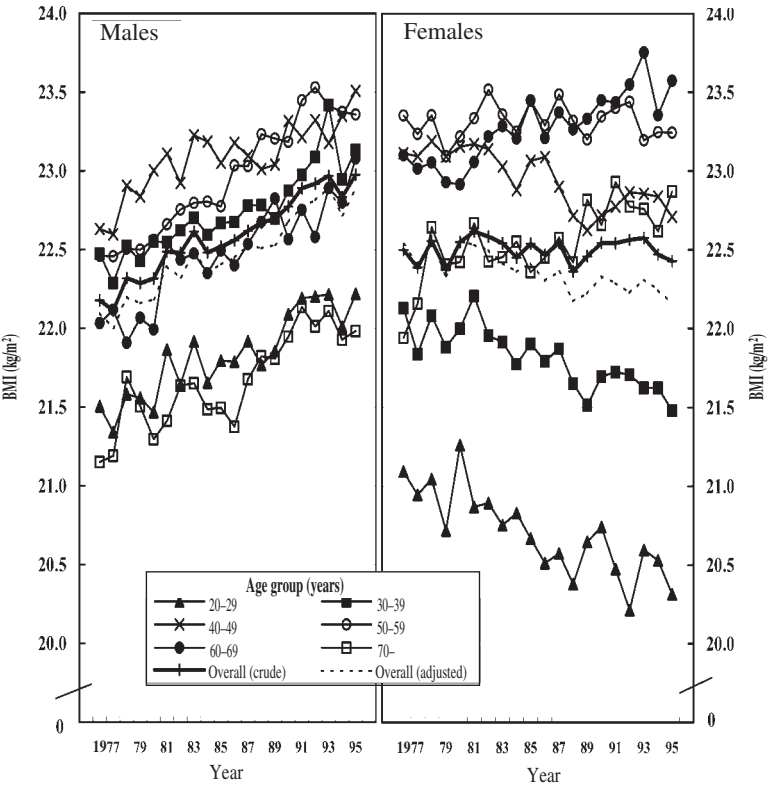
Country or area	*Mean BMI change per decade*
Sub-Saharan Africa	0.207
South and South-East Asia	0.169
India	0.048
Australasia	–0.295
Polynesia and Micronesia	0.957
Latin America and the Caribbean	0.112
China	0.106

obesity at BMIs of ≥30 kg/m^2 (see Figure 8.2) proved too inaccurate, given the data provided on annual increment in obesity and the need to extrapolate to 2030.

There is, however, a series of recent analyses that attempt to look at changes in body weight over the last 10–40 years. Some of these analyses, for example from Brazil (Monteiro et al. 1995), several European countries (Molarius et al. 2000), India (Shetty 2002), Japan (Yoshiike 2002), and the United States (Flegal and Troiano 2000), are based on repeated national representative data and allow some preliminary subregional estimates to be developed. To these data it was possible to add background data sets while recognizing that many were not representative of a country, let alone a subregion and that sex-specific rates may well be very different in different societal settings.

The uncertainty associated with assuming that both sexes and all age groups are likely to respond in the same way over the next 30 years is illustrated by Figure 8.18, which sets out the detailed age- and sex-based time trends in mean BMI for the Japanese population (Yoshiike 2002). It is evident that whereas the mean BMI for men has increased in a linear fashion between 1977 and 1995 in all age groups, the mean BMI for women aged <50 years has shown a progressive reduction amounting to about 0.3 BMI units per decade. Given that the mean BMI is now 20.4 kg/m^2 for young women aged 20–29 years and 22.2 kg/m^2 for women aged 30–49 years, it is clear that predicting a continuation of this trend for the subsequent 35 years, that is, to 2030, would mean that the average BMI of the younger women would fall to 19.1 kg/m^2; this in turn would imply an appreciable increase in the proportion of underweight women with potentially increased morbidity (Shetty and James 1994). Yet we know that Japanese children, both girls and boys, are becoming heavier. Therefore modelling the changes in BMI by age and sex for the next 30 years presents difficulties. What is clear, however, is that the age-related changes in BMI were

Figure 8.18 Annual changes in mean BMI, by age and sex



Note: Adjusted for age distribution by use of the new world population (WHO 1993).
Source: The National Nutrition Survey, Japan, 1976–1995.

maintained from 1976 to 1995 so these are unlikely to change, unless the weights of Japanese children have shown a particularly acute recent increase.

Bearing in mind the uncertainty of these predictions, the assumption was made that the current trends in any subregion would persist in unremitting manner for the next 30 years. It was assumed that data obtained from a country within a subregion would apply to the whole adult population within that subregion. Where more than one data set was available, the values were adjusted not only for the intervals of study and the date on which measurements were made, but also for the differential population numbers in the countries being measured, with appropriate adjustments to the overall population numbers in the subregion. Only adult data were considered; no allowances were made for

children's BMIs since this would have additionally required the prediction of the probability that the current BMIs of children, expressed in percentiles of BMI for age and sex, would be maintained on the same percentiles into adult life on the basis of the BMI percentile charts produced by the IOTF (Cole et al. 2000). Where the data only covered adults aged ≤60 years, it was assumed that the same delta changes were also occurring in the older age groups, thus maintaining the current age-related differences in mean BMI within the subregion.

Different subregional approaches

Given the paucity of data, the following extrapolations proved necessary for certain subregions.

AFR-D: The data relied predominantly on the observed changes in BMI in Ghanaian women, with additional data being available from Mauritius and the Seychelles. The latter two island data sets for both sexes were then used in conjunction with data from South Africa to assess the differential sex-specific trends. These differentials were then used in conjunction with the Ghanaian female data to derive male values for the subregion.

AFR-E: Here it proved necessary to use data on the secular increases found in rural South African men and women (Temple et al. 2001). It was expected that urban trends might have been greater, but given categorical analyses suggesting more modest increases in the prevalence of overweight in the United Republic of Tanzania and Zambia, the rural South African data were applied to the whole subregion.

AMR-A: This was relatively straightforward given the availability of data sets from both Canada (Tremblay et al. 2002) and the United States (Flegal and Troiano 2000). The changing prevalence of overweight and obesity in Cuba was noted (Rodriguez-Ojea et al. 2002), but it was not possible to obtain suitable data on mean BMI changes. Therefore the North American data sets were applied to the whole subregion.

AMR-B: There are ample Brazilian data on secular trends and a series of unpublished analyses from other countries, but the only published and available Brazilian data were those giving overall adult values (Monteiro et al. 1995). However, new analyses from a national survey carried out in Mexico in 2000 were made available (Sánchez-Castillo et al. 2003), together with earlier representative Mexican data provided by Arroyo and colleagues (Arroyo et al. 2000).

AMR-D: In the absence of satisfactory data, the equations of Pelletier and Rahn (1998) were applied.

EMR-B: Kuwaiti data (al-Isa 1997) were applied to the whole subregion, it being recognized that there were numerous published data sets from small clinical and other studies available, as well as several recent unpub-

lished national data sets which highlight the marked secular trends in the subregion.

EMR-D: Moroccan categorical analyses (Benjelloun 2002) having suggested marked BMI increases, the Kuwaiti data were also applied to this subregion.

EUR-A: There were ample data sets from Belgium, Denmark, Finland, Germany, the Netherlands and the United Kingdom that could be used to derive an overall set of population-adjusted, sex-specific and age-related predictions.

EUR-B: Reliance was placed on data on secular trends in Poland.

EUR-C: Although some categorical Hungarian trends data were available, it was concluded from subregional cross-sectional data that the Polish trends data should be applied to this subregion.

SEAR-B: In the absence of data on mean BMI trends, Pelletier and Rahn's equation (1998) for south and east Asia was used.

SEAR-D: Indian data from the repeated nationally-representative surveys were used for this subregion.

WPR-A: Both Australian and the extensive Japanese representative data could be used for this subregion.

WPR-B: Although new data sets are becoming available for the numerically dominant country (China), comparable data sets with mean BMIs from a variety of adult men and women were only available from Zhou (2002). To these data sets were added information from Malaysia, the Republic of Korea and Samoa.

The predicted mean BMIs for the different subregions in 2030 are set out in Table 8.44. As already indicated, these predicted values encompass considerable uncertainties but do suggest that if current trends continue then in some subregions (e.g. AMR-A, AMR-B, EUR-A and EUR-B) half of the adults in each of several age groups will have estimated BMIs of >30 kg/m^2, that is, the WHO classification of obesity. Given that there is now intense concern about escalating rates of obesity, it is very likely that new public health measures will be adopted to limit this rise, but so far the efforts of millions of people in affluent societies to either slim or limit weight gain seems to have been of only modest success. The challenge is to arrest the current trends towards increases in BMI and, if possible, to reverse the public health burden associated with weight gain. Although traditionally the prevalence rates of overweight and obesity are used as an index of the health burden, the current analyses show that the full range of BMIs should be considered. This in turn emphasizes the need to take population-based approaches to preventive strategies for minimizing the hazards of excess weight gain.

Table 8.44 Predicted mean BMIs in each subregion in 2030, by sex and age

		BMI (kg/m²) Age (years)				
Subregion	*Sex*	*30–44*	*45–59*	*60–69*	*70–79*	*≥80*
AFR-D	Male	21.9 (3)[a]	22.4 (3)	21.2 (3)	21.2 (3)	20.7 (4)
	Female	23.3 (5)	22.2 (6)	22.2 (6)	21.4 (6)	22.0 (6)
AFR-E	Male	23.1 (10)	23.3 (10)	22.9 (10)	24.1 (10)	21.9 (11)
	Female	28.0 (22)	27.9 (22)	27.4 (23)	25.5 (25)	24.0 (27)
AMR-A	Male	28.8 (9)	31.1 (13)	31.4 (15)	28.0 (5)	28.4 (13)
	Female	29.5 (13)	32.0 (16)	30.3 (10)	29.2 (9)	27.3 (9)
AMR-B	Male	28.0 (12)	28.0 (9)	27.9 (9)	28.5 (9)	27.1 (10)
	Female	29.2 (12)	30.0 (10)	28.9 (7)	28.7 (7)	27.5 (8)
AMR-D	Male	25.7 (2)	26.3 (2)	26.4 (2)	26.7 (2)	26.7 (2)
	Female	26.2 (2)	27.0 (2)	27.2 (2)	27.0 (2)	26.6 (2)
EMR-B	Male	27.8 (13)	27.8 (10)	24.9 (3)	23.7 (3)	24.1 (3)
	Female	29.0 (12)	28.9 (9)	26.1 (2)	23.9 (3)	26.6 (2)
EMR-D	Male	23.3 (7)	23.4 (7)	23.1 (7)	22.6 (8)	21.6 (8)
	Female	25.8 (8)	24.6 (8)	24.1 (8)	23.1 (9)	20.7 (10)
EUR-A	Male	28.2 (7)	29.9 (10)	30.5 (10)	30.2 (10)	28.5 (9)
	Female	25.7 (2)	30.2 (10)	31.1 (10)	30.5 (10)	28.5 (11)
EUR-B	Male	28.0 (12)	29.5 (11)	30.3 (11)	28.8 (12)	27.8 (12)
	Female	27.2 (6)	29.6 (6)	30.6 (6)	30.4 (5)	28.7 (4)
EUR-C	Male	28.2 (12)	28.1 (8)	28.9 (12	28.2 (12)	26.8 (8)
	Female	27.3 (3)	28.2 (–1)	27.7 (–4)	27.0 (–1)	26.6 (5)
SEAR-B	Male	23.2 (3)	24.0 (3)	23.6 (3)	23.2 (3)	23.2 (3)
	Female	23.3 (3)	24.5 (3)	24.9 (3)	23.1 (3)	23.1 (3)
SEAR-D	Male	21.6 (10)	20.8 (10)	21.0 (9)	19.8 (10)	21.2 (9)
	Female	21.7 (4)	22.0 (3)	19.5 (3)	20.2 (3)	16.6 (4)
WPR-A	Male	25.1 (6)	25.8 (8)	25.2 (8)	24.4 (7)	23.5 (7)
	Female	21.5 (–4)	23.5 (–1)	25.1 (5)	24.5 (5)	23.8 (5)
WPR-B	Male	26.1 (15)	26.4 (14)	26.1 (15)	25.6 (15)	23.6 (14)
	Female	26.1 (15)	26.8 (14)	26.9 (14)	25.9 (15)	24.4 (16)

[a] Figures in parentheses refer to the percentage increase over the 2000 estimate.

8.2 The implications of excessive weight gain among children

The foregoing analyses of the disease burden associated with increases in BMI relate only to adults aged ≥30 years. However, there is now rapidly mounting concern regarding the increasing prevalence of overweight and obesity in children (Ebbeling et al. 2002). The impact of psychosocial, physical and metabolic problems has not been incorporated in the current analyses, but may well have to be considered within the next 5–10 years. Overweight children in many countries are handicapped by the social stigma of being considered overtly fat by their peers. In

addition, these children have a propensity to bone and joint deformation during their growing phase, as well as breathlessness and, in more extreme cases, sleep apnoea arising from the mechanical handicap of being heavy. It is now clear that overweight children also have higher blood pressure, serum lipid abnormalities and increasing insulin resistance, all of which are hallmarks of early metabolic disease and susceptibility to atherosclerosis and other cardiovascular problems (DiPietro et al. 1994; Must et al. 1992; Unger et al. 1990). Within the last 5 years, paediatricians have observed that clinics for children with type I diabetes now include a remarkably increasing number of very overweight children with type II diabetes. In many parts of the world, type II diabetes occurring in adolescence is now a more prevalent condition than type I diabetes (Rosenbloom et al. 1999). As with adult type II diabetes, these children show a remarkable improvement in their glucose-handling capacity and in other risk factors if they lose weight, but many soon develop a need for insulin therapy and their condition becomes difficult to control. Children with poorly controlled diabetes are known to have accelerated atherosclerosis with microvascular disease leading to blindness and renal failure in their early 30s. Concern regarding obesity and type II diabetes in children is now being accentuated by the recognition that mothers who develop gestational diabetes produce larger babies who are then very susceptible to pre-adolescent obesity and to the development of type II diabetes in adolescence (Silverman et al. 1998).

People who are overweight in childhood are more prone to be obese when they enter adult life and these individuals are also likely to continue to gain weight in adulthood. Such individuals then have up to a 100-fold increased risk of developing type II diabetes compared with normal weight children who do not gain excessive weight once they are adults (Colditz et al. 1995). Early data from insurance companies also showed that heavy young adults had a much greater likelihood of suffering premature deaths (Blair and Haines 1966) and this is now recognized to be related to their greater propensity to hypertension, dyslipidaemia, glucose intolerance with insulin resistance and accelerated cardiovascular disease. These observations are relevant to any assessment of the future burden of disease arising from high BMIs in adults because the rapidly rising prevalence of overweight and obesity in children is likely to amplify the currently predicted risks of excess weight in young adults.

Acknowledgements

We are most grateful for the support of numerous individuals and institutions in gathering data for this analysis. In addition we wish to acknowledge the assistance of Mrs Jean James in preparing the manuscript for this chapter.

The following contributed invaluably by providing re-analysed data to assist in the IOTF's analyses: N. Adra, P. Arroyo, K. Babinska, G. Biro, A. Deev, T.P. Gill, N. Hwalla, M. Lahti-Koski, B. Lajos, A. Ferro-Luzzi, A. Kaic-Rak, G. Mensink, J.C. Montero, A. Nielsen, I. Palczewska, G. Pekcan, N. Rak, C.P. Sánchez-Castillo, G. Whitlock and the Asia-Pacific Cohort Studies Collaboration.

In addition, the following assisted by allowing access to data or suggesting sources:
T. Athanassouli, W. Becker, Z. Brazdova, W. Chen, W. Freire, V. Gudnason, K. Habibullah, U. Hagman, B. Heitman, N. Katsilambros, J. Luthy, V. Lambert, A.O. Musaiger, A. Neilson, L. Ovesen, R. Prattala, W. Rand, K.S. Reddy, A. Robertson, J. Seidell, L. Steingrimsdottir, M.Q.-K. and K. Talukder, R. Uauy, and M. Vasconcellos.

Notes

1 See preface for an explanation of this term.

2 The standard deviation (SD) was sometimes missing for different sexes and age groups. This problem was dealt with as follows:

SD missing for one sex; for example, there were some subregions where SDs were available only for females

For all subregions with SDs available for both men and women, SDs were in general higher in females than males. The mean difference between the SDs of females and males varied from 0.24–2.25 units, with the largest differences observed between higher SD values. For example, subregions with SDs of >5–6 units in women tended to have SDs in these women which were >1 unit higher than those in men, whereas with female SD values of 3–4.5, the sex differences in SDs were <1.

The mean of the differences when the SD values were <1 unit was 0.6 units, and for those >1 unit was 1.63. As a result, to estimate male SD from female SD in subregions where SD values were <5 in most age groups, a simple subtraction of 0.6 from the SD for females was used to estimate the male SD. In other subregions where SDs were higher in females, a value of 1 was subtracted from the female SD to estimate the corresponding male SD. The same approach was applied to all age groups. These differences in SDs by sex are then reflected in the differences in prevalences of overweight and obesity in men and women with the same mean BMI.

For example, in AMR-D an SD was available for females only and the values varied from 3.8–4.6 units. Subtracting 0.6 from these gave the corresponding values for men of 3.2, 3.8 and 4.0. In EMR-D, again only SDs for women were obtained with values varying from 6.4 to 8.5. In this case, the correction factor of 1.6 was applied to obtain the estimates for men.

SD missing for some age groups

There was no clear pattern of SD by age group. For the age groups where no SDs were available, it was assumed that the SD was equal to an average

of the observed SD for the subregion. For example, in AMR-D, the mean SD for females aged 18–59 years was (3.4+4.4+4.6)/3=4.13.

This figure was then applied to the remaining age groups where no SDs for females were available.

References

Acheson RM, Kelsy JL, Ginsburg GN (1974) The New Haven survey of joint diseases. XVI. Impairment, disability and arthritis. *British Journal of Preventive Medicine*, **27**:168–176.

Ajlouni K, Jaddou H, Batieha A (1998) Obesity in Jordan. *International Journal of Obesity and Related Metabolic Disorders*, **22**:624–628.

al-Isa AN (1995) Prevalence of obesity among adult Kuwaitis. *International Journal of Obesity*, **19**:431–433.

al-Isa AN (1997) Temporal changes in body mass index and prevalence of obesity among Kuwaiti men. *Annals of Nutrition and Metabolism*, **41**:307–314.

Allison DB, Fontaine KR, Manson JE, Stevens J, VanItallie TB (1999) Annual deaths attributable to obesity in the United States. *Journal of the American Medical Association*, **282**:1530–1538.

al-Mannai A, Dickerson JW, Morgan JB, Khalfan H (1996) Obesity in Bahraini adults. *Journal of the Royal Society of Health*, **116**:30–32, 37–40.

al-Nuaim AR, al-Rubeaan K, al-Mazrou Y, al-Attas O, al-Daghari N, Khoja T (1996) High prevalence of overweight and obesity in Saudi Arabia. *International Journal of Obesity*, **20**:547–552.

Anderson JJ, Felson DT (1998) Factors associated with osteoarthritis of the knee in the first national health and nutritional examination survey (NHANES I). *American Journal of Epidemiology*, **128**:179–189.

Anonymous (1999) *Victorian burden of disease study: morbidity*. Public Health Division, Department of Human Services, Melbourne.

Arroyo P, Loria A, Fernández V et al. (2000) Prevalence of pre-obesity and obesity in urban adult Mexicans in comparison with other large surveys. *Obesity Research*, **8**:179–185.

Ascherio A, Rimm EB, Giovannucci EL et al. (1992) A prospective study of nutritional factors and hypertension among US men. *Circulation*, **86**:1475–1484.

Barker DJP (1998) *Mothers, babies and health in later life*. Churchill Livingstone, London.

Bell AC, Swinburn BA, Simmons D, Wang W, Amosa H, Gatland B (2001) Heart disease and diabetes risk factors in Pacific Islands communities and associations with measures of body fat. *New Zealand Medical Journal*, **114**:364–365.

Benjelloun S (2002) Nutrition transition in Morocco. *Public Health Nutrition*, **5**:135–140.

Bergmann KE, Mensink GBM (1999) Korpermasse und Ubergewicht. *Gesundheitswesen*, **62**:S115–120.

Bergström A, Pisani P, Tenet V, Wolk A, Adami HO (2001) Overweight as an avoidable cause of cancer in Europe. *International Journal of Cancer*, **91**:421–430.

Berkham LF, Breslow L (1983) *Health and ways of living: the Alameda county studies*. Oxford University Press. New York.

Björntorp P, Rosmond R (1999) The hypothalamic origin of the metabolic Syndrome X. *Annals of the New York Academy of Sciences*, **892**:297–307.

Blair BF, Haines LW (1966) Mortality experience according to build at the higher durations. *Transactions of the Actuarial Society,* **18**:35–41.

Bovet P, Shamlaye C, Kitua A et al. (1991) High prevalence of cardiovascular risk factors in the Seychelles (Indian Ocean). *Arteriosclerosis Thrombosis*, **11**:1730–1736.

Calle EE, Thun MJ, Petrelli JM, Rodriguez C, Heath CW Jr (1999) Body-mass index and mortality in a prospective cohort of US adults. *New England Journal of Medicine*, **341**:1097–1105.

Carey VJ, Walters EE, Colditz GA et al. (1997) Body fat distribution and risk on non-insulin dependent diabetes mellitus in women. The nurses' health study. *American Journal of Epidemiology*, **145**:614–619.

Chaichareon P, Leeiahagul P, Tanphaichitr V (1992) Body mass index in adults with dental diseases. *Internal Medicine*, **8**:113–117.

Chan JM, Rimm EB, Colditz GA, Stampfer MJ, Willett WC (1994) Obesity, fat distribution, and weight gain as risk factors for clinical diabetes in men. *Diabetes Care*, **17**:961–969.

Chilima DM, Ismail SJ (1998) Anthropometric characteristics of older people in rural Malawi. *European Journal of Clinical Nutrition*, **52**:643–649.

Cicuttini FM, Baker JR, Spector TD (1996) The association of obesity with osteoarthritis of the hand and knee in women: a twin study. *Journal of Rheumatology*, **23**:1221–1226.

Cicuttini FM, Spector TD (1998) Obesity, arthritis and gout. In: *Handbook of obesity.* Bray GA, Bouchard C, James WPT, eds. Marcel Dekker Inc, New York.

Colditz GA, Willett WC, Rotnitzky A, Manson JE (1995) Weight gain as a risk factor for clinical diabetes mellitus in women. *Annals of Internal Medicine*, **122**:481–486.

Colditz GA, Willett WC, Stampfer MJ et al. (1990) Weight gain as a risk factor for clinical diabetes mellitus in women. *American Journal of Epidemiology*, **132**:501–513.

Cole TJ, Bellizzi MC, Flegal KM, Dietz WH (2000) Establishing a standard definition for child overweight and obesity worldwide: international survey. *British Medical Journal*, **320**:1240–1243.

Cooper RS, Ford E (1992) Comparability of risk factors for coronary heart disease among blacks and whites in the NHANES-I Epidemiologic Follow-up Study. *Annals of Epidemiology*, **2**:637–645.

Dattilo AM, Kris-Etherton PM (1992) Effects of weight reduction on blood lipids and lipoproteins: a meta-analysis. *American Journal of Clinical Nutrition*, **56**:320–328.

Dawber TR, Meadors GF, Moore FE (1951) Epidemiological approaches to heart disease: the Framingham study. *American Journal of Public Health*, **41**:279–286.

de Onis M, Blössner M (2000) Prevalence and trends of overweight among preschool children in developing countries. *American Journal of Clinical Nutrition*, **72**:1032–1039.

Despres JP, Lemieux I, Prud'homme D (2001) Treatment of obesity: need to focus on high risk abdominally obese patients. *British Medical Journal*, **322**:716–720.

Deurenberg P, Deurenberg-Yap M, Guricci S (2002) Asians are different from Caucasians and from each other in their body mass index/body fat percent relationship. *Obesity Reviews*, **3**:141–146.

Deurenberg P, Yap M, van Staveren WA (1998) Body mass index and percent body fat: a meta-analysis among different ethnic groups. *International Journal of Obesity*, **22**:1164–1171.

Diabetes Prevention Program Research Group (2002) Reduction in the incidence of Type 2 diabetes with lifestyle intervention or Metformin. *New England Journal of Medicine*, **346**:393–403.

Dietz WH, Bellizzi MC (1999) Assessment of childhood and adolescent obesity. *American Journal of Clinical Nutrition*, **70**:S117–175.

DiPietro L, Mossberg HO, Stunkard AJ (1994) A 40-year history of overweight children in Stockholm: life time overweight, morbidity and mortality. *International Journal of Obesity and Related Metabolic Disorders*, **18**:585–590.

Dobson AJ, Evans A, Ferrario M et al. (1998) Changes in estimated coronary risk in the 1980s: data from 38 populations in the WHO MONICA project. World Health Organization. Monitoring trends and determinants in cardiovascular diseases. *Annals of Medicine*, **30**:199–205.

Drivsholm T, Ibsen H, Schroll M, Davidsen M, Borch-Johnsen K (2001) Increasing prevalence of diabetes mellitus and impaired glucose tolerance among 60-year-old Danes. *Diabetic Medicine*, **18**:126–132.

Dyer, AR, Elliot P (1989) The INTERSALT study: relations of body mass index to blood pressure. INTERSALT Co-operative Research Group. *Journal of Human Hypertension*, **3**:299–308.

Eason RJ, Pada J, Wallace R, Henry A, Thornton R (1987) Changing patterns of hypertension, diabetes, obesity and diet among Melanesians and Micronesians in the Solomon Islands. *Medical Journal of Australia*, **146**:465–469, 473.

Ebbeling CB, Pawlak DB, Ludwig DS (2002) Childhood obesity: public-health crisis, common sense cure. *The Lancet*, **360**:473–482.

Edelstein SL, Knowler WC, Bain RP et al. (1997) Predictors of progression from impaired glucose tolerance to NIDDM: an analysis of six prospective studies. *Diabetes*, **46**:701–710.

Eichholzer M, Luthy J, Gutzwiller F (1999) Epidemiology of overweight in Switzerland: results of the Swiss National Health Survey 1992–3. *Schweizerische Medizinische Wochenschrift*, **129**:353–361.

el Mugamer IT, Ali Zayat AS, Hossain MM, Pugh RN (1995) Diabetes, obesity and hypertension in urban and rural people of Bedouin origin in the United Arab Emirates. *Journal of Tropical Medical Hygiene*, **98**:407–415.

Epstein FH, Napier JA, Block WD et al. (1970) The Tecumseh study: design, progress, and perspectives. *Archives of Environmental Health*, **21**:402–407.

Eriksson KF, Lindgarde F (1991) Prevention of type 2 (non-insulin-dependent) diabetes mellitus by diet and physical exercise. The 6-year Malmo feasibility study. *Diabetologia*, **34**:891–898.

Flegal KM, Troiano RP (2000) Changes in the distribution of body mass index of adults and children in the US population. *International Journal of Obesity*, **24**:807–818.

Ford ES, Giles WH, Dietz WH (2002) Prevalence of the metabolic syndrome among US adults: findings from the third National Health and Nutrition Examination Survey. *Journal of the American Medical Association*, **287**: 356–359.

Franklin MF (1999) Comparison of weight and height relations in boys from 4 countries. *American Journal of Clinical Nutrition*, **70**:S157–162.

Gerhardsson de Verdier M, Hagman U, Steineck G, Rieger A, Norell SE (1990) Diet, body mass and colorectal cancer: a case-referent study in Stockholm. *International Journal of Cancer*, **46**: 832–838.

Giay T, Khoi HH (1994) Use of body mass index in the assessment of adult nutritional status in Vietnam. *European Journal of Clinical Nutrition*, **48**:S124–130.

Giovannucci E, Ascherio A, Rimm EB, Colditz GA, Stampfer MJ, Willett WC (1995) Physical activity, obesity, and risk for colon cancer and adenoma in men. *Annals of Internal Medicine*, **122**:327–334.

Goldstein DJ (1992) Beneficial health effects of modest weight loss. *International Journal of Obesity*, **16**:397–415.

Gonzales GF, Villena A, Gonez C, Zevallos M (1994) Relationship between body mass index, age, and serum adrenal androgen levels in Peruvian children living at high altitude and at sea level. *Human Biology*, **66**:145–153.

Gorzelniak K, Engeli S, Janke J, Luft FC, Sharma AM (2002) Hormonal regulation of the human adipose-tissue renin-angiotensin system: relationship to obesity and hypertension. *Journal of Hypertension*, **20**:965–973.

Hanley AJ, Harris SB, Gittelsohn J, Wolever TM, Saksvig B, Zinman B (2000) Overweight among children and adolescents in a Native Canadian community: prevalence and associated factors. *American Journal of Clinical Nutrition*, **71**:693–700.

Harris ML, Goldstein DA, Flegal KM et al. (1998) Prevalence of diabetes, impaired fasting glucose, and impaired glucose tolerance in U.S. adults. *Diabetes Care*, **21**:518–524.

Harris TB, Ballard-Barbasch R, Madans J, Makuc DM, Feldman JJ (1993) Overweight, weight loss, and risk of coronary heart disease in older women. The NHANES I Epidemiologic Follow-up Study. *American Journal of Epidemiology*, **137**:1318–1327.

Harris TB, Launer LJ, Madans J, Feldman JJ (1997) Cohort study of effect of being overweight and change in weight on risk of coronary heart disease in old age. *British Medical Journal*, **314**:1791–1794.

Hart DJ, Spector TD (1993) The relationship of obesity, fat distribution and osteoarthritis in women in the general population: the Chingford study. *Journal of Rheumatology*, **20**:331–335.

Helmrich SP, Ragland DR, Leung RW, Paffenbarger RS Jr (1991) Physical activity and reduced occurrence of non-insulin-dependent diabetes mellitus. *New England Journal of Medicine*, **325**:147–152.

Hernandez RE, Cardonnet LJ, Libman C, Gagliardino JJ (1987) Prevalence of diabetes and obesity in an urban population of Argentina. *Diabetes Research and Clinical Practice*, **3**:277–283.

Higgins M, Kannel W, Garrison R et al. (1988) Hazards of obesity—the Framingham experience. *Acta Medica Scandinavica, Supplementum*, **723**:23–36.

Hill AB (1965) The environment and disease: association or causation? *Proceedings of the Royal Society of Medicine*, **58**:295–300.

Hochberg MC, Lawrence RC, Everett DF, Coroni-Huntley J (1989) Epidemiological associations of pain in osteoarthritis of the knee: data from the National Health and Nutrition Examination Survey and Nutrition Examination-I Epidemiological Follow-up Survey. *Seminars in Arthritis and Rheumatism*, **18**:4–9.

Huang Z, Hankinson SE, Colditz GA et al. (1997) Dual effects of weight and weight gain on breast cancer risk. *Journal of the American Medical Association*, **278**:1407–1411.

IARC (2002) *IARC Handbook of cancer prevention, Vol. 6. Weight control and physical activity*. Vainio H, Bianchini F, eds. International Agency for Research on Cancer, Lyon.

James WPT, Ferro Luzzi A, Waterlow JC (1988) Definition of chronic energy deficiency in adults. Report of a Working Party of the International Dietary Energy Consultative Group. *European Journal of Clinical Nutrition*, **42**:969–981.

James WPT, Ralph A, Ferro-Luzzi A (1989) Energy needs of the elderly: a new approach. In: *Human nutrition. Ageing and the elderly.* Munro HN, Danford DE, eds. Plenum Press, New York.

James WPT, Francois P (1994) The choice of cut-off point for distinguishing normal body weight from underweight or 'chronic energy deficiency' in adults. *European Journal of Clinical Nutrition*, **48**:S179–184.

James WPT, Norum K, Smitasiri S et al. (2000) *Ending malnutrition by 2020: an agenda for change in the millennium*. (Final report to the ACC/SCN by the Commission on the nutrition challenges of the 21st century. Supplement to the Food and Nutrition Bulletin, September/October 2000.) UNU International Nutrition Foundation, USA.

James WPT, Reeds PJ (1997) Nutrient partitioning. In: *Handbook on obesity.* Bray GA, Bouchard C and James WPT, eds. Marcel Dekker Inc, New York.

Jarrett RJ, Shipley MJ, Rose G (1982) Weight and mortality in the Whitehall study. *British Medical Journal*, **285**:535–537.

Jimenez JT, Palacios M, Canete F et al. (1998) Prevalence of diabetes mellitus and associated cardiovascular risk factors in an adult urban population in Paraguay. *Diabetic Medicine*, **15**:334–338.

Jones DW, Kim JS, Andrew ME, Kim SJ, Hong YP (1994) Body mass index and blood pressure in Korean men and women: the Korean National Blood Pressure Survey. *Journal of Hypertension*, **12**:1433–1437.

Jousilahti P, Vartiainen E, Tuomilehto J et al. (1999) Sex, age, cardiovascular risk factors, and coronary heart disease: a prospective follow-up study of 14 786 middle-aged men and women in Finland. *Circulation*, **99**:1165–1172.

Kain J, Uauy R, Vio F, Albala C (2002) Trends in overweight and obesity prevalence in Chilean children: comparison of three definitions. *European Journal of Clinical Nutrition*, **56**:200–204.

Keys A, Aravanis C, Blackburn H et al. (1972) Coronary heart disease: overweight and obesity as risk factors. *Annals of Internal Medicine*, **77**:15–27.

Khor GL, Yusof AM, Siong Tee E, Kandiah M, Lee Huang MS (1999) Prevalence of overweight among Malaysian adults from rural communities. *Asia Pacific Journal of Clinical Nutrition*, **8**:272–279.

Kromeyer-Hauschild K, Zellner K, Jaeger U, Hoyer H (1999) Prevalence of overweight and obesity among school children in Jena (Germany). *International Journal of Obesity*, **23**:1143–1150.

Kune GA, Kune S, Watson LF (1990) Body weight and physical activity as predictors of colorectal cancer risk. *Nutrition and Cancer*, **13**:9–17.

Lahti-Koski M, Vartiainen E, Mannisto S, Pietinen P (2000) Age, education and occupation as determinants of trends in body mass index in Finland from 1982 to 1997. *International Journal of Obesity*, **24**:1669–1676.

Lapidus L, Bengtsson C, Larsson B, Pennert K, Rybo E, Sjöström L (1984) Distribution of adipose tissue and risk of cardiovascular disease and death: a 12 year follow up of participants in the population study of women in Gothenburg, Sweden. *British Medical Journal*, **289**:1257–1261.

Larsson B, Svärdsudd K, Welin L, Wilhelmsen L, Björntorp P, Tibblin G (1984) Abdominal adipose tissue distribution, obesity and risk of cardiovascular disease and death: 13 year follow up of participants in the study of men born in 1913. *British Medical Journal*, **288**:1401–1404.

Lean ME, Powrie JK, Anderson AS, Garthwaite PH (1990) Obesity, weight loss and prognosis in type 2 diabetes. *Diabetic Medicine*, **7**:228–233.

Lee I-M and Paffenbarger RS Jr (1992) Quetelet's index and risk of colon cancer in college alumni. *Journal of the National Cancer Institute*, **84**:1326–1331.

Le Marchand L, Wilkens LR, Mi MP (1991) Early-age body size, adult weight gain and endometrial cancer risk. *International Journal of Cancer*, **48**: 807–811.

Lew EA, Garfinkel L (1979) Variations in mortality by weight among 750 000 men and women. *Journal of Chronic Diseases*, **32**:563–576.

Lindsted K, Tonstad S, Kuzma JW (1991) Body mass index and patterns of mortality among Seventh-day Adventist men. *International Journal of Obesity*, **15**:397–406.

Macdonald SM, Reeder BA, Chen Y, Despres JP (1997) Obesity in Canada: a descriptive analysis. Canadian Heart Health Surveys Research Group. *Canadian Medical Association Journal*, **157**:S3–9.

MacMahon S, Cutler J, Brittain E, Higgins M (1987) Obesity and hypertension: epidemiological and clinical issues. *European Heart Journal*, **8**:S57–70.

Manson JE, Colditz GA, Stampfer MJ et al. (1990) A prospective study of obesity and risk of coronary heart disease in women. *New England Journal of Medicine*, **322**:882–889.

Marshall JA, Shetterly S, Hoag S et al. (1994) Dietary fat predicts conversion from impaired glucose tolerance to NIDDM. The San Luis Valley Diabetes study. *Diabetes Care*, **17**:50–56.

Martinez ME, Giovannucci E, Spiegelman D, Hunter DJ, Willett WC, Colditz GA (1997) Leisure-time physical activity, body size and colon cancer in women. Nurses' Health Study Research Group. *Journal of the National Cancer Institute*, **89**:948–955.

Martorell R, Schroeder DG, Rivera JA, Kaplowitz HJ (1995) Patterns of linear growth in rural Guatemalan adolescents and children. *Journal of Nutrition*, **125**:S1060–1067.

Mathers C, Vos T, Stevenson C (1999) The Burden of disease and injury in Australia, 1999. (AIHW Catalogue No. PHE 17.) AIHW, Canberra.

McCarthy SN, Harrington KE, Kiely M et al. (2001) Analyses of the anthropometric data from the North/South Ireland Food Consumption Survey. *Public Health Nutrition*, **4**:1099–1106.

McGarvey ST (1991) Obesity in Samoans and a perspective on its etiology in Polynesians. *American Journal of Clinical Nutrition*, **53**:S1586–1594.

Micozzi MS, Albanes D, Jones DY, Chumlea WC (1986) Correlations of body mass indices with weight, stature, and body composition in men and women in NHANES I and II. *American Journal of Clinical Nutrition*, **44**:725–731.

Molarius A, Seidell JC, Sans S, Tuomilehto J, Kuulasmaa K (2000) Educational level, relative body weight, and changes in their association over 10 years: an international perspective from the WHO MONICA project. *American Journal of Public Health*, **90**:1260–1268.

Monteiro CA, Conde WL (1999) Time trends in overweight prevalence in children, adolescents and adults from less and more developed regions of

Brazil. In: *Progress in obesity research*. Guy Grand B, Ailhaud G, eds. John Libbey & Company, London.

Monteiro CA, Mondini L, de Souza AL, Popkin BM (1995) The nutrition transition in Brazil. *European Journal of Clinical Nutrition*, **49**:105–113.

Moreno LA, Sarria A, Fleta J, Rodriguez G, Bueno M (2000) Trends in body mass index and overweight prevalence among children and adolescents in the region of Aragon (Spain) from 1985 to 1995. *International Journal of Obesity*, **24**:925–931.

Morris JN, Everitt MG, Pollard R et al. (1980) Vigorous exercise in leisure-time: protection against coronary heart disease. *The Lancet*, **2**:1207–1210.

Mulrow DC, Chiquette E, Angel L et al. (2000) Dieting to reduce body weight for controlling hypertension in adults. *Cochrane Database System Review*, **2**:CD000484 [online database].

Musaiger AO, al-Mannai MA (2001) Weight, height, body mass index and prevalence of obesity among adult population in Bahrain. *Annals Human Biology*, **10**:346–350.

Musaiger AO, Gregory WB (2000) Profile of body composition of school children in Bahrain. *International Journal of Obesity*, **24**:1093–1096.

Must A, Jacques PF, Dallal GE, Bajema CJ, Dietz WH (1992) Long-term morbidity and mortality of overweight adolescents. A follow-up of the Harvard Growth Study of 1922 to 1935. *New England Journal of Medicine*, **327**:1350–1355.

Must A, Spadano J, Coakley EH (1999) The disease burden associated with overweight and obesity. *Journal of the American Medical Association*, **282**:1523–1529.

National Audit Office (2001) *Tackling obesity in England. A report by the Comptroller and Auditor General*. National Audit Office, London.

Niedhammer I, Bugel I, Bonenfant S, Goldberg M, Leclerc A (2000) Validity of self-reported weight and height in the French GAZEL cohort. *International Journal of Obesity Related Metabolic Disorders*, **24**:1111–1118.

NIH (1998) *Clinical guidelines on the identification, evaluation and treatment of overweight and obesity in adults: the evidence report*. US Department of Health and Human Services, National Institutes of Health, National Heart, Lung and Blood Institute, USA.

Nishizawa H, Shimomura I, Kishida K et al. (2002) Androgens decrease plasma adiponectin, an insulin-sensitizing adipocyte-derived protein. *Diabetes*, **51**:2734–2741.

Njolstad I, Arnesen E, Lund-Larsen PG (1998) Sex differences in risk factors for clinical diabetes mellitus in a general population: a 12-year follow-up of the Finnmark study. *American Journal of Epidemiology*, **147**:49–58.

Norgan NG (1994a) Population differences in body composition in relation to the body mass index. *European Journal of Clinical Nutrition*, **48**:S10–27.

Norgan NG (1994b) Relative sitting height and the body mass index. *Annals of Human Biology*, **21**:78–82.

Ogden CL, Kuczmarski RJ, Flegal KM et al. (2002) Centers for Disease Control and Prevention 2000 growth charts for the United States: improvements to the 1977 National Center for Health Statistics version. *Pediatrics*, **109**:45–60.

Okesina AB, Oparinde DP, Akindoyin KA, Erasmus RT (1999) Prevalence of some risk factors of ischaemic heart disease in a rural Nigerian population. *East African Medical Journal*, **76**:212–217.

Okosun IS, Cooper RS, Rotimi CN, Osotimehin B, Forrester T (1998) Association of waist circumference with risk of hypertension and type 2 diabetes in Nigerians, Jamaicans, and African-Americans. *Diabetes Care*, **21**:1836–1842.

Okosun IS, Rotimi CN, Forrester TE et al. (2000) Predictive value of abdominal obesity cut-off points for hypertension in Blacks from West African and Caribbean islands nations. *International Journal of Obesity*, **24**:180–186.

Ornish D, Brown SE, Scherwitz LW et al. (1990) Can lifestyle changes reverse coronary heart disease? *The Lancet*, **336**:129–133.

Paffenbarger RS Jr, Laughlin MD, Gima AS, Black RA (1970) Work activity of longshoremen as related to death from coronary heart disease and stroke. *New England Journal of Medicine*, **282**:1109–1114.

Parsons TJ, Power C, Logan S, Summerbell CD (1999) Childhood predictors of adult obesity: a systematic review. *International Journal of Obesity*, **23**: S1–107.

Pelletier DL and Rahn M (1998) Trends in body mass index in developing countries. *Food and Nutrition Bulletin*, **19**:223–239.

Pishdad GR (1996) Overweight and obesity in adults aged 20–74 in southern Iran. *International Journal of Obesity*, **20**:963–965.

Popkin BM (2002) An overview on the nutrition transition and its health implications: the Bellagio meeting. *Public Health Nutrition*, **5**:93–103.

Reilly JJ, Dorosty AR, Emmett PM, ALSPAC Study Team (2000) Identification of the obese child: adequacy of the body mass index for clinical practice and epidemiology. *International Journal of Obesity*, **24**:1623–1627.

Rexrode KM, Buring JE, Manson JE (2001) Abdominal and total adiposity and risk of coronary heart disease in men. *International Journal of Obesity*, **25**:1047–1056.

Rexrode KM, Hennekens CH, Willett WC et al. (1997) A prospective study of body mass index, weight change, and risk of stroke in women. *Journal of the American Medical Association*, **277**:1539–1545.

Rhoads GG, Kagan A (1983) The relation of coronary disease, stroke, and mortality to weight in youth and in middle age. *The Lancet*, **1**:492–495.

Rimm EB, Stampfer MJ, Giovannucci E et al. (1995) Body size and fat distribution as predictors of coronary heart disease among middle-aged and older US men. *American Journal of Epidemiology*, **141**:1117–1127.

Rissanen A, Heliövaara M, Knekt P et al. (1990) Risk of disability and mortality due to overweight in a Finnish population. *British Medical Association*, **301**:835-837.

Roberts SB, Dallal GE (2001) The new childhood growth charts. *Nutrition Reviews*, **59:**31–36.

Rodriguez-Ojea A, Jimenez S, Berdasco A, Esquivel M (2002) The nutrition transition in Cuba in the nineties: an overview. *Public Health Nutrition*, **5**:129–133.

Rose G, Shipley M (1990) The population mean predicts the number of deviant individuals. *British Medical Journal*, **301**:1031–1034.

Rosenbloom AL, Joe JR, Young RS, Winter WE (1999) Emerging epidemic of type 2 diabetes in youth. *Diabetes Care*, **22**:345–354.

Rosengren A, Wedel H, Wilhelmsen L (1999) Body weight and weight gain during adult life in men in relation to coronary heart disease and mortality. A prospective population study. *European Heart Journal*, **20**:269–277.

Rotimi CN, Cooper RS, Ataman SL et al. (1995) Distribution of anthropometric variables and the prevalence of obesity in populations of West African origin. *Obesity Research*, **3**:S95–105.

Roubenoff R, Klag MJ, Mead LA, Liang KY, Seidler AJ, Hochberg MC (1991) Incidence and risk factors for gout in white men. *Journal of the American Medical Association*, **266**:3004–3007.

Sargeant LA, Bennett FI, Forrester TE, Cooper RS, Wilks RJ (2002) Predicting incident diabetes in Jamaica: the role of anthropometry. *Obesity Research*, **10**:792–798.

Sarkkinen E, Schwab U, Niskanen L et al. (1996) The effects of monounsaturated-fat enriched diet and polyunsaturated-fat enriched diet on lipid and glucose metabolism in subjects with impaired glucose tolerance. *European Journal of Clinical Nutrition*, **50**:592–598.

Sanchez-Castillo CP, Velázquez-Monroy O, Berber A et al. (2003) Anthropometric cut-off points for predicting chronic diseases in the Mexican National Health Survey 2000. *Obesity Research*, **11**:442–451.

Schouten JSAG, van den Ouweland FA, Valkenburg HA (1992) A twelve year follow-up study in the general population on prognostic factors of cartilage loss in osteoarthritis of the knee. *Annals of the Rheumatic Diseases*, **51**:932–937.

Schroeder DG, Martorell R, Flores R (1999) Infant and child growth and fatness and fat distribution in Guatemalan adults. *American Journal of Epidemiology*, **149**:177–185.

Seckl JR, Walker BR (2001) Minireview: 11 β-hydroxysteroid dehydrogenase Type 1: a tissue-specific amplifier of glucocorticoid action. *Endocrinology*, **142**:1371–1376.

Seidell JC, Kahn HS, Williamson DF, Lissner L, Valdez R (2001a) Report from a Centers for Disease Control and Prevention Workshop on use of adult anthropometry for public health and primary health care. *American Journal of Clinical Nutrition*, **73**:123–126.

Seidell JC, Perusse L, Despres JP, Bouchard C (2001b) Waist and hip circumferences have independent and opposite effects on cardiovascular disease risk

factors: the Quebec Family study. *American Journal of Clinical Nutrition*, **74**:315–321.

Shaper AG, Wannamethee SG, Walker M (1997) Body weight: implications for the prevention of coronary heart disease, stroke and diabetes mellitus in a cohort study of middle aged men. *British Medical Journal*, **314**:1311–1317.

Shetty PS (2002) Nutrition transition in India. *Public Health Nutrition*, **5**: 175–182.

Shetty PS, James WPT (1994) *Body Mass Index. A measure of chronic energy deficiency in adults*. (FAO Food and Nutrition Paper No. 56.) Food and Agriculture Organization of the United Nations, Rome.

Silverman BL, Rizzo TA, Cho NH, Metzger BE (1998) Long-term effects of the intrauterine environment. The Northwestern University Diabetes in Pregnancy Center. *Diabetes Care*, **21**:B142–149.

Singh RB, Niaz MA, Agarwal P, Beegum R, Rastogi SS, Singh NK (1995) Epidemiologic study of central obesity, insulin resistance and associated disturbances in the urban population of North India. *Acta Cardiologica*, **3**:215–225.

Sjöström CD, Lissner L, Wedel H, Sjöström L (1999) Reduction in incidence of diabetes, hypertension and lipid disturbances after intentional weight loss induced by bariatric surgery: the SOS intervention study. *Obesity Research*, **7**:477–484.

Sjöström CD, Peltonen M, Wedel H, Sjöström L (2000) Differentiated long-term effects of intentional weight loss on diabetes and hypertension. *Hypertension*, **36**:20–25.

Sjöström L, Larsson B, Backman L et al. (1992) Swedish obese subjects (SOS). Recruitment for an intervention study and a selected description of the obese state. *International Journal of Obesity Related Metabolic Disorders*, **16**:465–479.

Stamler J, Farinaro E, Mojonnier L, Hall Y, Moss D, Stamler R (1980) Prevention and control of hypertension by nutritional-hygienic means. Long-term experience of the Chicago Coronary Prevention Evaluation Program. *Journal of the American Medical Association*, **243**:1819–1823.

Stam-Moraga MC (1999) Sociodemographic and nutritional determinants of obesity in Belgium. *International Journal of Obesity*, **23**:S1–9.

Stampfer MJ, Willett WC, Colditz GA, Rosner B, Speizer FE, Hennekens CH (1985) A prospective study of post-menopausal estrogen therapy and coronary heart disease. *New England Journal of Medicine*, **313**:1044–1049.

Stevens J, Cai J, Juhaeri, Thun MJ, Williamson DF, Wood JL (1999) Consequences of the use of different measures of effect to determine the impact of age on the association between obesity and mortality. *American Journal of Epidemiology*, **150**:399–407.

Stevens J, Cai J, Panuk ER, Williamson DF, Thun MJ, Wood JL (1998) The effect of age on the association between body-mass index and mortality. *New England Journal of Medicine*, **338**:1–7.

Swinburn BA, Ley SJ, Carmichael HE, Plank LD (1999) Body size and composition in Polynesians. *International Journal of Obesity*, **23**:1178–1183.

Tate RB, Manfreda J, Cuddy TE (1998) The effect of age on risk factors for ischemic heart disease: the Manitoba Follow-up study, 1948–1993. *Annals of Epidemiology*, **8**:415–421.

Temple NJ, Steyn K, Hoffman M, Levitt NS, Lombard CJ (2001) The epidemic of obesity in South Africa: a study in a disadvantaged community. *Ethnic Disorders*, **11**:431–437.

Thun MJ, Calle EE, Namboodiri MM et al. (1992) Risk factors for fatal colon cancer in a large prospective study. *Journal of the National Cancer Institute*, **84**:1491–1500.

Tremblay MS, Katzmarzyk PT, Willms JD (2002) Temporal trends in overweight and obesity in Canada, 1981–1996. *International Journal of Obesity*, **26**:518–543.

Tretli S (1989) Height and weight in relation to breast cancer morbidity and mortality. A prospective study of 570 000 women in Norway. *International Journal of Cancer,* **44**:23–30.

Trichopoulou A, Gnardellis C, Lagiou A, Benetou V, Trichopoulos D (2000) Body Mass Index in relation to energy expenditure among adults in Greece. *Epidemiology*, **11**:333–336.

Troiano RP, Frongillo EA, Sobal J, Levitsky DA (1996) The relationship between body weight and mortality: a quantitative analysis of combined information from existing studies. *International Journal of Obesity*, **20**:63–75.

Tuomilehto J, Lindstöm J, Eriksson JG et al. (2001) Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *New England Journal of Medicine*, **344**:1343–1350.

Törnberg SA, Carstensen JM (1994) Relationship between Quetelet's index and cancer of breast and female genital tract in 47 000 women followed for 25 years. *British Journal of Cancer*, **69**:358–361.

Unger R, Kreeger L, Christoffel KK (1990) Childhood obesity. Medical and familial correlates and age of onset. *Clinical Pediatrics*, **29**:368–373.

van der Sande MA, Bailey R, Faal H et al. (1997) Nationwide prevalence study of hypertension and related non communicable diseases in the Gambia. *Tropical Medicine and International Health*, **12**:1039–1048.

Verbrugge LM, Gates, DM, Ike RW (1991) Risk factors for disability among US adults with arthritis. *Journal of Clinical Epidemiology*, **44**:167–182.

Vessby B, Uusitupa M, Hermansen K et al. (2001) Substituting dietary monounsaturated for saturated fat improves insulin sensitivity in healthy men and women—the KANWU study. *Diabetologia*, **44**:312–319.

Visscher TL, Kromhout D, Seidell JC (2002) Long-term and recent time trends in the prevalence of obesity among Dutch men and women. *International Journal of Obesity Related Metabolic Disord*ers, **26**:1218–1224.

Weiderpass E, Adami HO, Baron JA et al. (1999) Risk of endometrial cancer following estrogen replacement with and without progestins. *Journal of the National Cancer Institute*, **91**:1131–1137.

Whitlock G, Lewington S, Ni Mhurchu C (2002) Coronary heart disease and body mass index: a systematic review of the evidence from large prospective cohort studies. *Seminars and Vascular Medicine*, **2**:369–381.

WHO (1985) *Energy and protein requirements*. Report of a joint FAO/WHO/UNU expert consultation. (WHO Technical Report Series No. 724.) World Health Organization, Geneva.

WHO (1993) *World health statistics. Annual report*. World Health Organization, Geneva, 1994.

WHO (1995) *Physical status: the use and interpretation of anthropometry*. (Technical Report Series No. 854.) World Health Organization, Geneva.

WHO (2000) *Obesity: preventing and managing the global epidemic*. (WHO Obesity Technical Report Series No. 894.) World Health Organization, Geneva.

WHO/IASO/IOTF (2000) The Asia-Pacific perspective: redefining obesity and its treatment. February 2000. Full document available from: http://www.idi.org.au/obesity_report.htm.

Wilks R, McFarlane-Anderson N, Bennett F et al. (1996) Obesity in peoples of the African diaspora. In: *The origins and consequences of obesity*. Chadwick DJ, Cardew G, eds. John Wiley & Sons, New York.

Willett W, Manson J, Liu S (2002) Glycemic index, glycemic load, and risk of type 2 diabetes. *American Journal of Clinical Nutrition*, **76**:S274–280.

Willett WC, Manson JE, Stampfer MJ et al. (1995) Weight, weight change, and coronary heart disease in women: risk within the "normal" weight range. *Journal of the American Medical Association*, **273**:461–465.

Williamson DF, Pamuk ER (1993) The association between weight and increased longevity. A review of the evidence. *Annals of Internal Medicine*, **119**:731–736.

Williamson DF, Pamuk E, Thun M, Flanders D, Byers T, Heath C (1995) Prospective study of intentional weight loss and mortality in never-smoking overweight US white women aged 40–64 years. *American Journal of Epidemiology*, **141**:1128–1141.

Witteman JC, Willett WC, Stampfer MJ et al. (1989) A prospective study of nutritional factors and hypertension among US women. *Circulation*, **80**:1320–1327.

Xiao-Ren Pan, Guang-wei Li, Ying-Hua Hu et al. (1997) Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The Da Qing IGT and Diabetes study. *Diabetes Care*, **20**:537–544.

Yajnik C (2000) Interactions of perturbations in intrauterine growth and growth during childhood on the risk of adult-onset disease. *Proceedings of the Nutrition Society*, **59**:1–9.

Yoshiike N, Matsumura Y, Zaman MM, Yamaguchi M (1998) Descriptive epidemiology of BMI in Japanese adults in a representative sample from the National Nutrition Survey 1990–1994. *International Journal of Obesity*, **22**:684–687.

Yoshiike N, Seino F, Tajima S et al. (2002) Twenty-year changes in the prevalence of overweight in Japanese adults: the National Nutrition Survey 1976–1995. *Obesity Reviews*, **3**:183–190.

Zajkas G, Biro G (1998) Data on the prevalence of obesity in Hungarian adult population between 1985–88 and 1992–94. *Zeitschrift für Ernärungswissenschaft*, **37**:S134–135.

Zhou B-F (2002) Predictive values of body mass index and waist circumference for risk factors of certain related diseases in Chinese adults—study on optimal cut-off points of body mass index and waist circumference in Chinese adults. *Biomedical and Environmental Sciences*, **15**:83–95.

Zhou B, Wu Y, Yang J, Li Y, Zhang H, Zhao L (2002) Overweight is an independent risk factor for cardiovascular disease in Chinese populations. *Obesity Reviews*, **3**:147–156.

Zimmermann MB, Hess SY, Hurrell RF (2000) A national study of the prevalence of overweight and obesity in 6–12 year old Swiss children; body mass index, body-weight perceptions and goals. *European Journal of Clinical Nutrition*, **54**:568–572.