7. Overweight adults

7.1 Introduction

7.1.1 *Background*

When intake of energy exceeds energy expenditure, the excess is stored, in the form of triglycerides, in adipose tissue. Although energy storage is fundamental in allowing survival when food is scarce, excessive body fat, or obesity, is associated with increased mortality and morbidity. Obesity may be defined as the degree of fat storage associated with clearly elevated health risks. However, fat mass in the human body is difficult to measure under field conditions, and the practical definition of obesity is therefore based on the body mass index (BMI), also known as Quetelet's Index, which relates height to weight (weight (kg)/height^2 (m^2)). Because BMI does not measure fat mass or fat percentage and because there are no clearly established cut-off points for fat mass or fat percentage that can be translated into cut-offs for BMI, the Expert Committee decided to express different levels of high BMI in terms of degrees of overweight rather than degrees of obesity (which would imply knowledge of body composition).

For adults, the Expert Committee proposed classification of BMI with the cut-off points 25, 30, and 40 for the three degrees of overweight described in section 7.2.1. This classification is based principally on the association between BMI and mortality. These cut-off points of the body mass index can be translated into height and weight tables (see Annexes 2 and 3). The following points are important in interpreting the cut-offs:

- The recommended cut-offs are appropriate for identifying the extent of overweight in individuals and populations, but do not imply targets for intervention.
- The broad ranges of BMI do not imply that the individual can fluctuate within this range without consequence; for example, for an individual of height 1.75 m, the BMI range of 18.5–25 covers a weight range of 20 kg (see Annex 3, Table A3.10). Weight gain in adult life may be associated with increased morbidity and mortality independently of the original degree of overweight.
- The cut-off points for degrees of overweight should not be interpreted in isolation but always in combination with other determinants of morbidity and mortality (disease, smoking, blood pressure, serum lipids, glucose intolerance, type of fat distribution, etc.).

Many recommendations on overweight use similar cut-off points (*1*) and most focus on weight-loss therapy in individuals who have reached at least grade 2 overweight (BMI 30.00–39.99). However, long-term, sustained weight loss appears to be difficult to achieve; most overweight individuals who lose an appreciable amount of body weight later regain it. Repeated treatment of overweight may thus lead to "weight cycling", which may itself be associated with adverse health consequences. Although most intervention studies have demonstrated a reduction in cardiovascular risk with weight loss, there are some reports of increased mortality from all causes, as well as from coronary heart disease, in people who have lost weight. These studies are observational and do not discriminate between voluntary weight loss (by dieting) and involuntary loss (as a result of illness), but the fact remains that there are no long-term intervention studies that show a clear-cut decrease in morbidity and mortality as a consequence of sustained weight loss. The present state of knowledge may therefore be summarized as follows:

- Weight gain is associated with increased morbidity and mortality.
- Overweight is associated with increased morbidity and mortality.
- Weight cycling may be associated with increased morbidity and mortality.
- Weight loss in overweight is difficult to sustain, is still of uncertain benefit to health in the long term, and may lead to weight cycling.

In view of these findings, the primary prevention of overweight should be the main concern. Unfortunately, little is known about how excessive weight gain with age can be prevented in modern societies. In individuals who are already overweight, weight control should be undertaken with the aim of normalizing the disorders and metabolic risk factors associated with excess weight rather than of weight loss as a target *per se*.

Overweight is a major public health issue. Grade 2 overweight (see section 7.2.1) is relatively common in most industrialized societies and also in many less modernized cultures: data compiled recently show that the prevalence among 20- to 60-year-olds is about 10–20% among whites in the USA and most countries of Europe (*2*). Prevalence is high (20–40%) among women in eastern European and Mediterranean countries and black women in the USA. Even higher prevalences are observed among American Indians and Hispanic Americans, and on the Pacific islands (*3*), with probably the highest rates in the world among Melanesians, Micronesians, and Polynesians (Table 35). In some African and Asian countries prevalence is much lower but in countries of South America and the Caribbean the prevalence of grade 2 overweight may be close to that in many European countries (Table 36).

In addition to the large differences between countries, prevalence of overweight within countries can vary substantially. This can largely be linked to variations in socioeconomic status and/or degree of urbanization; for instance, the prevalence of overweight has been found to be relatively high in certain professional groups in Bombay, India (*8*).

The method used to establish BMI cut-off points has been largely arbitrary. In essence, it has been based upon visual inspection of the relationship between BMI and mortality: the cut-off of 30 is based on the point of flexion of the curve. Studies in this area have usually suffered from certain methodological drawbacks (*9*); moreover, most have been

Table 35
Age-standardized prevalence of body mass index ≥ 30 in adults aged 25–69 years in various island populations of the Pacific and Indian Oceans[a]

			Age-standardized prevalence[b] (%)	
Population		Year	Men	Women
Caucasian				
Australia	Urban	1989	11.1	12.7
Chinese				
Mauritius		1987	6.2	4.9
		1992	2.1	6.0
Creole				
Mauritius		1987	3.8	13.3
		1992	8.0	20.7
	Rodrigues	1992	9.8	31.1
Indian				
Fiji	Urban	1980	4.3	20.0
	Rural	1980	2.8	9.5
Mauritius		1987	3.3	11.3
		1992	5.1	16.2
Melanesian				
Fiji	Urban	1980	17.8	40.8
	Rural	1980	9.4	24.2
Loyalty Islands		1979	10.5	25.0
Papua New Guinea				
Coast	Urban	1991	36.3	64.3
	Rural	1991	23.9	18.6
Highlands		1991	4.7	5.3
Micronesian				
Kiribati	Urban	1981	29.8	34.5
	Rural	1981	11.8	13.1
Nauru		1975/76	61.7	69.4
		1982	67.5	76.4
		1987	64.8	70.3
Polynesian				
Loyalty Islands		1979	5.0	31.3
Niue		1980	21.0	36.0
Rarotonga		1980	39.0	49.6
Tuvalu		1977	24.0	47.5

Table 35 (*continued*)

Population		Year	Age-standardized prevalence[b] (%) Men	Women
Polynesian (continued)				
Wallis Islands	Urban	1980	35.9	65.4
	Rural	1980	24.1	48.1
Western Samoa	Urban	1978	38.8	59.1
		1991	58.4	76.8
	Rural	1978	17.7	37.0
		1991	41.5	59.2

[a] Adapted from references *4*, *5*, and *6*.
[b] Standardized to Segi's world population. (See: Segi M. *Cancer mortality for selected sites in 24 countries (1950–57)*. Sendai, Tohuku University School of Medicine, 1960.)

Table 36
Proportion of overweight adults in countries of Africa, South America/Caribbean, and Asia[a]

Country	Year	Proportion (%) of population of BMI: 25.00–29.99	≥ 30
Africa			
Congo (women)	1986/87	11.8	3.4
Ghana	1987/88	17.1	0.9
Mali	1991	6.4	0.8
Morocco	1984/85	18.7	5.2
Tunisia	1990	28.6	8.6
South America/Caribbean			
Brazil	1989	25.1	8.6
Cuba	1982	26.9	9.5
Peru	1975/76	24.8	9.0
Asia			
China	1982	7.2	1.0
India	1988/90	3.0	0.5

[a] Adapted from reference *7*.

conducted among people living in western Europe or the USA. It may therefore be necessary to revise the classification of overweight in terms of BMI based on health risk.

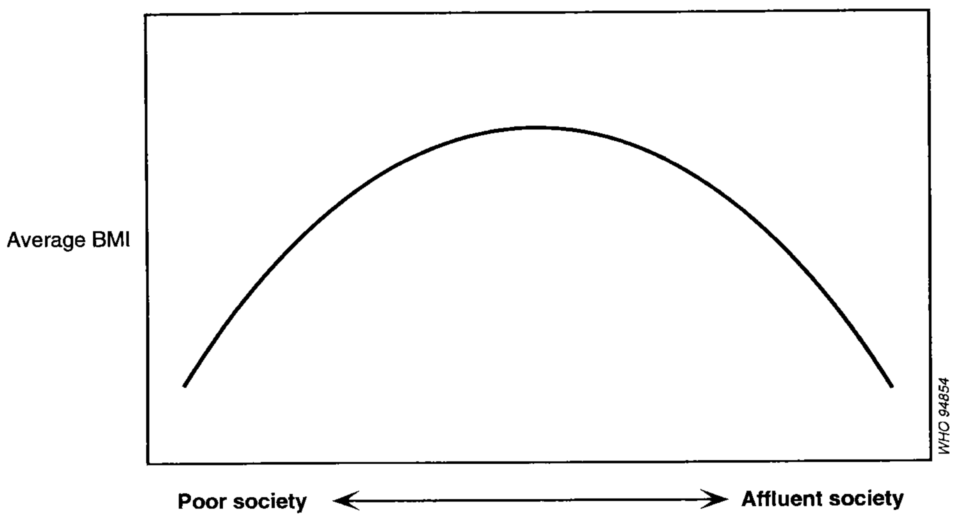
7.1.2 *Biological and social significance of overweight*

Biological and social determinants of overweight

Interaction of genetic and environmental factors. Overweight is always the product of a positive energy balance resulting from relatively low energy expenditure and/or relatively high energy intake. Social, cultural, and behavioural factors are important determinants of both components, but it is also clear that excess weight gain in affluent circumstances may result from a genetic predisposition. What remains unclear is the mechanism through which genetic factors exert their influence; it is probable that many genes are involved, affecting both energy expenditure and energy intake (*10*).

Figure 51

Schematic representation of the transition of a society from poverty to affluence and its relevance to changes in anthropometry



		Transition	
Average anthropometry	Short height	*Increase in height, weight, and abdominal fatness*	Tall height
Culturally desirable	Non-thinness		Thinness
Heaviest subgroup	High socioeconomic status		Low socioeconomic status

The expression of overweight requires a certain level of food availability above which the relative contributions of genetics and environment probably vary within and across populations. High-fat diets combined with low levels of physical activity play an important role in the increase of overweight that accompanies the transition from poverty to affluence (Fig. 51). In an affluent population of individuals with similar socio-economic values and resources, genetic factors become relatively more important in determining which individuals will become obese.

The "thrifty genotype" hypothesis (*11*) postulates that populations exposed to inadequate or fluctuating food supplies are genetically selected for a high level of efficiency in caloric utilization or fat storage. When more food becomes available, this efficiency may lead to an increase in the prevalence of overweight and non-insulin-dependent diabetes mellitus.

Secular trends in the prevalence of overweight. According to the NHANES study in the USA, the prevalence of overweight seems to have been stable in the white population during the period 1960–1980 but to have increased among non-whites, particularly black males (*12*). In the 1980s the prevalence of overweight remained stable or increased in Australia, Finland, the Netherlands, Sweden, and the UK (*13–15*). Since then, there seems to be no indication of a decrease in the prevalence of overweight in these affluent countries despite increased commercial and other interests in promoting leanness; on the contrary, the prevalence of overweight may be increasing further.

Trend analyses of populations among whom overweight is now common (e.g. Pima Indians in the USA, and Fijians, Maltese, Melanesians, Nauruans, and Samoans) are fragmentary (Table 35), but it is clear that the condition was uncommon before the adoption of sedentary lifestyles and high-fat diets. Data from Mauritius, for example, show that the prevalence of combined grade 2 and 3 overweight has increased by approximately 50%. Studies of Aborigines in Australia have shown that a return to their original lifestyle reduces the prevalence of overweight and other cardiovascular risk factors (*16*).

Biological determinants

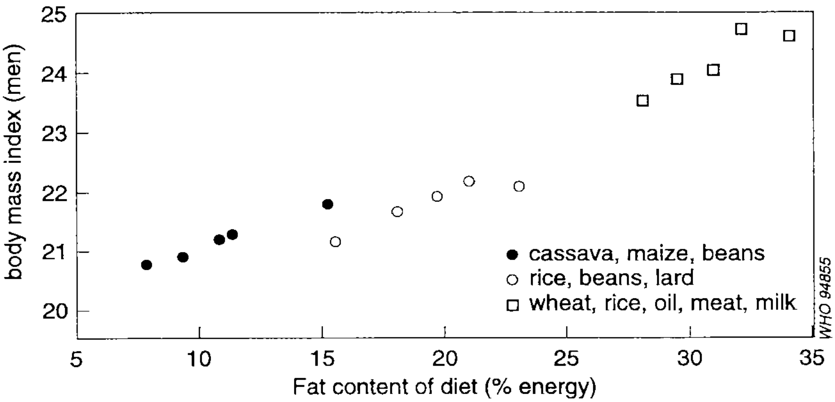
- *Age and sex.* In many affluent countries the prevalence of grades 1 and 2 overweight in men increases with age up to about 55 years, then levels off before finally decreasing somewhat in old age. In women, prevalence continues to rise until old age and then levels off. These observations generally come from cross-sectional studies and the age, period, and cohort effects have not usually been separated. In postmenopausal women, and to a lesser degree in men, BMI increases with age even when body weight remains stable, because of the age-associated decline in stature. Although age-specific mean BMI is usually lower in premenopausal women than in men, the overall prevalence of overweight is generally higher in women. Some studies report that obese adults gain most of their excess weight in early adult life (*17*); the incidence of substantial weight gain may be highest among those who are already overweight in early adulthood (*18*).

- *Pregnancy and lactation.* In cross-sectional studies from affluent countries, body mass index usually increases with the number of pregnancies. There are only a few longitudinal studies that include prepregnant weight as well as appropriate controls. A 1987 literature review revealed that mean body weight at different times after delivery was 0.5–2.4 kg higher than prepregnant weight. Among some 6000 Finnish women who were followed for 5 years, the mean (adjusted) weight gain for women with one child was 0.2 kg and for those with two or more children 0.6 kg (*20*). The relative risk of gaining 5 kg or more in these women depended on their level of education. Thus, the relative risk (compared with those of a high educational level and with no children) was 0.6 for women of high educational level and with two or more children, but 2.8 and 3.3 for similar mothers of intermediate and low educational level respectively. In a large prospective study of 2295 Swedish women, mean weight gain following one pregnancy was 1.5 kg (*21*); about 14% of the women gained more than 5 kg. Factors associated with a weight gain of more than 5 kg were higher prepregnancy BMI, greater weight gain during pregnancy, limited breast-feeding, and cessation of smoking during pregnancy. (The effects of lactation on weight are discussed in section 3.) It may therefore be that the influence of pregnancy in the development of overweight is preventable by educating women on optimal weight gain.

Sociocultural determinants

- *Socioeconomic status/educational level.* In most affluent societies, there is an inverse relationship between educational level and prevalence of overweight. Sobal & Stunkard (*22*) reviewed the relationship between socioeconomic status (usually measured as educational level and/or profession) and prevalence of overweight. Of 20 studies from Europe covering the period 1949–1988 and providing data on women, 16 showed an inverse association between socio-economic status and overweight, and only four showed no association. Of the 33 studies in men, 21 showed an inverse association between socioeconomic status and overweight prevalence, five showed no association, and seven a positive association. Most of the studies that showed a positive association in men were performed in the 1950s, 1960s, and early 1970s. For example, 19 studies were carried out in men in the UK up to 1988; the average year of publication of the four studies that showed a positive association between prevalence of overweight and socioeconomic status was 1961, but for the 11 that showed an inverse association it was 1975.

Figure 52
Household diet and adiposity in Brazil, according to dietary staples[a]



[a] Source: reference *23*.

As mentioned earlier, overweight may be seen as a visible indicator of wealth and status in societies where food is scarce. Brazil is an example of a country in which there is a clear positive association between socioeconomic status (measured as income rather than educational level) and average BMI (*23*). It has been suggested that this association is mediated by the fat content of the diet: see Fig. 52.

- *Marital status.* Longitudinal data from the USA showed that women who got married were more likely to gain weight subsequently, even after adjustment for their educational level and family income. Generally speaking, however, those who divorced lost some weight (*24*). Similarly, data from a 5-year study in Finland showed that men and women who got married during that period were at twice the risk of gaining 5 kg or more compared with men and women who were already married and stayed married during the period of study (*17*). These findings suggest that major changes in lifestyle connected with marriage may promote weight gain in affluent societies.

In some traditional societies, there are pressures on women both to gain weight and to remain overweight during reproductive life. An example of this is the custom of "fattening huts" for elite pubescent girls in certain communities in West Africa (*25*). Such practices reflect cultural perceptions and values related to overweight.

Behavioural determinants

- *Smoking habits.* Reports that the use of tobacco lowers body weight began to appear more than 100 years ago, but detailed studies have been reported only during the past 10 years or so (*26*). In most populations, smokers weigh somewhat less than ex-smokers;

individuals who have never smoked fall somewhere between the two. The physiological mechanisms involved include alterations in energy intake and expenditure, induced by changes in insulin homeostasis, lipoprotein lipase activity, the activity of the sympathetic nervous system, physical activity, and preferences in food consumption (*26*). The most important effects of smoking, however, seem to be those on basal metabolic rate and the thermic effect of food (*27*). In Finland, the inverse relation between smoking and body weight became considerably weaker among women and disappeared among men over the period 1982–1987 (*28*). A cluster of habits unfavourable to health, such as high intake of alcohol and saturated fat, was apparent among young Finnish smokers. Heavy smoking was also associated with increased BMI among young men in the Netherlands (*29*), and similar associations have been observed in the USA (*30*). These observations may suggest that, in populations in which there is growing health awareness and an increasing proportion of people who stop smoking, the remaining smokers are those whose lifestyles carry significant health risks.

Cessation of smoking is associated with weight gain. It has been reported that, after adjustment for age, average weight gain in men who stop smoking is about 3 kg and in women 4 kg (*31*). The same study reported risk of substantial weight gain (i.e. 10 kg or more) as much higher in people who stop smoking than in non-smokers.

Many recent studies have shown that smoking is also associated with an increased abdominal fatness at each BMI level (*32*). This relationship may be mediated by changes in the levels of sex hormones and glucocorticosteroids as a result of smoking; it may also be partly explained by a clustering of physical inactivity, smoking, and alcohol consumption.

- *Physical activity.* Individuals who are relatively inactive are more likely to gain weight than those who frequently engage in physical activity. The relative risk of gaining 5 kg or more during a 5-year follow-up study among inactive Finns was 1.6 in women and 1.9 in men (*20*). A prospective study of adults in the USA showed that remaining physically active is associated with the prevention of age-related weight gain (*33*). Moreover, there is now substantial evidence linking increased physical activity to a more favourable fat distribution (a lower proportion of visceral fat at a given BMI) (*32*).

- *Alcohol consumption.* A recent review of 31 studies (16 in the USA) concluded that the relationship between alcohol consumption and adiposity was generally positive for men and negative for women (*34*). Only three of these studies were prospective, but the two that examined changes in alcohol intake found that, as intake increased over a period of either 4 or 18 years, weight increased significantly.

This finding contrasts with the results of cross-sectional studies, which generally showed a negative association between overweight and alcohol intake, especially for women. The results were inconsistent both within and between the sexes, and the criteria usually employed to establish causality were not met. The data were not consistent across diverse populations in direction, strength, or gradation of association, which suggests diverse patterns of intake. On the one hand, some experimental studies suggested that metabolism of alcohol may not lead to formation of ATP (and therefore may have zero caloric value in the body); on the other hand it was shown that alcohol may reduce fat oxidation and thus contribute to excess fat storage in the body (*35*). It has also been suggested that there is a relationship between alcohol intake and abdominal fatness, although the evidence is inconclusive (*32*).

Biological and social consequences of overweight

Overweight and mortality. It has been widely concluded that the relationship between BMI and mortality is U-shaped or J-shaped. Some studies, however, usually small and of short duration, report no association between BMI and mortality (*36*). The minimum follow-up period necessary seems to be about 5 years and the sample size at least 7000 individuals for there to be sufficient power to detect the positive association between BMI and mortality (*36*). The causes of death at different ends of the U- or J-shaped curve are strikingly different: the high mortality at low BMI is dominated by digestive and pulmonary disease, but at high BMI it is related predominantly to cardiovascular disease, diabetes mellitus, and gallbladder disease. It is sometimes argued that the high mortality at low BMI is due to the confounding presence of smoking and disease and that the relationship between BMI and mortality may in fact be linear. A study of 8828 Seventh-Day Adventist men, among whom such confounding effects are minimal, demonstrated such a linear association, with the lowest mortality among the leanest men (BMI <20) (*37*).

It has been pointed out (*9*) that most studies published up to 1987 suffered from methodological drawbacks. These drawbacks include:

- Failure to control for cigarette smoking. Since smoking is a strong risk factor for mortality and is also more common among those with a low BMI, failure to control for smoking will lead to an overestimation of the importance of thinness to mortality. Stratification is recommended in analyses for smoking behaviour.
- Failure to eliminate early mortality from the analysis of prospective data may confound the weight/mortality association. Clinical or subclinical illness present before inclusion of an individual in a study could be the reason for, rather than a consequence of, reduced body weight. Failure to adjust for early mortality will thus result in underestimation of overweight-related mortality.

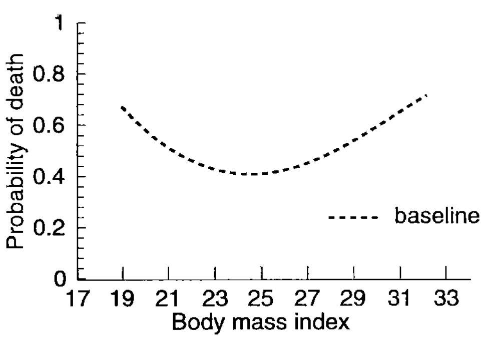
- Inappropriate adjustment for intermediate risk factors such as hypertension, hyperlipidaemia, and diabetes. Control for these intermediate risk factors is likely to lead to an underestimation of the risks associated with overweight.
- Most studies were carried out in predominantly white populations in Europe and the USA.

Troiano and colleagues (*38*) performed a meta-analysis on the relationship between BMI and mortality in adult Caucasian men and women (with elderly cohorts excluded). The Caucasian male sample was based on 17 studies with 37 sub-studies (17 of which were from the USA) representing more than 350 000 men and over 38 000 deaths. The female sample was based on six studies with 12 sub-studies representing about 250 000 women and 13 700 deaths. The authors concluded that the relationship between BMI and mortality is U-shaped and that mortality was increased at a high BMI (>29–30). Minimum mortality for white men who were 50 years of age at entry and were followed for 30 years was at BMI between 24 and 25 (Fig. 53). Although the meta-analysis did not stratify according to age, it can be assumed that the curvature may be even more pronounced at lower ages and minimum mortality shifted somewhat to the left in the acceptable BMI range of 18.5–25. The curves that included smokers and did not exclude individuals on the grounds of disease showed higher levels of mortality but no distinct minimum. The curvature was also much less pronounced for analyses with shorter follow-up (10 years).

For women, no studies have been reported that have both included long follow-ups and separated the effects of smoking. For combined smoking and non-smoking women other than in the USA, with 10 years of follow-up, the curvature was less pronounced and minimum mortality was at a

Figure 53

Predicted relationship between BMI and all-cause mortality for white men 50 years old at entry, with 30-year follow-up[a]



[a] Source: reference *38*.
Baseline = non-smoking US cohorts with exclusions for disease at entry.

BMI of approximately 25. Adjusting for the effect of smoking increased the nadir, which suggests that minimum mortality among non-smoking women would be at BMI less than 25.

A recent Japanese study showed a J-shaped relationship between BMI and mortality, remarkably similar to those in Europe and the USA, with minimum mortality at a BMI of around 22 (*39*).

Overweight and morbidity

- *Overweight and coronary heart disease.* Overweight is associated with an increased prevalence of cardiovascular risk factors such as hypertension, unfavourable blood lipid concentrations, and diabetes mellitus. Although there is a lack of controlled data on decreasing mortality after weight reduction, it is well known that overweight-related risk factors are improved by weight reduction. On the basis of changes in risk factors induced by spontaneous weight reductions in the Framingham study (*40*), it was estimated that a 10% reduction in body weight would correspond to a 20% reduction in the risk of developing coronary heart disease (CHD). Observational studies of weight loss and subsequent mortality, however, have been unable to confirm a reduction in the risk of CHD following weight loss (*41*). Weight cycling has been found to be associated with increased risk of CHD, and this may be particularly harmful in non-obese individuals (*42*).

  The relationship between BMI and CHD has usually been found to be linear, but the level of risk is modified by ethnicity, age, sex, and smoking habits. Elsewhere, it has been calculated that about 40% of the incidence of CHD was attributable to a BMI above 21 and was therefore potentially preventable (*43*).

  In prospective studies in which fat distribution was assessed by skinfolds or circumferences, abdominal fatness was a significant risk factor for CHD independently of BMI (*44*). Abdominal fatness is associated with increased levels of insulin and triglycerides and decreased levels of HDL-cholesterol (*45*). In addition, abdominal fatness may be associated with haemostatic and fibrinolytic factors, which also contribute to an increased risk of myocardial infarction.

- *Overweight and stroke.* Despite the clear relationship between overweight and hypertension (see below), it has been concluded that overweight is not among the major risk factors for stroke (*46*), although three prospective studies (two in women, one in men) have shown that abdominal fatness may be associated with increased risks for stroke independently of BMI. Barrett-Connor suggested that smoking habits and alcohol intake may be among the variables that partly explain the association between fat distribution and stroke (*47*).

- *Overweight and hypertension.* Increased body weight is associated with elevated blood pressure, and weight loss in hypertensive individuals is generally accompanied by a reduction in blood pressure. According to the results of a meta-analysis, a weight loss of 1 kg is associated with a decrease of 1.2–1.6 mmHg in systolic and 1.0–1.3 mmHg in diastolic blood pressure (*48*).[1] In individuals who regain weight after weight loss, it has been observed that blood pressures remain below baseline levels (*49*). The relationships between overweight and hypertension may sometimes be biased by artificially high readings of blood pressure resulting from increased arm circumference in overweight (*50*).

  A number of studies have suggested that lean hypertensive individuals may be at greater risk of CHD than obese hypertensives (*51*), which may partly reflect the different etiologies of hypertension in lean and obese people. Hazardous lifestyles, particularly involving smoking and high alcohol intake, which contribute to both leanness and risk of death, cannot be ruled out as causative factors in the excessive mortality among lean hypertensives.

  Weight loss is recommended for all obese hypertensive individuals. The beneficial effects of weight loss on both blood pressure and the need for antihypertensive medication are mediated through related changes such as reduced cardiac output and blood volume (*51*). Body fat distribution seems to predict hypertension independently of BMI. Increased insulin concentrations have been suggested as playing a role in both the association between BMI and abdomen:hip circumference ratio and blood pressure (*52*).

- *Overweight and non-insulin-dependent diabetes mellitus.* Overweight is a well-established risk factor for non-insulin-dependent diabetes mellitus (NIDDM). The duration of overweight seems to be important, and involuntary weight loss often precedes diagnosis of the condition. Although overweight is associated with poor glucose tolerance and hyperinsulinaemia (insulin resistance), which can be reversed in the short term with weight loss, the long-term benefits of weight loss for the risk of both NIDDM and complications have not been properly studied. During an 8-year follow-up of 113 861 women in the USA, aged 30–55 years, the risk of developing NIDDM increased with increasing BMI (*53*). Compared with women with a BMI below 22, risk was increased 20-fold for women with a BMI between 29 and 31, and more than 60-fold for those with a BMI above 35. Within the total cohort, 90% of diagnoses of NIDDM were attributable to a BMI greater than 22. Adult weight gain of more than 5 kg in 8 years was associated with a significantly increased risk of

[1] 1 mmHg = 0.133 kPa.

NIDDM, and weight gain after the age of 18 was also associated with an increased risk. However, risks were no different in women with and without a family history of diabetes mellitus.

The causal role of overweight in the development of NIDDM is supported by evidence that weight loss in NIDDM improves glucose tolerance and reduces the need for hypoglycaemic drugs. Moreover, experimental overweight in normal young men reduces insulin sensitivity, and the effect is reversible with weight loss (*54*).

In addition to many cross-sectional studies, an increasing number of prospective studies show that abdominal fatness is associated with an increased incidence of NIDDM (*52*). The most up-to-date hypothesis for a mechanism is increased accumulation of visceral fat leading to increased levels of free fatty acid in the portal vein which, in turn, diminish hepatic insulin clearance. Altered muscle morphology in abdominal fatness and relatively high concentrations of androgens in women with high abdomen:hip circumference ratios may also be involved, but the causal role of these factors remains unclear (*52*).

- *Overweight and gallbladder disease.* The risk of gallbladder disease is more pronounced in women than men. Overweight is a major risk factor for the development of gallstones, particularly those made of cholesterol rather than the pigmented stones containing bilirubin. The relationship between BMI and the risk of gallstones appears to be approximately linear. Supersaturation of the bile with cholesterol, which results from the relative hypersecretion of biliary cholesterol, is a necessary condition for the formation of gallstones, and this is more frequent in obese patients; it may also result as a short-term response to dieting (*55*). In the Nurses' Health Study (*56*), the relative risk of gallstone formation in women with a BMI above 32, compared with those with a BMI below 20, was 6. Independently of the degree of overweight, weight gain was associated with an increased risk. Long-term weight loss did not protect against the incidence of gallstones.

  A high abdomen:hip circumference ratio has been found to be related to cholecystectomy independently of BMI in American and Dutch women (*57, 58*). The relationship between abdominal fatness and gallbladder disease may be a result of the increase in the hypersecretion of biliary cholesterol that is associated with increased serum lipid levels, or an effect of hyperinsulinaemia associated with abdominal overweight, which stimulates hepatic cholesterol synthesis.

- *Overweight and osteoarthritis.* The pathogenesis of osteoarthritis and the risk factors for the condition are not well understood, but there is increasing evidence that overweight is associated with osteoarthritis in several joints (*59*). Specifically, an association has been found between overweight and osteoarthritis of the knee but not of the hips; the evidence is inconsistent for osteoarthritis of the feet. Overweight has also been shown to be associated with osteoarthritis of the non-weight-bearing joints, such as those in the hands, which may be a reflection of the metabolic consequences of overweight. Overweight-related conditions such as diabetes mellitus, hyperuricaemia, or hypercholesterolaemia, which may influence cartilage degradation, may act together with excessive mechanical stress on the joints in producing osteoarthritis (*59*). The results of cross-sectional studies should be interpreted with caution, because the limitations imposed by osteoarthritis on physical activity may, in turn, contribute to the development of overweight. Prospective studies have not substantiated this concern, however. The important health implications of the association between overweight and osteroarthritis were illustrated by the finding of a significantly increased risk of disability among obese Finnish men and women because of arthroses of the knee and hip (*60*).

- *Overweight and cancer.* A review of prospective and retrospective studies of the association between overweight and cancer of the colon, rectum, prostate, breast, ovaries, and endometrium (*61*) reached the following conclusions:

  - Overweight and the risk of endometrial cancer increase in direct proportion.
  - Overweight probably increases the risk of postmenopausal breast cancer; the case-control studies have yielded more consistent results than prospective studies. Weight gain after menopause may be a risk factor independent of the degree of overweight and further aggravate the problem.
  - The relationships between overweight and cancer of the colon, rectum, ovaries, and prostate are uncertain; reported associations are inconsistent between and within sexes and across populations.

  Fat distribution may be related to some types of cancer independently of overweight (*62*). Links between abdominal fatness and endometrial and breast cancer reported by some investigators have not been confirmed by others.

- *Overweight and other disorders.* Overweight is positively, and abdominal fatness negatively, associated with the presence of varicose veins (*58*). Abdominal fatness and overweight have been associated with some endocrine disorders and infertility (*63*). Overweight may also lead to important social and economic disadvantages (*64*) as well as psychosocial problems; however, the existence of a causal association and the direction of causality (i.e. the particular role played in the association by stigmatization of overweight and dieting) remain unclear, especially as regards the relationship between overweight and psychosocial problems. At least one epidemiological study suggested that overweight, particularly abdominal fatness in women, is associated with accident proneness and use of drugs for psychological problems (*65*).

Overweight has been found to increase the risk of reflux oesophagitis and hiatus hernia (*66*), although severe oesophagitis causes dysphagia and leads to weight loss. Sleep apnoea is common in overweight people and has been found to be an independent risk factor for CVD; it also causes daytime sleepiness, which increases the risk of traffic accidents (*67*).

7.1.3 *Anthropometry as an indicator of nutritional and health status*

Body mass index as a measure of body fatness

Body mass index appears to be a good indicator of the deposition of excess energy as fat in adult white men and women living in Europe and North America. It is probably less appropriate in other populations who differ in body build and body proportions. Using BMI to classify individuals according to fatness may result in misclassification because of the varying contributions of bone mass, muscle mass, and fluid to body weight. The percentage of body fat increases with aging and is higher in women than in men, but these differences may not be revealed by BMI. For instance, a BMI of 30 in Dutch men implies a body fat content of about 30% at age 20 years and about 40% at age 60; in women aged 20 and 60, these figures are 40% and 50%, respectively (*68*). Equations that included BMI, sex, and age for these same adults were shown to predict body fat percentage relatively accurately (r^2 about 0.8, SEE about 4%) (*68*); this prediction error is similar to values obtained with other more elaborate methods such as measurement of skinfold thickness or bioelectrical impedance.

Similar problems may occur in the classification of body fat distribution by abdomen:hip circumference ratio. Different contributions of muscle mass and bone structure as well as stature and abdominal muscle tone may lead to different associations between abdomen:hip ratio and visceral fat accumulation. Abdominal fatness may imply different health risks for different racial groups (*69*) and at different ages (*70*). These issues complicate the future development of universal cut-off points for abdomen:hip circumference ratio.

Modifiers of the relationship between overweight and health risks

Within populations, a BMI in excess of 30 is associated with elevated blood pressure and increased risk of coronary heart disease and non-insulin-dependent diabetes mellitus, as previously discussed. It seems, however, that some populations are more vulnerable to the effects of high BMI than others. This may be partly explained by differences in body composition, body build, and fat distribution, but also by the underlying cause of overweight and the genetic predisposition to the development of certain diseases associated with overweight. For instance, overweight resulting from the combination of inactivity with a diet high in saturated fat and low in antioxidants may have different consequences with respect to development of hyperlipidaemia and coronary heart disease from overweight that results from excess weight gain by individuals with relatively low metabolic rates but adequate physical activity and dietary habits. In addition, the combination of high BMI with one or more different risk factors (e.g. smoking, hypertension, hyperlipidaemia, diabetes mellitus) may lead to different risks of developing disease.

History of overweight may contribute to variations in health risks associated with overweight. Three issues should be considered: age of onset, duration, and weight fluctuation patterns. Overweight *per se* has no immediate effect on the occurrence of chronic disease despite short-term changes in metabolic profiles and mechanical consequences; with increasing duration of overweight, however, the impacts become greater. Early onset usually implies long duration of overweight: weight gain leading to overweight in adult life has been shown to be associated with increased risk of several chronic diseases. There are, however, a number of unresolved issues in this regard, particularly the modifying effects of abdominal fatness and of smoking cessation.

As mentioned earlier, data on the health effects of weight cycling are inconclusive but it may be that its adverse effects are more pronounced in people who are not overweight (*42*).

It has also been suggested that overweight may be less hazardous to health in certain populations, for instance black women (*3*). This is in accordance with the findings that abdominal fatness may be less strongly associated with risk factors for cardiovascular disease and diabetes in black women than in white (*69*). In other comparisons, for instance, between southern Asians and whites living in London and between Mexican-Americans and Anglo-Americans living in Texas, it was concluded that Asians and Mexican-Americans have higher risks of developing non-insulin-dependent diabetes mellitus than Caucasians of similar BMI (*71, 72*). This could be largely explained by differences in fat distribution (abdomen:hip circumference ratios are relatively high in Asians and Mexican-Americans). It has been further speculated that previous long-term malnutrition predisposes to increased abdominal fatness.

7.2 Using anthropometry in individuals

Body mass index and abdomen:hip circumference ratio are used to classify individuals in terms of overweight and abdominal fatness, respectively. Misclassification may occur but should be limited since anthropometric variables have to be interpreted in combination with other risk factors. For longitudinal assessment, variation in body weight or single (abdomen or hip) circumferences will be sufficient because – in contrast to children, adolescents, and the elderly – long-term weight change in adults will predominantly reflect change in fat mass. Furthermore, simple measurements may be the most informative for assessing the outcome of interventions in individuals.

7.2.1 *Screening for interventions*

When individuals have been classified according to BMI, their risk profile should be ascertained in terms of risk factors (abdomen:hip circumference ratio, smoking and dietary habits, physical activity, blood pressure, serum lipids, glucose) as well as of family history of certain disorders (premature coronary heart disease, non-insulin-dependent diabetes, hypertension). Because a large proportion of the adult population in industrialized societies will be overweight or obese, and because weight-loss therapy is ineffective unless closely supervised and followed up, not all overweight or obese individuals will qualify for intervention. Priorities should be given to those at highest risk, with the primary focus on reducing the risk profile rather than on weight loss *per se*. The following scheme may be adopted:

1. Measure height and weight and calculate body mass index.

2. Classify according to BMI:

normal range:	BMI 18.50–24.99
grade 1 overweight:	BMI 25.00–29.99
grade 2 overweight:	BMI 30.00–39.99
grade 3 overweight:	BMI ≥ 40.00

Annexes 2 and 3 of this report provide complete and more simplified versions, respectively, of BMI tables that will facilitate the use of BMI in the field; a nomogram is also provided in Fig. A2.1 (Annex 2).

3. For individuals with BMI 18.50–24.99: avoid becoming overweight. There are no recommendations for weight loss.

   For individuals with BMI 25.00–29.99: avoid weight gain. Before recommending any type of intervention, assess other risk factors. If there are additional risk factors (high abdomen:hip ratio, hypertension, hyperlipidaemia, glucose intolerance or NIDDM, strong family history of diabetes mellitus or premature coronary heart disease), recommend a healthy lifestyle that will contribute to improvement of the risk profile: cessation of smoking, increased physical activity, reduced intake of (saturated) fat. Moderate weight loss is recommended but weight loss *per se* should not be the primary target of intervention. A large proportion of the adult population will usually fall into this category, and most will receive advice on healthy nutrition and physical activity appropriate for the general population. Regular (yearly) weight measurement will be helpful in monitoring weight development, and weight histories should be noted. Individuals who have continued to gain weight (e.g. >5 kg during the previous 2 years) should be identified for weight maintenance programmes designed to halt the weight gain.

For individuals with BMI 30.00–39.99: the same recommendations as for grade 1 overweight, although the prevalence of risk factors and of overweight-associated disorders that require medical attention is usually markedly higher and moderate weight loss is therefore more urgently recommended. In many populations, the proportion of adults falling into this category is still considerable, and treatment priorities will have to be set on the basis of, among other things, the prevalence of health problems in the community concerned. The higher the prevalence of chronic diseases such as diabetes and CVD, the greater is the need for individuals with BMI 30.00–39.99 to lose weight. In other words, the potential impact of weight modification in preventing these problems is likely to be influenced by the disease rates in the population. The risks related to grade 2 overweight in adults depend on other, coexisting, risk factors for chronic noncommunicable diseases. Obese individuals with no additional risk factors or conditions that require medical supervision may be referred to self-help organizations. Such organizations are effective if their leaders have sufficient training in the principles of healthy weight loss (a maximum of about 0.5 kg/week) and of balanced nutrition. For individuals with conditions that do require medical supervision, the focus should be on normalizing the risk factors or alleviating health problems (e.g. improving respiratory function or arthritis in weight-bearing joints) rather than on achieving weight loss *per se*.

For individuals with BMI ≥ 40: intensive action to reduce weight. The proportion of adults with grade 3 overweight is small; for these individuals, weight loss *per se* may be the primary target and options such as surgical treatment for obesity should be considered (*73*).

7.2.2 *Assessing response to an intervention*

Until recently, the response to intervention was usually assessed in terms of attaining "ideal body weight" or reducing weight to below a certain BMI cut-off or "percentage over ideal body weight". It is no longer clear that such goals are optimal. For some people they are unrealistic: in most obese individuals they imply large sustained weight losses, which few are able to achieve unless they are enrolled in long-term programmes with extensive follow-up (e.g. >5 years). Moreover, substantial improvements in risk-factor profiles have been documented in obese individuals who lost only moderate amounts of weight and would still be classified as overweight or obese (*74*). Finally, there is no evidence that large weight losses either have beneficial effects or reduce mortality, and the more extreme diets needed to produce large weight losses may increase the likelihood of relapse (*75*). More realistic responses would therefore be:

- The normalization of risk factors or health conditions that is associated with any amount of weight loss in overweight individuals.

- Prevention of (further) weight gain in obese and non-obese subjects. Weight should be assessed at regular intervals (e.g. once a year), perhaps at routine check-ups.

7.3 Using anthropometry in populations

Overweight is an excellent indicator of energy imbalance caused by a combination of excessive energy intake and insufficient energy expenditure. Even small daily deviations from ideal energy balance can lead to substantial increases in body weight over time; for example, a consistent average daily excess energy intake of 168 kJ (39 $kcal_{th}$) (equivalent to 1 dl of carbonated soft drink) will theoretically lead to a weight gain of 15.6 kg over 10 years. Overweight is thus a sensitive indicator of a chronic energy imbalance that would probably not be detected by cross-sectional surveys of energy intake and energy expenditure in individuals. Moreover, it is a widespread condition in many industrialized societies and notoriously difficult to treat; interventions should therefore aim largely towards its prevention. In the community setting, treatment of all obese adults by physicians is neither rational nor feasible. To establish the need for, and priorities of, preventive interventions, the magnitude of the problem in a particular population must be defined with data derived from representative population surveys; the prevalence of BMI $\geq$ 30 can be used as the principal indicator.

7.3.1 *Targeting interventions*

Anthropometric data can be used to estimate the population-specific risks for various noncommunicable diseases. However, the genetic differences between different populations give rise to two major concerns in the use of anthropometric variables. First, they influence the degree of risk associated with excess body weight, and second, they are also important in determining the kinds of disease that may occur as a result of overweight. For instance, hypertension and hyperlipidaemia are common in obese Caucasian populations, but less common in American Indians and peoples of the Pacific islands, among whom diabetes is more frequent.

7.3.2 *Assessing response to an intervention*

Anthropometry is also used in populations to evaluate programmes of health promotion and disease prevention, in which the prevention and control of overweight may play an important role. While simple indicators may be useful for targeting communities, more detailed information is usually desirable for evaluation of prevention programmes. Since overweight in adult populations is a result of dietary habits and other aspects of lifestyle, and is associated with other metabolic abnormalities, it is important that due attention is given to these associated factors in the evaluation of prevention programmes. Age-specific and age-standardized proportions of the population above a certain BMI cut-off can be used for evaluation, as can trends in the median or other percentiles of BMI. (BMI is usually not normally distributed; distribution is skewed to the right, so means may not be useful for evaluation without logarithmic transformation.)

To evaluate interventions designed to prevent the development of overweight in populations who are not yet obese, it is essential to assess longitudinal weight development. The efficacy of a specific prevention programme can be judged by comparison of populations who have received the intervention with similar populations who have not. Evaluation of treatment interventions (designed to treat obesity or overweight associated with risk factors or medical conditions that improve with weight loss), weight development of treated and untreated individuals should be followed on a population basis. For both types of intervention, the follow-up period should be at least 5 years.

7.3.3 *Ascertaining determinants of overweight*

If secular trends in overweight are to be understood, information about possible determinants is desirable. The prevalence of overweight is affected by the overall stage of socioeconomic development of a given population. There is a link between low socioeconomic status and overweight in societies that have an abundance of food and in which other basic needs are fulfilled, and between high socioeconomic status and overweight in societies in which food is scarce (see Fig. 51).

The most obvious determinants of overweight are physical inactivity and high energy intake (generally in high-fat diets). Other factors known to be associated with, or to modify the effect of, significant weight gain are ethnicity/race, family history of overweight and its consequences, socioeconomic factors such as educational level (a low level is associated with higher risk for weight gain with aging), smoking (cessation may be associated with major weight gain), and parity (high parity may be associated with major weight gain).

7.3.4 *Ascertaining consequences of overweight*

The consequences of overweight are partly determined by the patterns of disease in the population. Among populations in which overweight is associated with relative affluence, mortality is dominated by infectious disease, and life-expectancy is low (as in most societies in Europe at the turn of the 19th century and in many developing countries at present), overweight may be relatively advantageous and associated with comparatively low mortality. Conversely, in societies in which affluence predisposes to high rates of diabetes mellitus and coronary heart disease, overweight will be associated with increased mortality risks.

Overweight is also associated with an increased incidence of coronary heart disease, diabetes mellitus, gallbladder disease, and musculoskeletal disorders. More immediate consequences are the increased levels of risk factors for these noncommunicable diseases, for instance dyslipidaemia, glucose intolerance, hypertension, and hyperuricaemia.

Sample size and follow-up time should be adequate for the purposes of the study. Study of the relationship between BMI and mortality, for example, may require a sample of at least 7000 individuals followed for at least 5 years.

7.3.5 *Nutritional surveillance*

Problem identification

The magnitude of the problem of overweight in populations and its geographical, sex, racial, ethnic, and socioeconomic distribution can be identified by the stratification of sufficiently large random population samples according to sex, age, and race. Details of urbanization, ethnicity, and socioeconomic status should also be recorded, and it is important to document other risk factors such as hypercholesterolaemia, hypertension, and smoking habits in the sample population. In women, menopause influences weight and the pattern of fat distribution as well as their health consequences, and this too should be documented.

Anthropometry can be used for surveillance of risk factors for chronic diseases in populations. This requires repeated measurements in random population samples at regular intervals. Besides height and body weight, abdominal and hip circumferences should also be measured. In many populations, lifestyle and nutrition have undergone radical changes over a relatively short period of time; different birth cohorts may therefore have developed differently in terms of anthropometric indicators, which may complicate interpretation. Increases in abdominal fatness in populations undergoing such changes may provide sensitive indication of an emerging public health problem of overweight and its consequences. Cross-sectional data on height and body weight are available for most adult populations, but data for other indicators are limited.

Monitoring of overweight on a population basis should be performed using independent cross-sectional surveys, either continuously or at regular intervals. Detecting a reliable trend in risk factors requires analysis at many points in time. Five-yearly surveys may be used to detect a trend; continuous monitoring can be even more sensitive in revealing responses to interventions.

Long-term planning

The prevention and treatment of overweight calls for long-term follow-up of weight maintenance. Long-term planning should involve preventive strategies that focus on educating people in the principles of adequate nutrition and physical activity and their role in weight control, and about socioeconomic aspects of overweight and weight gain.

National policies for the treatment and prevention of overweight should be developed and complemented by plans for implementing weight control as an integral activity in the control of hypertension, dyslipidaemia, and diabetes. To be effective, prevention of overweight in the community requires a multisectoral approach rather than purely medically oriented programmes. This may include, for example, the development of economic incentives or disincentives for the purchase of certain foods, determining the availability of particular foods, controlling the layout of supermarkets and the content of advertising in the media, and ensuring that town planners make provision for facilities for safe and regular physical activity.

Timely warning

Surveillance of body weight and BMI in the population is essential to provide information that can be used for timely warning. "Timely" in this context is a long-term concept and not concerned with acute problems. The interpretation of the rate and magnitude of change in anthropometric indices of overweight depends on the prevalence of overweight and its consequences in the population in question. A steep increase in the prevalence of overweight and a steep slope of weight gain by age or time in the population as a whole is a general warning that overweight is becoming a public health problem. An increase in abdominal fatness can provide further evidence of unfavourable developments in the population that would justify an intensification of preventive measures.

For programme management

Effective programme management calls for population-specific data on the prevalence of overweight, the relationship between age and BMI, trends in mean BMI and the prevalence of overweight, and the association of overweight with other risk factors among programme participants. These data should be complemented with risk estimates for major diseases associated with overweight in the target population.

7.4 Population data management and analysis

Participation rates in population surveys vary markedly; usually they are about 90% at best and may often be as low as 50–60%. There is recognized bias resulting from lesser participation among lower socioeconomic groups, smokers, and heavy alcohol drinkers; in addition, people with chronic diseases are often less interested in taking part in population risk factor surveys. This will have an effect on the distribution of anthropometric parameters and on prevalence estimates of overweight, because these parameters correlate with the factors that govern participation. Survey participation bias must therefore be estimated as accurately as possible.

Variability between and within observers should be assessed by the repetition of measurements on the same subjects by the same and different observers under standardized conditions and at short time intervals. Outliers at the lower and upper ends of the distribution of the measured parameters must be confirmed; the criteria should be defined in advance and the validity of unusual values should be confirmed at the survey site. Last digit preference and preference/avoidance of certain values should be analysed for the whole sample and for each observer. Time trends in overall and observer-specific values should be documented, particularly if the survey period is long. Original raw data should remain available for analysis.

Mean values of anthropometric parameters, plus 95% confidence intervals and standard deviations, medians, percentiles, and proportion of subjects with BMI in the ranges 25.00–29.99, 30.00–39.99, and $\geq$ 40.00 should be reported by sex, age, and race. Age grouping will depend on the sampling frame and sample selection: for instance, if the sample is stratified by age at 5- or 10-year age intervals, data should be documented using the same age intervals.

Ideally, population data on anthropometric parameters in adults should be presented by sex and 5-year age groups (assuming that the sample selection allows for such grouping). Any other stratification used in the sample selection (geographical, ethnic, socioeconomic, etc.) should also be taken into account in the presentation of data. Summary statistics will require proper adjustment for age and other variables used in sample selection (effect modifiers and confounding factors).

The use of indices (BMI and abdomen:hip circumference ratio) may not be entirely appropriate for assessing the functional outcomes of overweight unless an analysis of primary data (weight, height, circumferences) is also provided.

It is important to analyse the association of anthropometric parameters and/or indicators of overweight with various outcome parameters such as morbidity variables (prevalence of diabetes, hypertension, coronary heart disease, etc.), disability (osteoarthritis, activities of daily living, occupational limitations), and levels of physiological risk factors (blood pressure, blood levels of glucose, insulin, lipids). It should be kept in mind, however, that selective mortality associated with either overweight or underweight can influence these relationships, particularly the age-relation of anthropometric parameters and outcome measures in cross-sectional assessments. It is therefore important to supplement cross-sectional data with longitudinal data on the health effects of overweight in different populations.

In analysing data, it is also important to pay attention to such major determinants of body weight as diet, physical activity, smoking, and alcohol consumption.

Secular changes in overweight are of interest for:

- anticipating and preventing increases in the level of overweight in a population;
- revealing a decline in the level of overweight in a population;
- evaluating the contribution of community-based educational or intervention programmes to any decline in overweight;
- assessing the extent to which trends in determinants of overweight are affecting average body mass index or the prevalence of overweight in the population;
- predicting trends in overweight-related morbidity and mortality (e.g. in conjunction with trends in other risk factors for cardiovascular disease);
- linking levels of overweight with changes in population composition (e.g. because of migration).

7.5 Potential development of reference data

Because the prevalence of overweight varies widely from country to country, and because there are no indications that different populations with the same distributions of BMI have similar relative and attributable risks of morbidity and mortality associated with different degrees of overweight, there is currently no obvious need for reference data for BMI in adults. If sufficient data are collected in the future, however, reference data or even standards could be developed.

In order to understand the distribution of BMI values in a healthy population, it is important that data are derived from populations with no problems of nutrition (underfeeding and overfeeding), and that individuals in the population do not smoke and do not suffer from any chronic or acute disease.

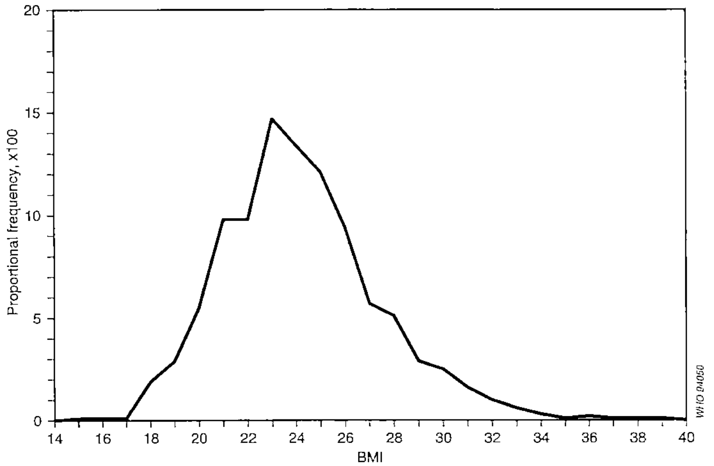
Figures 54–57 give examples of the presentation of such data derived from the WHO MONICA Project (*76*). The frequency and cumulative distribution of BMI values for non-smoking subjects aged 25–34 years in the eight populations with the lowest coronary heart disease mortality were plotted. The curves produced can be regarded as typical for a healthy, non-smoking young adult population with relatively long life-expectancy.

The recommended cut-off points of BMI refer to cross-sectional data only. It has been proposed in this section that weight fluctuations, weight gain, and weight loss are in themselves indicators of risk; it is not currently possible to recommend uniform cut-offs for such weight changes which would allow the identification of high-risk groups. Further research is needed in this area.

Anthropometric data for the potential development of reference data or standards should cover at least weight and height, plus age, sex, race, socioeconomic status, presence of disease, and smoking habits. If

Figure 54

Proportional frequency of body mass index values in eight MONICA populations: men aged 25–34 years[a]

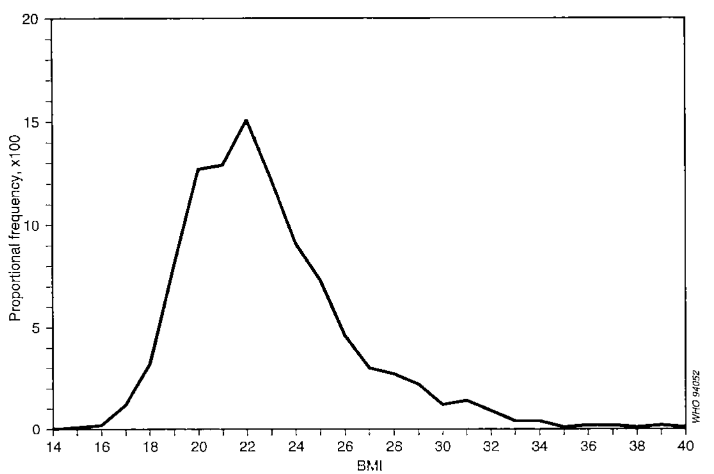


N = 1772 Mean = 24.21 SD = 3.30 Min. = 15 Max. = 49

[a] The WHO MONICA Project has provided these unpublished data from the database described in reference *76*. BMI data for the eight Caucasian populations with the lowest mortality from coronary heart disease were pooled.

Figure 55

Proportional frequency of body mass index values in eight MONICA populations: women aged 25–34 years[a]

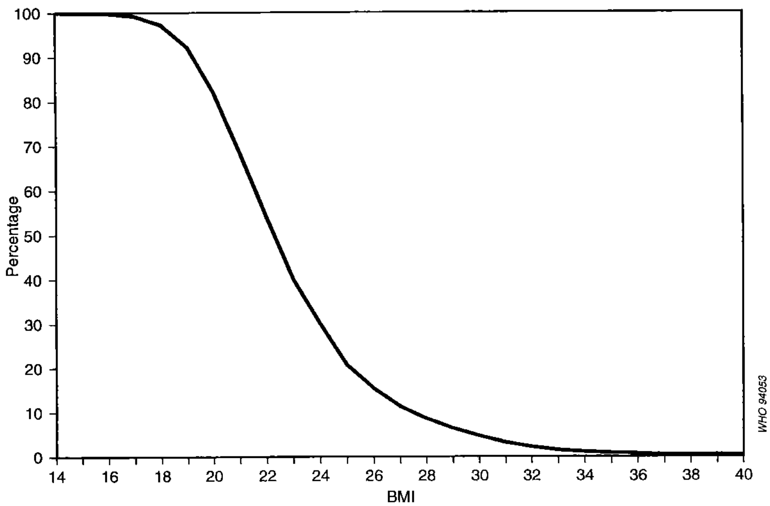


N = 1847 Mean = 22.90 SD = 3.57 Min. = 14 Max. = 44

[a] The WHO MONICA Project has provided these unpublished data from the database described in reference *76*. BMI data for the eight Caucasian populations with the lowest mortality from coronary heart disease were pooled.

Figure 56

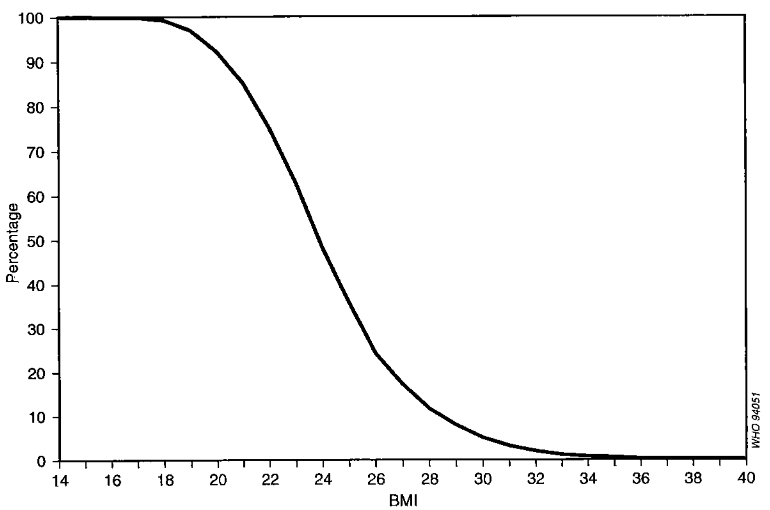
Inverse cumulative distribution of body mass index values in eight MONICA populations: men aged 25–34 years[a]



[a] The WHO MONICA Project has provided these unpublished data from the database described in reference *76*. BMI data for the eight Caucasian populations with the lowest mortality from coronary heart disease were pooled.

Figure 57

Inverse cumulative distribution of body mass index values in eight MONICA populations: women aged 25–34 years[a]



[a] The WHO MONICA Project has provided these unpublished data from the database described in reference *76*. BMI data for the eight Caucasian populations with the lowest mortality from coronary heart disease were pooled.

subjects measured are pregnant or receiving any kind of treatment that might influence weight (including dieting on their own initiative), this should be noted. Information on other (cardiovascular) risk factors should also be documented, as should weight history.

In the future, it may be possible to develop better anthropometric indicators of fatness and distribution in adults; indicators that would monitor change in visceral fat distribution would be particularly important.

7.6 Recommendations

7.6.1 *For practical implementation*

Recommendations to Member States

It is recommended that Member States employ anthropometric techniques to:

1. Assess and monitor levels and trends of adult overweight, BMI, and abdominal fatness and their association with health outcomes.
2. Establish national policies and guidelines for the prevention and control of overweight in adults and evaluate their implementation and outcome.
3. Develop and evaluate programmes for the primary prevention of overweight and abdominal fatness in adults, particularly as part of measures for the prevention of noncommunicable diseases.
4. Facilitate the research necessary to implement clinical and public health action; this should include comparative studies of health consequences of overweight in adults.

Recommendations to WHO

WHO should foster the collection and analysis of existing data (and, where necessary, of new data) on height, weight, and abdomen and hip circumferences to establish the prevalence and trends in overweight and abdominal fatness and their associations with health outcomes.

7.6.2 *For future research*

The following areas were considered to be of particular importance for future research.

1. Development of anthropometric indicators and cut-off points for total body fatness and visceral fat in relation to health outcomes, appropriate for certain subgroups of age, sex, and race.
2. Comparison and surveillance (monitoring) of standardized anthropometric parameters and their distribution among different populations.

3. Identification of the genetic and environmental determinants, and their interactions, of overweight and fat distribution in different populations.
4. Validation of new and existing techniques recommended for use in researching overweight, fatness, and abdominal fatness.
5. Cohort studies of anthropometric indicators and their subsequent risk for noncommunicable diseases and premature mortality, to be carried out in representative samples of populations in different ethnic groups.
6. Development of methods for the assessment of the health implications of various indicators of overweight, fatness, and abdominal fatness.
7. Research on the health effects of voluntary and involuntary weight changes and weight fluctuation.
8. Intervention studies to prevent and control overweight, and evaluation of their feasibility, effects, and impact.

References

1. Garrow JS. *Treat obesity seriously – a clinical manual.* London, Churchill Livingstone, 1981.
2. Seidell JC, Deerenberg I. Obesity in Europe – prevalence and consequences for use of medical care. *Pharmacoeconomics*, 1994, 5(Suppl. I):38-44.
3. Kumanyika SK. Special issues regarding obesity in minority populations. *Annals of internal medicine*, 1993, 119:650-654.
4. Hodge AM et al. Prevalence and secular trends in obesity in Pacific and Indian Ocean island populations. *Obesity research,* 1995, 3(Suppl. 2):77-87.
5. Hodge AM et al. Dramatic increase in the prevalence of obesity in Western Samoa over the 13-year period 1978-1991. *International journal of obesity and related metabolic disorders*, 1994, 18:419-428.
6. *Mauritius Non-Communicable Disease Intervention Program: report on 1992 disease and risk factor prevalence and incidence study.* Melbourne, International Diabetes Institute, 1993.
7. Shetty PS, James WPT. *Body mass index: a measure of chronic energy deficiency in adults.* Rome, Food and Agriculture Organization of the United Nations, 1994 (Food and Nutrition Paper No. 56).
8. Dhurandhar NV, Kulkarni PR. Prevalence of obesity in Bombay. *International journal of obesity and related metabolic disorders*, 1992, 16:367-375.
9. Manson JE et al. Body weight and longevity. A reassessment. *Journal of the American Medical Association*, 1987, 257:353-358.
10. Bouchard C. Genetics of obesity and its prevention. *World review of nutrition and dietetics*, 1993, 72:68-77.
11. Neel JV. Diabetes mellitus: a "thrifty" genotype rendered detrimental by "progress"? *American journal of human genetics*, 1962, 14:353-362.

12. Kuczmarski RJ. Prevalence of overweight and weight gain in the United States. *American journal of clinical nutrition*, 1992, 55(2 Suppl.):495S-502S.

13. Blokstra A, Kromhout D. Trends in obesity in young adults in The Netherlands from 1974 to 1986. *International journal of obesity*, 1991, 15:513-521.

14. Kuskowska-Wolk A, Bergström R. Trends in body mass index and prevalence of obesity in Swedish men 1980-1989. *Journal of epidemiology and community health*, 1993, 47:103-108.

15. Jalkanen L et al. Changes in body mass index in a Finnish population between 1972 and 1982. *Journal of internal medicine*, 1989, 226:163-170.

16. O'Dea K. Westernization and non-insulin-dependent-diabetes in Australian Aborigines. *Ethnicity and disease*, 1991, 1:171-187.

17. Rissanen AM et al. Overweight and anthropometric changes in adulthood: a prospective study of 17,000 Finns. *International journal of obesity*, 1988, 12:391-401.

18. Williamson DF et al. The 10-year incidence of overweight and major weight gain in US adults. *Archives of internal medicine*, 1990, 150:665-672.

19. Rookus MA et al. The effect of pregnancy on the body mass index 9 months postpartum in 49 women. *International journal of obesity*, 1987, 11:609-618.

20. Rissanen AM et al. Determinants of weight gain and overweight in adult Finns. *European journal of clinical nutrition*, 1991, 45:419-430.

21. Ohlin A, Rossner S. Maternal body weight development after pregnancy. *International journal of obesity*, 1990, 14:159-173.

22. Sobal J, Stunkard AJ. Socioeconomic status and obesity: a review of the literature. *Psychological bulletin*, 1989, 105:260-275.

23. *Diet, nutrition, and the prevention of chronic diseases. Report of a WHO Study Group.* Geneva, World Health Organization, 1990 (WHO Technical Report Series, No. 797).

24. Kahn HS, Williamson DF, Stevens JA. Race and weight change in US women: the roles of socioeconomic and marital status. *American journal of public health*, 1991, 81:319-323.

25. Brown PJ. *Cultural perspectives on the etiology and treatment of obesity*. In: Stunkard AJ, Wadden TA, eds. *Obesity: theory and therapy*, 2nd ed. New York, Raven Press, 1992:179-193.

26. Grunberg NE. Cigarette smoking and body weight: current perspectives and future directions. *Annals of behavioural medicine*, 1989, 11:154-157.

27. Hofstetter A et al. Increased 24-hour energy expenditure in cigarette smokers. *New England journal of medicine*, 1986, 314:79-82.

28. Marti B et al. Smoking and leanness: evidence for change in Finland. *British medical journal*, 1989, 298:1287-1290.

29. Baecke JAH et al. Obesity in young Dutch adults: II, Daily life-style and body mass index. *International journal of obesity*, 1983, 7:13-24.

30. Schoenborn CA et al. *Advance data from vital and health statistics*, No. 154. Hyattsville, MD, United States Department of Health and Human Services, 1985.

31. **Williamson DF et al.** Smoking cessation and severity of weight gain in a national cohort. *New England journal of medicine*, 1991, **324**:739-745.

32. **Seidell JC.** Environmental influences on regional fat distribution. *International journal of obesity*, 1991, 15(Suppl. 2):31-35.

33. **Williamson DF et al.** Recreational physical activity and ten-year weight change in a US national cohort. *International journal of obesity and related metabolic disorders*, 1993, **17**:279-286.

34. **Hellerstedt WL, Jeffery RW, Murray DM.** The association between alcohol intake and adiposity in the general population. *American journal of epidemiology*, 1990, **132**:594-611.

35. **Suter PM, Schutz Y, Jequier E.** The effect of ethanol on fat storage in healthy subjects. *New England journal of medicine*, 1992, **326**:983-987.

36. **Sjöström L.** *Impacts of body weight, body composition, and adipose tissue distribution on morbidity and mortality.* In: Stunkard AJ, Wadden TA, eds. *Obesity: theory and therapy*, 2nd ed. New York, Raven Press, 1992.

37. **Lindsted K, Tonstad S, Kuzma JW.** Body mass index and patterns of mortality among Seventh-day Adventist men. *International journal of obesity*, 1991, **15**: 397-406.

38. **Troiano RP et al.** The relationship between body weight and mortality: a quantitative analysis of combined information from existing studies. *International journal of obesity and related metabolic disorders* (in press).

39. **Tokunaga K et al.** Ideal body weight estimated from the body mass index with the lowest morbidity. *International journal of obesity*, 1991, **15**:1-5.

40. **Ashley FW Jr, Kannel WB.** Relation of weight change to changes in atherogenic traits: the Framingham Study. *Journal of chronic diseases*, 1974, **27**:103-114.

41. **Williamson DF, Pamuk ER.** The association between weight loss and increased longevity. A review of the evidence. *Annals of internal medicine*, 1993, **119**: 731-736.

42. **Blair SN et al.** Body weight change, all-cause mortality, and cause-specific mortality in the Multiple Risk Factor Intervention Trial. *Annals of internal medicine*, 1993, **119**:749-757.

43. **Manson JE et al.** A prospective study of obesity and risk of coronary heart disease in women. *New England journal of medicine*, 1990, **322**:882-889.

44. **Larsson B.** Regional obesity as a health hazard in men – prospective studies. *Acta medica Scandinavica supplementum*, 1988, **723**:45-51.

45. **Despres JP et al.** Regional distribution of body fat, plasma lipoproteins, and cardiovascular disease. *Arteriosclerosis*, 1990, **10**:497-511.

46. **Dyken ML et al.** Risk factors in stroke: a statement for physicians by the Subcommittee on Risk Factors and Stroke of the Stroke Council. *Stroke,* 1984, **15**:1105-1111.

47. **Barrett-Connor EL.** Obesity, hypertension and stroke. *Clinical and experimental hypertension [A]*, 1990, **12**:769-782.

48. **Staessen J et al.** Body weight, sodium intake and blood pressure. *Journal of hypertension supplement*, 1989, **7**:S19-S23.

49. **Schotte DE, Stunkard AJ.** The effects of weight reduction on blood pressure in 301 obese patients. *Archives of internal medicine*, 1990, **150**:1701-1704.

50. **Karvonen M et al.** Cigarette smoking, serum cholesterol, blood pressure and body fatness. Observations in Finland. *Lancet*, 1959, **1**:492-494.

51. **Stamler R, Ford CE, Stamler J.** Why do lean hypertensives have higher mortality rates than other hypertensives? Findings of the Hypertension Detection and Follow-up Program. *Hypertension*, 1991, **17**:553-564.

52. **Björntorp P.** "Portal" adipose tissue as a generator of risk factors for cardiovascular disease and diabetes. *Arteriosclerosis*, 1990, **10**:493-496.

53. **Colditz GA et al.** Weight as a risk factor for clinical diabetes in women. *American journal of epidemiology*, 1990, **132**:501-513.

54. **Sims EAH et al.** Endocrine and metabolic effects of experimental obesity in man. *Recent progress in hormone research*, 1973, **29**:457-496.

55. **Liddle RA, Goldstein RB, Saxton J.** Gallstone formation during weight-reduction dieting. *Archives of internal medicine*, 1989, **149**:1750-1753.

56. **Maclure KM et al.** Weight, diet, and the risk of symptomatic gallstones in middle-aged women. *New England journal of medicine*, 1989, **321**:563-569.

57. **Hartz AJ, Rupley DC, Rimm AA.** The association of girth measurements with disease in 32,856 women. *American journal of epidemiology*, 1984, **119**:71-80.

58. **Van Noord PAH et al.** The relationship between fat distribution and some chronic diseases in 11,825 women participating in the DOM-project. *International journal of epidemiology*, 1990, **19**:564-570.

59. **Davis MA.** Epidemiology of osteoarthritis. *Clinical geriatric medicine*, 1988, **4**: 241-255.

60. **Rissanen A et al.** Risk of disability and mortality due to overweight in a Finnish population. *British medical journal*, 1990, **301**:835-837.

61. **Osler M.** Obesity and cancer. A review of epidemiological studies on the relationship of obesity to cancer of the colon, rectum, prostate, breast, ovaries, and endometrium. *Danish medical bulletin*, 1987, **34**:267-274.

62. **Filipovsky J et al.** Abdominal body mass distribution and elevated blood pressure are associated with increased risk of death from cardiovascular disease and cancer in middle-aged men. The results of a 15- to 20-year follow-up in the Paris prospective study I. *International journal of obesity and related metabolic disorders*, 1993, **17**:197-203.

63. **Zaadstra BM et al.** Fat and female fecundity: prospective study of effect of body fat distribution on conception rates. *British medical journal*, 1993, **306**:484-487.

64. **Gortmaker SL et al.** Social and economic consequences of overweight in adolescence and young adulthood. *New England journal of medicine*, 1993, **329**:1008-1012.

65. **Lapidus L et al.** Obesity, adipose tissue distribution and health in women – results from a population study in Gothenburg, Sweden. *Appetite*, 1989, **13**: 25-35.

66. **Stene-Larsen G et al.** Relationship of overweight to hiatus hernia and reflux oesophagitis. *Scandinavian journal of gastroenterology*, 1988, **23**:427-432.

67. **Partinen M, Telakivi T.** Epidemiology of obstructive sleep apnea syndrome. *Sleep*, 1992, **15**(6 Suppl.):S1-S4.

68. **Deurenberg P, Weststrate JA, Seidell JC.** Body mass index as a measure of body fatness: age- and sex-specific prediction formulas. *British journal of nutrition*, 1991, **65**:105-114.

69. **Dowling HJ, Pi-Sunyer FX.** Race-dependent health risks of upper body obesity. *Diabetes*, 1993, 42:537-543.

70. **Seidell JC et al.** The sagittal diameter and mortality in men: the Baltimore Longitudinal Study on Aging. *International journal of obesity and related metabolic disorders*, 1994, **18**:61-67.

71. **McKeigue PM, Shah B, Marmot MG.** Relation of central obesity and insulin resistance with high diabetes prevalence and cardiovascular risk in South Asians. *Lancet*, 1991, **337**:382-386.

72. **Haffner SM et al.** Incidence of type II diabetes in Mexican Americans predicted by fasting insulin and glucose levels, obesity, and body-fat distribution. *Diabetes*, 1990, **39**:283-288.

73. NIH conference. Gastrointestinal surgery for severe obesity. Consensus Development Conference Panel. *Annals of internal medicine*, 1991, **115**: 956-961.

74. **Goldstein DJ.** Beneficial health effects of modest weight loss. *International journal of obesity and related metabolic disorders*, 1992, **16**:397-415.

75. Methods for voluntary weight loss and control. NIH Technology Assessment Conference Panel. Consensus Development Conference, 30 March to 1 April 1992. *Annals of internal medicine*, 1993, **119**:764-770.

76. WHO MONICA Project. Geographic variation in the major risk factors of coronary heart disease in men and women aged 35-64 years. *World health statistics quarterly*, 1988, **41**:115-138.