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OBESITY

PREVENTING AND MANAGING THE GLOBAL EPIDEMIC

**Report of a WHO
Consultation on Obesity**

Geneva, 3-5 June 1997



**World Health Organization
Geneva**

Division of
Noncommunicable Diseases

Programme of Nutrition
Family and Reproductive Health

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List of abbreviations

AIHW	Australian Institute of Health and Welfare
ALCO	Anonymous Fighters Against Obesity
BED	Binge-eating disorder
BMI	Body mass index
BMR	Basal metabolic rate
CHD	Coronary heart disease
CHNS	China Health and Nutrition Survey
CHO	Carbohydrate
CINDI	Community Interventions in Noncommunicable Diseases
CVD	Cardiovascular disease
DALY	Disability Adjusted Life Year
DEXA	Dual-Energy X-ray Absorptiometry
DNA	desoxyribonucleic acid
ENDEF	Estudo Nacional da Despesa Familiar in Brazil
EPI	Expanded Programme on Immunization
EPOC	Excess post-exercise oxygen consumption
FAO	Food and Agriculture Organization of the United Nations
FDA	Food and Drug Administration (USA)
HCG	Human chorionic gonadotrophin
HDL	High density lipoprotein
HMR	Health Management Resources
HPA	Hypothalamic pituitary axis
IGT	Impaired glucose tolerance
INTERHEALTH	Integrated Programme for Community Health in Noncommunicable Diseases
INTERSALT	International Cooperative Study on the Relation of Blood Pressure to Electrolyte Excretion in Population
IOTF	International Obesity Task Force
LDL	Low density lipoprotein
LDL-apoB	Low density lipoprotein apo B
LMS	Least mean square
LPL	Lipoprotein lipase
MONICA	Monitoring of trends and determinants in cardiovascular diseases (i.e. the WHO MONICA study)

NCD	Noncommunicable disease
NCHS	National Center for Health Statistics in the USA
NEFA	Non-esterified fatty acid
NES	Night-eating syndrome
NGO	Nongovernmental organization
NHANES	National Health and Nutrition Examination Survey in the USA
NHES	National Health Examination Survey in the USA
NIDDM	Non-insulin dependent diabetes mellitus
NNS III	The 1992 third Nationwide Nutritional Survey in China
OA	Overeaters Anonymous
PAF	Population-attributable fractions
PAL	Physical activity level
PNSN	Pesquisa Nacional de Saúde e Nutrição
POP	Pound of Prevention
REDP	Reduced energy diet programme
RMR	Resting metabolic rate
SBW	Standard body weight
SES	Socioeconomic status
SHBG	Sex hormone binding globulin
SOS	Swedish Obese Subjects
SSRI	Selective serotonin re-uptake inhibitors
STD	Sexually transmitted disease
TEF	Thermic effect of food
TOPS	Taking Off Pounds Sensibly
UK	United Kingdom of Great Britain and Northern Ireland
UN	United Nations
UNDP	United Nations Development Programme
UNICEF	United Nations Children's Fund
USA	United States of America
VLCD	Very low calorie diet
WHO	World Health Organization
WHR	Waist-hip circumference ratio or waist-hip ratio

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Executive summary

An expert consultation on Obesity was convened by WHO in Geneva from 3 to 5 June 1997 with the aim of reviewing current epidemiological information on obesity, and drawing up recommendations for developing public health policies and programmes for improving the prevention and management of obesity which is emerging as a global public health problem.

The specific objectives of the Consultation were:

1. To review global prevalence and trends of obesity among children and adults; factors contributing to the problem of obesity; and associated consequences of obesity, such as chronic noncommunicable diseases;
2. To examine health and economic consequences of obesity and their impact on development;
3. To develop recommendations to assist countries in developing comprehensive public health policies and strategies for improving the prevention and management of obesity;
4. To identify the issues which need further research.

The Consultation immediately recognized that overweight and obesity represent a rapidly growing threat to the health of populations and an increasing numbers of countries worldwide. It recognized obesity as a disease, which is prevalent in both developing and developed countries and affects children and adults alike. Indeed, overweight and obesity are now so common that they are replacing the more traditional public health concerns such as undernutrition and infectious diseases as some of the most significant contributors to ill health.

The Consultation noted that a coherent system for classifying overweight and obesity in adults is now available and should be adopted internationally. This is based on the Body Mass Index (BMI), calculated as the weight in kilograms divided by the square of the height in metres, with $BMI \geq 25$ denoting "overweight" and $BMI \geq 30$ denoting "obesity". The health burden of obesity would be more easily predicted if the hazards of accumulating intra-abdominal fat were also documented by simple measures such as waist circumference or waist/hip ratio. New criteria and methods for documenting obesity in children are now being developed, and the particular susceptibility of some ethnic groups to weight gain and abdominal fat accumulation is being explored. Many dietary, lifestyle and possibly ethnic factors may also prove to be important in determining the magnitude of the metabolic and mechanical complications associated with obesity. They include non-insulin-dependent diabetes mellitus, cardiovascular diseases, cancers, gastrointestinal diseases and arthritis which are major medical conditions that have hitherto been considered as unrelated to overweight and obesity.

The Consultation concluded that the fundamental causes of the obesity epidemic are sedentary lifestyles and high-fat, energy-dense diets. The rising epidemic reflects the profound changes in society and in the behavioural patterns of communities although some individuals may become obese, partly because they have a genetic or other biological predisposition to gain weight more readily when they are exposed to an unfavourable environment. Identifying environmental and behavioural factors that contribute to weight gain is particularly difficult. There are two principal factors which tend to overwhelm an individual's normal subconscious adjustments in food intake and metabolism that occur as part of a biological capacity to maintain energy balance. These are a fall in spontaneous and work-related physical activity, and a readiness to overconsume high-fat, energy-dense foods.

Because of the interaction of these two principal factors, people who sustain moderately high levels of physical activity throughout life may be able to tolerate high-fat diets, e.g. fat constituting between 30% to 40% of total energy intake. However, the widespread decline in physical activity in most societies, combined with rising fat intake, are associated with rapidly rising rates of obesity. These observations, together with physiological studies, suggest that lower fat intakes—around 20–25% of energy—are needed to minimize energy imbalance and weight gain in relatively sedentary individuals and societies. Other factors, for example, dietary energy density and behavioural aspects of eating, may also contribute to the obesity epidemic, but they need to be evaluated further.

To limit the impact of obesity on individuals, the Consultation called for the development of both preventive and therapeutic strategies. A systematic assessment and management approach is proposed based on evidence available from scientific studies and clinical trials, with primary care playing a central role. Therapeutic interventions among overweight and obese individuals, where support to patients is critical to achieving success, include dietary change, alterations in physical activity, and behaviour modification. These are long-term therapies that emphasize weight management, rather than relying on short-term extreme weight reduction. Strategies for supporting patients depend on individual health care systems. High-risk patients may benefit from carefully monitored drug therapy as part of a comprehensive management scheme, thereby limiting to extreme cases only the need for new forms of gastrointestinal surgery to slow or reverse life-threatening conditions.

The Consultation concluded that global epidemic projections for the next decade are so serious that public health action is urgently required. Analyses show that merely concentrating on children and adults who have a high BMI and associated health problems will not stem the escalating numbers of people entering the medically defined categories of ill health. It is thus essential to develop new preventive public health strategies which affect the entire society. Without societal changes, a substantial and steadily rising proportion of adults will succumb to the medical complications of obesity; indeed, the medical burden of obesity already threatens to overwhelm health services. The spectrum of problems seen in both developing and developed countries is having so negative an impact that obesity should be regarded as today's principal neglected public health problem.

The Consultation's report is divided into five sections. Section A examines the definition and classification of obesity and presents up-to-date regional data on the prevalence of and secular trends in obesity. Section B considers the true costs of obesity both in terms of physical and mental ill health, and the human and financial resources being diverted to deal with it. Section C examines the etiological factors implicated in weight gain and obesity. While most of the information about risk factors comes from studies in developed countries, findings have worldwide relevance for predicting the impact in developing countries. Section D lays the foundation for a comprehensive strategy for preventing and managing of obesity through health care services and public health policy. Section E presents the Consultation's conclusions and recommendations.

1 OBESITY – A MAJOR GLOBAL PUBLIC HEALTH PROBLEM

Throughout most of human history, weight gain and fat storage have been viewed as signs of health and prosperity. In times of hard labour and frequent food shortages, securing an adequate energy intake to meet requirements has been the major nutritional concern.

Today, however, as standards of living continue to rise, weight gain and obesity are posing a growing threat to the health of inhabitants from countries all over the world. Obesity is a chronic disease, prevalent in both developed and developing countries, and affecting children as well as adults. Indeed, it is now so common that it is replacing the more traditional public health concerns, including undernutrition and infectious disease, as one of the most significant contributors to ill health. Furthermore, as obesity is a key risk factor in the natural history of other chronic and noncommunicable diseases (NCDs), it is only a matter of time before developing countries are likely to experience the same high mortality rates for such diseases that industrialized countries with well-established market economies exhibited 30 years ago.

Obesity is not a recent phenomenon. Its historical roots can be traced to the Palaeolithic era more than 25 000 years ago, since stone age artefacts of corpulent women have been found in several sites across Europe. However, the prevalence of obesity has never before reached such epidemic proportions as today.

Clinical evidence of obesity can be dated as far back as Greco-Roman times, but little scientific progress was made towards understanding the condition until the 20th century. In the 19th century, the work of Lavoisier and others indicated that metabolism was similar to slow combustion, and that obese and lean humans obeyed the laws of thermodynamics. Also, the discovery that fat is stored in “cells”, the basic unit of biology, led to the idea that obesity could be caused by too many fat cells. Interestingly, the 19th century also saw the publication of the first diet book entitled *Letter on Corpulence Addressed to the Public* by a Mr Banting.

With the turn of the century, analysis of life insurance data indicated that obesity was associated with an increased death rate. In the 1920s, a familial basis for obesity was suggested along with descriptions of Cushing’s Disease and hypothalamic obesity. Later, the introduction of thyroid hormone, dinitrophenol and amphetamine as pharmacological treatments of obesity opened the door to the use of drugs, and the field of genetics improved understanding of several specific forms of obesity resulting from genetic defects.

Considerable advances have been made in diet, exercise and behavioural approaches to treatment for obesity since they began to be introduced in the first half of the 20th century, and new drugs with ever better profiles of pharmacological activity continue to be introduced on a regular basis. Gastric surgery has had the most effective long-term success in treating the severely obese. Despite this progress, however, obesity prevalence continues to increase sharply as we approach the end of the millennium. The challenge to public health workers and scientists in this area has never been greater.

This report provides an assessment of current data on the prevalence of obesity, its health consequences and its economic costs. Strategies for implementing a systematic approach to the prevention and management of obesity in different health service systems, and recommendations from leading international obesity experts, are also provided. It is hoped that these recommendations will be used in the development of new policies to address the escalating public health problem of obesity.

1.1 The structure of the report

The report is divided into five sections, four of which deal with different aspects of the global epidemic of obesity. The final section (E) outlines the conclusions and recommendations from the WHO Consultation on Obesity (Figure 1.1).

Section A of this report examines issues surrounding the definition and classification of obesity, and sets out the most recent data on the global prevalence and secular trends in all regions of the world. Defining and identifying the extent of the problem of obesity is a critical first step in a coherent approach to its prevention and management.

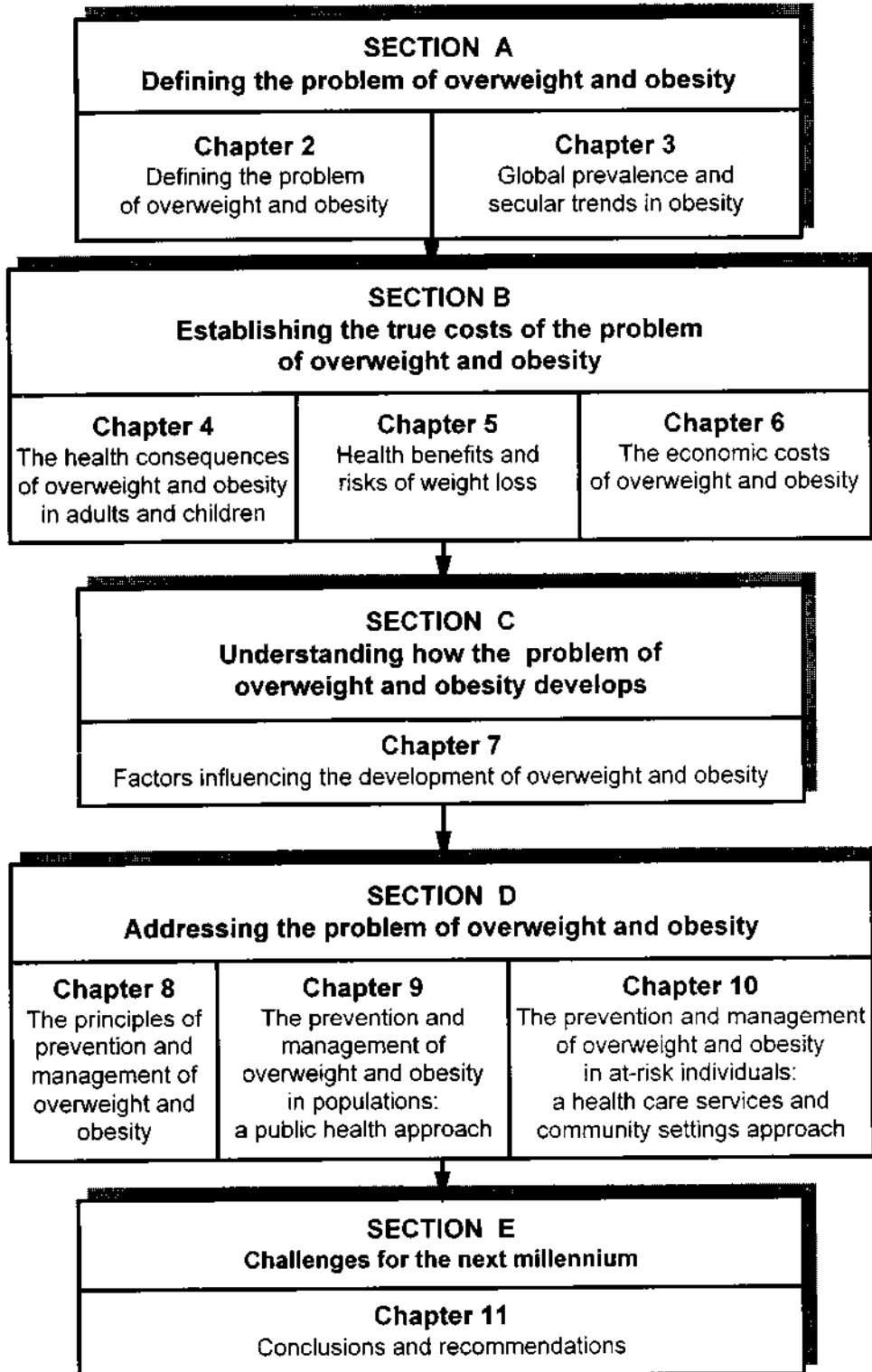
Section B covers the true costs of obesity in terms of physical and mental ill health, and the human and financial resources being diverted to deal with it. The amount of suffering, and the money expended by health agencies to deal with the problem, are staggering and reinforce the need for urgent action.

Section C examines what we know about this complex, multifactorial disease and identifies the major factors which are implicated in its development. Most of the information about risk factors for weight gain and obesity comes from studies in developed countries because developing countries have only recently seen a rise in chronic diseases and therefore have little experience in carrying out research on this area. Examination of the factors implicated in weight gain and obesity in developed countries, however, is of worldwide relevance for predictions about the future impact in countries at early stages of the transition, and provides a unique opportunity for taking preventive action. It is also important that these factors should be addressed in any coordinated strategy to tackle the problem of obesity.

Section D takes account of the issues raised in the preceding three sections to present the foundations of a comprehensive strategy for the prevention and management of obesity through health care services and public health policy. The challenge is there for policy-makers, health professionals and the community at large to join forces in an effort to tackle this major global public health problem.

Section E outlines the final conclusions and recommendations from the WHO Consultation on Obesity. Priority areas for further research are identified, and recommendations on strategies and actions for the effective prevention and management of the global epidemic of obesity are made.

Figure 1.1 Structure of the report



1.2 Themes of the report

Obesity is a complex and incompletely understood disease. This report aims to highlight key issues which are central to the development of a coherent strategy for the effective management and prevention of obesity on a worldwide basis. In producing the report, a number of important themes have dictated the content and style. These include:

- Obesity is a serious disease, but its development is not inevitable. It is largely preventable through lifestyle changes.
- The health risks associated with excessive body fat occur with a relatively small increase in body weight, and are not present only in those who are markedly obese. Effective management of obesity cannot be separated from prevention.
- Obesity is not just an individual problem. It is a population problem and should be tackled as such. Effective prevention and management of obesity will require an integrated approach, with actions across all sectors of society.
- Obesity is a chronic disease that requires the application of long-term strategies for its effective prevention and management.
- Obesity affects all age groups. The effective prevention of adult obesity will require attention to the prevention and management of childhood obesity.
- Obesity is a global problem. Prevention and management strategies that are applicable to all regions of the world should be developed.
- Obesity can be seen as the tip of the iceberg of a defined cluster of NCDs now observed in both developed and developing nations. The global epidemic of obesity is a reflection of the massive social, economic and cultural problems now facing developing and newly industrialized countries, as well as the ethnic minorities and the disadvantaged in developed countries.
- Examination of the factors implicated in weight gain and obesity in developed countries is crucial for predictions about the future impact in countries at early stages of the transition, and provides a unique opportunity for taking preventive action.
- In countries with developing economies, the problem of obesity is emerging at a time when undernutrition remains a significant problem. Strategies which take account of both these important nutritional problems will need to be developed, particularly when dealing with children whose growth may be stunted.

Section A

Defining the problem of overweight and obesity

2 DEFINING THE PROBLEM OF OVERWEIGHT AND OBESITY

2.1 Introduction

Obesity is often defined simply as a condition of abnormal or excessive fat accumulation in adipose tissue, to the extent that health may be impaired (1). The underlying disease is the process of undesirable positive energy balance and weight gain. However, obese individuals differ not only according to the degree of excess fat which they store, but also in the regional distribution of that fat within the body. The distribution of fat induced by weight gain affects the risks associated with obesity, and the kinds of disease that result. Indeed, excess abdominal fat is as great a risk factor for disease as is excess body fat *per se*. There is value, therefore, in having a method of distinguishing those at increased risk as a result of "abdominal fat distribution", or "android obesity" as it is often known, from those with the less serious "gynoid" fat distribution, in which fat is more evenly and peripherally distributed around the body.

Classifying obesity during childhood or adolescence has the added complication that height is still increasing and body composition is continually changing. Furthermore, there are substantial international differences in the age of onset of puberty and in the differential inter-individual rates of fat accumulation.

This chapter outlines the most appropriate methods for (a) classifying overweight and obesity in adults, and (b) identifying abdominal fat distribution. It also briefly discusses the use of additional tools for more detailed characterization of obese individuals. The final section outlines the current lack of consistency and agreement between studies over the classification of obesity in childhood and adolescence, and highlights the need for a globally standardized classification system.

Key issues covered include:

- Obesity can be defined simply as a disease in which excess body fat has accumulated to an extent that health may be adversely affected. However, the degree of excess fat, its distribution within the body and the associated health consequences vary considerably between obese individuals.
- The graded classification of overweight and obesity allows: (a) meaningful comparisons of weight status within and between populations, (b) the identification of individuals and groups at increased risk of morbidity and mortality, (b) the identification of priorities for intervention at individual and community levels, and (d) a firm basis for evaluation of interventions.
- Body mass index (BMI) provides the most useful, albeit crude, population-level measure of obesity. It can be used to estimate the prevalence of obesity within a population and the risks associated with it. However, BMI does not account for the wide variation in body fat distribution, and may not correspond to the same degree of fatness or associated health risk across different individuals and populations.

- Obese individuals with excess fat in the intra-abdominal depots are at particular risk of the negative health consequences of obesity. Therefore, measurement of waist circumference provides a simple and practical method of identifying overweight patients at increased risk of obesity-associated illness due to abdominal fat distribution.
- Ethnic populations differ in the level of risk associated with a particular waist circumference, and a globally applicable grading system of waist circumference has not yet been developed.
- Additional tools available for the more detailed characterization of the obese state include measures of body composition (e.g. underwater weighing), anatomical distribution of body fat (e.g. magnetic resonance imaging), energy intake (e.g. prospective dietary record) and energy expenditure (e.g. doubly labelled water). However, the cost and practical difficulties involved in such techniques limit their usefulness to research.
- The classification of weight status of children and adolescents is complicated by the fact that height and body composition are continually changing; such changes often occur at different rates and times in different populations, making simple universal indices of adiposity of little value. To date, there has not been the same level of agreement over the classification of obesity for children or adolescents as there is for adults.

2.2 Why classify overweight and obesity?

The graded classification of overweight and obesity is valuable for a number of reasons. In particular, it allows:

- Meaningful comparisons of weight status within and between populations.
- The identification of individuals and groups at increased risk of morbidity and mortality.
- The identification of priorities for intervention at individual and community levels.
- A firm basis for evaluating interventions.

2.3 Body mass index

BMI is a simple index of weight-for-height that is commonly used to classify overweight and obesity in adults. It is calculated as the weight in kilograms divided by the square of the height in metres (kg/m^2).

For example, an adult who is 70 kg in weight and 1.75 m in height will have a BMI of 22.9:

$$\text{BMI} = 70 \text{ (kg)} / 1.75^2 \text{ (m}^2\text{)} = 22.9$$

The classification of overweight and obesity, according to BMI, is shown in Table 2.1. Obesity is classified as a BMI ≥ 30.0 . The classification shown in Table 2.1 is in agreement with that recommended by WHO (2), but includes an additional subdivision at BMI 35.0–39.9 in recognition of the fact that management options for dealing with obesity differ above a BMI of 35. The WHO classification is based primarily on the association between BMI and mortality (see Chapter 3).

Table 2.1 Classification of overweight in adults according to BMI

Classification	BMI (kg/m^2)	Risk of co-morbidities
Underweight	< 18.5	Low (but risk of other clinical problems increased)
Normal range	18.5–24.9	Average
Overweight	≥ 25	
Pre-obese	25–29.9	Increased
Obese class I	30.0–34.9	Moderate
Obese class II	35.0–39.9	Severe
Obese class III	≥ 40.0	Very severe

Note that these BMI values are age-independent and the same for both sexes. However, BMI may not correspond to the same degree of fatness across different populations due, in part, to different body proportions (see section 2.3.2).

This table shows a simplistic relationship between BMI and risk of co-morbidity which can be affected by a range of factors, including nature of the diet, ethnic group and activity level. The risks associated with increasing BMI are continuous and graded, and begin at a BMI below 25. Interpretation of BMI gradings in relation to risk may differ for different populations.

Note that both BMI and a measure of fat distribution (waist circumference or waist-hip ratio (WHR)) are important in calculating the risk of obesity co-morbidities. BMI < 18.5 signifies an increased risk of developing other clinical problems.

2.3.1 The value of consistency in the global classifications of obesity

A BMI of 30 or more is now widely accepted as denoting the classification of obesity. Some studies, however, have used alternative BMI cut-off points both above and below 30. For instance, in the USA, obesity is routinely classified on the basis of the distribution of BMI in the

National Health and Nutrition Examination Survey (NHANES), a representative study of the US population: 27.8 in men and 27.3 in women are taken as the 85th percentile (3). Such differences in cut-off points have a great impact on estimates of the prevalence of obesity in given populations. Furthermore, by choosing a percentile value to determine the cut-off point, any public health analyses of the health burden of obesity will be minimized because the cut-off point will increase as a population gains weight. For meaningful comparisons between or within populations it is therefore advisable to apply the single BMI cut-off points proposed in Table 2.1.

2.3.2 Variation in the relationship between BMI and body fatness

Although it can generally be assumed that individuals with a BMI of 30 or above have an excess fat mass in their body, BMI does not distinguish between weight associated with muscle and weight associated with fat. As a result, the relationship between BMI and body fat content varies according to body build and proportion, and it has been shown repeatedly that a given BMI may not correspond to the same degree of fatness across populations. Polynesians, for example, tend to have a lower fat percentage compared to Caucasian Australians at an identical BMI (4). In addition, the percentage of body fat mass increases with age up to 60–65 years in both sexes (5,6), and is higher in women than in men of equivalent BMI (7). For cross-sectional comparisons, therefore, BMI values should be interpreted with caution if estimates of body fat are required.

Differences in body proportions and in the relationship between BMI and body fat content can affect the BMI range that is considered to be healthy. Calculations based on the ratio of sitting height to standing height that allow BMI to be corrected to take account of unusual leg lengths are now available. Thus, very tall and lean Australian Aboriginals tend to have a deceptively low BMI; a healthy BMI range for this population appears to be between 17 and 22, with metabolic complications developing rapidly as BMI increases above this. Recalculating Aboriginal data to allow for their unusual body proportions increases both the mean BMI and the BMI distribution, so that the percentage with a BMI > 25 increases from 8% to 15% (8).

2.3.3 The use of BMI to classify obesity

BMI can be considered to provide the most useful, albeit crude, population-level measure of obesity. The robust nature of the measurements and the widespread routine inclusion of weights and heights in clinical and population health surveys mean that a more selective measure of adiposity, such as skin fold thickness measurements, provide additional rather than primary information. BMI can be used to estimate the prevalence of obesity within a population and the risks associated with it. It does not, however, account for the wide variation in the nature of obesity between different individuals and populations.

2.4 Waist circumference and waist-hip ratio

Abdominal fat mass can vary dramatically within a narrow range of total body fat or BMI. Indeed, for any accumulation of total body fat, men have on average twice the amount of abdominal fat than that which is generally found in pre-menopausal women (9). Additional alternative methods to the measurement of BMI would therefore be valuable in identifying individuals at increased risk from obesity-related illness due to abdominal fat accumulation.

Over the last 10 years or so, a high WHR (WHR > 1.0 in men and > 0.85 in women) has become accepted as the clinical method of identifying patients with abdominal fat accumulation (10). However, recent evidence suggests that waist circumference alone—measured at the midpoint between the lower border of the rib cage and the iliac crest—may provide a more practical correlate of abdominal fat distribution and associated ill health (10-12).

Waist circumference is a convenient and simple measurement which is unrelated to height (13), correlates closely with BMI and WHR (12), and is an approximate index of intra-abdominal fat mass (14-16) and total body fat (17). Furthermore, changes in waist circumference reflect changes in risk factors for cardiovascular disease (CVD) (18) and other forms of chronic disease, even though the risks seem to vary in different populations.

Some experts consider that the hip measurement contains additional valuable information related to gluteofemoral muscle mass and bone structure (19). The WHR may therefore remain a useful research tool but individuals can be identified as being at increased risk of obesity-related illness by using the waist circumference alone as an initial screening tool.

2.4.1 Threshold values of waist circumference

Populations differ in the level of risk associated with a particular waist circumference, and so globally applicable cut-off points cannot be developed. For instance, abdominal fatness has been shown to be less strongly associated with risk factors for CVD and non-insulin dependent diabetes mellitus (NIDDM) in black women than in white women (20). Also, people of South Asian (Indian, Pakistani and Bangladeshi) descent living in urban societies have a higher prevalence of many of the complications of obesity than other ethnic groups (21). These complications are seen to be associated with abdominal fat distribution which is markedly higher for a given level of BMI than in Europeans. Finally, although women have an almost equivalent absolute risk of coronary heart disease (CHD) to men at the same waist-hip ratio (22,23), they show increases in relative risk of CHD at lower waist circumferences than men. Thus, there is a need to develop sex-specific waist circumference cut-off points appropriate for different populations.

The sex-specific waist circumferences outlined in Table 2.2 denote enhanced relative risk for a random sample of 2183 men and 2698 women aged 20–59 from the Netherlands (23).

Table 2.2 Sex-specific waist circumferences that denote “increased risk” and “substantially increased risk” of metabolic complications associated with obesity in Caucasians

	Risk of obesity-associated metabolic complications	
	Increased	Substantially increased
Men	≥ 94 cm (~37 inches)	≥ 102 cm (~40 inches)
Women	≥ 80 cm (~32 inches)	≥ 88 cm (~35 inches)

Note that this table is an example only. The identification of risk using waist circumference will be population-specific and will depend on levels of obesity and other risk factors for CVD and NIDDM. This issue is currently under investigation.

2.5 Additional tools for the assessment of obesity

In addition to the anthropometric assessment methods outlined previously, there are various other tools that are useful for measuring body fat in certain clinical situations and also in obesity research. These tools are particularly useful when trying to identify the genetic and environmental determinants of obesity and their interactions, as they enable the variable and complex nature of obesity to be split into separate components. Thus, obese individuals can be characterized by measuring body composition, anatomical distribution of fat, energy intake and insulin resistance, among others.

A list of those characteristics considered suitable for measuring in genetic studies has recently been agreed (24) and is summarized in Table 2.3. Measures in a given category do not necessarily have equal validity.

Table 2.3 Currently recommended characteristics for assessment in genetic studies

Characteristic of obesity measured	Examples of measurement tools
Body composition	BMI; waist circumference; Underwater weighing; dual-energy X-ray absorptiometry (DEXA); isotope dilution; bioelectrical impedance; skin fold thickness
Anatomical distribution of fat	Waist circumference; WHR; computer tomography; ultrasound; magnetic resonance imaging
Partitioning of nutrient storage	¹³ C palmitic acid; extended overfeeding challenge
Energy intake	“Total” by prospective dietary record or recall; “macronutrient composition” by prospective dietary record or recall or dietary questionnaire
Energy expenditure	“Total” by doubly labelled water; “resting” by indirect calorimetry; physical activity level (PAL) by questionnaire, motion detector, heart rate monitor, etc.

2.6 Classifying obesity in childhood

To date, there has not been the same level of agreement over the classification of overweight and obesity in children and adolescents as there has been in adults. There has been confusion both in terms of a globally applicable reference population and the selection of appropriate cut-off points for designating a child as obese.

2.6.1 Use of growth charts

Many countries have produced reference charts for growth based on weight-for-age and height-for-age. However, these measures are a collection of the child's size (small or large) and provide no indication of relative fatness. The close correlation between height and weight during childhood means that an index of weight adjusted for height can provide a simple measure of fatness.

2.6.2 International childhood reference population

The most widely used growth reference, which WHO has recommended for international use since the late 1970s (25,26), was developed by the US National Center for Health Statistics (NCHS). However, a WHO Expert Committee (2) recently drew attention to a number of serious technical and biological problems with the growth reference. WHO is therefore currently undertaking the development of a new growth reference for infants and children from birth to 5 years. This will be based on a sample of infants and children from different parts of the world whose caregivers follow internationally recognized health recommendations. A similar reference will also be required for older children and adolescents.

2.6.3 BMI-for-age reference curves

Adult BMI increases very slowly with age, so age-independent cut-off points can be used to grade fatness. In children, however, BMI changes substantially with age, rising steeply with infancy, falling during the pre-school years, and then rising again into adulthood. For this reason, child BMI needs to be assessed using age-related reference curves.

Such curves have been produced for a number of countries (6,27–29). However, many are imperfect either because the data are old or the age range is restricted. More recent BMI-for-age charts have been developed for Swedish, British and Italian children (30–32) using the least mean square (LMS) method of Cole (33), which adjusts BMI distribution for skewness and allows BMI in individual subjects to be expressed as an exact centile or standard deviation score. The use of BMI-for-age is currently being explored, in parallel with other potential techniques, by an expert working group in order to determine the best method for classifying overweight and obesity in childhood. A common standard should allow the comparative evaluation of childhood obesity internationally.

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3 GLOBAL PREVALENCE AND SECULAR TRENDS IN OBESITY

3.1 Introduction

Evidence is now emerging to suggest that the prevalence of overweight and obesity is increasing worldwide at an alarming rate. Both developed and developing countries are affected. Moreover, as the problem appears to be increasing rapidly in children as well as in adults, the true health consequences may only become fully apparent in the distant future.

The value of estimating the prevalence of, and secular trends in, overweight and obesity cannot be over-emphasized. Knowledge about the level and changing distribution of overweight and obesity can be used to:

- Identify populations at particular risk of obesity and its associated health and economic consequences.
- Help policy-makers and public health planners in the mobilization and reallocation of resources for the control of the disease.
- Provide baseline data for monitoring the effectiveness of national programmes for the control of obesity.

This chapter provides a global overview of secular trends in obesity among adults. It begins with a note of caution in making comparisons between different studies, and then outlines the comprehensive WHO MONICA (MONItoring of trends and determinants in CARDIOvascular diseases) study. The bulk of the chapter, however, reviews secular trends over the last 10 to 20 years and the most current prevalence data available within each of the six WHO regions—Africa, the Americas, South-East Asia, the Eastern Mediterranean, Europe and the Western Pacific—and highlights where more data are needed.

Despite the limited availability of nationally representative data (particularly secular trend data), the following key points are evident:

- Obesity prevalence is increasing worldwide at an alarming rate in both developed and developing countries.
- In many developing countries, obesity co-exists with undernutrition (BMI < 18.5). Obesity is still relatively uncommon in African and Asian countries, but is more prevalent in urban than in rural populations. In economically advanced regions, prevalence rates may be as high as in industrialized countries.

- Women generally have higher rates of obesity than men although men may have higher rates of overweight.
- The current lack of consistency and agreement between different studies over the classification of obesity in children and adolescents makes it difficult to give an overview of the global prevalence of obesity for younger age groups. Nevertheless, whatever classification system has been used, studies investigating obesity during childhood and adolescence have generally reported increasing prevalence of obesity.

3.2 A note of caution

Several factors can make comparisons of data between different cross-sectional studies problematic. These are:

- **Classification of obesity:** a number of studies have not used the recommended WHO international classification of obesity, i.e. $BMI \geq 30$.
- **Age group:** the age group chosen affects the proportion of obese cited.
- **Age-standardization:** many studies have failed to standardize the age structure of the population according to a reference such as the new standard world population data (1).
- **Time-period/year of data collection:** there is need for continuous monitoring of programmes so that current data are always available.
- **Measured versus self-reported weight and height:** self-reported weight and height are notoriously unreliable, especially in the obese.

Many studies have been excluded from this review because of problems generated by the factors listed above, or because they were conducted several years ago without any follow-up and are therefore of limited value. The prevalence data cited in this chapter are those most recently available and illustrate the global nature of the prevalence of obesity. Prevalence data have generally been derived from representative national surveys. However, due to limited availability of longitudinal data, secular trends have often been illustrated with data from representative samples.

In each of the following tables, obesity is classified as $BMI \geq 30$ unless otherwise stated.

3.3 The WHO MONICA study

The most comprehensive data on the prevalence of obesity worldwide are from the WHO MONICA study (2). Although the populations are not necessarily representative of the countries in which they are located, the 48 populations shown in Figure 3.1 and Figure 3.2 can be compared because the data were collected in the same time-period, are age-standardized, and are based on weights and heights measured with identical protocols. The data presented were collected in the first round between 1983 and 1986, but more recent data are expected to be published in the near future. The majority of the data are from European populations.

Figure 3.1 and Figure 3.2 show the BMI distributions in 48 MONICA populations for men and women, respectively (3). Although this report focuses on data relating to highlighted obesity, i.e. BMI ≥ 30 , it is important to note that a BMI between 25 and 29.9 is responsible for the major part of the impact of overweight on certain obesity co-morbidities; it has been estimated, for example, that about 64% of male and 77% of female cases of NIDDM could theoretically be prevented if no person had a BMI ≥ 25 . These figures are compared with preventing only 44% and 33% if keeping below a BMI cut-off point of 30 is chosen (4,5).

Figure 3.1 and Figure 3.2 show that in all but one male population, and in the majority of female populations, between 50% and 75% of adults aged 35–64 were either overweight or obese during the period 1983–1986. In a few populations, this figure was over 75%. Thus, between 1983 and 1986, the majority of adults in these populations were at increased risk of illness due to being overweight or obese. Based on the evidence of increasing prevalence of obesity on a global basis, the current situation is likely to be even worse.

Figure 3.1 BMI distribution: age-standardized proportions of selected categories in MONICA populations, age group 35-64 years (men)

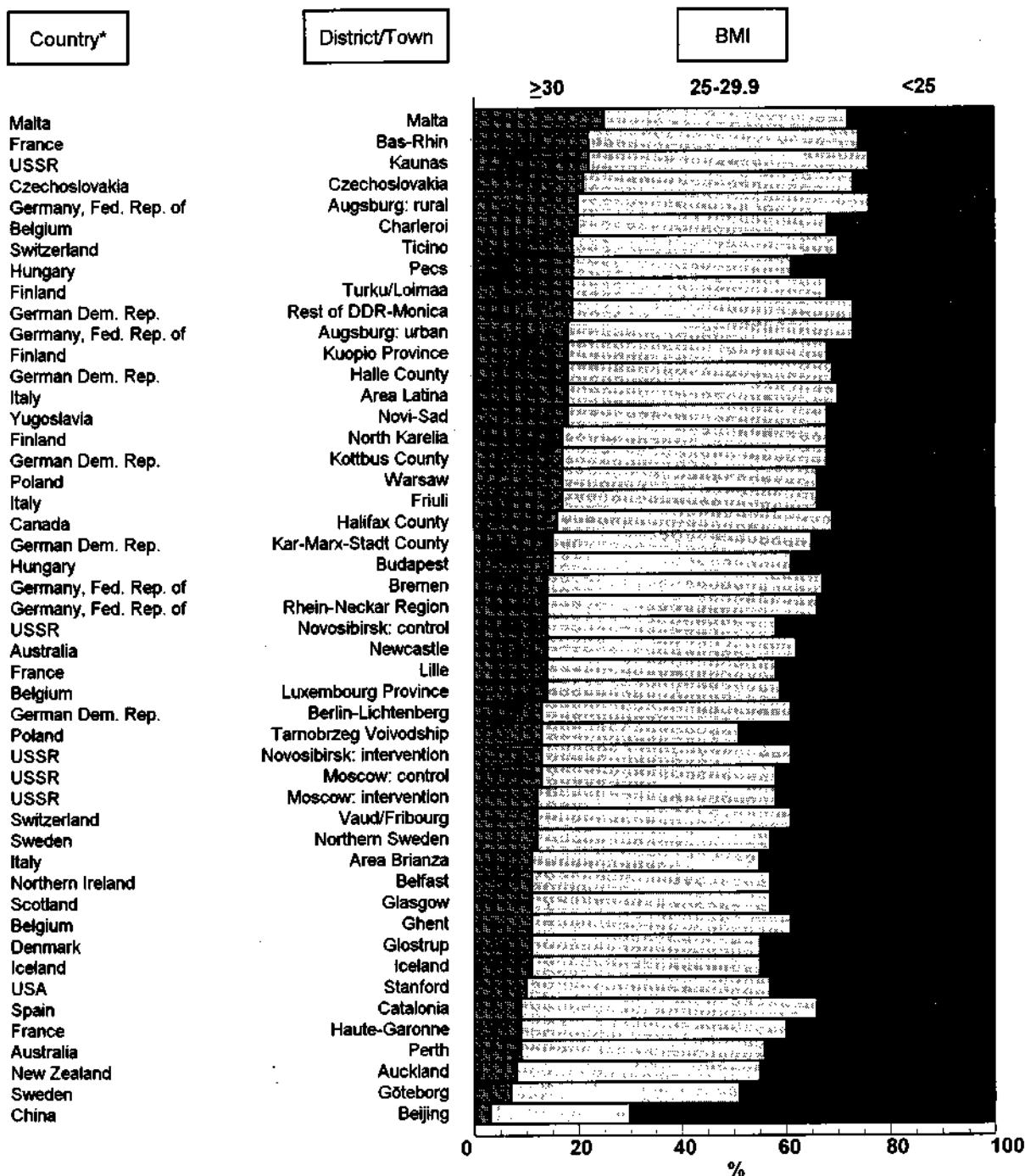


Figure 3.1 shows the proportions of men who were classified as obese, overweight and normal weight in 48 populations (mainly European) taking part in the WHO MONICA study. Although the 48 populations shown are not necessarily representative of the countries in which they are located, they can be compared because the data were collected in the same time-period, are age-standardized, and are based on heights and weights measured with identical protocols. The WHO MONICA study has generated one of the most comprehensive data sets on the prevalence of obesity worldwide. Data collected 1983-1986 (3).

* Names of countries are those that were valid at the time of data collection.

Figure 3.2 BMI distribution: age-standardized proportions of selected categories in MONICA populations, age group 35–64 years (women)

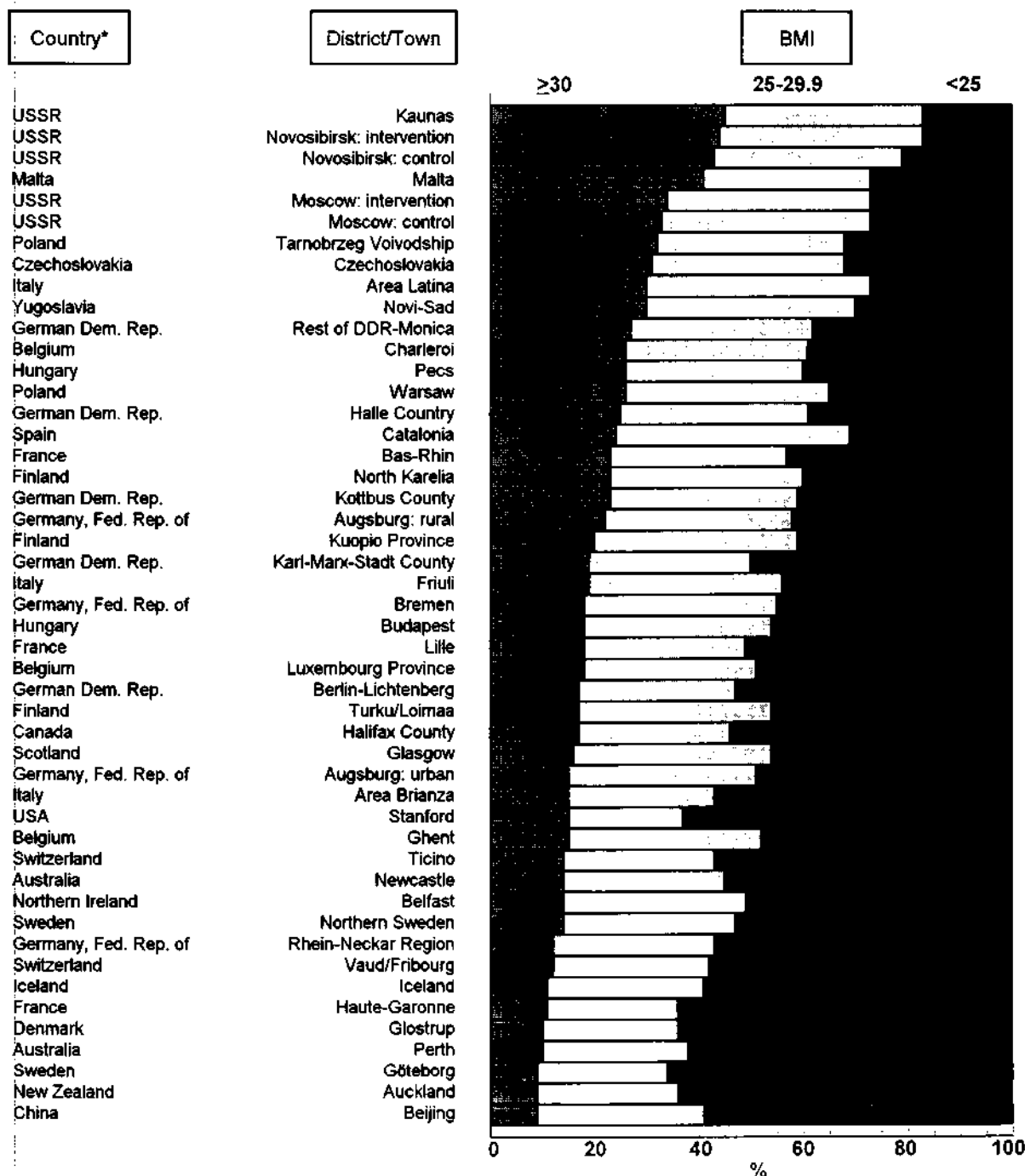


Figure 3.2 shows the proportions of women who were classified as obese, overweight and normal weight in 48 populations (mainly European) taking part in the WHO MONICA study. Although the 48 populations shown are not necessarily representative of the countries in which they are located, they can be compared because the data were collected in the same time-period, are age-standardized, and are based on heights and weights measured with identical protocols. The WHO MONICA study has generated one of the most comprehensive data sets on the prevalence of obesity worldwide. Data collected 1983–1986 (3).

* Names of countries are those that were valid at the time of data collection.

3.4 African Region

3.4.1 Secular trends in obesity

In contrast to most industrialized nations, the focus in many countries in WHO's African Region has necessarily been undernutrition and food security. As a result, there are few African countries or populations for which trends in obesity have been documented.

However, one recent study in Mauritius has shown the trend which is in line with the other five WHO regions—a dramatic increase in obesity prevalence over only a five-year period in both men and women aged 25–74. The proportion of obese men increased from 3.4% in 1987 to 5.3% in 1992, while the proportion of obese women increased from 10.4% to 15.2% in the same period. This increase was seen in all age groups and ethnic groups (6,7). Although it could be argued that Mauritius is not typical of other countries in the Region, this study highlights the adverse effects of lifestyle change in rapidly modernizing populations and how quickly obesity can become a public health problem.

3.4.2 Current prevalence of obesity

From the fragmentary and limited prevalence data available, it is evident that obesity does exist in developing as well as in the more developed countries in the Region, particularly among women. Table 3.1 shows data from a selection of studies carried out in African countries.

Table 3.1 Obesity prevalence (BMI \geq 30) in selected countries in Africa

Country	Year	Ages (years)	Prevalence of obesity (%)	
			Men	Women
Ghana ^a	1987/8	20+		0.9
Mali ^a	1991	20+		0.8
Mauritius ^b	1992	25–74	5	15
Rodrigues ^c Creoles	1992	25–69	10	31
South Africa (Cape Peninsula) ^d Blacks	1990	15–64	8	44
Tanzania ^e	1986/89	35–64	0.6	3.6

^a Reference 11

^b Reference 7

^c Reference 12

^d Reference 10

^e Reference 13

In developing countries, rural adults living traditionally gained little or no weight with age until relatively recently. This was formerly the case in Africa, and still is today in the few remaining hunter-gatherer populations, such as San, in northern Botswana (8). However, with the rise in socioeconomic status (SES) and increasing changes due to rapid urbanization, the prevalence of obesity among some groups of black women has risen markedly to levels exceeding those in populations in industrialized countries (9). In fact, approximately 44% of African women living in the Cape Peninsula were estimated to be obese in 1990 (10).

3.5 Region of the Americas

3.5.1 Secular trends in obesity

Secular trend data are available for Brazil, Canada and the USA, and are summarized in Table 3.2. These data indicate that obesity rates for both men and women are increasing not only in developed countries, but also in developing countries and in countries in transition, such as Brazil.

The most comprehensive data of national trends in the prevalence of obesity for a developed country in the Region come from the USA. These are based on comparisons of data from the NHES I (1960–1962), NHANES I (1971–1974), NHANES II (1976–1980), and NHANES III (1988–1994) and, as detailed earlier, are usually quoted for a BMI > 27.8 in men and BMI > 27.3 in women (14). However, the figures for the USA presented in Table 3.2 are particularly valuable as they have been recalculated from the above-noted NHES and NHANES surveys according to the WHO classification of obesity, i.e. BMI \geq 30. These suggest that obesity is an escalating problem in the USA; there was a slight increase in the overall estimated prevalence of obesity during the period covered by the first three surveys, but a much larger increase between the third and the fourth surveys.

Data from Brazil provide the most valuable information on obesity prevalence and trends for a country in transition in the Region; two comparable, nationally representative, random nutrition surveys made 15 years apart provide for a detailed investigation of patterns of contemporary changes in the nutritional status of children and adults, men and women, rich and poor. These studies, which were undertaken by the Brazilian agency in charge of national statistics in 1974/75 (the Estudo Nacional da Despesa Familiar (ENDEF) survey) and in 1989 (the Pesquisa Nacional de Saúde e Nutrição (PNSN) survey), show that increases in adult obesity have been occurring among all groups of men and women. However, a greater increase has been observed among lower income families. Brazil is rapidly shifting from a problem of dietary deficit to one of dietary excess (15).

Table 3.2 Trends in obesity (BMI \geq 30) in selected countries in the Americas

Country	Year	Ages (years)	Prevalence of obesity(%)	
			Men	Women
Brazil ^a	1975	25-64	3.1	8.2
	1989		5.9	13.3
Canada	^b 1978	20-70	6.8	9.6
	^c 1981	20-70	8.5	9.3
	^d 1988	20-70	9.0	9.2
	^e 1986-92	18-74	13.0	14.0
USA ^f	1960	20-74	10.0	15.0
	1973		11.6	16.1
	1978		12.0	14.8
	1991		19.7	24.7

^a Reference 15

^b Reference 16

^c Reference 17

^d Reference 18

^e Reference 19

^f Prevalence figures recalculated from US NCHS, Centers for Disease Control and Prevention, with BMI \geq 30

3.5.2 Current prevalence of obesity

The most recent data for the prevalence of obesity in the USA stem from the third NHANES (1988-1994). For global comparisons, a recent re-analysis of the data using a BMI \geq 30 to classify obesity is particularly valuable; it revealed that around 20% of all men and 25% of all women in the USA are obese. Table 3.3 indicates that, in the early 1990s, obesity was more widespread in the USA than in Canada. Detailed subgroup analysis of the USA data shows that black women and other minority populations in the USA tend to have particularly high rates of obesity.

The only Latin American country to have had a nationally representative survey conducted in the last 10 years is Brazil. The PNSN survey indicated that obesity is prevalent in Brazil, affecting 6% of women and 13% of men in 1989 (15).

Evidence from the Caribbean, specifically Barbados, Cuba, Jamaica and St. Lucia, indicate that obesity is a significant problem in this region. It is more common in those countries with a higher per capita GNP, affects women more than men, and is associated with a parallel increase in the prevalence of hypertension and NIDDM (20). However, as the supporting data use an unusual classification system (obese males: BMI \geq 31.1; obese females: BMI \geq 32.3), the study is not cited in Table 3.3.

Table 3.3 Obesity prevalence (BMI \geq 30) in selected countries in the Americas

Country	Year	Ages (years)	Prevalence of obesity (%)	
			Men	Women
Brazil ^a	1989	25–64	6	13
Canada ^b	1991	18–74	15	15
USA ^c	1991	20–74	19.7	24.7

^a Reference 15^b Reference 19^c Prevalence figures recalculated from US NCHS, Centers for Disease Control and Prevention, with BMI \geq 30

3.6 South-East Asian Region

3.6.1 Secular trends in obesity

Good quality, nationally representative, secular trend data for countries in the South-East Asian Region were not identified. However, data from two studies conducted by the same research centre in Thailand do suggest that diet-related chronic diseases, including obesity, are on the increase in affluent urban populations. The first study was conducted in 1985 among 35- to 54-year-old Thai officials and found that 2.2% of the 2703 men, and 3.0% of the 792 women, had a BMI \geq 30 (21). The second study in 1991 was smaller (66 men and 453 women), and had a broader age range (19–61 years), but also assessed nutritional factors in affluent urban Thais. It found that 3.0% of men and 3.8% of women had a BMI \geq 30. Prevalence figures for BMI 25–29.9 were considerably higher (15.2% in men and 23.2% in women) (22).

3.6.2 Current prevalence of obesity

Only limited obesity prevalence data are available for South-East Asian countries. Various studies assessing nutritional status have been carried out, particularly in India, but these have generally focused on undernutrition, have been for selected population groups, and have not used the WHO classification of obesity. As many countries in South-East Asia are presently undergoing the so-called “nutrition transition”, there is special need to collect good quality, nationally representative obesity prevalence data. The nutrition transition is associated with a shift in the structure of the diet, reduced physical activity and rapid increases in the prevalence of obesity (23).

3.7 Eastern Mediterranean Region

3.7.1 Secular trends in obesity

Good quality nationally representative secular trend data for countries in the Eastern Mediterranean Region have not been identified.

3.7.2 Current prevalence of obesity

With the exception of Saudi Arabia, data on the prevalence of adult obesity in the Eastern Mediterranean Region have not been well-documented at a national level. Various surveys have been conducted but these have tended to be only for specific population groups within a country, such as women attending an infertility clinic, and/or have not classified obesity as a BMI ≥ 30 . Nevertheless, the limited data available, a selection of which is shown in Table 3.4, indicate that the prevalence of adult obesity in countries in the Region is high, and women in particular are affected. In general, the prevalence of obesity among women is higher than that reported for women in most industrialized countries.

Table 3.4 Obesity prevalence (BMI ≥ 30) in selected Eastern Mediterranean countries

Country	Year	Ages (years)	Prevalence of obesity (%)		
			Men	Women	
Bahrain ^a	1991–92	20–65	9.5	30.3	
			6.5	11.2	
Cyprus ^b	1989/90	35–64	19	24	
Iran, Islamic Republic of (south) ^c	1993/94	20–74	2.5	7.7	
Kuwait ^d	1994	18+	32	44	
Saudi Arabia ^e	1990–93	15+			
			Total	16	24
			Urban	18	28
Rural	12	18			
United Arab Emirates ^f	1992	17+	16	38	

^a Reference 26

^b Reference 13

^c Reference 27

^d Reference 28

^e Reference 24

^f Reference 25

A nationally representative, cross-sectional survey was conducted between 1990 and 1993 to study the effects of sex, age and the regional distribution on the prevalence of overweight and obesity among 13 177 randomly selected adult Saudi subjects. The prevalence of obesity among the female subjects was several fold higher than the reported prevalence from more industrialized countries, and was higher than among male subjects for all regions of Saudi Arabia (24).

In the United Arab Emirates, obesity is recognized as a major public health problem which may play an important role in the increasing occurrence of other chronic diseases. Data from the National Nutrition Survey revealed that 38% of married women and 15.8% of married men were obese (25). In Bahrain, obesity was more common in urban than in rural areas, especially in women (26).

Finally, a recent study in the south of the Islamic Republic of Iran revealed that obesity is prevalent in the adult population, and is more frequent among women than men (27).

3.8 European Region

3.8.1 Secular trends in obesity

Although the most comprehensive data on the prevalence of obesity in Europe are from the WHO MONICA study (2), the 38 centres chosen across Europe are not necessarily representative of their host countries, and only data from the first cycle have so far been published. The best picture of secular trends in obesity prevalence among European countries should therefore be provided by data from national surveys. Population-level trend data on obesity prevalence in Europe are available for several countries including England, Finland, Germany, the Netherlands and Sweden. A selection of these are summarized in Table 3.5.

From Table 3.5 it can be seen that the prevalence of obesity has increased by about 10–40% in the majority of European countries in the past 10 years. The most dramatic increase has been observed in England, where it has more than doubled during this period (29). There is some evidence, however, that there has been less of an increase among women in recent years, at least in some Scandinavian countries (30).

Table 3.5 Trends in obesity (BMI \geq 30) in selected European countries

Country	Year	Ages (years)	Prevalence of obesity (%)	
			Men	Women
England ^a	1980	16–64	6.0	8.0
	1986/7		7	12
	1991		12.7	15.0
	1994 ^b		13.2	16.0
	1995 ^c		15.0	16.5
Finland ^d	1978/9	20–75	10	10
	1985/7		12	10
	1991/3		14	11
Former East Germany ^e	1985	25–65	13.7	22.2
	1989		13.4	20.6
	1992		20.5	26.8
Netherlands ^f	1987	20–59	6.0	8.5
	1988		6.3	7.6
	1989		6.2	7.4
	1990		7.4	9.0
	1991		7.5	8.8
	1992		7.5	9.3
	1993		7.1	9.1
	1994		8.8	9.4
	1995		8.4	8.3
Sweden ^g	1980/81	16–84	4.9	8.7 ¹
	1988/89		5.3	9.1 ¹

¹ Obesity defined as BMI > 28.6

^a Reference 31

^b Reference 32

^c Reference 29

^d Reference 33

^e Reference 34

^f Reference 35

^g Reference 36

3.8.2 Current prevalence of obesity

Obesity is relatively common in Europe, especially among women and in Southern and Eastern European countries. The average prevalence of obesity among European centres participating in the WHO MONICA study between 1983 and 1986 was about 15% in men and 22% in women, although there was great variability both within and between countries. The lowest prevalence was found in Göteborg, Sweden (men: 7%, women: 9%) and the highest prevalence in Kaunas, USSR (current Lithuania) (men: 22%, women: 45%).

The most recent data from individual national studies suggest that the prevalence of obesity in European countries is currently in the range of 10–20% in men and 10–25% in women (Table 3.6). In agreement with the MONICA data, the prevalence of obesity is generally higher in women than in men.

Table 3.6 Obesity prevalence (BMI \geq 30) in selected European countries

Country	Year	Ages (years)	Prevalence of obesity (%)	
			Men	Women
Czech Republic ^a	1988	20–65	16	20
England ^b	1995	16–64	15	16.5
Finland ^c	1991/3	20–75	14	11
Former West Germany ^d	1990	25–69	17	19
Former East Germany ^e	1992	25–69	21	27
Netherlands ^f	1995	20–59	8	8

^a Reference 37, 38

^b Reference 29

^c Reference 33

^d Reference 39

^e Reference 34

^f Reference 35

3.9 Western Pacific Region

3.9.1 Secular trends in obesity

Trend data on the prevalence of overweight and obesity in countries in the Western Pacific Region are available for Australia, Japan, Samoa and China. These are summarized in Table 3.7 and show increasing prevalence of obesity among Australians and Samoans. The Australian data are from three National Heart Foundation studies conducted in the six state capitals in 1980 and 1983, with two extra cities added in 1989 (40). Rural residents were not included.

Detailed analysis of data from the National Nutrition Survey in Japan conducted by the Japanese Ministry of Health and Welfare ($n = 5000$ per year) has shown that there has been a secular increase in obesity in both men and women during the last 20 years (1976 to 1993). The increase in obese men was about 2.4 times whereas the increase in obese women, which was restricted to the 20–29 year age group, was about 1.8 times (41).

Data from the 1989 and 1991 China Health and Nutrition Survey (CHNS) show an increase in the proportion of adult men, but not women, who are severely overweight (BMI \geq 27) and obese (BMI \geq 30) (42). This longitudinal survey, which is now under way, is considered to be representative of all provinces in China. As successive surveys are planned for every two years, the CHNS should prove a valuable source of data for documenting the secular trends in obesity for a country in economic transition. The 1993 survey has been completed and the data will be published shortly.

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Secular trends have also been observed in Samoa where there has been a marked increase in the prevalence of obesity between 1978 and 1991, especially among men living in rural areas. Obesity is not new to Pacific populations and has long been regarded by Polynesian and Micronesian societies as attractive and a symbol of high social status and prosperity (43). However, there is evidence that these traditional notions are being replaced by an image of small body size (44).

Table 3.7 Trends in obesity (BMI \geq 30) in selected Western Pacific countries

Country	BMI cut-off	Year	Ages (years)	Prevalence of obesity (%)	
				Men	Women
Australia ^a		1980	25-64	9.3	8.0
		1983		9.1	10.5
		1989		11.5	13.2
China ^b	27	1989	20-45	1.7	4.3
		1991		2.9	4.3
China ^c	30	1989	20-45	0.29	0.89
		1991		0.36	0.86
Japan ^d	26.4	1976	20+	7.1	12.3
		1982		8.4	12.3
		1987		10.3	12.6
		1993		11.8	13.0
Japan ^d	30	1976	20+	0.7	2.8
		1982		0.9	2.6
		1987		1.3	2.8
		1993		1.8	2.6
Samoa (urban) ^e	30	1978	25-69	38.8	59.1
		1991		58.4	76.8
Samoa (rural) ^e	30	1978	25-69	17.7	37.0
		1991		41.5	59.2

^a Reference 40

^b Reference 42

^c Reference 45

^d Reference 41

^e Reference 43

3.9.2 Current prevalence of obesity

Table 3.8 shows the most recent estimates of obesity rates in a selection of Western Pacific countries. The prevalence of obesity in the general population of both New Zealand and Australia appears to be in the range of 10–15%. Studies of Aborigines living in different regions of Australia are not consistent with this finding; depending on the degree of “westernization” of Aboriginal communities, they have either a much higher or a substantially lower prevalence of obesity than the general Australian population (46).

Interim data from the Japanese National Nutrition Survey show that the prevalence of obesity in Japan is around 2% in males and 3% in females. When a BMI cut-off point of 26.4 is used ($\geq 120\%$ of standard body weight (SBW)), the figures are around 12% and 13%, respectively. Various studies have also been conducted on specific population groups and centres within Japan (41).

The current prevalence of obesity in China is probably best documented by the 1992 third Nationwide Nutritional Survey (NNS III). This survey was conducted throughout urban and rural provinces, and collected data from a larger representative sample of men ($n = 14\,964$) and women ($n = 14\,590$) aged 20–45 than the CHNS cohort ($n = 5000$ approximately). Data from the NNS III show that obesity does exist in China, albeit at a low prevalence, is more common in women than in men (Table 3.8), and is more prevalent in urban than in rural areas. These findings are supported by a study in 11 478 randomly selected Chinese adults aged 40 years and older, although this study reported slightly higher rates than in the younger age group studied in the NNS III (47). A number of other data sets are available but these rarely use the WHO classification of obesity, are not age-standardized and tend not to be nationally representative.

The most striking feature of Table 3.8 is the extremely high age-standardized prevalence of obesity observed in the Pacific island populations of Melanesia, Polynesia and Micronesia. In urban Samoa, for example, the prevalence of obesity has been estimated to be over 75% of adult women and almost 60% of adult men. However, Swinburn et al. (48) recently concluded that Polynesians seem leaner than Caucasians at any given body size, and so the prevalence of obesity in Polynesian populations may not be quite as high as is currently estimated using Caucasian-derived classifications based on BMI. The prevalence in rural populations is also extremely high, but lower than in urban areas.

Among adults aged 18–60 years in Malaysia, a total of 4.7% males and 7.9% females were found to have a BMI > 30 . In the females, overweight and obesity problems were more serious in the Indian population; 17.1% of Indian women had a BMI > 30 compared to 8.8% of Malay and 4.3% of Chinese women. Among the Malay population, a considerably higher proportion of both men and women had a BMI > 30 in the urban areas (males: 5.6% urban, 1.8% rural; females: 8.8% urban, 2.6% rural) whereas the reverse was true for undernutrition; prevalence rates of undernutrition for males and females were 7% and 11% in urban areas and 11% and 14% in rural areas, respectively. Overall, overweight (BMI ≥ 25) was more prevalent than undernutrition in both urban and rural settings (49).

Table 3.8 Obesity prevalence (BMI \geq 30) in selected Western Pacific countries

Country	Year	Ages (years)	Prevalence of obesity (%)	
			Men	Women
Australia ^a	1989	25–64	11.5	13.2
China ^b	1992	20–45	1.20	1.64
Japan ^c	1993	20+	1.7	2.7
New Zealand ^d	1989	18–64	10	13
Melanesia ^e				
Papua New Guinea				
Coastal urban	1991	25–69	36.3	54.3
Coastal rural	1991	25–69	23.9	18.6
Highlands	1991	25–69	4.7	5.3
Micronesia ^e				
Nauru	1987	25–69	64.8	70.3
Polynesia ^e				
Samoa				
Urban	1991	25–69	58.4	76.8
Rural	1991	25–69	41.5	59.2

^a Reference 40

^b Reference 47

^c Reference 41

^d Reference 50

^e Reference 43

3.10 Body mass index distribution in adult populations

BMI distribution varies significantly according to the stage of transition of a country. As the proportion of the population with a low BMI decreases, there is an almost symmetrical increase in the proportion with a BMI above 25 (Figure 3.3). This indicates a tendency for a population-wide shift as socioeconomic conditions improve, with overweight replacing thinness.

In the first stages of transition, the wealthier sections of society show an increase in the proportion of people with a high BMI, whereas thinness remains the main concern among the less wealthy. Thus, in countries in the early stage of the transition, overweight can co-exist with underweight, presenting a double burden of disease.

The distribution of BMI tends to change again in the later phases of transition, with an increase in the prevalence of high BMI among the poor.

Figure 3.3 BMI distribution of various adult populations worldwide (both sexes)

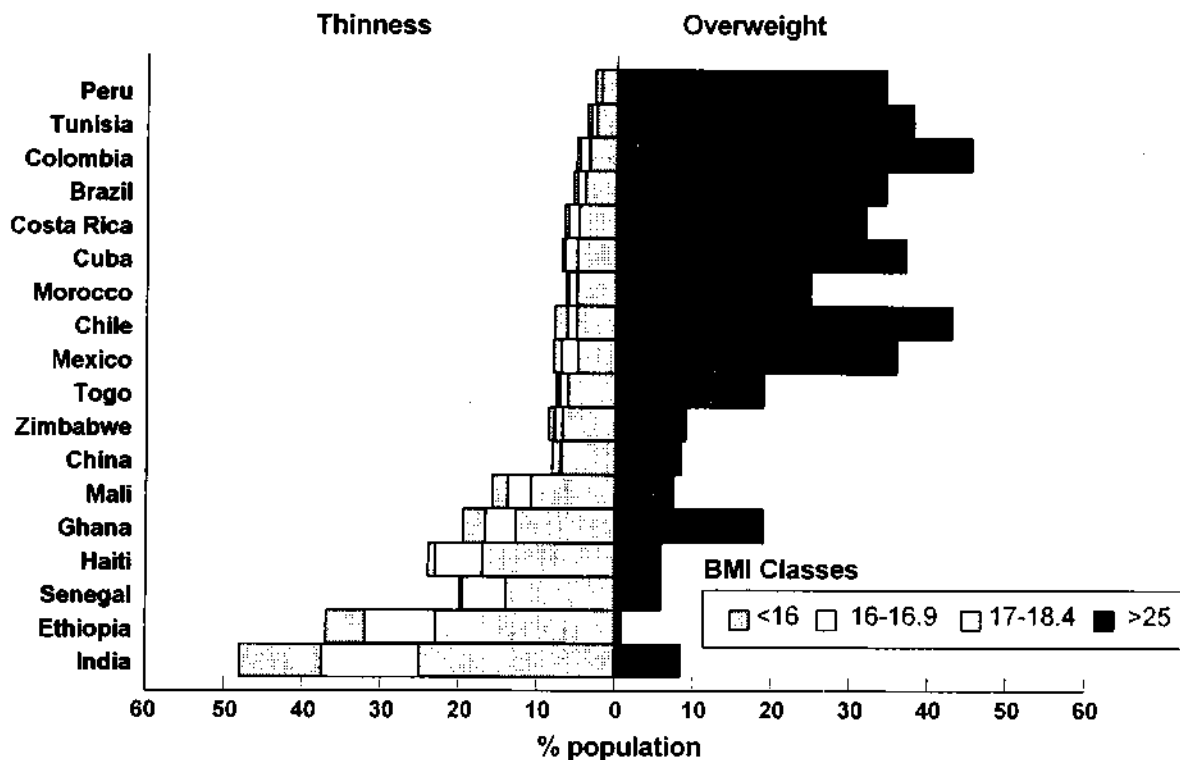


Figure 3.3 illustrates the tendency for a symmetrical increase in the proportion of a population with high BMI as the proportion of the population with low BMI decreases.

Source: Reference 11

3.11 Obesity during childhood and adolescence

The lack of consistency and agreement between different studies over the classification of obesity in children and adolescents has already been acknowledged (Chapter 2). For this reason, it is not yet possible to give an overview of the global prevalence of obesity for these younger age groups.

Nevertheless, whatever method is used to classify obesity, studies investigating obesity during childhood and adolescence have generally reported a high prevalence of obesity, and that rates are on the increase. In the USA, for example, the prevalence of overweight (defined by the 85th percentile of weight-for-height) among 5- to 24-year-olds from a biracial community of Louisiana (total $n = 11\,564$) increased approximately twofold between 1973 and 1994. Furthermore, the yearly increases in relative weight and obesity during the latter part of the study period (1983 through 1994) were approximately 50% greater than those between 1973 and 1982 (51). A similar trend has been observed in Japan; the frequency of obese schoolchildren ($>120\%$ SBW) aged 6–14 years increased from 5% to 10%, and that of extremely obese ($>140\%$ SBW) children from 1% to 2% during the 20 years between 1974 to 1993. The increase was most prominent in male students at ages of 9–11 years. Early obesity leads to an increased likelihood of obesity in later life, as well as an increased prevalence of obesity-related disorders. In the Japanese study, approximately one third of obese children grew into obese adults (52).

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Childhood obesity is not only confined to the industrialized countries, as high rates are already evident in some developing countries. The prevalence of obesity among schoolchildren aged 6–12 years in Thailand, as diagnosed by weight-for-height > 120% of the Bangkok reference, rose from 12.2% in 1991 to 15.6% in 1993 (53), and in a recent study of 6- to 18-year-old male schoolchildren in Saudi Arabia, the prevalence of obesity was found to be 15.8% (54).

The only integrated data currently available that give an overview of the global prevalence of obesity during childhood are those compiled by the WHO Programme of Nutrition (55,56). In the WHO analysis, children were classified as obese when they exceeded the NCHS median weight-for-height plus two standard deviations or z-scores. The reported prevalence of obese children for the age group 0–4.99 years is listed in Figure 3.4. It should be noted, however, that some children classified as obese under this system may actually have a higher relative weight due to stunting rather than as a result of excess adiposity. This is of particular significance in developing countries undergoing the nutrition transition, where a higher risk of obesity in stunted children has been described (57).

There is an urgent need to evaluate existing and future data sources concerning children and adolescents from across the world based on a standardized obesity classification system.

Figure 3.4 Prevalence of obese pre-school children in selected countries

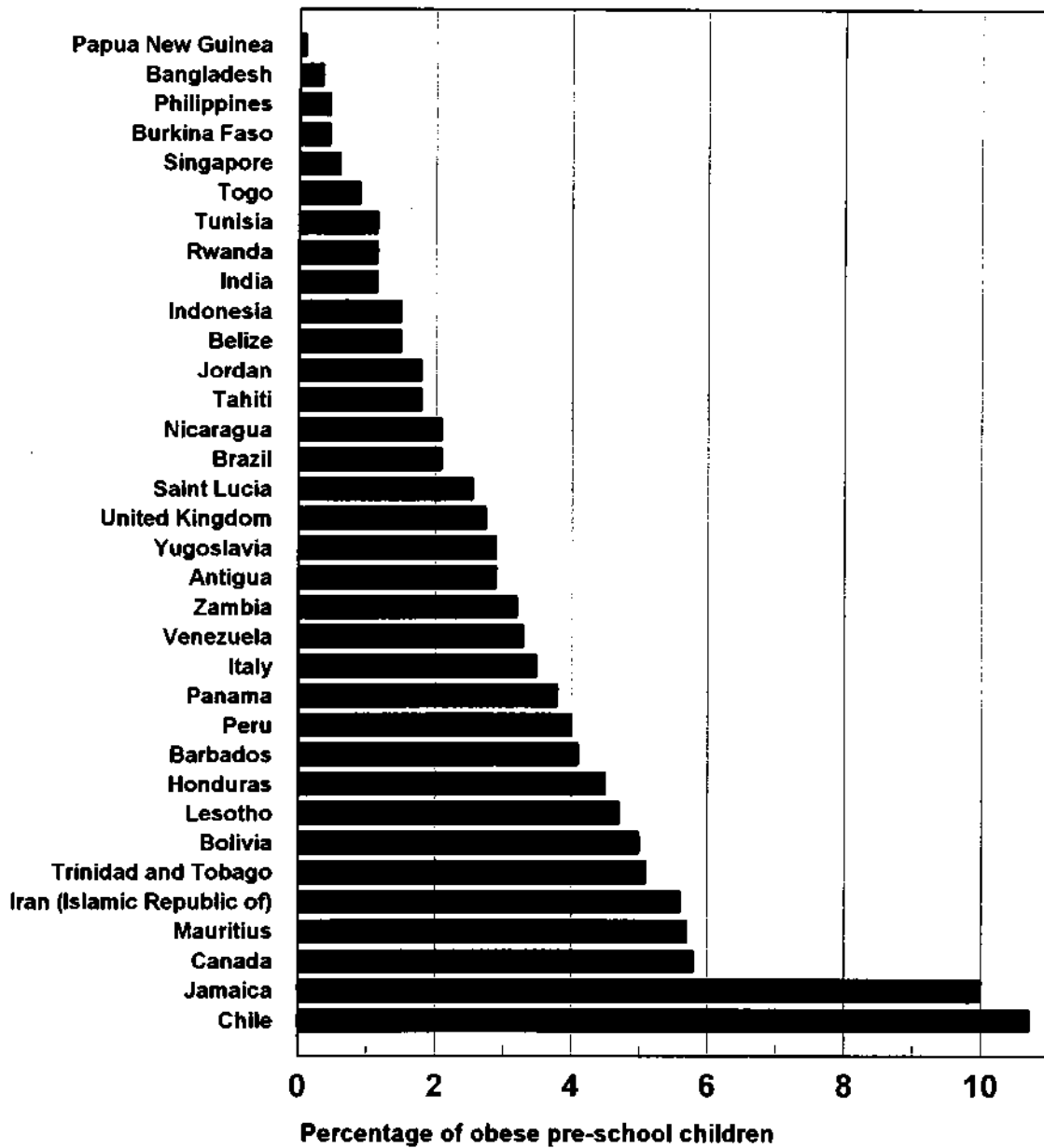


Figure 3.4 illustrates the prevalence of obesity among pre-school aged children (0–59 months) in selected countries. Obesity is defined as more than two standard deviations above the reference median weight-for-height (NCHS reference population).

Source: Reference 58

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Section B

**Establishing the true costs
of the problem of
overweight and obesity**

4 THE HEALTH CONSEQUENCES OF OVERWEIGHT AND OBESITY IN ADULTS AND CHILDREN

4.1 Introduction

The health consequences of obesity are many and varied, ranging from an increased risk of premature death to several non-fatal but debilitating complaints that impact on immediate quality of life. Obesity is also a major risk factor for NCDs such as NIDDM, CVD and cancer, and in many industrialized countries it is associated with various psychosocial consequences. Abdominal obesity is a particular area of concern as it is associated with elevated risks to health in comparison to a more peripheral fat distribution.

This chapter examines the health consequences of overweight and obesity in both adults and children. The effect of weight loss on these conditions is considered in Chapter 5.

Key issues covered are:

- The major health consequences associated with overweight and obesity are NIDDM, CHD, hypertension, gallbladder disease, psychosocial disturbances and certain types of cancer.
- Detailed relative risk data of the various health problems associated with obesity are limited to a few industrialized countries. This work has shown that the risks of suffering from NIDDM, gallbladder disease, dyslipidaemia, insulin resistance and sleep apnoea are greatly increased in the obese (relative risk much greater than 3). The risks of CHD and osteoarthritis are moderately increased (relative risk 2–3) and the risks of certain cancers, reproductive hormone abnormalities and low back pain are slightly increased (relative risk 1–2).
- When biases such as failure to control for cigarette smoking and unintentional weight loss are removed from the analysis of mortality data, there is an almost linear relationship between BMI and death. The longer the duration of obesity, the higher the risk. Severe obesity is associated with a 12-fold increase in mortality in 25- to 35-year-olds when compared to lean individuals. This highlights the importance of preventing weight gain throughout adult life.
- Excess abdominal fat is an independent predictor for NIDDM, CHD, hypertension, breast cancer and premature death.

- Weight gain during early adulthood, most of which is body fat, increases health risks.
- There are many non-fatal but debilitating conditions which affect the obese. These conditions are responsible for a much reduced quality of life in overweight patients and are often the primary reason for consultations with the health care system. Most of these conditions are improved with modest weight loss.
- The psychosocial consequences of obesity have important implications for disease management. These problems are compounded by the fact that health professionals often view obese individuals as weak-willed and unlikely to benefit from counselling.
- Obesity is associated with a number of conditions in children and adolescents, the most prevalent being the psychosocial consequences in adolescence and the persistence of obesity into adulthood.

4.2 Obesity as a risk factor for noncommunicable diseases

Although obesity should be considered as a disease in its own right, it can also be recognized as one of the key risk factors for other NCDs, such as NIDDM and CHD. The key risk factors for NCDs are smoking, obesity, high blood pressure and hypercholesterolaemia (1). The detrimental health consequences of obesity are influenced to a greater or lesser extent by body weight, the location of body fat, the magnitude of weight gain during adulthood, and a sedentary lifestyle (2).

As a chronic disease, obesity has many similarities to hypertension and hypercholesterolaemia. Figure 4.1 shows the positive relationship between relative risk of mortality and (a) BMI (as an index of obesity), (b) cholesterol, and (c) diastolic blood pressure. In the “moderate risk” category, which represents the ranges between widely accepted cut-off points for lower and higher risk levels, an increase in any of the three variables raises the risk of mortality sharply. The increase is even steeper in the “high risk” category implying greater individual risk. However, from a population perspective, the middle range is of most concern as this encompasses the greatest number of people (2).

Figure 4.1 Relationship of (a) BMI, (b) cholesterol and (c) diastolic blood pressure to relative risk of mortality

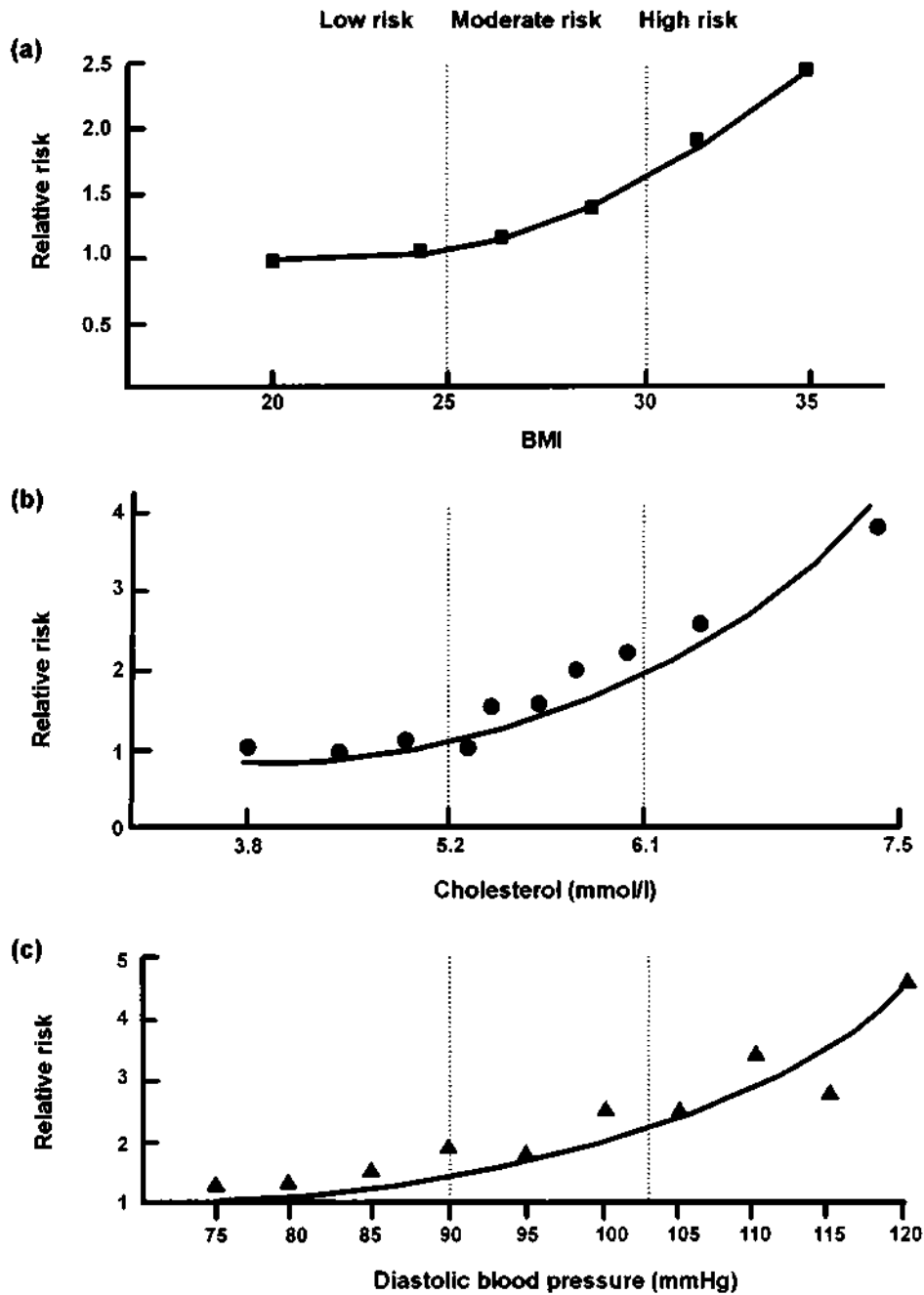


Figure 4.1 is based on data from papers by Stamler et al. (3,4) for the construction of the blood pressure and cholesterol plots, and from the Nurses' Health Study (5) for the BMI plot. This figure illustrates the similar continuous graded increases in relative risk of mortality as BMI, blood pressure and cholesterol increase. However, the RR rises more rapidly for cholesterol and blood pressure than it does for BMI. The rise in RR of mortality is notably steeper from BMI > 30, cholesterol > 6 mmol/l, and diastolic blood pressure > 100 mmHg.

4.3 Difficulties in evaluating the health consequences of obesity

Most of the evidence for associating health problems with obesity comes from prospective and cross-sectional population-based studies, although there is additional information from community interventions and clinical trials. Some confusion over the consequences of excess weight may arise because studies have used different BMI cut-off points for defining obesity, and because the presence of many medical conditions which are involved in the development of obesity may confound the effects of obesity itself.

Specific problems in evaluating the health consequences of obesity include:

- ***The recognized continuous relationship between gradations of excess weight and morbidity.*** This means that individuals who have gained weight but still lie within the normal range will be assigned to a normal weight category even though they may be at increased risk of co-morbidity due to excess weight gain.
- ***Present health status and health behaviours (such as smoking).*** These may have an impact on current weight and confuse its association with future health or even current well-being. For example, smoking is associated with a reduced BMI so the incidence of lung cancer caused by smoking appears to decrease with increased body weight.
- ***The duration and design of epidemiological studies.*** This will influence the strength of association between weight and morbidity. Long-term monitoring is required to identify the range of health consequences of obesity, whereas shorter duration studies with a large study population can be useful in identifying the major impact of obesity. Longer-term studies are also required where the outcome, e.g. cancer, is the result of a multistage process, with obesity having an effect on some but not necessarily all the stages. Most epidemiological studies measure prevalence rather than incidence, with the result that they are often confounded by survival bias and post-morbid modification of risk.
- ***The age group studied.*** This affects the relationship between obesity and health. For example, if the incidence of CHD in men is being analysed, obesity is a much more important predictor at younger rather than older ages. In contrast, if total mortality is the end point, then the reverse is true. The reason for this may be that obesity at an earlier age affects intervening risk factors much more strongly than in later life.

- **The use of initial weight criteria.** Most epidemiological studies use (by necessity) a static approach to classifying people's weight. People are generally placed in a weight group at the beginning of the study, and the association with future illness or events is based on that initial classification even if weight is gained or lost in the intervening time. This gives the impression that there is a risk-free zone up to a BMI 27 or 28, which is misleading: weight gain independent of BMI is an important risk factor as is the distribution of the fat gained.

4.4 Relative risk of obesity-associated health problems

The non-fatal but debilitating health problems associated with obesity include respiratory difficulties, chronic musculo-skeletal problems, skin problems and infertility.

The more life-threatening, chronic health problems associated with obesity fall into four main areas: (a) cardiovascular problems including hypertension, stroke and CHD; (b) conditions associated with insulin resistance, namely NIDDM; (c) certain types of cancers, mainly the hormonally related and large-bowel cancers; and (d) gallbladder disease.

It is important to recognize that ethnic differences will have a bearing on the prevalence of a particular disease; some minority populations in the USA have a higher prevalence of certain obesity-related diseases (particularly NIDDM but, for black Americans, also CVD, stroke and osteoarthritis of the knee) compared with the white population (6). Nevertheless, although the absolute prevalence may vary, the relative risk of any particular disease (whether the risk is slightly, moderately or greatly increased for an obese person compared to a lean person) is fairly consistent throughout the world (Table 4.1).

Table 4.1 Relative risk of health problems associated with obesity

Greatly Increased (relative risk much greater than 3)^a	Moderately increased (relative risk 2–3)^a	Slightly increased (relative risk 1–2)^a
NIDDM	CHD	Cancer (breast cancer in postmenopausal women, endometrial cancer, colon cancer)
Gallbladder disease	Hypertension	Reproductive hormone abnormalities
Dyslipidaemia	Osteoarthritis (knees)	Polycystic ovary syndrome
Insulin resistance	Hyperuricaemia and gout	Impaired fertility
Breathlessness		Low back pain due to obesity
Sleep apnoea		Increased anaesthetic risk
		Fetal defects associated with maternal obesity

^a All relative-risk estimates are approximate.

4.5 Intra-abdominal (central) fat accumulation and increased risk

Compared with subcutaneous adipose tissue, intra-abdominal adipose tissue has

- More cells per unit mass
- Higher blood flow
- More glucocorticoid (cortisol) receptors
- Probably more androgen (testosterone) receptors
- Greater catecholamine-induced lipolysis.

These structural differences make intra-abdominal adipose tissue more susceptible to both hormonal stimulation and changes in lipid accumulation and metabolism. Furthermore, intra-abdominal adipocytes are located upstream from the liver in the portal circulation. This means that there is a marked increase in the flux of non-esterified fatty acid (NEFA) to the liver via the portal blood in patients with abdominal obesity.

There is good evidence that abdominal obesity is important in the development of insulin resistance (see section 4.8.1), and in the metabolic syndrome (hyperinsulinaemia, dyslipidaemia, glucose intolerance, hypertension) which links obesity with CHD (see section 4.8.2). Some non-Caucasian populations appear to be especially susceptible to this type of syndrome, in which lifestyle changes may play a particularly important etiological role (7).

Premenopausal women have quantitatively more lipoprotein lipase (LPL) and higher LPL activity in gluteal and femoral subcutaneous regions which contain larger fat cells than men, but these differences disappear after the menopause (8). In contrast, men show minimal regional variations in LPL activity or fat cell size. These differences may explain the tendency for premenopausal women to deposit fat preferentially in lower body fat depots. The higher level of intra-abdominal adipose tissue found in men compared with pre-menopausal women seems to explain, in part, the greater prevalence of dyslipidaemia and CHD in men than in pre-menopausal women.

4.6 Obesity-related mortality

There has been much controversy about the relationship between obesity and mortality. While a number of studies have shown a U- or J-shaped association, with higher mortality rates at both the upper and lower weight range, some have shown a gradual increase in mortality with increasing weight, while others have reported no association at all.

Many studies relating obesity and mortality have included biases in their design that have led to a systematic under-estimation of the impact of obesity on premature mortality. These include the failure to control for cigarette smoking (producing an artificially high mortality in leaner subjects),

inappropriate control for conditions such as hypertension and hyperglycaemia which were assumed to be confounding factors but are to a large degree the effects of obesity (hence some factorial analyses distort the true association between obesity and mortality), failure to control for weight loss associated with illness (leading to an underestimate of the impact of obesity on mortality), and failure to standardize for age (9,10).

The Nurses' Health Study (5) in the USA found that when biases are removed from the analysis, an almost linear, continuous relationship between BMI and mortality is found, with no specific lower threshold (see Figure 4.2). This is not surprising, given the largely linear relationship between body weight and conditions such as CHD, hypertension and NIDDM when BMI increases from 20 to 30 (11-13). Similar results and conclusions have been reached by others (10,14) but a follow-up study of NHANES has continued to show U-shaped curves after control of the pertinent variables. Nevertheless, whatever the shape of the curve, it appears that the lowest mortality risk is associated with a BMI between 18 and 25. This conclusion was reached by the American Institute of Nutrition (15) after analysing numerous studies of obesity and mortality risk.

Although the increase in mortality rate with increased relative body weight is steeper for both men and women under age 50, the effect of overweight on mortality persists well into the ninth decade of life. The increased risk observed in younger people is linked to the duration of overweight and implies that particular efforts should be made to control the weight of younger adults (14,16,17).

Figure 4.2 Relation between BMI and relative risk of overall mortality

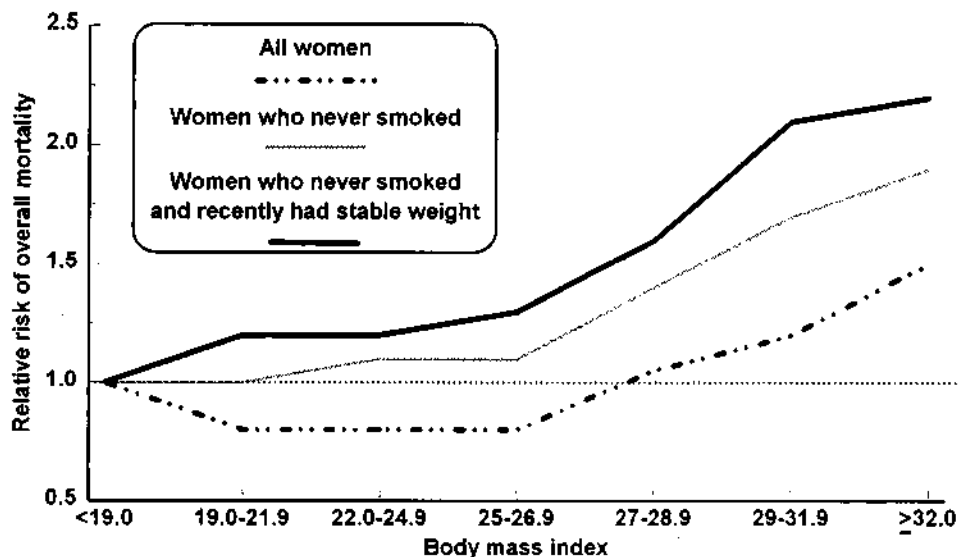


Figure 4.2. The apparent excess risks associated with leanness in the all-women study (4726 deaths during the 16-year follow-up) were found to be artefacts as they were eliminated after accounting for smoking (leaving 1499 deaths) and subclinical disease (leaving 531 deaths). By excluding former and current smokers, women with BMI < 22 were found to have the lowest mortality. When disease-related health loss was also accounted for, the leanest women (BMI < 19) had the lowest mortality. This analysis is based on professional middle-aged women and so may not be representative of all population groups (5).

Finally, if obesity is associated with increased risk of premature mortality, it is paradoxical that obesity rates are escalating in many countries at a time when overall death rates in these same countries are actually falling. This can be explained primarily by the declining death rates which have resulted from reductions in CVD. The reductions in CVD can, in turn, be attributed to two main elements: falling rates of smoking and improved dietary quality from higher intakes of fruits and vegetables, reduced salt intake, and reduced intake of saturated fat and cholesterol. The incidence of NIDDM, however, is increasing and there is evidence that this is a consequence of the rise in the prevalence of obesity. The rise in the prevalence of obesity cannot be explained in total by reduced rates of smoking, which appear to be associated with only small increases in the average body weight of the population. The expected effect over time is an increase in mean BMI worldwide which will lead to a further increase in NIDDM, gallbladder disease, hypertension and atherosclerosis. Although these effects may not be reflected in overall mortality rate figures, they will surely lead to a higher frequency of the debilitating and prolonged morbidity from NCDs which require expensive health care.

4.7 Chronic diseases associated with obesity

4.7.1 Cardiovascular disease and hypertension

Cardiovascular disease

CVD encompasses CHD, stroke and peripheral vascular disease. CHD and stroke account for a large proportion of deaths in men and women in most industrialized countries, and the incidence is increasing in developing countries.

Obesity predisposes an individual to a number of cardiovascular risk factors including hypertension, raised cholesterol and impaired glucose tolerance. However, longer-term prospective data now suggest that obesity is also important as an independent risk factor for CHD-related morbidity and mortality (18). The Framingham Heart Study ranked body weight as the third most important predictor of CHD among males, after age and dyslipidaemia (19). Similarly, in women, a large-scale prospective study in the USA found a positive correlation between BMI and the risk of developing CHD. Weight gain substantially increased this risk (20). These findings are consistent with data from other countries. A 15-year follow-up study of sixteen thousand men and women in Eastern Finland concluded that obesity is an independent risk factor for CHD mortality in men and contributes to the risk of CHD in women (21).

On the basis of the Framingham Heart Study and other studies, it can be concluded that the degree of overweight is related to the rate of development of CVD (22). The CHD risk associated with obesity is more acute in younger age groups and it is higher in people with abdominal obesity than in those with excess fat around the hips and thighs (23) (see section 4.5). In addition, mortality from CHD has been shown to be increased in the overweight, even at body weights only 10% above the average (24).

Interestingly, Asian Indians have the highest rates of CHD of any ethnic group studied, despite the fact that nearly half of this group are life-long vegetarians. CHD occurs at an early age and generally follows a severe and progressive course. Although the prevalence of classic risk factors is relatively low, there is a substantial prevalence in this population of high triglyceride and low high-density lipoprotein (HDL) cholesterol levels, high lipoprotein (a) levels, hyperinsulinaemia and abdominal obesity (25). These appear to constitute weight-related risk factors in this population which, in particular, may reflect the central distribution of body fat.

Hypertension and stroke

The association between hypertension and obesity is well documented. Both systolic and diastolic blood pressure increase with BMI, and obese individuals are at higher risk of developing hypertension than are lean subjects (4,26). Community-wide surveys in the USA (NHANES II) show that the prevalence of hypertension in overweight adults is 2.9-fold higher than that for non-overweight adults (27). The risk in those aged 20 to 44 years is 5.6 times greater than that in those aged 45 to 74 years (28), which in turn is two-fold higher than for non-overweight adults (29). The risk of developing hypertension increases with the duration of obesity, especially in women, and weight reduction leads to a fall in blood pressure (see section 5.3.1).

A 7.5 mmHg difference in diastolic pressure within the range 70–110 mmHg is accompanied by a 29% difference in CHD risk and a 46% difference in the risk of stroke, irrespective of sex, age group or ethnicity (30).

While many large studies have examined the relationship between obesity and CHD, there has not been the same emphasis on stroke. One study which examined 1163 non-smoking men aged between 55 and 68 years in Honolulu found that elevated BMI was associated with increased risk of thromboembolic stroke (31). However, preliminary results obtained from women in the Swedish Obese Subjects (SOS) study were not conclusive (32). Other studies found that a high WHR rather than BMI was the risk factor associated with stroke and that this relationship was stronger than for any other anthropometric variable tested (33,34). It was suggested that a lifelong history of obesity rather than weight in middle age is more important for assessing risk of stroke (13).

The reason for the association between increased body weight and elevated blood pressure is unclear. One possibility is that obesity is associated with higher circulating levels of insulin (a consequence of insulin resistance) which enhances renal retention of sodium, resulting in increased blood pressure (35). As exercise is known to improve insulin sensitivity, this would perhaps explain why exercise also reduces blood pressure. Other possible etiological factors include elevated plasma renin or enhanced catecholamine activity (36).

4.7.2 Certain types of cancer

A number of studies have found a positive association between overweight and the incidence of cancer, particularly those cancers which are hormone-dependent or gastrointestinal (Table 4.2).

Table 4.2 Cancers with a higher incidence reported in obese persons

Hormone dependent	Gastrointestinal/hepatic/renal
Endometrial	Colorectal
Ovarian	Gallbladder
Breast	Pancreatic
Cervical	Liver
Prostate	Renal

Greater risks of endometrial, ovarian, cervical and post-menopausal breast cancer have been documented for obese women while there is some evidence for an increased risk of prostate cancer among obese men. The increased incidence of these cancers in the obese is more prominent for those with abdominal fat distribution at lower degrees of obesity, and is thought to be a direct consequence of hormonal changes (37). The incidences of gastrointestinal cancers, such as colorectal and gallbladder cancer, have also been reported to be positively associated with body weight or obesity in some but not all studies, and renal cell cancer has consistently been associated with overweight and obesity, especially in women (38,39).

In addition to overall obesity, intra-abdominal fat distribution and adult weight gain have been independently associated with increased risk of breast cancer. For example, it has been reported that an increase in intra-abdominal fat accumulation increases the risk of postmenopausal breast cancer, independent of relative weight and particularly when there is a family history of the disease. Furthermore, weight gain during adulthood has consistently been associated with increased risk of breast cancer, even in cohort studies that showed no association between baseline relative weight and subsequent risk of breast cancer (40,41).

One major prospective study which followed 750 000 men and women for 12 years found that the mortality ratio for any cancer was 1.33 and 1.55 for obese men and women, respectively (42). It should be noted, however, that in some studies of gastrointestinal and breast cancer, it has been difficult to determine whether it is the effect of dietary components that promote weight gain, such as a high dietary fat content, or the effect of obesity *per se* that is the important association. This area needs to be studied further.

High levels of physical activity have been shown to decrease the risk of colon cancer in men in the majority of studies, and in women in half the studies. However, the effect of physical activity on rectal cancer was not significant for most cases. Breast cancer and cancers of the reproductive system were less prevalent in women who had been athletes at college (43) compared with less active women. NHANES I data indicate that a high level of non-recreational activity is important in reducing the risk of cancer, but that recreational exercise has little relation to cancer with the exception of prostate cancer (44).

4.7.3 Diabetes mellitus

A positive association between obesity and the risk of developing NIDDM has been repeatedly observed in both cross-sectional (45–57) and prospective studies (53,58–66). The consistency of the association across populations despite different measures of fatness and criteria for diagnosing NIDDM reflects the strength of the relationship. When women aged 30 to 55 years were monitored for 14 years, the additional risk of developing NIDDM for those who were obese was over 40 times greater than for women who remained slim (BMI < 22) (61). The risk of NIDDM increases continuously with BMI and decreases with weight loss. Analysis of data from two recent large prospective studies illustrates the impact of overweight and obesity on NIDDM; about 64% of male and 74% of female cases of NIDDM could theoretically have been prevented if no person would have had a BMI over 25 (61,66).

Detailed analyses of the relationship between obesity and NIDDM have identified certain characteristics of obese persons that further increase the risk of developing NIDDM, even after controlling for age, smoking and family history of NIDDM. These include obesity during childhood and adolescence, progressive weight gain from 18 years, and intra-abdominal fat accumulation. In particular, intra-abdominal fat accumulation has been implicated as an independent risk factor for NIDDM in a variety of populations and ethnic groups around the world and, in some studies, has been an even stronger predictor of NIDDM than overall fatness (52,56,60).

Lack of physical activity and an unhealthy diet, both of which are associated with a lifestyle in industrialized countries, are also important modifiable risk factors for overweight and obesity. The prevalence of NIDDM is 2- to 4-fold higher in the least physically active individuals compared to the most physically active (67,68), an effect which is independent of the level of body mass, and a healthy diet can reverse the deterioration in glucose tolerance commonly seen with diets high in fat and low in carbohydrate and fibre (69).

Intra-abdominal fat accumulation, as well as obesity *per se*, are also associated with an increase in the risk of pre-diabetic conditions such as impaired glucose tolerance and insulin resistance. The benefits of weight loss in controlling NIDDM are discussed in Chapter 5.

4.7.4 Gallbladder disease

In the general population, gallstones are more common in women and the elderly. However, obesity is a risk factor for gallstones in all age groups and in both men and women, gallstones occur three to four times more often in obese compared with non-obese individuals, and the risk is even greater when excess fat is located around the abdomen. The relative risk of gallstones increases with BMI and data from the Nurses' Health Study suggest that even moderate overweight may increase the risk (70).

Supersaturation of the bile and reduced motility of the gallbladder, both of which are present in the obese, are thought to be underlying factors for gallstone formation. Furthermore, since gallstones enhance the propensity to gallbladder inflammation, acute and chronic cholecystitis is

also more common in the obese. Biliary colic and acute pancreatitis are other potential complications from gallstones.

Paradoxically, gallstones are also a common clinical problem in those losing weight (see Chapter 5).

4.8 Endocrine and metabolic disturbances associated with obesity

4.8.1 Endocrine disturbances

Recent research has emphasized that adipocytes (fat cells) are more than just fat depots. They also function as endocrine cells, producing many local and distantly acting hormones, and as target cells for a great many hormones. Altered hormonal patterns have been observed in obese patients, especially in those with a central fat distribution (71,72). Common hormonal abnormalities associated with intra-abdominal fat accumulation are outlined in Table 4.3.

Table 4.3 Common hormonal abnormalities associated with intra-abdominal fat accumulation

-
- insulin resistance and increased insulin secretion
 - increased free testosterone and free androstenedione levels associated with decreased sex hormone binding globulin (SHBG) in women
 - decreased progesterone levels in women
 - decreased testosterone levels in men
 - increased cortisol production
 - decreased growth hormone levels
-

Insulin resistance

There is a large range of sensitivity to insulin among any group of people, but insulin resistance is very frequently associated with obesity. It is especially pronounced with intra-abdominal fat accumulation and, since abdominal fat mass increases with increasing adiposity, it is universally found in very severe obesity (BMI \geq 40).

It has been suggested by some investigators that insulin resistance is an adaptation to obesity which tends to limit further weight deposition (73). In insulin resistance the oxidation of fat tends to be favoured over its storage and over the oxidation of glucose. Thus, if an individual who is gaining weight continues to eat the same amount, there will come a time at which net fat oxidation will, through insulin resistance, equal dietary fat intake and the subject will be in fat balance. A corollary, shown to be true in prospective studies (74), is that the more insulin resistant among

a group of normal body-weight individuals will be protected from future weight gain. However, this is only a theory and is by no means universally accepted (75). Also insulin resistance is clearly maladaptive in terms of risk of CVD and other chronic diseases.

Insulin normally inhibits fat mobilization from adipose tissue and activates LPL. These are both metabolic processes which become insulin resistant in obesity. However, in contrast to the direct regulation of insulin secretion by plasma glucose concentration, the regulation of insulin secretion by fat metabolites is relatively weak. This means that over-secretion of insulin (due to insulin resistance) compensates for defects in glucose metabolism to a much greater degree than for defects in lipid metabolism. Disruption of the postprandial response by insulin leads to the dyslipidaemic state (section 4.7.3). Differential insulin resistance of specific organs or tissues may account for regional fat accumulation. For instance, relative insulin sensitivity of intra-abdominal fat is thought to be required for central fat accumulation.

Physical activity improves insulin sensitivity through weight reduction and increased cardio-respiratory fitness. However, it also improves insulin sensitivity independently of these factors (76).

Hormones affecting reproductive function

Significant associations are seen in reproductive endocrinology between excess body fat, particularly abdominal obesity, and ovulatory dysfunction, hyperandrogenism and hormone sensitive carcinomas (77). Distinct changes in circulating sex hormones appear to underlie these abnormalities. Androstenedione and testosterone concentrations are commonly elevated whereas SHBG is reduced. The plasma ratio of oestrone to oestradiol is also increased in obesity. A decrease in SHBG is associated with an increased clearance of free testosterone and oestradiol, resulting in a disturbed sex hormone equilibrium.

Moderate obesity is frequently associated with polycystic ovary syndrome which is the most common endocrine disorder of reproduction (78). Obesity contributes to or worsens, and weight loss generally improves, the associated hormonal abnormalities and menstrual function of obese women with polycystic ovary syndrome (79).

Adrenocortical function

Obese subjects have a normal circulating plasma cortisol concentration with a normal circadian rhythm, and normal urinary free cortisol. However, cortisol production rate is increased in obesity to compensate for an accelerated rate of cortisol breakdown (80,81). Cortisol inhibits the antilipolytic effect of insulin in human adipocytes, an effect which may normally be particularly pronounced in abdominal fat because it contains a high density of glucocorticoid receptors. This mechanism may contribute to the manifestations of insulin resistance (82).

Studies have shown that patients with central fat distribution have increased cortisol secretion, probably because they have increased activity of the hypothalamic pituitary axis (HPA). Stress, alcohol and smoking have all been shown to stimulate the activity of the HPA (83).

4.8.2 Metabolic disturbances

Dyslipidaemia

Obese individuals are frequently characterized by a dyslipidaemic state in which plasma triglycerides are raised, HDL cholesterol concentrations are reduced and low density lipoprotein apo B (LDL-apoB) levels are raised. This metabolic profile is most often seen in obese patients with a high accumulation of intra-abdominal fat and has consistently been related to an increased risk of CHD (84).

Excessive intra-abdominal fat accumulation is also associated with a greater proportion of small, dense low density lipoprotein (LDL) particles. These small dense LDL particles may be caused by metabolic disturbances related to the accompanying high triglyceride or low HDL levels. Indeed, the hypertriglyceridaemic state may be the combined result of an increased production and a reduced break-down of triglyceride-rich lipoproteins (84,85). This process results in lower HDL cholesterol levels and favours the triglyceride enrichment of LDL. The triglyceride-rich LDL are then enzymically degraded by hepatic lipase to produce small, dense LDL particles. An elevated proportion of these particles cannot be identified simply by the measurement of total or LDL cholesterol levels because these cholesterol levels are frequently in the normal range in such patients. A better indicator of small, dense LDL particle levels is an elevated ratio of LDL-apoB to LDL cholesterol.

Impaired fat tolerance (i.e. prolonged and/or exaggerated lipaemia following fat ingestion) is now also recognized as a component both of insulin resistance and of the atherogenic lipoprotein phenotype (86).

The metabolic syndrome and obesity

The common occurrence of obesity with other CVD risk factors is well recognized. This clustering has been given several labels, including Syndrome X and the Insulin Resistance syndrome, but the term Metabolic Syndrome is now favoured. There is no internationally agreed definition for the syndrome, but a suitable working definition would include two or more of the following:

- impaired glucose tolerance
- elevated blood pressure
- hypertriglyceridaemia and low HDL cholesterol
- insulin resistance
- central obesity

Insulin resistance and/or hyperinsulinaemia have been put forward as the underlying cause(s) linking these conditions (87). Each individual component of the syndrome conveys increased CVD risk but, in combination, they interact to increase risk in a synergistic fashion.

Epidemiological studies confirm that the Metabolic Syndrome occurs commonly in a wide variety of ethnic groups including Europids, Afro-Americans, Mexican Americans, Asian Indians and Chinese, Australian Aborigines, Polynesians and Micronesians. However, there is some evidence that the patterns of risk factors observed vary between and even within populations (88).

4.9 Debilitating health problems associated with obesity

Before chronic, life-threatening illness develops, overweight and obese patients usually present to primary care physicians with a range of conditions that affect their immediate quality of life. These are often mechanical in origin, due to the bearing of large amounts of excess weight. Though often perceived as less serious, they are none the less debilitating, sometimes painful, and may be costly in terms of consuming health resources and absence from work. Sleep apnoea can have fatal consequences associated with cardiac arrhythmias. Unfortunately, there are few data available on the economic costs of these conditions attributable to obesity.

4.9.1 Osteoarthritis and gout

Obesity is associated with the development of osteoarthritis and gout and, in obese middle-aged women at or after menopause, pain at the medial aspect of the knee (*adiposa dolorosa juxta-articularis*). Possible mechanisms underlying the relationship between obesity and osteoarthritis include mechanical stresses related to increased load of obesity, metabolic changes associated with increased fatness, and dietary elements that are related to the development of obesity. The strength of the data supports mechanical damage above other causes. The increased risk of gout associated with obesity may be related to the accompanying hyperuricaemia although central fat distribution may also be involved, particularly in women (89–91).

4.9.2 Pulmonary diseases

Obesity impairs respiratory function and structure, leading to physiological and pathophysiological impairments. The work of breathing is increased in obesity. This is mainly due to the extreme stiffness of the thoracic cage, resulting from the accumulation of adipose tissue in and around the ribs, abdomen and diaphragm (92). Hypoxaemia (low levels of oxygen in the blood) is common, partly due to a low relaxation volume causing ventilation to occur at volumes below the closing volume (93,94). Hypoxaemia is exacerbated when lying down due to a lower functional residual capacity (95).

Sleep apnoea occurs in more than 10% of men and women with a BMI of 30 or above, and 65% to 75% of individuals with obstructive sleep apnoea are obese. In one study, sleep apnoea occurred in 77% of those with a BMI above 40. In addition to BMI, however, obstructive sleep apnoea is related to central obesity and to neck size, probably due to narrowing of the upper airway when lying down. The nocturnal disruption of sleep is associated with daytime somnolence, hypercapnia, morning headaches, pulmonary hypertension and, eventually, right ventricular failure (96,97).

4.10 Psychosocial problems associated with obesity

The SOS study found that the proportion of individuals on pensions for medical reasons was over twice as high in obese patients as in population controls. Psychological problems in the obese (with women more affected than men) were found to be worst in those who were also chronically ill or injured, for example, suffering from rheumatoid arthritis, cancer or spinal injury (98). Therefore, the true social as well as economic costs of the non-fatal health consequences of obesity could be seriously underestimated.

Other data examining the psychosocial aspects of obesity are mainly from the USA. These reflect cultural differences, which may be irrelevant to other countries, especially as there appear to be ethnic differences in attitudes towards obesity. Black women in the USA, for instance, are two to three times more likely than white women to be obese, yet black women have been shown to experience less social pressure about their weight, initiate dieting later in life, and be significantly less likely to diet at each developmental milestone (99). Nevertheless, as the prevalence of obesity rises in developing countries, and with populations being increasingly affected by cultural values more common in industrialized countries, these findings are likely to become increasingly applicable to the overall health profile of the obese.

It is important to note that the mechanisms leading to impaired psychological health are different from those of physical illness. The untoward psychosocial consequences of obesity are not the inevitable consequences of obesity but derive from culture-bound values by which people view body fat as "unhealthy" and "ugly". Stunkard and Sobal (100) noted that:

... obesity does not create a psychological burden. Obesity is a physical state. People create the psychological burden.

4.10.1 Social bias, prejudice and discrimination

There can be little doubt that obesity is a highly stigmatized physical state in many industrialized countries, both in terms of the perceived undesirable bodily appearance and in terms of its purported significance in designating individuals as having a number of character defects. Even children as young as 6 years of age describe the silhouette of an obese child as "lazy, dirty, stupid, ugly, liar and cheat" more often than drawings of other body shapes (101).

Obese people have to contend with discrimination. Analyses of large surveys have shown that, compared with their non-obese peers, those who are obese are less likely to complete as many years at school, to be accepted into prestigious schools or to enter desirable professions. Furthermore, overweight young women in the UK and the USA earn significantly less than healthy women who are not overweight, or than those with other chronic health problems (102).

The negative stereotypes and attitudes of health professionals (including doctors, medical students, nutritionists and nurses) towards obesity are of particular importance. Awareness of these negative attitudes may make the obese reluctant to seek medical assistance for their condition (103). Doctors may be less interested in managing overweight patients, believing that

the obese are weak-willed and less likely to benefit from counselling. The prescription of lipid-lowering agents by British general practitioners was noted to be less likely for overweight people (as for smokers), with doctors explicitly stating that this was their policy (104). Although the potential to improve the stereotypes and attitudes of health professionals has received little attention so far, Wiese et al. (105) found that educational intervention was associated with a more positive attitude to the obese among first-year medical students.

4.10.2 Psychological effects

Research in this area has produced inconclusive results. Scores on standard psychological tests have been shown to differ little if at all between obese and non-obese people; and the evaluation of self-esteem in obese children and adolescents has not revealed a consistent picture (106). However, the implication that obesity has no psychological consequences conflicts with reports from overweight individuals and with a consistent literature showing strong cultural bias and negative attitudes towards obese people. Friedman and Brownell (107) suggest that the "paradox" can be explained by the manner in which these first-generation studies have been conducted and that new studies should be set up to examine risk factors within the obese population.

4.10.3 Body shape dissatisfaction

Many obese people have an altered body image, viewing their bodies in a negative light and believing that others wish to exclude them from social interaction. The disturbance occurs most often in young women of middle and upper SES, groups in which obesity is less prevalent and in persons who have been obese since childhood.

4.10.4 Eating disorders

Binge-eating disorder (BED) is a recognized psychological condition (108) that occurs with increased frequency among obese persons and accounts for approximately 30% of obese people asking for medical help. In particular, the disorder is associated with severe obesity, a high frequency of weight cycling, and pronounced psychiatric co-morbidity. The main characteristic of BED is uncontrolled binge-eating episodes which predominantly take place in the early evening or at night.

Obese binge-eaters have worse moods and more psychopathology than obese people who do not binge-eat, and are more likely to drop out of weight-control programmes based on behaviour modification. Although binge-eaters may regain weight faster than non-binge eaters, both short- and long-term weight loss among binge eaters and non-binge eaters appear to be quite similar (109).

The night-eating syndrome (NES) is characterized by the consumption of at least 25%, although more recent opinion suggests up to 50%, of total energy intake after the evening meal. This syndrome seems to be more common in morbidly obese patients and is related to sleep

disturbances such as sleep apnoea. It is thought to be due to alterations in the circadian rhythm, encompassing food intake and mood.

Nocturnal sleep-related disorder is a newly delineated night-eating pattern characterized by eating on arousal from sleep. It may be a variant of BED but its relationship with NES is unclear.

There is no clear evidence that these eating disorders are the primary cause of people gaining weight. Some suggest that the increasing incidence of eating disorders is associated with psychological pressure to slim. The failure to find these disorders in societies where obesity is not stigmatized is strong evidence for the cultural basis of its inception. Once established in a patient, however, it is a serious medical condition which limits the chances of therapeutic success.

4.11 Health consequences of overweight and obesity in childhood and adolescence

4.11.1 Prevalence of the health consequences of overweight and obesity

Obesity-related symptoms in children and adolescents include psychosocial problems, increased CVD risk factors, abnormal glucose metabolism, hepatic-gastrointestinal disturbances, sleep apnoea and orthopaedic complications (Table 4.4).

The most significant long-term consequence of childhood obesity is its persistence into adulthood, along with all the associated health risks. The persistence of obesity is more likely when its onset is in late childhood or adolescence and when the obesity is severe (110,111). Overweight in adolescence has also been shown to be significantly associated with long-term mortality and morbidity (112).

Table 4.4 Health consequences of childhood obesity

High Prevalence	Intermediate Prevalence	Low Prevalence
Faster growth	Hepatic steatosis	Orthopaedic complications
Psychosocial	Abnormal glucose metabolism	Sleep apnoea
Persistence into adulthood (for late onset and severe obesity)	Persistence into adulthood (depending on age of onset and severity)	Polycystic ovary syndrome
Dyslipidaemia		Pseudotumour cerebri
Elevated blood pressure		Cholelithiasis
		Hypertension

4.11.2 Psychosocial effects

The most common consequence of obesity in children in industrialized countries is psychosocial functioning. Pre-adolescent children associate an overweight body related to shape (or silhouette) with poor social functioning, impaired academic success and reduced fitness and health (113). However there is little evidence to suggest that self-esteem is significantly affected in obese young children (106,114).

On reaching the teenage years, however, cross-sectional studies consistently show an inverse relationship between body weight and both overall self-esteem and body image (106). Adolescence is a period when a marked self-awareness of body shape and physical appearance develops. Therefore, it is perhaps not surprising that the pervasive, negative social messages associated with obesity in many communities have an impact at this stage. Overweight in adolescence may also be associated with later social and economic problems. A large prospective study from the USA has shown that women who were overweight in late adolescence and early adulthood are more likely to have lower family incomes, higher rates of poverty and lower rates of marriage than women with other forms of chronic physical disability but who were not overweight in childhood (102).

4.11.3 Cardiovascular risk factors

Dyslipidaemia, hypertension and insulin resistance are frequently present in obese children (115,116), and dyslipidaemia appears to be related to increased abdominal fat distribution (117). Caprio and co-workers (118) suggest that insulin resistance in children may also be associated with abdominal obesity.

Although NIDDM is very rare, it accounts for one-third of all new cases of diabetes seen in some institutions in the USA (119).

Serum lipid and lipoprotein levels, blood pressure and plasma insulin all follow from childhood into young adulthood, with obesity at baseline being a significant predictor of adult values (120,121).

4.11.4 Hepatic and gastric complications

Hepatic complications in obese children have been reported, particularly hepatic steatosis which is characterized by raised serum transaminase levels (122). Abnormal liver enzymes may be associated with cholelithiasis but this condition is rare in children and adolescents.

Gastro-oesophageal reflux and gastro-emptying disturbances which affect a minority of obese children may be a consequence of raised intra-abdominal pressure due to increased abdominal fat.

4.11.5 Orthopaedic complications

It is well-documented that obese children can suffer from orthopaedic complications. The more serious of these include slipped capital femoral epiphysis (123) and Blount's disease (a bone deformity resulting from overgrowth of the tibia) (124,125), while the more minor abnormalities include knock knee (genu valgum) and increased susceptibility to ankle sprains.

4.11.6 Other complications of childhood obesity

Obstructive sleep apnoea is another complication of childhood obesity which has been reported to cause hypoventilation and even sudden death in severe cases (126,127).

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5 THE HEALTH BENEFITS AND RISKS OF WEIGHT LOSS

5.1 Introduction

While the effects of obesity on the functioning, health and quality of life of obese subjects has been studied in great detail, the impact of weight loss on these same measures is less well-documented. Short-term studies have demonstrated clear benefits from a modest weight loss on most of the associated consequences of obesity but there are very few well-designed studies which examine the benefits of long-term weight loss.

The health benefits and risks of weight loss and maintenance are examined in this chapter with particular reference to mortality, general health, obesity-related co-morbidities including chronic diseases, endocrine and metabolic disturbances, and psychosocial functioning. It also notes two distinct hazards of weight loss, namely gallstones and reduced bone density, and considers the issue of weight cycling. The chapter concludes with a brief section on the effects of weight loss in obese children and adolescents.

Some key points include:

- Well-designed studies which examine the effects of long-term (>2 years) weight loss are few in number. Specific difficulties associated with such studies include the difficulty of maintaining long-term weight loss, and the need to separate intentional from unintentional weight loss.
- Intentional weight loss results in marked improvements in NIDDM, dyslipidaemia, hypertension, cardiovascular risk and ovarian function. It also improves measures of general health including breathlessness, sleep quality, sleep apnoea, back and joint pain, and osteoarthritis.
- Hazards of weight loss are limited to an increased incidence of gallstones (when weight loss is rapid) and possibly a reduction of bone density.

5.2 Difficulties in examining the effects of weight loss

Specific problems in evaluating the benefits of long-term weight loss include:

- The difficulty of maintaining weight loss in adults over a long period.
- Whether weight cycling is taken into account, and how it is defined when assessing a study's outcome.
- The important separation of "unintentional" weight loss, which may reflect underlying disease, from "intentional" weight loss.

- The separation of the beneficial effects of weight loss *per se* from those of the dietary and physical activity change necessary to achieve that weight loss.

The distinction between “intentional” and “unintentional” weight loss is of major importance for studies investigating the relationships between weight loss and morbidity or mortality. If weight loss occurs unintentionally as a result of underlying disease or serious illness, then the association between weight loss and morbidity or mortality will be artificially increased. A bias from misclassification may also occur if studies include only two weight measurements, especially if weight loss is temporary and due to a minor acute illness. For this reason it is recommended that a minimum of three—and preferably more—weight measurements are carried out throughout the period of study.

5.3 Weight loss and general health

5.3.1 Modest weight loss

Data from a number of studies have shown that modest weight loss (as defined by a weight loss of up to 10%) improves glycaemic control, reduces blood pressure and reduces cholesterol levels (1). Modest weight loss also improves lung function and breathlessness, reduces the frequency of sleep apnoea, improves sleep quality, and reduces daytime somnolence. However, the degree of improvement often depends on the length of time that the condition has been present. Modest weight loss will also alleviate osteoarthritis, depending on the degree of structural damage, as well as back and joint pain.

5.3.2 Extensive weight loss

Following surgically banded gastroplasty, severely obese patients who lose 20–30 kg in weight, at a rate of 4.5 kg per month for the first 6 months, gain substantial health benefits. They show a marked fall in blood lipids within the first two years of follow-up, and the conditions of 43% of hypertensive patients and 69% of NIDDM patients are improved. Furthermore, at a population level, the incidences of hypertension, hyperlipidaemia and NIDDM are reduced to about a sixth of those seen in obese patients who maintain their excess weight (2,3).

5.4 Weight loss and mortality

Unfortunately, most studies investigating weight loss and mortality have not controlled for unintentional weight loss nor for cigarette smoking. In one large study of overweight white women in the USA where these variables were evaluated, intentional weight loss consistently reduced mortality in women with obesity-related co-morbidities such as NIDDM or CVD. However, the effects in women without co-morbidities were inconsistent with the association between intentional weight loss and reduction in mortality. Thus the benefit of intentional weight loss was best seen in those with a poorer health status (4).

In a randomized controlled dietary intervention trial of post-infarct patients in India, the effect of the dietary intervention on cardiac mortality was greatest among those patients who had also lost around 10% of their body weight (5). Thus, more longer-term, well-controlled studies are clearly needed to define accurately the benefits of weight loss on mortality.

5.5 The impact of weight loss on chronic disease, and on endocrine and metabolic disturbances

5.5.1 Cardiovascular disease and hypertension

A number of cardiovascular risk factors related to blood clotting (haemostatic, rheological and fibrinolytic) have been associated with overweight (6–8). In particular, coagulation factors VII and X, which are directly associated with BMI, are involved with thrombosis (9) and increased risk of myocardial infarction (10). Weight loss in overweight subjects has been shown to reduce red blood cell aggregation and to improve fibrinolytic capacity.

Weight loss induces a fall in blood pressure. Short trials lasting a few weeks show that each 1% reduction in body weight leads, on average, to a fall of 1 mmHg systolic and 2 mmHg diastolic pressure (11–14). Marked falls in blood pressure can occur with very low energy diets, although modest dietary restrictions are also beneficial. Anti-hypertensive drug therapy, reducing a high alcohol intake, lowering salt intake (15, 16), and altering the fatty acid content of the diet in favour of one with less saturated fat (17, 18) all further reduce blood pressure independent of weight loss. It is estimated that a 10 kg weight loss can produce a fall of 10 mmHg systolic and a fall of 20 mmHg diastolic blood pressure (19).

Longer trials, with a 10-year follow-up of patients identified originally as mildly hypertensive, show that positive dietary change together with smoking cessation and an increase in isotonic exercise (e.g. running) reduce both body weight and blood pressure. These levels can be sustained for 10 years and significantly limit the need for drug therapy (12).

5.5.2 Diabetes mellitus and insulin resistance

Studies of weight loss in NIDDM patients have consistently shown that weight reduction of 10% to 20% in obese individuals with NIDDM results in marked improvements in glycaemic control and insulin sensitivity. These improvements can last from 1 to 3 years even if the weight is subsequently regained. Of the 75% of newly diagnosed NIDDM patients who are overweight, a 15% to 20% weight loss in the first year after diagnosis seems to reverse the elevated mortality risk of NIDDM (20). However, not all Type II diabetic patients respond to weight loss with metabolic improvements; the loss of abdominal adipose tissue may be more important for improvements in diabetic control than loss of weight *per se*.

Hyperglycaemia frequently decreases as soon as a low energy diet is initiated, suggesting that dietary energy restriction has a beneficial effect independent of weight loss. Exercise training also improves glucose tolerance and insulin sensitivity independent of weight loss. The American

Diabetes Association (21) recommends that aerobic exercise be performed at moderate intensity for 20 to 45 minutes, three days per week. However, although epidemiological studies have emphasized the value of vigorous activity, mainly because it is easy to assess, the total level of energy expenditure may be the important factor in limiting NIDDM rather than the periods of intense physical activity (22).

5.5.3 Dyslipidaemia

The adverse changes in blood lipids associated with obesity, namely high triacylglycerides, high cholesterol and low HDL cholesterol can also be expected to return towards normal after modest weight loss. LDL cholesterol has been estimated to reduce by 1% for every 1 kg lost (23).

A 10 kg weight loss can produce a fall of 10% in total cholesterol levels, 15% decrease in LDL levels, 30% decrease in triacylglycerides and an increase of 8% in HDL cholesterol (19). Also, it has been found that serum triglyceride and HDL cholesterol levels show the most favourable changes after weight loss in those with a high waist-hip ratio (24).

5.5.4 Ovarian function

Overweight and obese women with hirsutism and polycystic ovaries show improved insulin sensitivity and ovarian function when more than 5% weight loss occurs. In addition, some women with amenorrhoea may be restored to normal menstrual function after weight loss.

5.6 Weight loss and psychosocial functioning

Most studies on the quality of life of obese patients before and after weight loss have been conducted on patients following surgery for obesity. These studies all indicate dramatic improvements in the overall quality of life. The SOS study in Sweden (25), for example, showed significant improvements in social interaction, anxiety, depression and mental well-being which were sustained two years after weight loss surgery. Although it is unclear whether these improvements will be seen with modest weight loss following non-surgical intervention, Klem et al. (26) recently reported that reduced-obese subjects indicated substantial improvements in quality of life following weight loss. While this is based on self-reports of individuals who were maintaining weight losses for periods of over one year, it provides additional evidence of the benefits of weight loss.

Dieting is often perceived to have untoward psychological effects including depression, nervousness and irritability. However, studies have shown that weight loss is associated with a decrease in depression score, particularly when weight loss is achieved by behaviour modification (27,28).

A dramatic example of how severely overweight persons perceive their disorder has been provided by Rand and MacGregor (29,30) who studied a group of severely obese patients before and after losing weight as a result of gastric surgery. Prior to surgery, all patients felt unattractive

and a great majority felt that people talked about them behind their backs at work. They also felt that they had been discriminated against when applying for jobs and treated disrespectfully by the medical profession. Having achieved a weight loss of 50 kg, all the patients said they would prefer to be deaf, dyslexic, diabetic, or to suffer very bad heart disease or acne than to return to their previous weight. Given a hypothetical choice, all preferred to be of normal weight than to have “a couple of million dollars”, a choice that they made within less than one second!

5.7 Hazards of weight loss

Weight loss resulting from crash dieting may result in acute attacks of gout. However, for intentional and controlled weight loss resulting from medical intervention, only two distinct hazards of weight loss have emerged from a variety of prospective studies:

- **Gallbladder disease.** Women who lose 4–10 kg have a 44% increased risk of clinically relevant gallstone disease, and greater weight loss enhances this risk. Mobilization of cholesterol from adipose tissue stores is increased during weight loss and so the risk of supersaturation of bile is greater at this time than when weight is stable. Pre-menopausal women are at particular risk because of an oestrogen-induced enhanced biliary secretion of cholesterol.
- **Reduced bone density.** Bone density is typically increased in obese patients and reduced after weight loss. In white women, weight loss beginning at age 50 was found to increase the risk of hip fracture (31). Whether there is restitution of bone mass with weight regain following slimming, however, is uncertain; Compston et al. (32) found this to be the case whereas Avenell et al. (33) did not. There is little information about the impact of weight cycling on bone density.

It should also be noted that weight loss in some societies, where overweight and obesity are seen as a sign of affluence, may be interpreted as a sign of personal misfortune.

5.8 Weight cycling and stable weights

Weight cycling refers to the repeated loss and regain of weight which can occur as a result of recurrent dieting. However, there is no standard definition of weight cycling and so comparison between different studies is difficult (34).

It has been suggested that weight cycling is associated with negative health outcomes, makes future weight loss more difficult and results in a decrease in lean-to-fat tissue ratio (35). However, supporting evidence is conflicting; weight variability was associated with increased risk of CVD and all-cause mortality in men, particularly in those who continued to smoke, but the association between weight change and death was not seen in the heaviest men (36). Recently in the USA, the National Task Force on the Prevention and Treatment of Obesity (37) concluded

that the evidence available at the time was not sufficiently compelling to override the potential benefits of moderate weight loss in obese patients.

5.9 The effects of weight loss in obese children and adolescents

Weight loss of just 3% significantly decreased blood pressure in obese adolescents, and blood pressure was further improved if exercise was added to the weight-loss programme (38). A weight loss of nearly 16% in obese children resulted in a parallel decrease in serum triacylglycerides and plasma insulin in the first year, and HDL cholesterol increased. These changes remained stable in the second year of the study; after 5 years, weight loss was still 13% below the initial weight, peripheral hyperinsulinaemia was reduced, and HDL cholesterol remained higher (39).

The symptoms of hepatic steatosis in obese children eventually disappear when excess weight is lost (40).

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6 THE ECONOMIC COSTS OF OVERWEIGHT AND OBESITY

6.1 Introduction

The economic costs of overweight and obesity are important issues to health care providers and policy-makers alike. To date, there have been only a few attempts to quantify the economic burden of obesity-related morbidity and mortality. This is in marked contrast to smoking and alcohol consumption, where a large number of international studies have been undertaken to identify the magnitude of the economic burden they impose on the community. In addition, few studies have assessed the relative cost-effectiveness of alternative interventions aimed at either preventing or treating obesity.

This chapter reviews the limited evidence available on the economics of overweight and obesity. It first summarizes the use and limitations of cost-of-illness studies on obesity-related disease and outlines the basic steps in undertaking such a study. A brief overview of the few studies from different countries that have estimated the economic costs of obesity is then given; key findings as well as limitations of the methodologies employed are highlighted. The next section reviews the cost-effectiveness of alternative interventions aimed at either preventing or treating obesity. Finally, the implications from current understanding of the economics of obesity for public policy decision-making are highlighted and priorities for future research in this area are discussed.

Some key points include:

- The economic cost of obesity has three main components: “direct costs” (the costs to the individual and service providers associated with treating obesity itself), “intangible costs” (the opportunity cost of the disease to the individual because of the impact on the individual of ill health), and “indirect costs” (usually measured as lost production due to work-related absenteeism and premature death).
- The economic impact of obesity-related disease is usually estimated from cost-of-illness studies. These are useful in the development of public health policy but their limitations need to be recognized; intangible costs and many of the direct costs of disease management and prevention, especially those outside the formal health care system, tend to be ignored. A number of studies have therefore focused on the impact of obesity on broader economic issues such as attainment of social class and the frequency of long-term sick leave.
- The economic costs of obesity have been assessed from several developed countries and range from 2% to 7% of total health care costs. These are conservative estimates from variable criteria yet they are a clear indication that obesity represents one of the largest expenditures in national health care budgets.

- Although there have not been any studies of the economic impact of obesity in developing countries, the escalating economic burden of adult NCDs in such countries has already been recognized by a number of international agencies such as the WHO and the World Bank. The real costs of therapy in developing countries exceed those in developed countries because of the extra burden associated with importing expensive equipment with scarce foreign exchange as well as the need for specialized training of staff. In view of the existing burdens of endemic deficiency and infectious diseases, obesity prevention is therefore not only crucial, but it is also the only sensible approach to planning public health policies in developing countries.
- Preliminary data suggest that a large proportion of the economic costs of obesity can be saved by efficient prevention or intervention strategies against obesity.

6.2 Cost-of-illness studies

“Cost-of-illness” or “disease-costing” is a technique used to estimate the cost impact of disease on a community. The economic costs of a health condition such as obesity can be defined as:

- **Direct costs:** the cost to the *community* because of the diversion of resources to the diagnosis and treatment of diseases directly related to obesity, as well as the cost of obesity treatment itself (including the cost of providing health care services to patients and their families, and the cost of service providers).
- **Intangible costs:** the cost to the *individual* because of the impact of obesity on quality of life generally, and on health specifically.
- **Indirect costs:** the forgone welfare and economic benefits to other members of society through a reduction in goods and services produced, in other words, the impact of the reduced quality of life on the productive potential available to the rest of society. Usually these costs are measured as lost production due to work-related absenteeism and premature death.

Most cost-of-illness studies focus on measuring direct and indirect costs, while less attention is given to the more difficult task of quantifying the intangible costs.

6.2.1 Uses of cost-of-illness studies

These cost-of-illness studies are useful in the development of public policy because they can:

- Identify and analyse how resources are currently being allocated between different types of costs, services and diseases.
- Help to identify potential improvements in health status, in the case of a specific disease, that can be achieved through the application of effective preventive programmes, or to identify a risk factor for a disease. A knowledge of the incidence, prevalence, consequential utilization of health services and costs can allow a calculation of the potential savings to a community that can be achieved through effective preventive programmes, which may (or may not) be in excess of the costs of prevention.
- Assist health planners to make comparisons between the relative economic burden of different diseases that may assist in setting priorities for prevention, if taken together with information on the costs and effectiveness of prevention strategies.
- Provide data on the cost side of the cost-effectiveness equation for a later economic appraisal.
- Provide an advocacy role through highlighting to policy-makers and politicians the magnitude of the health problem in a language that they understand, i.e. money.

6.2.2 Limitations of cost-of-illness studies

The major criticism of cost-of-illness studies is that they are subject to misuse. A cost-of-illness study may indicate that a disease is costly to treat. It may also suggest that a disease has a high social cost relative to other diseases or social problems, implying that society would be relatively better off without that disease. While this is obviously true it does not imply that a higher priority should be given to treating that high-cost disease. Treatment (or prevention) may be relatively ineffective or expensive, so priority-setting should be based on the relative cost-effectiveness of interventions and not on the cost of the disease alone. This criticism is best set out by Davey and Leeder (1):

... Instead of answering the question, "Where should I put the next health care dollar to achieve the greatest health gain?" cost-of-illness studies provide information only about the burden of illness. They concentrate on cost and say nothing about the effectiveness of treatment and value for money invested.

Some economists have argued that while cost-of-illness studies do not indicate where resources should be allocated in the short term, they do indicate where the greatest potential health improvements and health care resource savings could be made if effective interventions were available.

A further criticism concerns their focus on direct health care costs and indirect costs of lost production, with less emphasis on the primary burden of disease, premature death and reduced quality of life. Because these latter intangible costs are less easy to measure in monetary terms, they tend to be ignored. Diseases associated with high health care costs but relatively low morbidity and mortality (such as dental disease) may therefore be identified as imposing a far greater burden than other diseases posing high costs in terms of premature death and loss in quality of life but low direct health care costs (such as youth suicide).

The definition of health care incorporated in cost-of-illness studies tends to be narrow, ignoring many of the direct costs of disease management and prevention, especially those outside the formal health care system. This is particularly the case with obesity, as the highest direct cost category is most likely to be personal expenditure on weight-loss programmes incurred by overweight and obese individuals. The impact of the narrow range of direct costs included in studies is likely to be inconsistent across disease types and risk factors.

6.2.3 Steps in undertaking a cost-of-illness study

Using standardized WHO criteria for overweight (i.e. BMI 25–29.9) and obesity (BMI \geq 30), a number of basic steps need to be followed for a cost-of-illness study on obesity-related disease:

- Identify those diseases related to overweight and obesity.
- Quantify the relationship between obesity and the associated disease morbidity and mortality using standard criteria (i.e. the population-attributable fractions (PAFs)).
- Identify the relevant economic cost categories to be estimated.
- Quantify the total costs associated with diet-related disease.
- Use the PAFs to apportion that share of total costs directly attributed to overweight and obesity.
- Undertake sensitivity analysis of key epidemiological and economic parameters (or assumptions) to provide a range of cost estimates.

Population-attributable fractions

The epidemiological statistic needed to quantify the direct relationship between a risk factor of interest and disease (and thus quantify its associated economic costs) is the PAF. This has been defined as the proportion of total events (e.g. deaths or morbidity) in a population that could be prevented if a particular risk factor (e.g. obesity) could be eliminated.

The PAF reflects the overall impact of morbidity and mortality from a factor (e.g. obesity) in the specified population. It can be interpreted from an etiological viewpoint (causal outcomes attributed to a particular risk factor) or from a prevention viewpoint (the maximum number of events that could be prevented). Many epidemiologists use the concept of "preventable proportion" as a useful generalization of the concept of PAF.

For the situation where only one category of exposure (e.g. obese or non-obese) is presented, the formula for the PAF is:

$$\text{PAF} = \frac{p(\text{RR}-1)}{1 + p(\text{RR}-1)}$$

where p = prevalence of risk factor (e.g. obesity) in a population

RR = relative risk = incidence of disease in an obese person (I_e) divided by the incidence of disease in a non-obese person (I_o) = I_e/I_o

The PAF can be presented as a fraction or as a percentage. Thus a PAF of 0.73 means that 73% of the incidence of the disease could be eliminated by removal of the risk factor (or conversely, that the risk factor contributes to 73% of the incidence of the disease).

A number of epidemiological studies have assessed the relative risk of specific diseases associated with excess body weight. Most have used BMI as the risk factor, with only a few studies quantifying the risk of disease relating to body fat distribution (e.g. by the use of the waist circumference). Studies have shown a positive relationship between BMI and the development of CHD (2-4), hypertension (5), stroke (6), NIDDM (2), gallbladder disease (7), sleep apnoea (8), and a number of cancers including breast cancer (9,10) and colon cancer (11). In addition, further studies have shown a relationship between excess body weight and obstetrical complications in women (12), progression of osteoarthritis (13), and rheumatoid arthritis (14).

There is need for a comprehensive systematic review (e.g. a meta-analysis) to provide a clearer understanding of the relationships between excess weight and the diseases found in existing studies. Once these data are available, relative risk estimates can be combined with country-specific overweight and obesity prevalence data to determine PAFs for use in cost-of-illness studies.

6.2.4 The Disability Adjusted Life Year

An alternative tool to the cost-of-illness study for economic evaluation of the diverse consequences of obesity and overweight is the Disability Adjusted Life Year (DALY). This measure provides estimates of the burden imposed by death and disability due to any health disorder and enables comparison of populations in different geographical and social settings. The proportion of chronic diseases which are attributable to overweight and obesity, and the costs of their management, vary across populations and between social classes within populations. It is of value, therefore, to utilize a common combined measure of the loss of life expectancy and prolonged morbidity in national, regional and global estimates of the economic effects of overweight and obesity.

Obesity and overweight, analogous to tobacco, contribute to several NCDs. Thus, the total DALY loss attributable to obesity and overweight would represent the attributable fraction of the total loss of DALYs due to NCDs associated with excess body weight. Such estimates have been derived for tobacco, and facilitate national and regional comparisons. A similar effort should be made to generate valid global estimates of the health effects of obesity and overweight.

6.3 Review of international estimates of the cost of obesity

6.3.1 Studies in developed countries

At present, only a few studies have estimated the economic burden of obesity-related diseases. Some of the data available from developed countries are reviewed below and summarized in Table 6.1. The scope and methodology of the various studies vary considerably in terms of the diseases costed, the definition of obesity, the cost categories used and the epidemiological assumptions on the relationship between obesity and disease. This makes it difficult to compare costs across countries and to extrapolate the results from one country to another. The limited data available suggest that anywhere between 2% and 7% of total health care expenditure in a country may be directly attributable to overweight and/or obesity.

Table 6.1 Economic costs of obesity

Country	Year	Study	Obesity definition (BMI)	Estimated direct costs	% National health care costs
Australia	1989/90	NHMRC (15)	> 30	AUD\$ 464 million	> 2%
France	1992	Lévy et al. (16)	≥ 27	FF 12 000 million	2%
Netherlands	1981–89	Seidell & Deerenberg (17)	> 25	Guilders 1000 million	4%
USA	1990	Wolf & Colditz (18)	> 29	US\$ 458 000 million	6.8%

United States

The first national study undertaken on the economic cost of obesity was by Colditz (19) in the USA. Diseases included in the cost estimate were NIDDM, CVD, hypertension, gallbladder disease, and colon and post-menopausal breast cancer. Obesity was defined as a BMI greater than 29. Total costs attributable to obesity, calculated according to both direct and indirect costs for 1986, were estimated to be US\$ 39 300 million. This figure represented 5.5% of the overall costs of illness for the USA in that year. The PAFs used for particular diseases were NIDDM 0.57; CVD 0.19; hypertension 0.26; breast cancer 0.06; and colon cancer 0.02. However, original estimates of relative risks by Colditz are currently being revised by a number of groups to bring PAF and economic cost estimates into line with agreed classification criteria for overweight and obesity.

Colditz's original estimate should be considered conservative because estimates for many obesity-related diseases and for several relevant economic cost categories were excluded. Colditz points out that the addition of musculo-skeletal disorders to his estimate would have raised the figure to US\$ 56 300 million, or 7.8% of the cost of illness for the USA in 1986.

Wolf and Colditz (18) later revised the estimate of economic costs of obesity in the USA by extending the range of obesity-associated diseases included in the analysis and updating their calculations. They estimated that the total cost of obesity in 1990 was US\$ 68 800 million, of which US\$ 45 800 million was due to the direct cost of obesity-associated disease. The remaining US\$ 23 000 million was an estimate of the indirect costs of obesity due to lost productivity (about US\$ 4000 million or 52 591 480 annual work-days) and premature mortality from diseases associated with obesity (about US\$ 19 000 million). These figures should still be considered conservative.

Australia

The National Health and Medical Research Council (NHMRC) replicated the 1992 Colditz study using the same obesity-related diseases and the same estimates of relative risk, but applying Australian estimates of obesity prevalence (based on a BMI of greater than 30). The NHMRC estimated the direct cost of obesity to be AUD\$ 464 million (1989–1990), with indirect costs amounting to an additional AUD\$ 272 million. CHD and hypertension combined accounted for approximately 60% of the total economic costs of obesity. With respect to hypertension, the largest "cost categories" were medical services and pharmaceuticals, whereas CHD hospital costs and the indirect costs associated with premature mortality were the most significant (15).

Within the total cost-of-obesity estimate, the NHMRC also estimated the costs of obesity treatment within the formal health care system in Australia. This figure accounted for approximately 10% of the total economic cost of obesity.

The estimate provided by the NHMRC should be considered conservative for the same reasons as the Colditz study in the USA. Of interest is the fact that, while the costs of obesity treatment within the health care sector amounted to less than AUD\$ 80 million, a 1992 survey by the

Consumer Advocacy and Financial Counselling Association of Victoria (20) estimated that 300 000 consumers purchased a weight-loss programme in Australia each year from a variety of weight-loss centres, and estimated that the industry turnover was in excess of AUD\$ 500 million per annum. This highlights the fact that a substantial proportion of the economic cost of obesity lies outside the formal health care sector.

The Netherlands

The excess use of medical care and associated costs due to obesity in the Netherlands was estimated using the data on 58 000 participants in the Health Interview Surveys from 1981 to 1989 (17). The health care costs included reported consultations of general practitioners and medical specialists, hospital admissions and the use of prescribed drugs. Obese (BMI ≥ 30) and overweight (BMI 25–30) persons had an increased likelihood of having consulted a general practitioner. The total general practitioner costs attributable to obesity/overweight were equivalent to 3% to 4% of that country's total general practitioner expenditure. For hospitalizations, the fraction attributable to obesity was 3% and for overweight 2%. The excess use of medications by obese and overweight people was, however, very conspicuous. Compared to non-obese people, obese persons were five times more likely to use diuretics and 2.5 times more likely to take drugs for CVD. It was estimated from these data that the direct costs of overweight and obesity were about 4% of the total health care costs in the Netherlands. This is of the same order of magnitude as the health care costs attributable to all forms of cancer.

While the study did not include all potential cost categories relevant to obesity, it was the first cost estimate to include the impact of overweight, with this category accounting for about 48% of the total costs of excess weight gain.

France

To estimate the direct costs of obesity-related diseases in France for 1992, Lévy et al. (16) identified the direct costs of personal health care, the need for hospital care, physician services and drugs for diseases with well-established relationship with obesity. These included NIDDM, hypertension, hyperlipidaemia, CHD, stroke, venous thromboembolism, osteoarthritis of the knee, gallbladder disease and certain cancers. The proportion of these diseases attributable to obesity (defined by the cut-off point of BMI ≥ 27) ranged from about 25% for hypertension and stroke to about 3% for breast cancer. The direct costs of obesity were estimated to be almost 12 000 million French francs, which corresponded to approximately 2% of the total health care expenditure in France in 1992. Costs of hypertension represented 53% of the total direct costs of obesity.

Finland

The impact of obesity on several indicators of health care utilization was assessed among 10 000 adult Finns in the National Survey on Health and Social Security in 1987 (21). The costs of medicines, physician consultations and hospital inpatient stay increased with increasing BMI. The excess health care utilization was mainly due to an increased need for medication, the cost of

which increased by about 120% when BMI increased from 25 to 40. It was estimated on the basis of these data that if all Finns were of normal weight the annual savings would be of the same order of magnitude as if all smokers in Finland were to stop smoking permanently.

6.3.2 Studies on the broader economic issues

A number of studies have used methods other than cost-of-illness studies to describe the economic impact of obesity-related diseases. These studies have generally focused on the influence of obesity on either social class attainment, or on the impact on pension and disability payments.

It is important to note that indirect costs of disease relate to the loss of worker productivity (e.g. worker absenteeism, staff turnover and reduced worker productivity) as a result of obesity-related morbidity, together with lost earnings due to premature death from an obesity-related disease. A common misconception among health professionals is that sickness, unemployment and other social welfare benefits should be included as the indirect costs of diseases. Economists do not include these in cost-of-illness studies as they are viewed as a transfer payment from the tax-paying population to the benefit recipient. There is further debate among health economists about whether to include indirect costs in a study and how to measure these costs reliably.

Attainment of social class

Cross-sectional studies in many affluent societies show an inverse relationship between educational level and the prevalence of obesity. However, in addition to indications that low SES leads to obesity, there are also indications that obesity may lead to poorer SES. Obese subjects may also have economic disadvantages such as higher premiums for life insurance policies.

One study of Danish draftees showed that, after adjustment for parental social class, level of education and intelligence, fewer obese men attained relatively high social class compared to non-obese men (22). Similarly, a prospective study in young women from the USA showed that those who were obese were less likely to marry, had fewer years of schooling, and had lower income compared to non-obese women (23). These results are supported by a number of other prospective studies showing that obese young adults do not reach the same social class level as their non-obese peers. Although such data should be interpreted with caution, it has been suggested that societal discrimination may limit the socioeconomic potential in the obese.

Frequency of long-term sick leave

In the SOS study (24) in Sweden the frequency of long-term sick-leave (over 6 months) was reported to be 1.4 and 2.4 times higher in obese men and women, respectively, compared with the general Swedish population. Similarly, the rate of premature disability pensions was reported to be increased 1.5- to 2.8-fold among the SOS. The total loss of productivity due to obesity was estimated to be about 7% of the total cost of losses of productivity due to sick-leave and disability pensions in Sweden.

Premature work disability

In a large prospective Finnish study (25), obesity was associated with a two-fold increased risk of premature work disability in men and a 1.5-fold greater risk in women. Most of the premature pensions attributable to obesity were due to cardiovascular and musculo-skeletal diseases. One-quarter of all disability pensions for these diseases in women were solely attributable to overweight and obesity.

6.3.3 Studies in developing countries

Although there have not been any comparable studies of the economic impact of obesity in developing countries, both the WHO and the World Bank have recently highlighted the escalating burden associated with the rapidly emerging adult NCDs in this environment (26,27). These diseases now account for more deaths than infection. In developing countries, about 50% of deaths in 1990 were caused by NCDs, but by 2020 that proportion is expected to climb to almost 77%. In 1990, some 42% of deaths were attributed to infectious and reproductive conditions, while by 2020 that proportion is expected to decline to about 12%. In contrast, in developed countries 87% of deaths in 1990 were from NCDs and the proportion is expected to rise only slightly—to 90%—by 2020.

The treatment needs of the rapidly expanding urban populations and of the increasingly affluent middle classes in developing countries are already overwhelming many medical services. Furthermore, the real costs of therapy associated with NCDs in developing countries exceed those found in developed countries; the need to import expensive equipment and drugs with scarce foreign exchange and to divert limited numbers of trained specialists to medical care creates an extra burden.

In recent World Bank studies, e.g. in Chile (28), the burden of disease has been expressed in terms of DALYs lost. NCDs account for a 5- and 9-fold greater rate of premature death than communicable diseases in men and women, respectively, and 10- and 5-fold greater rates of disability. The DALYs lost in men are 15-fold, and in women 20-fold, greater for NCD than for infections. So far, the burden of disease attributable to excess weight gain and obesity has not been calculated but cancers impose a substantial burden as do NIDDM and CVD. Thus there is a need to apply the new economic analyses of the proportion of those diseases attributable to excess weight gain so that the impact of one of the principal contributors to NCDs can be recognized in developing countries.

6.3.4 Summary

International studies on the economic costs of obesity have shown that they contribute between 2% and 7% to total health care costs, the level depending on the range of diseases and the cost categories included in the analysis. The figures are based mainly on cross-sectional data, and should be considered a conservative estimate of the true cost of obesity-related diseases for a number of reasons:

- Most studies have costed only a limited range of obesity-related diseases.
- Most studies have excluded some relevant direct-cost categories from the analysis.
- In the majority of cases only the economic costs associated with obesity (BMI \geq 30) have been included in the analysis. The inclusion of costs associated with overweight (i.e. BMI 25 to 29.9) would substantially increase the attributed cost because the number of overweight individuals in a community is generally greater than those who are obese by a factor of 3 to 4; the economic cost of drug use, for example, was increased by 65% if the overweight category was included (17).

Although there have not been any comparable studies of the economic impact of obesity in developing countries, the real costs of therapy associated with NCDs in developing countries are likely to exceed those found in developed countries.

6.4 The economic costs and benefits of obesity treatment

6.4.1 Analyses of obesity control trials

Unfortunately, very little information is available on the economic benefits of treatment, but some extrapolations might be derived from preliminary and early data from the large-scale SOS intervention study of 1743 obese men and women in Sweden (24).

After two years of follow-up, Sjöström and his colleagues found a number of benefits in the subjects who were surgically treated and who individually lost between 30 kg and 40 kg. First, quality of life was markedly improved and several cardiovascular risk factors were substantially decreased. The prevalence of NIDDM—13% in controls and 16% in the intervention group before treatment—decreased by 68% in the intervention group and by only 16% in the controls. In other words, two-thirds of NIDDM was cured by the obesity intervention. Furthermore, the incidence of NIDDM was only 0.5% in the intervention group but 7% in the controls. A 4- to 5-fold risk reduction was observed in the development of hypertension, hypertriglyceridaemia and the lowering of HDL cholesterol. During two years of follow-up, the incidence of NIDDM was very low in the intervention group but 30-fold higher in controls. Data on other disease end points are not yet available.

In order to try to estimate the economic consequences of this controlled study, one may compare the results of treatment and associated costs with the estimated costs of non-treated obese subjects. If NIDDM is taken as an example, then the risk reduction of 14-fold in the treatment group suggests that NIDDM was prevented to a large extent. In addition, two-thirds of patients with established NIDDM were “cured”. Overlaying these results on the estimations of costs for obesity-related NIDDM in France would decrease the total costs of obesity in that country by approximately 3%. Utilizing the same approach for the USA reveals that costs could be reduced

by almost 20%. Similar calculations with change in cardiovascular risk factors are not easy, but a large fraction of obese subjects who would usually be eligible for treatment for hypertension and hyperlipidaemia would not be needing treatment and thus in French terms this would equate to a 25% reduction in costs.

Little published information is yet available on the potential impact of obesity treatment in the SOS trial on sick-leave and pensions, which are the other major fraction of costs of obesity. Such factors are difficult to evaluate from the current short-term follow-up of treated patients, but the initial data indicated that the number of lost working days increased faster in the control than in the intervention groups (24). Furthermore, the marked improvements in the quality of life of treated patients not only constitute an important outcome in themselves but also suggest that other major benefits relevant to health costs would be expected after longer follow-up.

However, it is important to balance the costs by including the expense of the intervention (i.e. surgery) and follow-up review. The actual cost of the surgical procedure is not available, but follow-up figures suggest that, in spite of the surgical intervention, the frequency of visits to a doctor was not different between controls and intervention subjects by the second year after surgery.

The SOS study is the only fully controlled, large-scale, long-term study of the effects of radical treatment of obesity with substantial weight loss. The results of this study will provide valuable information on the medical and economic consequences of effective intervention against obesity within a limited period. Preliminary results are very promising.

6.4.2 Potential cost savings associated with a reduction in the prevalence of obesity

A small number of studies have estimated the potential impact on health care costs of a reduction in the population prevalence of obesity.

A study in the USA (29) estimated the medication cost saving for obese patients with NIDDM who were assigned to a 12-week weight-loss programme involving an 800 kcal diet. Subjects lost an average of 15.3 kg over the 12 weeks, but at a one-year follow-up had regained 9.0 kg. The authors estimated that average saving in prescription costs per subject over one year was US\$ 442.80. While the study showed a significant prescription cost saving, sample sizes were small and the energy intake associated with the weight-loss programme was very restrictive. It would thus be unwise to generalize these results to a wider setting.

As an extension of the cost-of-obesity study discussed earlier in this chapter, the NHMRC estimated the potential annual saving to the Australian health care system that would result if the prevalence of obesity were reduced by 20% by the year 2000 (baseline 1989), as specified by the National Health Goals and Targets (30). The methodology used in this study was to re-calculate the PAF based on the target prevalence of obesity (and assuming relative risk estimates remain constant) for each obesity-related disease. The 1989–1990 estimated cost for each obesity-related disease was multiplied by the change in PAF to estimate the potential annual saving. The

NHMRC estimated that an annual saving of AUD\$ 59 million in health care expenditure and a potential 2300 life years could be gained if the obesity goal were met.

While the NHMRC figure provides the potential cost saving that might be achieved if the target obesity prevalence was achieved, it does not provide information regarding the amount of public and private expenditure that would be required to fund programmes to achieve such targets. The analysis therefore does not inform decision-makers on whether investing resources into preventing or treating overweight and obesity represents an efficient use of scarce community resources. Such decisions should be based on an evaluation of both the costs and outcomes (effectiveness) of alternative interventions for both the prevention and treatment of overweight and obesity which is the focus of economic evaluation.

6.4.3 Cost-effectiveness of obesity prevention and treatment

Few studies have addressed the economic evaluation of the prevention and treatment of overweight and obesity and, of these, the emphasis has been mostly on treatment rather than primary prevention. A limited number of studies have addressed the cost-effectiveness of non-drug versus drug treatment of hypertension, where weight loss has been included in this equation. In addition, a number of studies have focused on the financial benefits of workplace wellness programmes (including the benefit of weight loss) in reducing employee absenteeism. The worksite studies have been criticized on the basis of their poor methodologies (31,32). In some cases authors have over-generalized and used optimistic estimates of the health benefits of risk factor modification. Other studies have specified incorrectly the relevant programme cost categories and have used rather dubious methods of valuing and measuring those costs. The results seem in some instances to be biased in favour of finding worksite health promotion to be a good investment.

The following discussion summarizes the results of two studies that have assessed the cost-effectiveness of alternative interventions for weight control. The first study, by Segal et al. (33), models the potential costs and outcomes of a variety of prevention and treatment interventions for NIDDM. The second study, by Spielman et al. (34), focuses only on commercial weight-loss programmes.

Cost-effectiveness of obesity management in the prevention of NIDDM

A recent study by Segal et al. (33) attempted to model the potential cost-effectiveness of a range of interventions for the prevention and treatment of NIDDM in Australia. Interventions included: a population approach using mass media programmes focusing on lifestyle changes (including diet and exercise); a behaviour modification programme for the seriously obese; a group programme targeting overweight men (based on an established programme called GutBusters); gastric surgery for the morbidly obese; and a behaviour modification programme for women who had gestational NIDDM.

The study estimated both the costs and outcomes of the various interventions. Net costs (or savings) were derived by adding programme costs and the potential savings in future health care

costs from the prevention of cases of NIDDM. Outcomes were expressed as NIDDM years deferred and life-years saved. Costs were based on reports in the literature, discussions with service providers and published health service cost data. The effectiveness of various programmes in preventing NIDDM was based on epidemiological data reported in the literature. A range of estimates was calculated using different assumptions on programme success, programme costs and other important variables.

The study estimated that the most cost-effective interventions were the GutBusters Programme (a commercial 6-week group session programme for men) and the mass media lifestyle modification programme. Both interventions were estimated to result in downstream or future cost savings from reduced incidence of NIDDM, and these savings were greater than the programme costs. For example, the cost-effectiveness ratio for the mass media programme was estimated to be a net saving of AUD\$ 1400 per NIDDM year deferred or a net saving of AUD\$ 2200 per life-year gained. Table 6.2 summarizes key results from the study.

Table 6.2 Summary of the estimated cost-effectiveness of a range of interventions for the prevention of NIDDM

Intervention^a	Net cost per NIDDM years avoided (AUD\$)	Net cost per life year gained (AUD\$)
Surgery for seriously obese		
• all IGT ^a	1200	4600
• 10% IGT 90% normal ^b	3500	12 300
Diet/behaviour modification for seriously obese		
• all IGT	saving	saving
• 10% IGT 90 % normal	1600	2600
Group programme overweight men		
• all IGT	saving	saving
• 10% IGT 90% normal	saving	saving
Diet/behavioural programme women with previous gestational NIDDM		
• all IGT	800	1200
• % IGT, 70% normal	2100	2400
Media programme	saving	saving

^a "IGT" refers to programmes targeted at those with impaired glucose tolerance

^b "normal" relates to normal glucose tolerance

Source: Reference 33

Although the results presented in Table 6.2 are very much dependent on the assumption of long-term success in the various weight-loss programmes, a wide range of costs-effectiveness estimates indicate that they are robust. Indeed, while the analysis incorporates the estimated effect of

weight loss on all-cause mortality, and not just that associated with NIDDM, the likely impact of a successful prevention programme on other risk factors (such as cholesterol or blood pressure) has not been taken into account. In addition the expected savings in future health care costs relate to NIDDM only, ignoring possible savings in the management of other obesity-related diseases. For these reasons the results may well be conservative.

The authors of the study concluded that the prevention of NIDDM, through appropriate interventions, can represent a highly efficient use of community resources. Such programmes can achieve a substantial improvement in health status at little cost or indeed with the possibility of a net saving in the utilization of health care resources.

Cost-effectiveness of commercial weight-loss programmes

A study by Spielman et al. (34) analysed the cost of losing weight in commercial weight-loss programmes in the Boston Metropolitan area. It reviewed 11 commercial diet programmes and estimated the out-of-pocket cost (over a 12-week period) paid to the clinic by the participant to lose one kg on each programme. The diet programmes were classified into three groups:

- medically supervised very low-calorie diets (VLCDs) that provide < 800 kcal/day.
- Nutrient-balanced reduced energy diet programmes (REDPs) with the client consuming 800 kcal/day to 1200 kcal/day (50% carbohydrate, 15% to 20% protein, < 30% fat).
- Support groups which may/may not offer individual dietary advice and act as a self-help programme with volunteer staff.

Table 6.3 Cost per kg of active weight loss (12 weeks)

Programme type	Initial weight	
	80 kg	136 kg
Nutrient-balanced REDPs	US\$	US\$
• Jenny Craig	23.00	13.50
• Nutri-System	19.00	12.00
• Registered Dietitian	15.00	9.00
• Weight Watchers	2.50	1.50
VLCDs programmes		
• Health Management Resources (HMR)	17.50	10.00
• Medifast	14.00	8.00
Support Groups		
• Taking Off Pounds Sensibly (TOPS)	0.07	0.04

Source: Reference 34

The study found that the cost of a 12-week commercial weight-loss programme varied enormously from US\$ 2120 for the most expensive VLCD to US\$ 108 for the least expensive REDP. A summary of the analyses is shown in Table 6.3.

This short-term analysis suggests that support groups such as TOPS and lower-cost REDPs such as Weight Watchers were the most cost-effective interventions. Dietitians were only marginally better value for money than other REDPs, particularly when the expected reduction in usual supermarket expenditures is subtracted. However, this study can be criticized on a number of grounds:

- It did not measure weight loss from a sample of programme recipients. Rather, “expected” weight loss based on the literature was generalized to the programmes assuming excellent compliance throughout the duration of the weight-loss programme. This may substantially over-estimate weight loss, and in practice there would be significant differences in weight loss achieved by the alternative programmes. The analysis also ignores potential drop-out rates.
- The study was based on a 12-week programme, did not consider the costs and impacts of weight maintenance programmes, and could not consider the longer-term impact of the competing programmes.
- The financial “costs” measured were restricted to programme initiation fees, any food supplements purchased as a result of the programme (e.g. liquid protein formulae for VLCD, or pre-prepared foods, for example from Jenny Craig) and to medical monitoring and/or associated behaviour modification programmes. Additional costs (or savings) associated with daily food purchases, reduced-energy beverages, etc. were not factored in. Thus, for a programme such as Jenny Craig, the cost of prepared food plus additional staple items that are purchased from the supermarket have to be weighed up against usual food bills.
- The “time” costs of attending the programmes were also not factored in. These costs may be significant and would vary among the various programmes.

If all costs had been included and the effectiveness of interventions measured, the costs of the programmes may in fact change.

The economic costs and benefits of obesity treatment in developing countries

No economic analyses have been made of the economic costs of obesity treatment in developing countries. However, other analyses of the costs of health interventions show that prevention is a more cost-effective strategy than treatment once disease is diagnosed. Table 6.4 compares the costs of a variety of public health packages (including education, information, surveillance and monitoring) with the costs of some primary care clinical services for developing countries where major needs are the treatment of trauma and infection. Low-income developing countries do not

have any resources to cope with measures other than public health and essential clinical services. In middle-income developing countries, the high costs of discretionary clinical services mean that coping with the chronic diseases exceeds the costs of all other health cases. Thus, it would appear to be more cost-effective for money spent on obesity and other NCDs to go towards prevention than on expensive treatments during the advanced stage of disease.

Table 6.4 Allocation of public expenditure on health in developing countries, 1990

	Allocation in developing countries (US\$ per person per year) ^a		Contents of health-related packages	Cost per DALY
	Actual	Proposed		
Public health package	1	5	EPI plus; school health programmes; tobacco and alcohol control; health, nutrition and family planning information; vector control; STD prevention; monitoring and surveillance.	25
Essential clinical services	4–6 ^b	10	Treatment of tuberculosis, STD; infection and minor trauma; management of the sick child; prenatal and delivery care; family planning; assessment, advice, and minor pain alleviation.	25–75
Discretionary clinical services ^c	13–15	6	All other health services, including low-cost treatment of cancer, CVD, other chronic conditions, major trauma, and neurological and psychiatric disorders.	> 1000
Total	21	21		

^a Estimates are for all developing countries, i.e. an average of costs in low-income countries (estimated from an income level of US\$ 350 per capita) and middle-income countries (estimated from an income level of US\$ 2500 per capita). The numbers reported should be regarded as approximations.

^b On the basis of estimates in World Bank health sector reports, current spending on essential clinical services is estimated to be 20% to 30% of total public expenditure on health.

^c Estimated as total cost of overall health package minus cost of public health and essential clinical services packages.

Source: References 35,36

A further benefit of prevention is that these public health costs are based on establishing social and physical structures within a society which can have long-term and even intergenerational effects. Treatment systems, however, are likely to demand recurrent expenditure as new cases of obesity emerge together with the need for either long-term or repeated treatments. At present most individuals with excess weight gain in developing countries are not treated, and the demand for medical and dietetic help is expected to rise rapidly. In addition, limited resources will be diverted to pay for slimming diets and other aids to weight loss.

WHO Consultation on Obesity

In developing countries where NCD epidemics are emerging or accelerating, a large proportion of NCD deaths occur in the productive middle years of life, at ages much younger than are witnessed in developed countries. The health burdens attributable to excess weight in transitional societies are likely to be huge due to the absolute numbers at risk, the large loss in life expectancy and the fact that the problem affects, in particular, individuals with a key role in promoting economic development.

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Section C

**Understanding how the
problem of overweight and
obesity develops**

7 FACTORS INFLUENCING THE DEVELOPMENT OF OVERWEIGHT AND OBESITY

7.1 Introduction

In simple terms, obesity is a consequence of an energy imbalance where energy intake has exceeded energy expenditure over a considerable period. Numerous different complex and diverse factors can give rise to a positive energy balance, but it is the interaction between a number of these influences, rather than any single factor acting alone, that is thought to be responsible. In contrast to the widely held perception among the public and parts of the scientific and medical communities, it is clear that obesity is not simply a result of overindulgence in highly palatable foods, or of a lack of physical activity.

An overview of the major influences on energy balance and weight gain is provided in Figure 7.1.

Figure 7.1 Influences on energy balance and weight gain

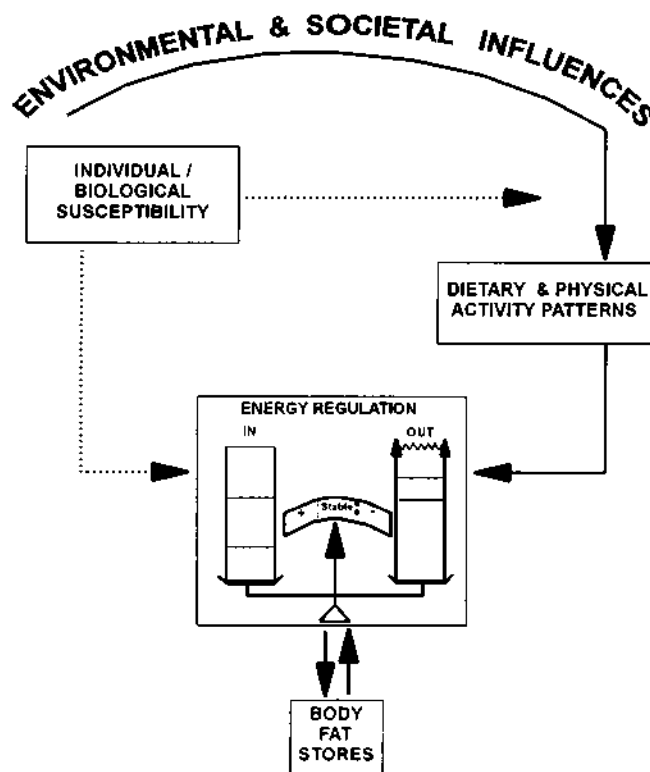


Figure 7.1 shows that powerful societal and environmental forces influence energy intake and expenditure, and may overwhelm the physiological regulatory mechanisms that operate to keep weight stable. The susceptibility of individuals to these forces is affected by genetic and other biological factors, such as sex, age and hormonal activities, over which they have little or no control. Dietary factors and physical activity patterns are considered to be the modifiable intermediate factors through which the forces that promote weight gain act.

This chapter examines the various influences on energy intake and expenditure that are considered to be important in weight gain and the development of obesity. Section 7.2 gives an overview of the fundamental principles of energy balance, the physiological regulation of body weight, and the dynamics of weight gain. Section 7.3 examines the role of dietary factors and physical activity patterns in weight gain. Section 7.4 discusses the multitude of environmental and societal forces which adversely affect food intake and physical activity patterns, and thereby may overwhelm the normal regulatory processes controlling long-term energy balance in man. Finally, section 7.5 reviews the various genetic, physiological or medical factors which can determine an individual's susceptibility to those forces and which put that person at higher risk of weight gain and obesity.

Key points raised include:

- Obesity can result from a minor energy imbalance which leads to a gradual but persistent weight gain over a considerable period. Once the obese state is established, physiological processes tend to defend a new weight.
- Body weight is primarily regulated by a series of physiological processes but is also influenced by external societal and cognitive factors.
- Recent epidemiological trends in obesity indicate that the primary cause of the global obesity problem lies in environmental and behaviour changes. The rapid increases in obesity rates have occurred in too short a time for there to have been significant genetic changes within populations.
- The increasing proportion of fat and the increased energy density of the diet, together with reductions in the level of physical activity and the rise in the level of sedentary behaviour, are thought to be major contributing factors to the rise in the average body weight of populations. Dealing with these issues would appear to be the most effective means of combating rises in the level of overweight and obesity in the community.
- The global obesity problem can be viewed as a consequence of the massive social, economic and cultural problems now facing developing and newly industrialized countries, as well as ethnic minorities and the disadvantaged in developed countries. Escalating rates of obesity, NIDDM, hypertension, dyslipidaemia and CVD, coupled with cigarette smoking and alcohol abuse, are frequent outcomes of the modernization/acculturation process.
- Epidemiological, genetic and molecular studies in many populations of the world suggest that there are people who are more susceptible to weight gain and the development of obesity than others. Genetic, biological and other personal factors such as smoking cessation, sex and age interact to determine an individual's susceptibility to weight gain.

- Certain ethnic groups appear to be especially liable to the development of obesity when exposed to an affluent lifestyle, although susceptibilities to obesity co-morbidities are not uniform across these groups.

7.2 Energy balance and the physiological regulation of body weight

Figure 7.2 Influences on energy balance and weight gain (energy regulation)

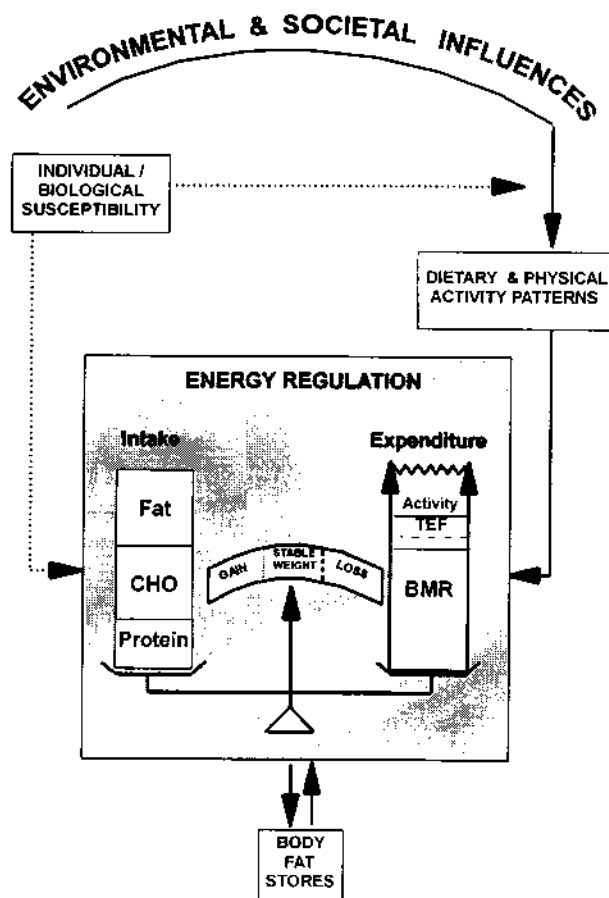


Figure 7.2 shows the fundamental principles of energy balance and regulation. Positive energy balance occurs when energy intake is greater than energy expenditure and promotes weight gain. Conversely, negative energy balance promotes a decrease in body fat stores and weight loss. Body weight is regulated by a series of physiological processes which have the capacity to maintain weight within a relatively narrow range (stable weight). It is thought that the body exerts a stronger defence against undernutrition and weight loss than it does against over-consumption and weight gain. TEF = thermic effect of food; BMR = basal metabolic rate; CHO = carbohydrate.

7.2.1 Fundamental principles of energy balance

The fundamental principle of energy balance is:

$$\text{Changes in energy stores} = \text{Energy Intake} - \text{Energy Expenditure}$$

Positive energy balance occurs when energy intake is greater than energy expenditure; it promotes an increase in energy stores and body weight. Conversely, a negative energy balance occurs when intake is less than expenditure, promoting a decrease in energy stores and body weight.

Under normal circumstances, energy balance oscillates from meal-to-meal, day-to-day and week-to-week without a persistent change in body stores or weight. Multiple physiological mechanisms act within each individual to equate overall energy intake with overall energy expenditure and to keep body weight stable in the long term. Thus, it is only when there has been a positive energy balance for a considerable period that obesity is likely to develop.

Energy intake

Total energy intake refers to all energy consumed as food and drink that can be metabolized inside the body. Table 7.1 shows the constituent macronutrients present in food and drink that provide energy. Fat provides the most energy per unit weight, and carbohydrate and protein the least. Fibre undergoes bacterial degradation in the large intestine to produce volatile fatty acids which are then absorbed and utilized as energy. The size of the energy contribution from fibre is thought to be 6.3 kJ/g (1.5 kcal/g) (1).

Table 7.1 Energy content of macronutrients

Macronutrient	Energy contribution	
	(kcal/g)	(kJ/g)
Fat	9	37
Alcohol	7	29
Protein	4	17
Carbohydrate	4	16

Energy expenditure

The second element of the energy balance equation, total energy expenditure, has three main components:

- BMR
- Dietary thermogenesis (meal-induced heat production)
- Physical activity

The proportion that each component contributes to total energy expenditure varies according to the regularity and intensity of physical activity. In sedentary adults BMR accounts for nearly 60% of total energy output, the dietary thermogenic response around 10%, and physical activity the remaining 30%. In those engaged in heavy manual work, total energy expenditure increases and the proportion of energy expenditure provided by physical activity may rise to about 50%. Dietary thermogenesis appears to remain constant at 10%, leaving BMR to account for 40% of the total energy expenditure. Although the BMR may vary intrinsically between individuals of similar weight by $\pm 25\%$, within each individual it is tightly controlled (2). The key variable of energy output in an individual is the degree of physical activity.

7.2.2 The physiological regulation of body weight

Societal and cognitive factors can influence the control of body weight to a certain extent, but it is a series of physiological processes that are primarily responsible for body weight regulation. In traditional societies where people tend to be more physically active and where food supplies are not limited, few adults are either underweight or overweight despite the interaction of seasonal cycles of work, festivities, individual susceptibilities to obesity for physiological or genetic reasons, and the wide range of varying physical demands within a society. These extensive physiological mechanisms represent a fundamentally important biological process which can be observed throughout the animal kingdom. It is thought that the body exerts a stronger defence against undernutrition and weight loss than it does against overconsumption and weight gain (3).

The physiological mechanisms responsible for body weight regulation

The physiological mechanisms responsible for body weight regulation are incompletely understood. However, there is increasing evidence of a range of mechanisms within the intestine, the adipose tissue and brain, and perhaps within other tissues, which sense the inflow of dietary nutrients, their distribution and metabolism and/or storage. These signalling mechanisms are coordinated within the brain and lead to behavioural changes in eating, in physical activity and in body metabolism so that body energy stores are maintained. The recent discovery of the hormone leptin, which is secreted by adipocytes in proportion to their triglyceride stores and binds with receptors in the hypothalamus, provides interesting insights into possible regulatory signal systems which act to maintain energy balance. However, much remains to be elucidated about such systems. Some of these mechanisms are illustrated in Figure 7.3.

Figure 7.3 Physiological processes involved in body weight regulation

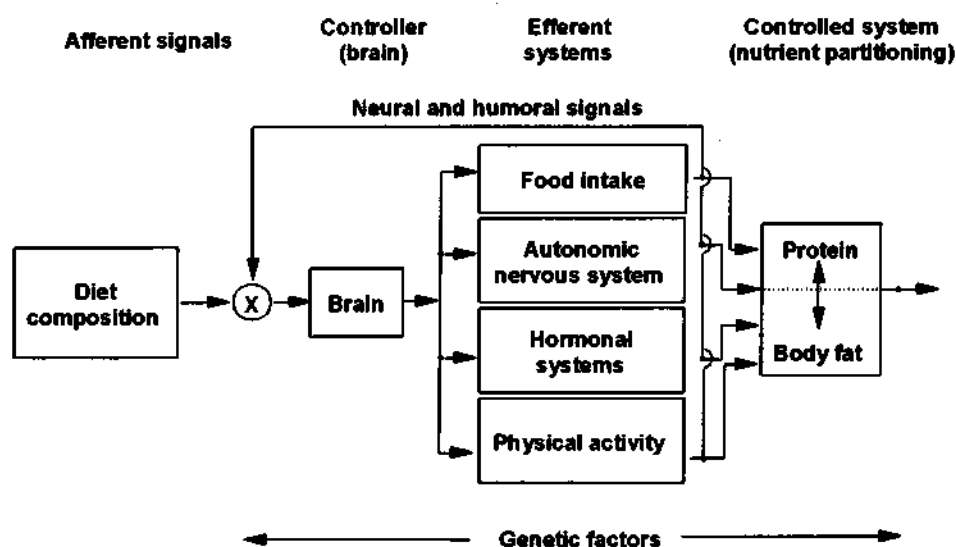


Figure 7.3 presents a model of the interaction between different mechanisms which affect energy and body weight regulation within individuals. The brain integrates an array of afferent signals (nutrient, metabolic, hormonal and neuronal) and responds by inducing changes in food intake, autonomic nervous system activity, hormonal responses or in spontaneous physical activity. The different components then directly or indirectly determine the partition of fat and protein.

7.2.3 The dynamics of weight gain

Despite the extensive physiological regulation of body weight outlined above, positive energy balance can lead to weight gain if it persists in the long term. The initiation of a chronic positive energy balance is due to an increase in energy intake relative to requirements, either as a result of an increase in total energy intake, a decrease in total energy expenditure, or a combination of the two. Currently there is little information about the fluctuations in energy balance which lead to weight gain and obesity. It is possible that large deviations from energy balance at regular intervals may contribute to weight gain, but it is believed that a small consistent deviation over a long period is also capable of producing large increases in body weight.

Figure 7.4 shows that weight gain can be divided into three phases:

- The *pre-obese static phase*, when the individual is in long-term energy balance and weight remains constant.
- The *dynamic phase*, during which the individual gains weight as a result of energy intake exceeding energy expenditure over a prolonged period.
- The *obese static phase*, when energy balance is regained but weight is now higher than during the pre-obese static phase.

Figure 7.4 Effect on energy expenditure, energy balance and body weight of an increase in energy intake relative to requirements

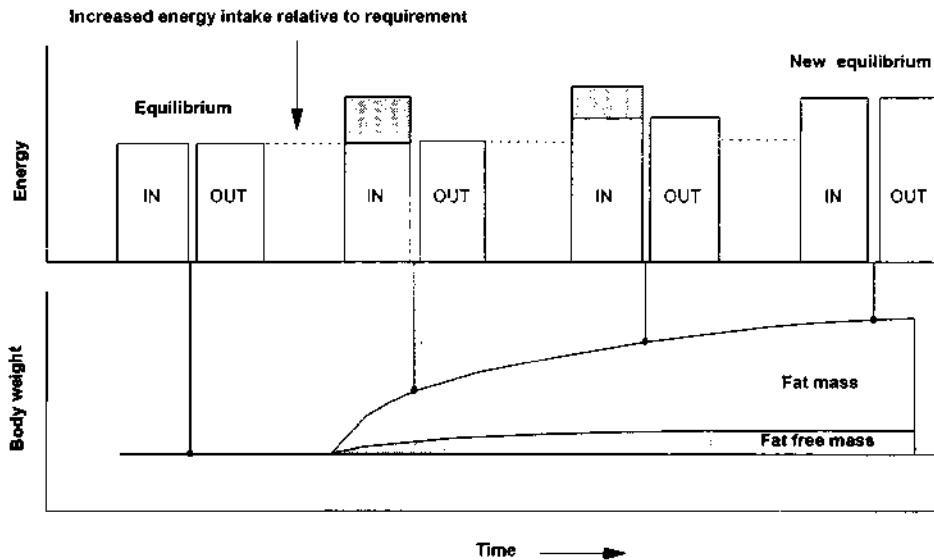


Figure 7.4 shows that a persistent increase in energy intake above requirements will lead to a gradual gain in body weight. However, the size of the energy imbalance progressively diminishes as weight is gained, because of an increase in metabolism associated with the expanded fat free mass and adipose tissue. A new higher equilibrium weight is eventually established which is again defended by physiological mechanisms. Thus, it is harder to lose the weight gained than it is to experience a second cycle of increasing body weight should, for example, a fall in physical activity occur with a further period of prolonged positive energy balance.

The dynamic phase can last for several years and often involves considerable fluctuations in weight (weight cycling) as a result of conscious efforts by the individual to return to a lower weight. However, in the absence of intervention, the difference between energy intake and energy expenditure progressively diminishes. This is due to an increase in BMR from the larger fat-free mass (including that in the expanded adipose tissue) as well as to an additional energy cost of activity imposed by the extra weight (4). There may also be an increase in RMR with overfeeding (5).

Once the obese static phase is established, the new weight appears to be defended. This can best be shown by the response of obese individuals to underfeeding; they show a fall in metabolic rate as the body recognizes the loss of energy (6) and an unconscious physiologically driven increase in energy intake (7).

7.2.4 Implications for public health

Given the global epidemic of obesity, the public health issue should be one of identifying:

- The environmental factors, including societal changes, which have overwhelmed the physiological regulatory processes outlined above.

- Whether some individuals are more susceptible to those influences for medical, behavioural or genetic reasons.

7.3 Dietary and physical activity patterns

Dietary factors and physical activity patterns have a strong influence on the energy balance equation and can be considered to be the major modifiable factors through which many of the external forces promoting weight gain act (Figure 7.5). In particular, high-fat/energy-dense diets and sedentary lifestyles are the two characteristics most strongly associated with the increased prevalence of obesity worldwide.

Figure 7.5 Influences on energy balance and weight gain (dietary and physical activity patterns)

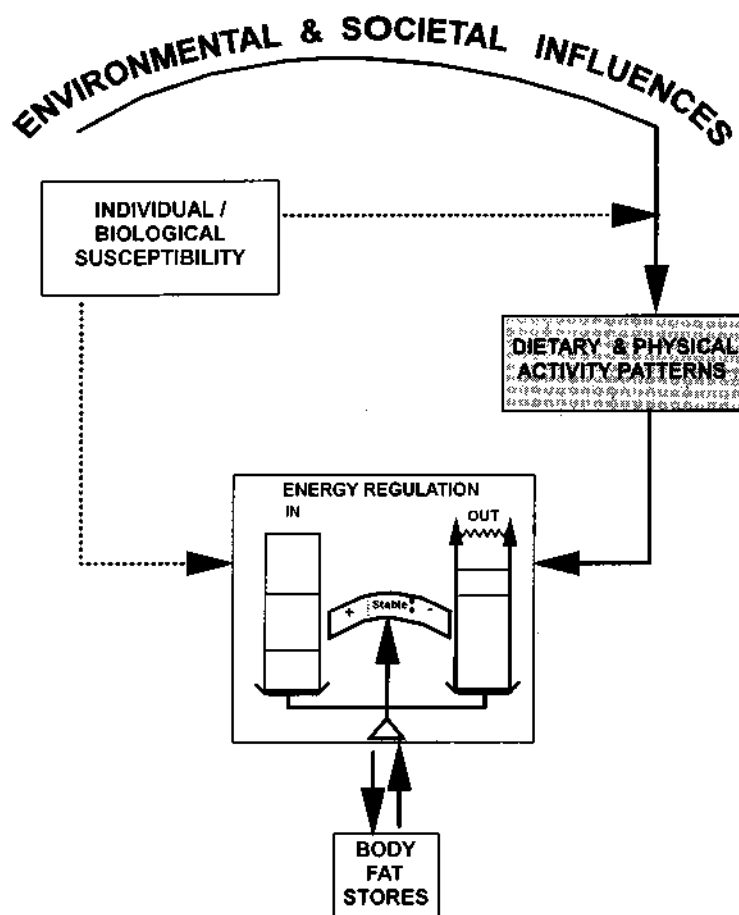


Figure 7.5 shows that dietary factors and physical activity patterns have a direct effect on the outcome of physiological regulation of body weight. They can be considered to be the intermediate modifiable factors through which many of the forces promoting weight gain act.

7.3.1 Dietary factors

This section examines how the macronutrient composition of the diet and eating patterns can affect energy balance.

Macronutrient composition

Laboratory experiments in animals and clinical studies in humans have repeatedly shown that dietary factors, particularly the level of fat and energy intake, are strongly and positively associated with excess body weight. By contrast, the population-based studies of diet and obesity have reported inconsistent results. Such inconsistencies have been attributed to a number of factors, including weaknesses in the study design, methodological flaws, confounders, and random and/or systematic measurement error in the data, especially dietary data (8). Thus, when population studies give careful attention to the determinants of obesity, a positive association is observed between dietary factors and obesity identical with those found in animal models and human clinical studies (9).

Energy intake

Dietary fat has a higher energy density than the other macronutrients (Tables 7.1 and 7.2). This quality is thought to be largely responsible for the overeating effect, or *passive overconsumption* as it is often known, experienced by many subjects who are exposed to high-fat foods (3). The stimulatory effect of fatty foods on energy intake may also be due to the pleasant mouth-feel of fat when eaten (10).

The body does compensate for the overconsumption of energy from high-fat foods to some extent, but the fat-induced appetite control signals are thought to be not strong enough, or too delayed, to prevent the rapid intake of the energy from a fatty meal. Episodic intakes of high-fat foods are therefore particularly likely to overwhelm these signals, and the control of food intake has to depend on long-term regulatory processes which seem much less able to respond to overfeeding than to underfeeding with weight loss. Fibre, by contrast, limits energy intake by lowering a food's density and allowing time for appetite control signals to occur before large amounts of energy have been consumed (3).

There is no clear evidence to suggest that high intakes of sugar overwhelm the appetite control signals in the same manner as fat. However, there is some indication from short-term feeding trials that *ad libitum* low-fat high-complex CHO diets with a low energy density induce weight loss, which does not occur on energy-dense diets, regardless of whether the energy density has been increased by modifying the fat content or the sugar content of the diet (11). Further studies are required before any conclusions can be drawn from this work.

Energy storage and macronutrient balance

The macronutrient composition of the diet also influences the extent to which excess energy is stored. Those macronutrients with a low storage capacity within the body are preferentially oxidized when intakes exceed requirements:

- **Alcohol:** no storage capacity within the body and so all ingested alcohol is oxidized immediately. This response dominates oxidative pathways and suppresses the rates at which other fuels are oxidized.
- **Protein:** limited storage capacity as body protein, which is only accessible through loss of lean body mass. Amino acid metabolism is tightly regulated to ensure the oxidation of any excess.
- **Carbohydrate:** small capacity for storage as glycogen. The intake and oxidation of carbohydrate are very tightly "autoregulated", with rapid and substantial changes in carbohydrate oxidation in response to alterations in carbohydrate intake. Excess carbohydrate can also be converted to fat, but human subjects do not use this metabolic pathway to any appreciable extent unless large excess of a low fat, high carbohydrate diet is consumed. When carbohydrate is oxidized, however, less fatty acid oxidation is required so dietary fat is stored and endogenous fat retained. About 60% to 80% of the excess energy may be stored on carbohydrate overfeeding (12).
- **Fat:** the capacity for fat storage in the body is virtually unlimited and excess dietary fat does not acutely increase fat oxidation. Excess dietary fat is readily stored in adipose tissue depots with a very high efficiency (about 96%).

Thus, the bulk of evidence suggests that carbohydrate and protein balances, but not fat balance, are well regulated. It is becoming clear that weight changes following challenges to body weight are due primarily to disruptions in fat balance, as these account for most of the imbalance produced in total energy (12–18).

In the long term, however, fat balance has to be regulated in order to achieve energy and macronutrient balance. Reachievement of fat balance following a perturbation in energy balance is thought to require a change in the body fat mass. This may be because fat oxidation varies directly with body fat mass (19), but the way in which fat mass and total fat oxidation are linked is not clear. As an example, an increase in dietary fat without a rapid change in fat oxidation will produce positive fat balance and hence lead to increases in body fat mass. As body fat mass increases, fat oxidation also increases. Fat mass will increase to the point at which fat oxidation matches fat intake and then the body fat mass will stabilize at the new, higher, level.

Food palatability and pleasure

The palatability of food has an important influence on behaviour (3). Food palatability tends to promote consumption and is one of the most powerful influences in inducing a positive rather than a negative energy balance. It increases both the rate of eating and the sense of hunger during and between meals. The presence of fat in food is particularly enjoyable, and is associated with a pleasurable mouth-feel. The food industry has capitalized on this phenomenon by developing foods with increasingly potent and pleasurable palatability. Moreover, pleasurable sensations from food can be viewed as a reward by those consuming them and can condition behaviour which favours overconsumption.

Sweetness is one of the most powerful, easily recognized and pleasurable tastes, so many foods are sweetened in order to increase their palatability and consumption. Consumption of sugars does, however, lead to a subsequent suppression of intake of energy by an amount roughly equivalent to the amount provided by the sugars (20). Nevertheless, sweetened foods which have a high fat content are expected to be conducive to an excess energy consumption since palatability is enhanced by both sweetness and mouth-feel, and fat has only a small suppression effect on appetite and intake. A preference for sweet-fat mixtures has been observed in obese women which may be a factor in promoting excess energy consumption (21).

Table 7.2 Characteristics of macronutrients

	Protein	Carbohydrate	Fat
Ability to bring eating to an end	High	Intermediate	Low
Ability to suppress hunger	High	High	Low
Contribution to daily energy intake	Low	High	High
Energy density	Low	Low	High
Storage capacity in body	Low	Low	High
Metabolic pathway to transfer excess intake to another compartment	Yes	Yes	No
Autoregulation (ability to stimulate own oxidation on intake)	Excellent	Excellent	Poor

Overview of macronutrient influence on body weight regulation

Table 7.2 summarizes the main characteristics of the macronutrients. Fat appears to be the key macronutrient which undermines the body's weight regulatory systems since it is very poorly regulated at the level of both consumption and oxidation. There is currently no consensus regarding the role of sugar intake on body weight regulation but there is some concern that the overconsumption of sweet-fat foods may be a problem, at least in certain subgroups of the

population. Finally, although high protein intakes may appear to be advantageous in controlling energy intake and contributing to good body weight regulation, high protein intakes (especially animal protein) have been associated with a number of adverse health consequences.

Dietary patterns

Daily eating pattern

Research into the relevance of eating patterns and health have mainly focused on fluctuations in blood glucose and blood lipid concentrations throughout the day, particularly in the context of control of NIDDM. There does appear to be some advantage in nibbling versus gorging under iso-caloric conditions with respect to glycaemic control and hypertriglyceridaemia (22). However, in at least one controlled study, there was no effect of meal patterns on energy metabolism and energy balance (23).

Under free-living conditions, meal patterns vary widely across populations and cultures. Regular (high-fat) snacking has been associated with increased overall dietary intake in affluent societies but this area remains controversial (24). Other evidence from affluent societies suggests that dietary restraint and slimming leads to skipping breakfast and that this may lead to overconsumption later in the day (25). Some people exhibit additional eating during the night (proposed by Stunkard et al. to be part of a NES) (26) which is associated with obesity, although causality in this association is not known. Recently, one study in obese people trying to lose weight found that the prognosis of weight loss was better in women who ate more smaller meals than those who ate fewer but larger meals (27).

Eating disorders

Eating disorders, particularly those which result in excess energy intake relative to requirements, have been implicated in the development of obesity. However, it is uncertain whether obesity is a direct result or an underlying cause of the eating disorder. For a more detailed discussion of eating disorders, including BED and NES, see Chapter 4 (section 4.10.4).

7.3.2 Physical activity patterns

This section examines the vital role played by physical activity in the regulation of body weight and fat stores, and outlines the evidence that modern inactive lifestyles are heavily implicated in the etiology of obesity.

Cross-sectional data often reveal an inverse relationship between BMI and physical activity (28–31) indicating that obese and overweight subjects are less active than their lean counterparts. However, such correlations do not provide cause and effect relationships and it is difficult to know whether obese individuals are less active because of their obesity or whether a low level of activity caused the obesity. Results from other types of study, however, suggest that low and decreasing levels of activity are primarily responsible; for instance, obesity is absent among elite athletes while those athletes who give up sports frequently experience an increase in

body weight and fatness (32–35). Furthermore, the secular trend in the increased prevalence of obesity seems to parallel a reduction in physical activity and a rise in sedentary behaviour. One of the best examples of this is provided by Prentice and Jebb (36), who used crude proxies for inactivity, such as amount of time viewing television or number of cars per household. These studies all suggest that decreased physical activity, and/or increased sedentary behaviour, play an important role in weight gain and the development of obesity. This conclusion is further supported by prospective data. Dietz and Gortmaker (37), for example, have documented that the amount of television watching in young children is predictive of BMI some years later, while Rissanen et al. (34) have shown that a low level of physical activity during leisure in adults is predictive of substantial weight gain (≥ 5 kg) in 5 years time. More prospective data will help clarify this relationship but it seems reasonable to link physical inactivity with future weight gain.

Physical activity patterns have an important influence on the physiological regulation of body weight. In particular, they affect total energy expenditure, fat balance and food intakes. Box 7.1 gives an outline of the different components of “physical activity” and defines “physical inactivity”. Box 7.2 introduces the concept of PALs.

Contribution of physical activity to total energy expenditure

Increased energy expenditure is an intrinsic feature of physical activity and exercise. Energy requirements increase from the basal levels immediately after the initiation of physical activity, and the increase persists for the duration of the activity. The total amount of energy expended depends on the characteristics of the physical activity (mode, intensity, duration and frequency) and of the individual performing the exercise (body size, level of habituation and fitness). These relationships have been extensively reviewed in the literature (43), and tables providing approximations of the energy costs of various physical activities are widely available.

If exercise is severe, oxygen consumption remains elevated above resting levels for some time after the cessation of activity. This metabolic response is termed “excess post-exercise oxygen consumption” (EPOC) and is due to the need to restore energy reserves, especially glycogen levels in liver and muscles. Compared with the energy cost of exercise itself, however, the contribution of EPOC is likely to be modest. A recent study estimated that after two hours exercising at a moderate intensity this accounted for an extra 200 kJ/d (48 kcal/d) when averaged over 24 hours (44). Although this is quite small in terms of total daily energy expenditure, it has the potential to help maintain energy balance if exercise is undertaken on a regular basis.

In addition to the immediate energy costs of increased physical activity and of the recovery period (i.e. EPOC), habitual exercise may influence several other components of energy expenditure including RMR. Although there is still controversy surrounding this area of research, several recent studies have provided evidence for a positive association between activity levels and RMR (45). As the increase in the RMR is lost after several days of inactivity, this highlights the benefit of regular and sustained exercise patterns (46). Moreover, resistance exercise such as weight training may contribute to the maintenance of, or to an increase in, muscle mass, thereby favouring an elevation of the RMR or preventing a decrease in metabolic rate in the presence of weight loss (47).

Box 7.1

Physical activity

A global term referring to "any bodily movement produced by skeletal muscle that results in a substantial increase over the resting energy expenditure". It has three main components (38):

- **Occupational work:** activities undertaken during the course of work.
- **Household and other chores:** activities undertaken as part of day-to-day living.
- **Leisure-time physical activity:** activities undertaken in the individual's discretionary or free time. Activity is selected on the basis of personal needs and interests. It includes exercise and sport:
 - **Exercise:** a planned and structured subset of leisure-time physical activity that is usually undertaken for the purpose of improving or maintaining physical fitness.
 - **Sport:** its definition varies around the world. In North America it implies a form of physical activity that involves competition, whereas in Europe it may also embrace general exercise and a specific occupation.

The division of time between each of the three components varies considerably between individuals and populations.

Physical inactivity (sedentary behaviour)

Physical inactivity, or sedentary behaviour as it is otherwise known, can be defined as "a state when body movement is minimal and energy expenditure approximates RMR" (39). However:

- Physical inactivity represents more than an absence of activity; it refers also to participation in physically passive behaviours such as television viewing, reading, working at a computer, talking with friends on the telephone, driving a car, meditation or eating (40).
- Physical inactivity may contribute to weight gain through other means than a reduction in energy expenditure. For example, recent studies in adolescents (41) and adults (42) have demonstrated significant relationships between inactivity and other adverse health practices, such as the consumption of less-healthy foods and an increased fat intake.

Box 7.2**Physical activity levels**

PAL values express daily energy expenditure as a multiple of BMR, thereby allowing approximate adjustment for individuals of different sizes. PALs are a universally accepted way of expressing energy expenditure and help to convey an easily understandable concept.

Individuals whose occupation involves regular physical activity are likely to be at PALs of 1.75 or more. Individuals whose lifestyle involves only light occupational and leisure time activity are likely to have a PAL of 1.55–1.60. Some individuals who engage in no activity whatsoever will have a PAL below this, at around 1.4.

In order to avoid obesity, populations should remain physically active throughout life at a PAL of 1.75 or more. Thus:

Lifestyle	Actual PAL	Target PAL
Sedentary	1.4	≥ 1.75
Limited activity	1.55 – 1.60	≥ 1.75
Physically active	≥ 1.75	≥ 1.75

The table below shows some ways in which PAL can be raised from 1.55–1.60 up to 1.75 or more by the equivalent of an extra hour of moderate activity each day. More strenuous activities require less time than one hour per day to bring the overall average daily PAL up to 1.75.

Equivalent of 1 hour's extra moderate physical activity daily:

Duration	Activity ratio*	Activity
1 hour	4–5	Brisk walk (6 km/hr); canoeing (5 km/hr); cycling (12 km/hr); gardening; baseball; volleyball
45 mins	6–7	Cross-country hiking; cycling (15 km/hr); skating (14 km/hr); water skiing; dancing; snowshoeing
30 mins	10–12	Any vigorous activity, e.g. soccer/football; hockey; running (13 km/hr); rugby; handball; basketball (competition)

*Activity ratio = multiple of BMR

Energy expenditure across the world

There is a widespread belief that daily life in less-developed countries demands a much greater physical effort; for instance, a Third World woman spends 30–150 minutes every day of her life simply fetching water (48), and walks while attending to her daily chores for up to 1.5 hours. However, it is difficult to get accurate assessments of energy expenditure in free-living conditions; where developed and developing countries have been compared, few differences have been found (49). One explanation offered for this apparent discrepancy is that adults in less developed countries compensate by being inactive whenever possible; in Ethiopia, for example, energy expended on physical activity decreases in the post-harvest season (50). Secondly, the curtailment of physical activity in order to spare energy represents the first line of defence when exposed to energy stress caused by insufficient dietary energy. Such a behavioural response can be illustrated by poorly nourished Rwandan women, who spend more time in low-cost activities than their better nourished counterparts (51). In the aggregate, however, it is reasonable to conclude that people in less developed countries who spend a considerable portion of their time on finding food for their next meal and for personal chores are expending more energy for work and physical activity for a given body size than people in more-developed countries.

Effect of physical activity on fat and substrate balance

Regular physical activity and substrate balance

One of the most important adaptations to regular exercise is the increased capacity to utilize fat relative to carbohydrate during moderate levels of physical activity. These differences become considerable when the exercise is maintained over a longer period; physically trained individuals metabolize more fat at equivalent levels of energy expenditure than the untrained. It has been shown, for example, that the rate of fat oxidation in a group of unfit individuals increased by approximately 20% after a 12-week fitness training programme (52).

Of particular relevance to this chapter is the observation that regular moderate physical exertion allows free-living volunteers to consume *ad libitum* a 40% fat diet without storing excess fat, whereas the same individuals, when sedentary, are in positive fat and energy balance and thus have a greater risk of becoming overweight and obese with time. If, however, they are offered a 20% fat diet, they remain in balance even when sedentary (53). Although these physiological studies should be interpreted with caution, they are of profound significance because they suggest a fundamental interaction between the level of physical activity and the proportion of dietary fat in determining whether energy balance can be sustained. It is not known precisely at what level dietary fat can overwhelm the body's capacity to preferentially oxidize fat by increased exercise nor how much this varies between individuals. However, it is thought that people who sustain moderate or high levels of physical activity throughout life may tolerate diets with a high fat content (e.g. 35% to 40% of energy) whereas lower fat intakes (20% to 25% of energy) may be needed to minimize energy imbalance and weight gain in sedentary individuals and societies. Thus, since most people in developed countries are sedentary, it is reasonable to assume that fat balance is achieved at a level of fat intake of 30% or less. In developing countries, the level of

dietary fat compatible with fat balance could be higher as a result of the amount of energy expended for work and personal chores.

Exercise intensity and substrate balance

The metabolic responses to low- and high-intensity physical activity are very different. The extent to which fat and carbohydrate contribute to energy metabolism depends on the intensity level of the activity; fat is preferentially oxidized during low-intensity activity whereas carbohydrate is the dominant fuel at high intensity. In theory, the highest relative level of fat oxidation occurs when adults are moderately active at around 50% to 60% of maximum. In addition, theoretical calculations suggest that multiple intense bouts of exertion are better stimuli for fat oxidation than the equivalent energy use through more prolonged low-activity levels (54). The important point to remember is that the number of grams of fat oxidized during activity increases with the intensity and the duration of the activity, despite the fact that the proportion of fat in the mixture of fuel oxidized for muscular contraction may decrease at higher intensities. It should also be kept in mind that fat is oxidized not only during the activity but also in the recovery period.

Impact of physical activity on food intake and preferences

Food intake

There is a common perception that exercise stimulates appetite, leading to an increased food intake that even exceeds the energy cost of the preceding activities. Nevertheless, supporting evidence for this from human studies is lacking; if a compensatory rise in intake does occur, this tends to be accurately matched to expenditure in lean subjects so that energy balance is re-established in the long term (54,55). However, Woo et al. (56) showed that obese women did not compensate the higher energy expenditure induced by exercise with increased intake, and thereby obtained a significant negative energy balance on exercise. This suggests that those who have an excess amount of energy stored may particularly benefit from exercise.

In the short term, hunger can be suppressed by intense exercise, and possibly by low-intensity exercise of long duration (54). The effect is short-lived, however, so the temporal aspects of exercise-induced anorexia may best be measured by the delay in eating rather than the amount of food consumed (57).

Food preference

Whether exercise influences the type of food and the mix of macronutrients chosen by free-living subjects remains uncertain. In a small number of longitudinal studies, a higher intake of carbohydrate-rich foods has been observed with an increase in PAL (58) and, recently, a significant positive relation was found between the level of PAL and carbohydrate intake in a diet intervention study (59). However, it is unknown whether dietary advice about optimum sport nutrition or physiological needs help to initiate such dietary changes (54).

More information is needed in order to evaluate the value of this change in food selection in the general population with relatively small changes in the level of physical activity.

Physical activity levels for prevention of excessive weight gain

Analyses of over 40 national physical activity studies worldwide show that there is a significant relationship between the average BMI of adult men and their PAL, with the likelihood of becoming overweight being substantially reduced at PALs of 1.8 or above (see Box 7.2 for description of PALs). The relationship for women, though not statistically significant, is similar, but their physical activity tends to be lower (mean PAL 1.6) (49). It has been suggested, therefore, that people should remain physically active throughout life and sustain a PAL of 1.75 or more in order to avoid excessive weight gain. Sedentary people living or working in cities typically have a PAL of only 1.55–1.60, and PALs in industrialized societies are drifting downwards.

Figure 7.6 Active leisure required to achieve an overall mean PAL of 1.76

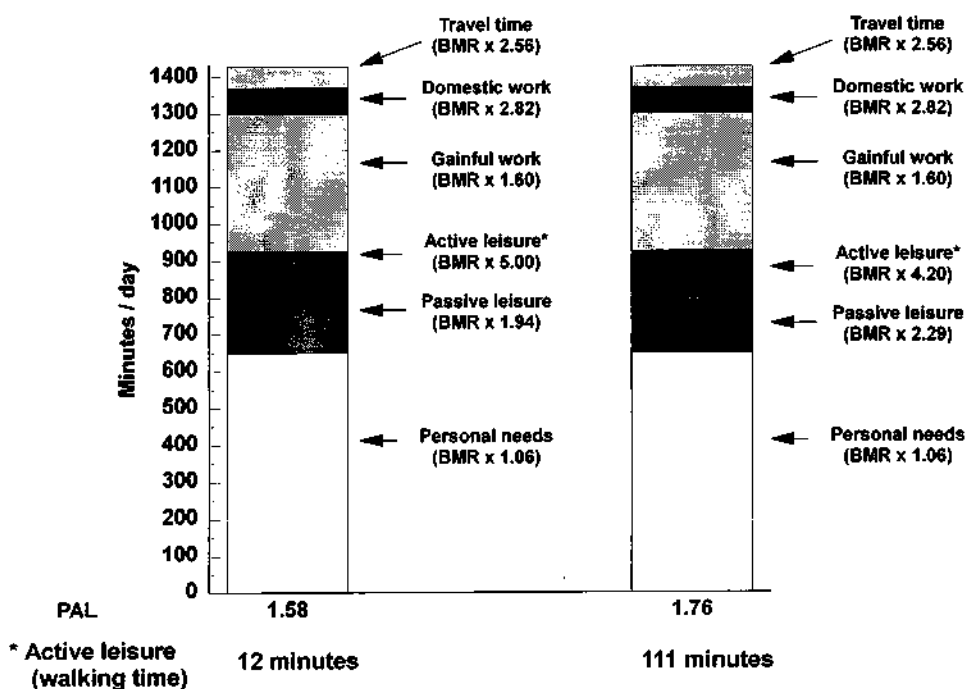


Figure 7.6. Modelling the nature, duration and timing of active leisure required to achieve an overall mean PAL of 1.76. The activity profile of the average Italian adult male, aged 30–60 years, is taken as the basis for the modelling exercise (60). He is assumed to weigh 70 kg and to have a predicted BMR of 1690 kcal/day. He is sedentary, being employed in a light-activity job (BMR factor = 1.60, Food and Agriculture Organization of the United Nations (FAO)/WHO Ad Hoc Expert Committee 1985), and he spends only 24 minutes per day in active leisure (of which 12 minutes in sports and 12 minutes in walking) at an overall BMR factor of 5.0. The other 252 minutes are spent in passive leisure (BMR x 1.94). Increasing his daily walking time (speed 4 km/hr, BMR x 4.0) to 111 minutes raises his daily PAL to 1.76. The extra 99 minutes of walking time have been taken from the 252 minutes of his passive leisure time; more specifically, it has been assumed that he would replace all of his time spent on TV watching (90 minutes), and another 9 minutes spent on reading, with walking.

People who make extensive and increasing use of motorized transport, automated work and sedentary leisure pursuits, may find it difficult to attain PAL levels at or above 1.75 simply by increasing activity during "leisure time". This is illustrated by the calculations of Ferro-Luzzi and Martino (49). They showed that, for an average 70 kg adult male, moving a PAL of 1.58 up to around 1.70 involves an average of 20 minutes a day of vigorous exercise, such as running or circuit training at an activity ratio of 11 (a level of activity achievable only by a physically fit person), or else one hour of extra walking every day. Moving a PAL of 1.58 up to 1.76 requires approximately 1 hour and 40 minutes of extra walking (at 4 km/hr) per day (Figure 7.6). As these activity prescriptions are *additional* to a 24-minute period of "active leisure" (12 minutes of sports and 12 minutes of walking) already required for a PAL of 1.58, it follows that urban sedentary populations are likely to attain a PAL of 1.75 or more only if supported by vigorous national policies that encourage physical activity. For example, these should encourage children to be active at play and school, and should create environments in which walking and cycling become the most common means of travel to work and for short journeys.

7.4 Environmental and societal influences

The rapid increases in obesity rates over recent years have occurred in too short a time for there to have been any significant genetic changes within populations. This suggests that the primary cause of the rapid global rise in obesity lies in environmental and societal changes that are now affecting a large proportion of the world's population.

This section assesses the environmental and societal factors which, through their effects on food intake and physical activity patterns, have overwhelmed the physiological regulatory processes that operate to keep weight stable in the long term (Figure 7.7). It also considers briefly the sectors which influence the impact of societal changes on food intake and physical activity.

Figure 7.7 Influences on energy balance and weight gain (environmental and societal influences)

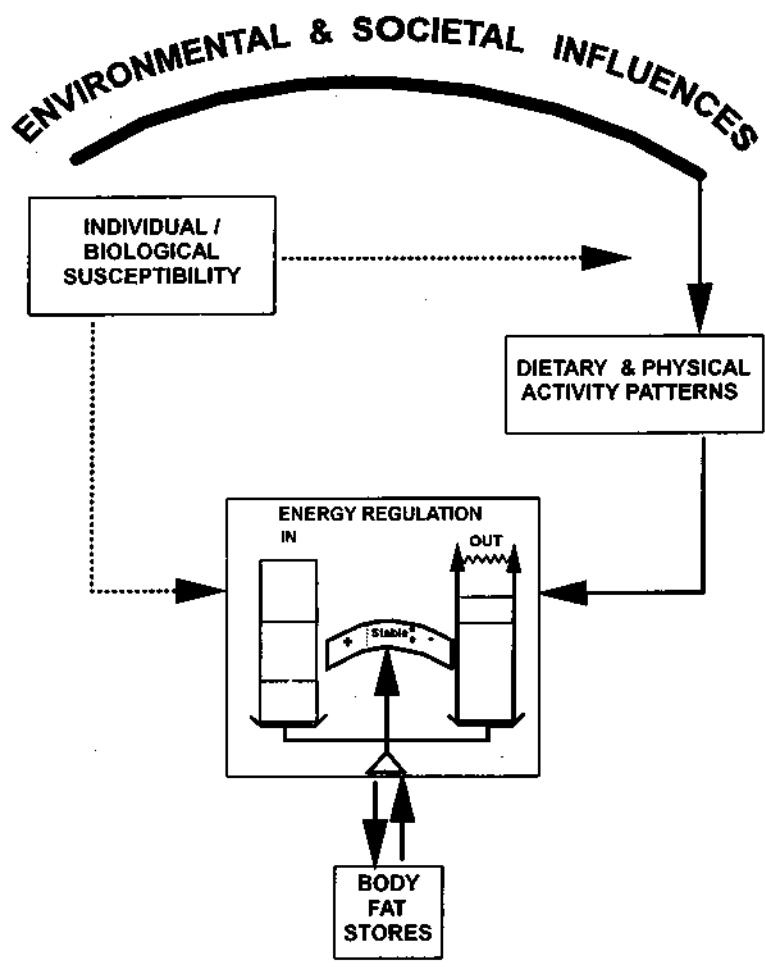


Figure 7.7 shows that powerful environmental and societal influences influence energy intake and expenditure through their effects on dietary and physical activity patterns. They may overwhelm the physiological regulatory mechanisms that operate to keep weight stable.

7.4.1 Changing societal structures

The process of modernization and economic transition has seen most countries of the world move towards industrialization and an economy based on trade within a global market. This has brought about a number of improvements to the standard of living and services available to people throughout the world. However, it has also had a number of negative consequences that have directly and indirectly led to deleterious nutritional and physical activity patterns that contribute to the development of obesity. Changing societal structures resulting from this transition have given rise to new problems associated with unemployment, urban crowding, and family and community breakdown. Social dislocation has often followed the loss by indigenous populations of traditional lands given over to production for the export market (61).

Table 7.3 Examples of energy-saving activity patterns in modern societies

Transport	The recent dramatic rise in car ownership means that many people now travel short distances by car, rather than walking or cycling to their destination.
In the home	Ready fuel supplies overcome the need to collect and prepare alternative sources for lighting and heating, and central heating reduces the need to expend energy for thermo-regulation. Energy expenditure is also reduced through the use of cooking equipment and ready-prepared foods/ingredients during food preparation. The use of washing machines and vacuum cleaners makes for easier and quicker cleaning.
In the workplace	Mechanization, robotics, computerization and control systems have markedly reduced the need for even moderate activity, and only a very small proportion of the population now engage in physically demanding manual work.
Public places	Elevators, escalators and automatic doors are all designed to save substantial amounts of time and energy.
Sedentary pursuits	Television viewing has been identified as a major cause of inactivity, especially in the obese (64). Data from the USA showed that television viewing was strongly related to the onset of new cases of obesity and to the lack of remission among obese children (37). These results are consistent with recent intervention research that found notable reductions in obesity when reductions in television viewing time were included in a dietary and activity intervention (65). The average person now watches over 26 hours of television a week in England, compared with 13 hours in the 1960s (66), and children in the USA can spend more time viewing television than attending school (67). Data are needed from other countries and for other sedentary pursuits such as video watching and computer work.
Urban residence	In urban areas of the more affluent countries, children, women and older people are dissuaded from going out either alone or at night because of fear for their personal security. Children also have difficulty in playing on local streets because cars use these to bypass areas of congestion (68). For active leisure pursuits, children and adults therefore usually travel by car to a sports facility or to the open country as a special outing, rather than taking exercise routinely as a part of their daily lives. Further research is needed in this area to determine the relative importance of such factors, and indeed whether or not they have an impact on obesity.

The food system that has emerged today uses an industrial approach to agriculture and food production, overcomes seasonality for most foods, and supplies highly processed outputs. While this may have contributed to improved food availability, it has not necessarily solved the problem of undernutrition in many of the poorer countries, nor has it improved the nutritional quality of the diets of the affluent (62). Indeed, some changes in the industrialization of food production have contributed to the consumption of a diet higher in protein and fat (particularly saturated fat) and lower in complex carbohydrate.

The decline in energy expenditure seen with modernization and other societal changes is associated with a more sedentary lifestyle in which motorized transport, mechanized equipment, and labour-saving devices both in the home and at work have replaced physically arduous tasks (Table 7.3). Work-related activity has declined over recent decades in industrialized countries, while leisure time dominated by television viewing and other physically inactive pastimes has

increased (49). In the UK, for instance, the average distance walked by English children 14 years and younger fell by 20% between 1985 and 1992, and the average distance cycled fell by 26%, while the average distance travelled by car increased by 40% (63). The dangers of traffic and fear for personal security have also influenced the decline of play in public areas.

This section outlines some of the key changes in societal structures that are thought to underlie the observed adverse changes in dietary and physical activity patterns implicated in the rapid global rise in obesity.

Modernization

Most adults who still live a "traditional" lifestyle appear to gain little or no weight with age. Anthropometric studies have reported an absence of obesity in the few remaining hunter/gatherer populations of the world where energy expenditure is generally high and food supplies are periodically scarce (69). For the majority of the world's population, however, the process of "modernization" has had a profound effect on the environment and lifestyles over the last 50 to 60 years.

Food is now more abundant and the overall energy demand of modern life has dropped appreciably. These changes have subsequently been associated with dramatic increases in obesity rates. Indeed, Trowell and Burkitt's 15 case-studies of epidemiological change in modernizing societies conclude that obesity is the first of the so-called "diseases of civilization" to emerge (70). While those earlier reports pointed out that obesity usually emerged first in middle-aged women and then in middle-aged men, particularly among the more affluent groups, in the last decade it is clear that obesity is increasingly seen in much younger age groups, e.g. in children and adolescents. Trend or longitudinal data generally indicate that steady increases in the rates of obesity are greater in urban areas (71,72). However, a recent report from Samoa noted a dramatic increase in obesity prevalence of 297% in men and 115% in women in a rural community (73). This was clearly apparent even in the 25- to 34-year age group in both sexes.

New World syndrome

Obesity can be seen as the first wave of a defined cluster of NCD now observed in both developed and developing nations. This has been described as the "New World syndrome" (74) and is already creating an enormous socioeconomic and public health burden in poorer nations. High rates of obesity, NIDDM, hypertension, dyslipidaemia and CVD, coupled with cigarette smoking and alcohol abuse, are closely associated with the modernization/acclimation process and growing affluence. The New World syndrome is responsible for disproportionately high levels of morbidity and mortality in newly industrialized countries, including Eastern Europe, as well as among the ethnic minorities and the disadvantaged in developed countries (74). Thus, while obesity is viewed by health professionals from a medical perspective, it also needs to be recognized as a symptom of a much larger global social problem.

Economic restructuring and transition to market economies

The world is going through a period of rapid economic transition. Economies based on a few primary commodities are no longer feasible, and a great deal of investment is often required to modernize existing industries and infrastructure in order to compete in a global market.

For many countries, economic transition has meant huge loans from international banks as well as the investment of large multinational companies on terms more favourable to the company than to the host country. Rising interest rates have crippled health, education and social services due to capital and interest repayments, and local economies have been restructured to rely on industries based on cheap labour (62). Thus, many developing countries are becoming increasingly reliant on imported non-traditional foods, have very high rates of unemployment, and have seen a population shift to urban areas in search of work which is becoming increasingly sedentary in nature (75,76).

Increasing urbanization

In less developed countries, urban residents are generally taller, heavier, and have a higher BMI than those who live in rural areas (71,72). This association between urban residence and obesity is of particular concern given the increasing numbers of people living in urban areas. Europe and North America are no longer the only major urban regions of the globe. In the post-World War II period, the proportion of people who live in the urban areas of less developed countries has increased from 16.7% in 1950 to 37% in 1994, and is predicted to grow to 57% in 2025 (77). Furthermore, there has been a shift towards concentrated population growth in a few large cities of more than 5 million, often called urban agglomerations, and a shift in poverty towards the urban areas, particularly into squatter and slum zones.

Urban residence is associated with a wide range of factors which in turn affect diet, physical activity and body composition. These include changes in transportation, access to and use of modern educational and health facilities, communications, marketing and availability of food, and large differences in occupational profiles, among others. In most countries, urban residents consume smaller proportions of carbohydrates and greater proportions of protein and fat, particularly saturated fat (78).

Changing occupational structures

In industrialized societies, an increasing number of women are entering the job market or are returning to full- or part-time paid employment within a few years of childbirth. They still tend to take responsibility for the health and well-being of the family but the more time- and energy-consuming domestic chores concerned with cleaning and the preparation and serving of food are declining.

Working in the labour force has given women relatively more economic influence, especially over domestic purchases, and has contributed to the demand for convenience foods and labour-saving devices such as the microwave oven. People in paid employment tend to allocate less time for

shopping, cooking and other household tasks, and so demand for food products with an inbuilt convenience component has risen accordingly. Indeed, it is predicted that by the year 2000, 90% of retail food products will be prepared to a certain degree in some countries, and it is quite conceivable that a proportion of the population will not know how to cook at all (79). People may no longer have the time, energy, motivation or skills to prepare food from the basic ingredients. In the USA, the percentage of food dollars spent outside the home increased by about 40% between 1980 and 1990 (80).

Changing societal structures have also led to an increasing proportion of the population working in service, clerical and other professional occupations that demand considerably less energy expenditure than the physically demanding manual work of more traditional societies.

Globalization of world markets

Food and food products are now commodities that are produced, traded and sold for profit in a market that has extended its reach from a largely local level to one which is increasingly global. Foods are less often seen as a matter of life and death, or of religious or cultural meaning. Manufacturers and retailers seek to minimize uncertainties and costs, and to maximize returns. Competition is high, both within and outside areas of operation (81).

Large companies have expanded to control ever-increasing shares of trade in agriculture, manufacturing and retailing, with smaller farms and groceries being squeezed out of business (82). The effects of the Third World debt crisis, the collapse of communism in Eastern Europe and the former Soviet Union, and the dominance of free-market ideologies are helping globalization and the development of market economies throughout the world, drawing even the most isolated self-provisioning peasants into a global market (62). The concentration of food power into a smaller number of multinational companies reduces the need for responsiveness to consumer or government pressure, and increases their influence on government policy (83).

7.4.2 Variation within societies

Socioeconomic status and obesity

SES is usually presented as a composite index combining income, education, occupation and, in some developing countries, place of residence (urban/rural). However, the individual components of SES may have independent and even antagonistic effects on dietary intake and physical activity patterns so it is often very difficult to make generalizations about the relationship between SES and obesity.

Despite the difficulties outlined above, studies have repeatedly shown that high SES is negatively correlated with obesity in developed countries, particularly among women, but is positively related with obesity in populations of developing countries (84,85). Further evidence suggests that, as the less developed countries attain higher levels of affluence, the positive relationship between SES and obesity is slowly replaced by the negative correlation seen in developed countries (78).

Developing countries

In developing countries, the lesser obesity rates observed in the lower SES populations are associated with a situation where people are limited in their ability to provide enough food, yet still engage in moderate to heavy manual work with little access to public transport. Hence, thin adults are considered poor, and overweight and obesity are a sign of affluence.

However, as per capita income increases, the nature of the diet in traditional societies tends to change in a pervasive and well-documented manner (86). In particular, intakes of:

- Animal fat and protein increase
- Vegetable fat and protein decrease
- Total, and particularly complex, carbohydrates decrease
- Sugar increases

The increase in income may be associated with increased away-from-home consumption of high-fat food items, as in the Philippines, or with increased consumption of meat, as in China. However, the overall effect tends to be a greater intake of total fat and an increased prevalence of obesity (78).

Developed countries

Developed countries tend to show an inverse relationship between obesity and SES, and between obesity and income, especially among women. A state of food deprivation is now very unusual in any substantial sector of industrialized countries and the proportion of adults engaged in physical activity at home has fallen substantially with modernization. Thus, the lower SES groups need to be no more physically active or short of food (in energy terms) than those in an upper SES category. In fact, studies suggest that families from the lower SES groups engage in much less physical activity than those in professional groups; for instance, they have developed obesity in parallel with rising car ownership and watch television for many more hours per day (36).

Studies indicate that change in income has little effect on dietary structure in countries where income levels are already quite high in relation to the basic food needs; instead, increases in income are spent on more elaborate packaging and processing, or on higher quality foods rather than on the quantity of food. In the poorest income groups, however, food demand is much more price- and income-sensitive, with many people struggling to obtain enough quality food for what is considered to be a healthy diet (87). The diet of lower SES households tends to be energy-dense, with high-fat intakes a prominent feature; the more expensive vegetables, fruit and whole-grain cereals are eaten more sparingly.

Education and health-related knowledge

Level of education appears to be inversely associated with body weight in industrialized countries. Surveys in France, the UK, and the USA all found that the proportion of obese men and women was higher among those with a lower educational level (88,89). The observed inverse relationship between education and body weight may be partly attributed to the fact that individuals with higher education levels are more likely to follow dietary recommendations and adopt other risk-avoidance behaviours than those with low educational attainment (90). In the USA, a trend has been emerging among the more educated segments of the population to adopt and adhere to dietary guidelines and other “healthy lifestyles” (78). Unfortunately, little is known about the relationship between education level and obesity in developing countries except that urban adults are more highly educated than those from rural areas.

The benefit of nutritional knowledge *per se* appears to be limited. Surveys indicate that although some people know how to follow a healthy diet, they prefer to consume a relatively unhealthy diet in practice (91,92). Obesity rates continue to climb, despite the increased percentage of dieting in obese people, suggesting that knowledge and frequent attempts to slim are insufficient for successful weight control (92). However, without these widespread attempts to control body weight the prevalence of obesity in industrialized countries might be much higher.

7.4.3 Cultural influences

It is also essential for any international review of obesity to recognize that at least two-thirds of the world’s population is comprised of communities of Chinese, Asian Indian or African origin, and from developing countries. In these societies, the risk factors and perceived causes for obesity often differ from those societies of European origin.

Culture affects both food intake and physical activity patterns, although the characterization and measurement of the “cultural attributes” responsible are not well developed at present. Cultural behaviours and beliefs are learned in childhood, are often deeply held, and are seldom questioned by adults who pass this knowledge to their offspring. Attitudes and beliefs may change over time, however, as shown by expectations in industrialized countries of body weight and shape which appear to be of particular importance in determining people’s behaviour. Substantial differences in obesity prevalence between relatively affluent populations indicate that cultural values and traditions may mediate or moderate the effects of affluence on obesity rates.

Cultural influences on food intake, selection and preparation

Cultural factors are among the strongest determinants of food choice. These include peer group pressures, social conventions, religious practices, the status value afforded to different foods, the influence of other members of the household and individual lifestyles. These are often reflected in children adhering to peer pressure by selection of high-fat food choices, and executives dining extravagantly with business colleagues.

Cultural explanations of obesity subsume aspects which are traditionally thought of as “learned” behaviours. For example, it is not uncommon for white American parents to encourage their children to eat particular foods by rewarding them with other food items. Recent research has shown that this culturally sanctioned pattern of rewards actually contributes to a dislike of the “good” foods and a preference for the “bad” foods (93). In some cultures, high-fat meals are provided for family entertainment and celebration.

Few foods are unique to particular cuisines, although some may be considered suitable for consumption by one culture but not by another. Human beings value food for a lot more than its nutrient content and food is used to express relationships between people as well as celebrating religious festivities, weddings and other important social occasions.

Attitudes towards health, fitness, and activity

The concept of engaging in physical activity during leisure time is not recognized in many cultures and communities in which energy conservation has historically been a prime concern during times of food shortage. The improvement in food availability has done little to change such attitudes to physical activity, which often persist across generations even though the original rationale for their adoption has been lost.

In contrast, the Nordic countries and some other cultures prize fitness and vitality and thus have a positive attitude to physical activity; they devote considerable amounts of leisure time to vigorous activity at the expense of more sedentary pursuits.

Body image

Throughout most of human history, an increased weight and girth have been viewed as a sign of health and prosperity. This is still the case in many cultures, especially where conditions make it easy to remain lean or where thinness in babies is associated with increased risk of infectious disease. Fat women are often viewed as attractive in Africa, for example, where some traditional communities have “fattening huts” for elite pubescent girls to ensure that they start their reproductive lives with a peripheral fat energy surplus (85). In Puerto Rican communities, weight gain after marriage is a positive reflection on the husband as a good provider and on the woman as wife, cook and mother. Weight loss is socially discouraged and there is a widespread fatalistic concept concerning the possibility of successful weight loss for the obese (94).

In many present-day Western cultures, the last three decades have witnessed a marked change in expectations of body shape and weight. Thinness in women has come to symbolize competence, success, control and sexual attractiveness, while obesity represents laziness, self-indulgence and a lack of will power (95). Ideals of thinness occur in a setting where it is easy to become fat, and tend to lead to inappropriate dieting, to a failure to achieve unrealistic weight goals and to weight cycling. Recent research suggests that, as many traditional cultures embrace the values and ideals of a politically or economically dominant culture, they too are likely to see an increase in eating disorders and unhealthy weight control practices (96,97). In the USA, concern about overweight is seen in a variety of ethnic groups (98) although the preferred “unhealthy” method of weight

control tends to vary; compared to white adolescent females, Hispanics reported greater use of diuretics, Asians reported more binge eating, and blacks reported higher rates of vomiting (99).

Cross-cultural research reveals that the male body ideal is most often related to “bigness” (large structure and muscularity), but not necessarily to fatness (69,100). In contrast to women, men generally do not recognize their increased size and adiposity as so much of a problem. This issue is of concern because men are more at risk of developing abdominal obesity yet tend not to present for needed treatment (27).

Television and popular magazines have been criticized for reinforcing the association of thinness with attractiveness (101,102), especially when they present conflicting messages in the form of abundant advertisements for energy-dense and high-fat foods. Media exposure and the presentation of thin female models as ideal increase in many women their dissatisfaction with their body shape and promote symptoms of eating disorders (103,104). As many societies have not stigmatized obesity or created eating disorders, efforts are required to ensure that the media do not induce or exacerbate such a situation.

7.4.4 Sectors influencing the impact of societal changes on food intake and activity patterns

A number of sectors, including governments, the food industry, the media and consumers, have the potential to influence, positively and negatively, the impact that societal and environmental factors, particularly modernization, have on the food supply and PALs. No one sector alone has been responsible for creating an obesity-promoting environment any more than, acting alone, a single sector can effect meaningful change. Thus a partnership between sectors is clearly required to deal positively with the situation. The influences of some of the sectors are discussed further below.

Government and regional authorities

Governments and regional authorities have a responsibility to protect and promote the health of the community through ensuring access to a safe, nutritious and affordable food supply as well as to facilities for performing regular physical activity. The increasing levels of modernization and competing demands of economic development and health have sometimes created a situation where actions by governments have contributed to a decrease in physical activity and an increase in the intake of an energy-dense diet contrary to their own health guidelines.

Development and adaptation of national dietary guidelines

Dietary recommendations and food-based dietary guidelines have often not kept pace with societal changes, with advances in nutrition science or with the specific nutritional problems of communities as countries move through nutrition transition.

Government nutrition programmes

Government feeding programmes in developing countries set up to deal with undernutrition often remain in place even when there is evidence to suggest that such nutrition priorities are no longer relevant. In some cases, these programmes may contribute to a worsening of the problem of over-consumption of energy which occurs with modernization.

Meals provided in government institutions

Governments and regional authorities have responsibility for the food served in schools, hospitals, day-care centres and government organizations. Even when provided by external organizations, they have the capacity to lay down firm guidelines for the quality and composition of such meals. Unfortunately, many have failed to provide and monitor guidelines for the provision of meals in such outlets.

Physical activity at school

Governments and regional authorities are in a position to ensure that regular physical activity is included in school curricula. However, many have allowed a reduction in regular physical activity at schools and have made land originally devoted to safe playing available for other uses.

Regulation of food quality, advertising and labelling

Many governments have failed to respond to the changing food supply by developing or adjusting food regulations that control food quality and safety, the labelling of foods and the advertising of foods. This has led to a situation where consumers are at risk of being ill-informed or confused by poor labelling or unregulated marketing of foods. A recent report by Consumers International (105) has shown that, even when regulations governing marketing and advertising exist, they are often not enforced and so adherence is poor.

Food production policies

Economic development and increasing involvement in free markets often result in governments abandoning a food production policy based on small regional food producers and changing to one that involves large-scale or centralized farming. Such policies often increase the movement of people from a rural to urban setting and can result in a loss of diversity of food production of traditional foodstuffs in favour of wide-scale production of cash crops demanded by export markets.

The focus in many developing countries is still on increasing the total food energy available to the population so that the problems of under-nutrition are avoided. However, concentration of food production on oil crops or meat products may add to problems associated with the rapidly increasing energy density of the national diet, especially when these products make their way into the local food supply and displace the traditional foods that are no longer widely available.

Food surpluses

For many decades, the primary objective of governments and the food industry has been to maintain a cheap supply of food so that even the poorest sections of society could purchase sufficient amounts. Use of tax concessions, direct subsidies and rebates from the producer to the retailer, however, have often led to an oversupply of commodities, and so economic strategies now tend to be directed at increasing consumer demand to meet supply. As a result, surplus cheaper foodstuffs are exported from developed countries to markets created in developing countries (106). This is illustrated by the export of cheap vegetable fats from Australia, the USA and Europe to neighbouring countries in the Pacific, South America, Asia and Eastern Europe (76,107).

The food industry

Advances in food technology and product development

Technological advances in cultivating, preserving, producing, transporting and storing foods have increased the availability of a wider variety of foods to a larger number of people year round. The continuing globalization of these processes means that such trends in food availability are extending from industrialized societies to developing countries.

Advances in food technology have also contributed to the consumption of diets which are increasingly dependent on processed foods. It is now possible to produce food products with an almost limitless combination of taste, textural quality and nutrient content. In fact, food characteristics are often so manipulated that it is difficult for individuals to associate visual, textural or taste cues with the energy content of meals. This is especially important given the increasing trends towards pre-packaged foods and the concomitant decline in use of natural and basic ingredients for food preparation in the home (79). Consumers are losing control over the preparation of the foods which they eat and food composition is being placed in the hands of manufacturers.

In order to survive in the competitive market economies of the 1990s, businesses cannot stand still but need to grow and maintain or increase profits for shareholders. With food, if this cannot be done by increasing sales of basic foodstuffs to those who can afford it, it can be done by turning basic foodstuffs into other, more expensive products (62).

Fast foods

Although it may be argued that "fast foods" have been available for centuries, the foods provided tended to be those of traditional diet and culture. Today, fast foods and snacks tend to be universal in nature, often provided by large multinational franchises, and are high in fat, low in complex carbohydrates, and energy-dense (108). These foods may be less satiating and are often used as regular additions to the diet instead of being consumed as the occasional meal or treat (27). Furthermore, beverages containing substantial amounts of sugar or alcohol are often consumed as part of the fast-food meal.

Modern fast foods have proliferated rapidly, are widely available and are intensively advertised. In 1991, Euromonitor reported that fast foods accounted for 19% of the global consumer catering market, then worth US\$ 730 000 million, and expected them to grow to 25% of the market by 2000. In the USA, the market for fast foods accounted for US\$ 78 000 million in 1992 (109) and more than 200 people are served a hamburger every second of the day. Changes in availability have been implemented by an increased number of outlets and increasing opportunities to eat outside the home; the number of fast-food outlets in the UK doubled in the 10 years between 1984 and 1993, while the number of restaurants and cafes remained stable (110).

Direct evidence that increased consumption of fast foods leads to overweight and obesity is lacking. However, it is widely perceived that this is the case and that obesity has increased in industrialized societies as families turn away from home-prepared meals and utilize more fast or take-away foods. The roles of the media and of the consumer in this process are discussed in the later section on consumers.

Marketing and advertising

The commercialization of food manufacturing and retail outlets has encouraged enthusiastic marketing. Larger portion sizes give the consumer an impression of "better value" for money, and marketing strategies such as "eat all you can for X dollars" represent an encouragement to eat beyond natural biological limits. Furthermore, these foods and outlets are backed by substantial advertising campaigns which, in stark contrast to public health or nutrition campaigns, are extremely persuasive and successful (27).

The media

The various communications media, including television, radio and print, play a major role in information dissemination in modern consumer societies. They are part of informal education and both reflect and influence public attitudes. However, the money spent on promoting high-fat/energy-dense foods has been considerably more than that spent on promoting healthier foods. For example, £80 million was spent on promoting chocolate consumption in the UK in 1992 compared to only £3 million spent on promoting fruit and vegetable consumption.

The media provide information on new and existing foods to consumers and have a pervasive influence on food choice; they have clearly been influential in changing dietary patterns over recent decades. Television, in particular, plays a major role in informing and influencing children. This development may not be helpful. For example, 91% of foods advertised during peak children's viewing time in the USA, and a similar proportion in the UK, were high in fat, sugar, and/or salt (111,112). Although the food and advertising industries consistently argue that food advertising has little influence or detrimental effect on children's eating habits, a considerable amount of evidence now suggests that it does influence food selection in children and adolescents, especially among susceptible groups (62,95,113). Television viewing appears closely linked to the consumption of foods advertised on television for children (114,115).

Consumers

Consumers play a role in fuelling a demand for a wide variety of products and services which are conducive to weight gain; they often demand processed and convenience meals which tend to be high in fat and energy-dense, as well as labour-saving devices both at home and in the workplace which require little energy expenditure. Although it is recognized that consumer demand is itself influenced by a number of factors, including marketing, advertising, culture, fashion and convenience, the product or service is unlikely to survive in its existing form if consumers do not want it. Better educated consumers can demand better products, especially with respect to improved nutritional quality.

Most societies have a preference for sweet foods and prize fatty foods the most (116). With increasing incomes and availability of such foods, there has been a marked increase in the intake of such foods. The ability to purchase labour-saving devices is widely welcomed by consumers in all societies and ownership of a car is seen as an important symbol of status. Consumers in emerging economies are likely to be reluctant to return to diets of traditional foods, to physical labour or to walking associated with poverty once a certain level of income has been achieved.

7.5 Individual/biological susceptibility

Epidemiological, genetic and molecular studies suggest that there are people who are more susceptible than others to becoming overweight and obese. These observations have been made in populations all over the world, indicating that susceptible individuals can be found across a wide range of lifestyle and environmental conditions.

Obesity is commonly recognized as a complex multifactorial disease; it is a condition resulting from a lifestyle which promotes a positive energy balance, but also one that becomes manifest more readily in people who have an inherited susceptibility to be in positive energy balance. Furthermore, no two obese individuals are the same; there are differences in both the degree and the regional distribution of excess body fat as well as in the fat topography response of individuals to factors which promote weight gain. Such differences are not only due to genetic variation but also to prior experiences and environments to which the individuals have been exposed. The evidence for this conclusion has been carefully reviewed (117). However, there remains considerable uncertainty about the genes and mutations that are involved, and how they operate and interact to enhance the susceptibility of some individuals to obesity.

This section discusses briefly the evidence for a role of genetic, biological and other factors in determining the susceptibility of individuals to weight gain and obesity (Figure 7.8).

7.5.1 Genetic susceptibility

The role of genetic factors in weight gain is currently the focus of much research, and the discovery of leptin has led to a renewed interest in genetic and metabolic influences in the development of obesity. While it is possible that single or multiple gene effects may cause

overweight and obesity directly, and indeed do so in some individuals, this does not appear to be the case in the majority of people. Instead, it is currently considered that the genes involved in weight gain increase the susceptibility or risk of an individual to the development of obesity when exposed to an adverse environment. Only in the case of certain genetic disorders are particular gene effects “necessary” for obesity expression.

Figure 7.8 Influences on energy balance and weight gain (individual/biological susceptibility)

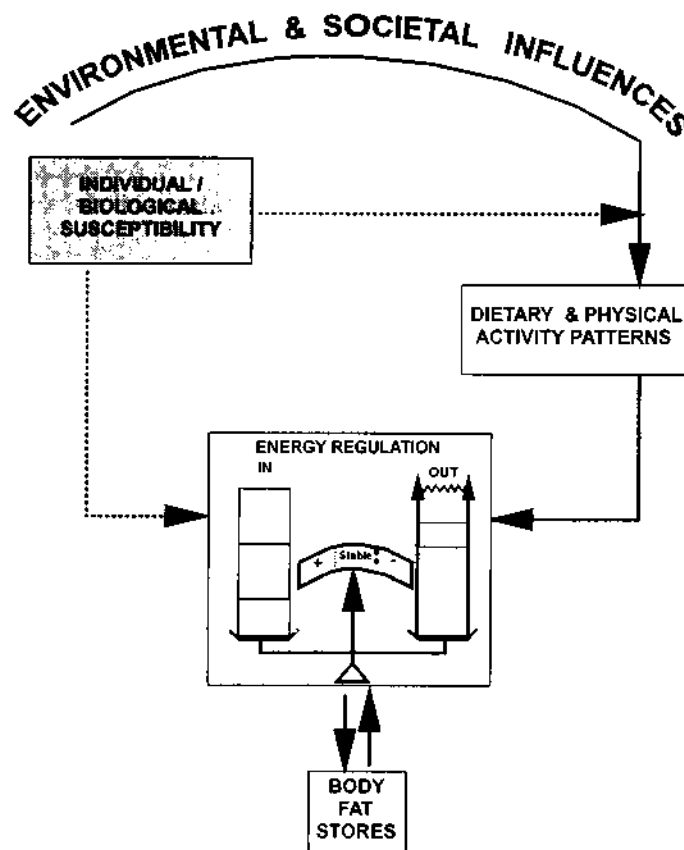


Figure 7.8 shows that the susceptibility of individuals to the obesity-promoting environmental and societal forces is affected by genetic and other biological factors such as sex, age and hormonal activities, over which they have little or no control.

Heritability

A large number of twin, adoption and family studies have attempted to establish the heritability of different measures of obesity. The level of heritability is the fraction of population variation in a trait (e.g. BMI) that can be explained by genetic transmission. Adoption studies tend to generate the lowest estimates and twin studies the highest levels. Recently, however, the application of complex analytical techniques to databases encompassing all three types of studies has led to the conclusion that the true heritability estimate for BMI in large sample sizes was likely to be from 25% to 40% (118,119). On the other hand, similar genetic epidemiological research

has shown that the profile of fat distribution was also characterized by a significant heritability level of the order of about 50% of the total human variation. Finally, recent studies have shown that the amount of abdominal fat was influenced by a genetic component attaining 50% to 60% of the individual differences (120,121).

Obesity tends to run in families, with obese children frequently having obese parents. However, there is a dearth of data concerning the level of risk of developing obesity for a first-degree relative of an overweight, a moderately obese or a severely obese person in comparison to the population prevalence of the condition (119). A first paper on this topic by Allison et al. (122) concluded that the risk was about 2 for overweight, increasing to about 3 to 4 for growing levels of obesity.

Gene-environmental interactions

While some individuals are prone to excessive accumulation of fat and find weight loss a continuous battle, others seem relatively protected against such a problem. Evidence to suggest that genetic factors are partially responsible for such differences in the sensitivity of individuals to gain fat when chronically exposed to a positive energy balance comes from studies in both animals and humans.

Using animal models, for example, scientists have shown that feeding a high-fat diet to different inbred strains of mice identifies sensitive as well as resistant strains (123). More recently, a prospective study found that high-fat intake in humans was correlated with subsequent weight gain only in those subjects who were overweight at baseline and had obese parents (124). These studies and others suggest that the genetic predisposition to obesity observed in animal models may also apply to humans, making some individuals particularly susceptible to a high-fat intake.

It is also quite clear that there are inbred strains of rodents that are particularly prone to becoming obese when exposed to overfeeding or to a highly palatable diet. Similarly, in a study with pairs of identical twins, the amount of body weight and the proportion of fat gained in response to controlled overfeeding was significantly more alike within pairs than between pairs of twins (125). This study and others based on the same design strongly suggest that there are individuals who are more likely to gain body mass and body fat than others when challenged by an energy overload. Thus, the responsiveness to energy intake and dietary composition is partly dependent on specific genetic factors that remain to be clearly identified.

Type of genetic effects

If the heritability estimates are correct—and the evidence is quite strong in that direction—the genes are exerting their influence on body mass and body fat as a result of desoxyribonucleic acid (DNA) sequence variation either in the coding sequence of the genes or in the segments that affect gene expression. It is obvious that most of the genes contributing to obesity do not qualify as necessary genes, that is, genes that cause obesity whenever one or two copies of the deficient allele are present. Indeed, the genetic susceptibility seems to be rather one caused by genes associated with an increase in the proneness to gain weight over time or, alternatively, to the

absence of genetic influences which protect against the development of a positive energy balance. In general, such genes exert smaller effects than necessary genes on the phenotype, a situation that makes the identification of these genes and of the responsible mutations much more difficult. Nonetheless, even though the genetic effect associated with the risk of obesity appears to be of the multigenic type, there is some indirect evidence supporting the notion that one or a few genes may play a more important role. In other words, obesity is truly a complex multifactorial phenotype with a genetic component that includes both polygenic and major gene effects.

A series of studies reported over the last five years or so strongly supports the view that many genes are involved in causing a susceptibility to obesity. Several types of research have been used to identify these genes and the specific DNA sequence variation responsible for the increase in risk of becoming obese. The evidence accumulated so far has been recently reviewed (126) and it indicates that statistical or experimental support is found for a role of about 70 genes, loci or markers. Many more years of research will be needed before the important genes and the critical mutations are finally defined for both excess body fat content and upper body and abdominal fat accumulation.

Possible mechanisms through which a genetic susceptibility may be operating include:

- **Low RMR:** e.g. studies in the Pima Indians have shown that the RMR clusters in families and those with lower RMR have a greater risk of gaining 10 kg in the following 5 years (127,128).
- **Low level of lipid oxidation rate:** e.g. a low ratio of fat to carbohydrate oxidation under standardized conditions is a risk for subsequent weight gain (17,129).
- **Low fat-free mass:** a low fat free mass for a given body mass constitutes a risk of subsequent weight gain as it tends to depress the level of RMR, thus favouring positive energy balance.
- **Poor appetite control:** e.g. if satiety is reached at a high level of energy intake, the net result is likely to be positive energy balance and weight gain. Here, many genes and molecules are currently under investigation. For instance, leptin, the hormone product of the *ob* or leptin gene, is an important satiety factor secreted by the adipose tissue in humans. An anomaly in the leptin receptor gene may potentially be associated with leptin resistance in humans. However, the genetic mutations which result in leptin insufficiency and lead to obesity in mice are not thought to exist in humans.

Many other potential mechanisms are currently under intensive investigation. The following table (Table 7.4) lists of several of these factors.

Table 7.4 Some factors involved in the development of obesity thought to be genetically modulated

Macronutrient-related
<ul style="list-style-type: none">• Adipose tissue lipolysis• Adipose tissue and muscle LPL activity• Muscle composition and oxidative potential• Free fatty acid and β-receptor activities in adipose tissue• Capacities for fat and carbohydrate oxidation (respiratory quotient)• Dietary fat preferences• Appetite regulation
Energy expenditure
<ul style="list-style-type: none">• Metabolic rate• Thermogenic response to food• Nutrient partitioning• Propensity for spontaneous physical activity
Hormonal
<ul style="list-style-type: none">• Insulin sensitivity• Growth hormone status• Leptin action

The place of genetic research into obesity

While research is important to identify potential genes for screening, and ultimately for therapy, the practical application of these discoveries will take many years to develop. The most important progress associated with genetic research into obesity at present is in understanding the pathophysiology of the disease.

7.5.2 Non-genetic biological susceptibility

In addition to the genetic influences discussed in section 7.5.1, a number of other biological factors have been shown to influence an individual's susceptibility to weight gain and the development of obesity. These are discussed below.

Sex

There are a number of physiological processes that are believed to contribute to an increased storage of fat in females. Such fat deposits are believed to be essential in ensuring female reproductive capacity. Studies in humans and animals indicate that girls exhibit a stronger preference for carbohydrate prior to puberty while boys prefer protein. However, after puberty, both males and females display a marked increase in appetite for fat in response to changes in the gonadal steroid levels. This rise in fat appetite occurs much earlier and to a greater extent in females (130).

Females have a tendency to channel extra energy into fat storage while males utilize more of this energy for protein synthesis. This pattern of energy usage, or "nutrient partitioning", in females contributes to further positive energy balance and fat deposition for two reasons. First, the storage of fat is far more energy-efficient than that of protein, and second, it will lead to a lowering of the lean-to-fat tissue ratio with the result that RMR does not increase at the same rate as body mass.

Ethnicity

Ethnic groups in many industrialized countries appear to be especially liable to the development of obesity and its complications. Evidence suggests that this may be due to a genetic predisposition for obesity which only becomes apparent once the individuals are exposed to a more affluent lifestyle. This is demonstrated graphically by the following:

- ***Pima Indians of Arizona:*** a tribe with a very high prevalence of obesity (131) who gained weight after abandoning their traditional lifestyle earlier this century.
- ***Australian Aboriginals:*** tend to have a high incidence of central adiposity, hypertension and NIDDM. These complications can be reduced or eliminated within a very short period by simply reverting to a more traditional lifestyle (132,133). Similar reductions in obesity and cardiovascular risk have been observed when natives in Hawaii have returned to a traditional diet from the usual modern diet (134).
- ***South Asians overseas:*** prevalence of NIDDM and mortality from CHD are higher in people of South Asian (Bangladeshi, Indian and Pakistani) descent living in urban societies than in other ethnic groups. This is related to a tendency to accumulate intra-abdominal fat compared to other populations for a given BMI (135).

It appears from the above that several ethnic groups are more prone to the risks of obesity when exposed to a lifestyle more common in industrialized countries. For the majority this problem seems to arise from the combination of genetic predisposition, a change from the traditional to a more affluent sedentary lifestyle and its accompanying diet. However, susceptibilities to the obesity co-morbidities are not uniform across groups. In Mexico, for example, NIDDM is more of a problem among the obese population than hypertension, whereas in other areas of the world CVD may be more common.

Other environmental factors may also be important in promoting obesity in ethnic minority groups in industrialized countries. This is illustrated in African Americans in the USA, where the highest rates of obesity are found in the poorest communities. In these populations, fat-rich energy-dense diets are likely to be cheapest, and reduced levels of activity stem from unemployment. Other factors associated with poverty may also be involved.

The problem of obesity in ethnic minorities demonstrates the need for targeted prevention and intervention strategies.

Vulnerable periods of life for weight gain

Although a general rise in body weight and a modest increase in percent body fat over the lifespan can be expected in developed countries, at least until 60–65 years of age (136), recent studies have shown the importance of nutrition during certain periods of life when an individual may be more vulnerable to the development of future obesity. However, until longitudinal studies have been completed, the contribution of each of the periods shown in Table 7.5 to the prevalence of obesity and its co-morbidities remains unclear (137).

Table 7.5 Vulnerable periods of life for the development of future obesity

Vulnerable period of life	Reason for increased risk
Prenatal	Nutrition during fetal life may contribute directly to the development of the size, shape and composition of the body, and to the metabolic competence to handle macronutrients. Close relationships exist between patterns of intra-uterine growth and the risk of abdominal fatness, obesity and their co-morbidities in later life (138–140).
Adiposity rebound (5–7 years)	BMI begins to increase rapidly after a period of reduced adiposity during pre-school years. This period coincides with increased autonomy and socialization and so may represent a stage when the child is particularly vulnerable to the adoption of behaviours that both influence and predispose to the development of obesity. There is controversy over whether early adiposity rebound is associated with an increased risk of persistent obesity later in life (141–143).
Adolescence	Period of increased autonomy which is often associated with irregular meals, changed food habits and periods of inactivity during leisure combined with physiological changes which promote increased fat deposition, particularly in females (144,145).
Early adulthood	Early adulthood usually correlates to a period of marked reduction in physical activity. In women this usually occurs between the ages of 15–19 years but in men it may be as late as the early 30s (146).
Pregnancy	It has been claimed that a mother's BMI increases with successive pregnancies. However, recent evidence suggests that this contribution is likely to be only an average of less than 1 kg per pregnancy although the range may be wide and is associated with total weight gained during pregnancy (147). Many study designs confound the changes in weight with ageing and changes in weight with parity (148). In many developing countries, consecutive pregnancies with short spacing are often associated with weight loss rather than with weight gain.
Menopause	In industrialized societies, weight generally increases with age but it is not certain why menopausal women are particularly prone to rapid weight gain. The loss of the menstrual cycle does affect food intake and reduces metabolic rate slightly, although most of the weight gain has been attributed to reduced activity (149).

7.5.3 Other factors promoting weight gain

An individual's sensitivity to weight gain may be amplified by certain factors such as smoking cessation, the development of a disease, or by therapy with drugs which promote weight gain as a side-effect. These are considered briefly below.

Smoking cessation

Smoking induces an acute rise in metabolic rate and tends to reduce food intake relative to non-smokers (2). It may also cause a longer-term increase in RMR although evidence for this is conflicting (150,151).

Smoking and body weight are inversely related (152), and smokers frequently gain weight when giving up the habit. Recently, Williamson et al. (153) studied a nationally representative cohort of smokers and non-smokers in the USA (1971 to 1984) and found that the mean weight gain attributable to smoking cessation was 2.8 kg in men and 3.8 kg in women. However, heavy smokers (more than 15 cigarettes per day) and younger people were at higher risk of major weight gain (>13 kg) after giving up smoking.

Notwithstanding the risk of gaining weight, it is important to understand that smoking cessation should be a higher priority than weight loss in smoking obese patients; a large number of prospective studies have shown that smoking has a larger impact on morbidity and mortality than any small rise in BMI (154–158). The beneficial effects of giving up smoking are unlikely to be negated by the weight gain that may follow.

Excess alcohol intake

The body is unable to store alcohol, and oxidation of ingested alcohol is given priority over oxidation of other macronutrients. Alcohol consumption therefore meets some of the body's energy needs, and allows a greater proportion of energy from other foods eaten to be stored (27). Alcohol intake is associated with increased risk of abdominal fat (157). However, in epidemiological studies, those with the greatest alcohol intakes tend to be thinner (158,159). This latter paradox may be the result of these people eating less and having much of their energy requirements met by alcohol (160).

Drug treatment

The use of certain therapeutic drugs can promote weight gain. These are listed in Table 7.6.

Adults on long-term corticosteroid therapy for rheumatoid arthritis may be at particular risk of weight gain, with the side-effects of the drug exacerbating the effects of limited physical activity.

Table 7.6 Drugs which may promote weight gain

Drug	Main condition treated
Tricyclic antidepressants, Lithium	Depression
Sulphonylureas	NIDDM
β -adrenergic blockers	Hypertension
Some steroid contraceptives	Contraception
Corticosteroids	Various diseases
Insulin	NIDDM
Cyproheptadine	Allergy, hay fever
Sodium Valproate, neuroleptics	Epilepsy
Phenothiazine	Psychosis
Pizotifen	Vasometric headache

Disease states

Certain genetic disorders, as well as some endocrinological conditions such as hypothyroidism, Cushing's Disease and hypothalamic tumours can cause weight gain. However, these are extremely rare causes of obesity, accounting for only a very small proportion of obesity in the population.

Major reduction in activity

In some individuals, a major reduction in activity without a compensatory decrease in habitual energy intake may be the major cause of increased adiposity. Examples include the weight gain often observed in elite athletes when they retire, in young people who sustain sports injuries, in young people in wheelchairs after accidents or in others who develop arthritis.

Change in social circumstances

Social factors such as marriage (161), birth of a child, a new job and climate change can all lead to negative changes of eating patterns and consequent weight gain.

Successful weight loss

Although many people are successful in losing weight, half to one-third of this weight loss is commonly regained over the following year (162). This weight regain is independent of the extent of initial weight loss or the techniques employed to assist weight loss. This is considered to be a particularly difficult period for weight regain prevention, because biological and behavioural processes act to drive body weight back to baseline levels (146). Despite the difficulty of successful long-term weight loss and maintenance, some people are successful over long periods (163). Studying these individuals may provide some clues as to their success in weight maintenance.

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