17

OBESITY
ITS PREVENTION

MARK L. WAHLQVIST

WHAT IS IT?

Obesity is that degree and distribution of body fatness which leads to an unacceptable morbidity and mortality. For healthy men, body mass as fat is about 15 to 21 per cent and for women about 24 to 30 per cent. The traditional 'gold standard' for assessment of body fatness has been whole body densitometry by underwater weighing, but even this has its limitations on account of small differences in the density of body fat from one site to another. It is also not readily accessible and is not applicable to the unwell.

Most usually, anthropometric techniques are used to assess the degree of body fatness. One such method is to examine the relationship between weight and height and to use a mathematical expression in which the effect of height-related tissue such as muscle and bone is minimised. Body Mass Index (BMI) is currently the most preferred mathematical expression; it is weight/height^2 in kg.m^{-2}, and the healthy range for men and women is similar, namely 20 to 25 kg.m^{-2}. It is a matter of judgement what constitutes obesity. On the basis of mortality rates, a BMI of 30 or more is taken to represent obesity. The range between 25 to 30, is regarded as overweight; there is not the same degree of confidence that adiposity is excessive, especially since some training methods, like weight building, can contribute to an increased BMI. In general, the BMI applies to individuals of average physical fitness (usually sedentary in industralised societies). An alternative anthropometric technique is to use skinfold thicknesses.[1] Grade of obesity, according to Garrow[2] are shown in Table 17.1. Durnin[3] has demonstrated that the sum of four skinfold thicknesses (triceps, biceps, subscapular, supra-iliac) is highly predictive of body fatness as judged by densitometry (Table 17.2). An advantage of making direct measurements of body fatness by skinfold is that there is less assumption about differences in

body composition for a particular weight/height relationship. However, the problems of measuring skinfolds include the co-existence of fluid retention, and the increased error of the method with increased degree of body fatness.

Table 17.1 Grade of obesity, according to Garrow

Grade	BMI	% body fat		Significance
		Men	Women	
0	20–24.9	15–21	24–30	Minimal mortality
I	25–29.9	21–27	30–40	Slight mortality increase, but important to prevent progress from this grade
II	30–40	–	–	At mid-point, mortality double that of Grade 0
III	> 40 > 45	– > 49	– > 57	Incompatible with normal employment or health

Table 17.2 General relationship between mean individual and sum of four skinfold measurements, age and percentage body weight as fat

Age	Skinfold (mm)					Fat (% of body-weight)
	Biceps	Triceps	Sub-scapular	Supra-iliac	Total	
Males						
17–19	4.6	11	11	14	40	15
20–29	4.8	9.8	12	16	42	15
30–39	7.1	11	19	24	62	23
40–49	7.1	11	17	19	55	25
50–72	6.6	11	21	21	61	28
Females						
16–19	8.7	16	14	16	55	26
20–29	11	21	18	18	66	29
30–39	15	25	24	21	85	33
40–49	14	23	22	19	78	35
50–68	15	25	24	23	86	39

Source: Durnin and Womersley (see Reference 3)

New technologies are now available for the assessment of body fatness, the most attractive and convenient of which are those based on electrical conductivity.[4] Bioelectric impedance is as convenient as the performance of an ECG.

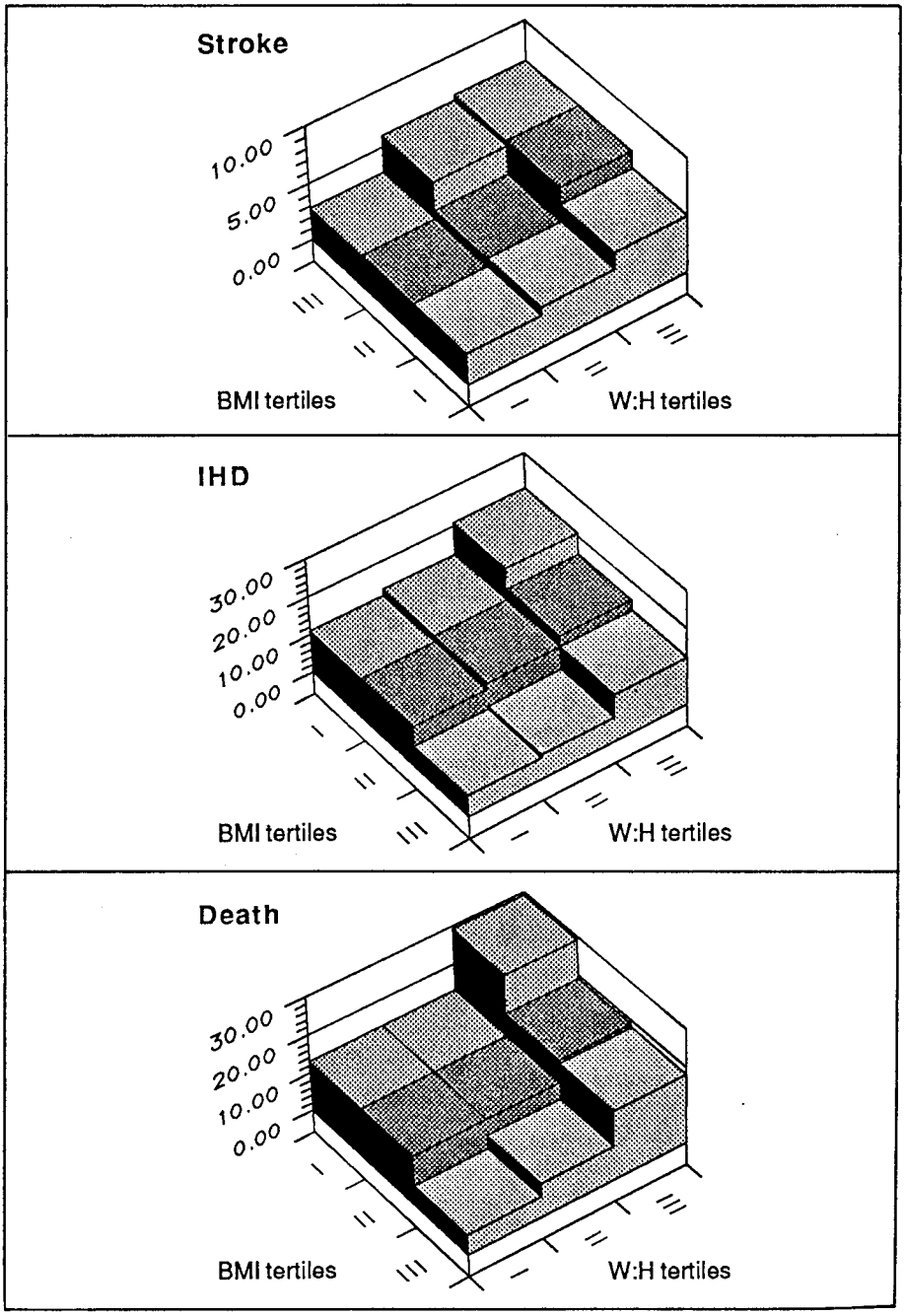

Figure 17.1

Contribution to abdominal fatness (waist/hip ratio), independent of BMI (body mass index) to various sources of mortality

In the assessment of significant adiposity, it is now clear that it is also important to assess the distribution of body fatness. Particularly persuasive have been studies from Gothenburg, Sweden (Figure 17.1[5]). For both men and women, they have shown that abdominal adiposity, in relation to hip circumference, is predictive, independent of BMI, of total mortality, ischaemic heart disease, stroke,[6] diabetes[7] and, in women, endometrial carcinoma.[8] How much of this effect of abdominal adiposity resides in subcutaneous fat and how much in omental fat is uncertain. There is some evidence from Björntorp's work that free fatty acid (FFA) flux from omental adipose tissue is itself a determinant of hyperinsulinaemia, a characteristic of Type II diabetes. On the other hand, in respect of ischaemic heart disease, Donahue and others have demonstrated that a subcutaneous measurement, subscapular skinfold thickness, is predictive, independent of BMI of coronary events.[9] Another outstanding question is the importance of waist circumference as reference in the assessment of abdominal adiposity. It could well be that there is intrinsic value in the degree of muscle mass at the hips for men and women, and, for women, fatness at the hips (given its potentially important energy storage function during reproductive life).

The prevalence of obesity depends on its definition. For a BMI greater than 30, 7 per cent of Australian men and 7 per cent of women are obese from the 1980 and 1983 NHF risk factor prevalence surveys.[10]

DOES IT MATTER?

Actuarial data and prospective studies have revealed considerable impact of obesity and its central distribution on mortality and morbidity rates (Table 17.3).

Table 17.3 Increased morbidity and mortality with obesity

Increased mortality
1 Cardiovascular Disease
2 Diabetes
3 Neoplastic disease (including leukaemia)
4 Gall bladder disease
5 Accidents
6 Suicide
Increased morbidity
1 Social penalties
2 Psychological penalties
3 Physical disability
a mechanical problems of mass
b cardiorespiratory demand
c joint disease
4 Medical problems
(as for mortality)

Source: Garrow (see Reference 50); Garrow (see Reference 1); Lapidus and others (see Reference 7).

Wilcken and colleagues in Sydney, have added to an understanding of left ventricular hypertrophy in obesity by showing that it is responsive to weight reduction, independent of changes in blood pressure.[11] This is of particular interest since, from the Framingham study of risk factors for coronary disease, we know that left ventricular hypertrophy is such a risk factor. It is possible that obesity operates, in part, through an increase in left ventricular mass to increase coronary risk.

MAKING OBESITY MORE OR LESS OF A PROBLEM

One obese person is not necessarily at the same risk as another. One possibility is that food intake patterns associated with lower risk for coronary heart disease, for example, may minimise the impact of obesity on that health problem.[12] Fish is a food which may decrease such risk.[13] Potentially protective dietary patterns include the traditional Mediterranean, Scandinavian, Chinese and Japanese.

Impaired glucose tolerance increases the risk of coronary death in obesity.[14]

The interaction between obesity and cigarette smoking is striking[15] (Figure 17.2[16]). In the American Cancer Society Study, for both men and women, the consumption of more than twenty cigarettes a day doubled mortality ratio in comparison with non-smokers of comparable adiposity. It is safer to be over-fat than to be a smoker. Although smokers are generally leaner than non-smokers, it does not make sense to use cigarette smoking as a way to lose fat – certainly not from a health point of view.

AGE AND OBESITY

Obesity can have its onset at any age. There is frequently resolution of infantile obesity with time,[17] but resolution of obesity later in childhood is less.[18] The likely significance of obesity in puberty is difficult to evaluate on account of the growth spurt and changing relations between weight and height; moreover, dietary restriction at this stage is hazardous because of potentially adverse effects on growth and development. The prevalence of obesity decreases in early to mid-adolescence, especially in males. In late adolescence and early adulthood, presumably because of a failure to reduce energy intake after the growth spurt (or to increase energy expenditure), it again increases.[19] A younger adult woman may have been obese since childhood, adolescence or after pregnancy. Failure to return to pre-pregnancy weight post-partum is a particular problem for women who may not have needed previously recommended increments in energy intake during pregnancy.[20] However, food restriction during pregnancy can be a risk for the unborn child.[21]

In later life, a reduction in energy expenditure may not be accompanied by a reduction in energy intake with a consequential increase in the level of adiposity.[22] However, weight may change little while adiposity increases at the expense of a decrease lean body mass.[23] The impact of obesity on morbidity

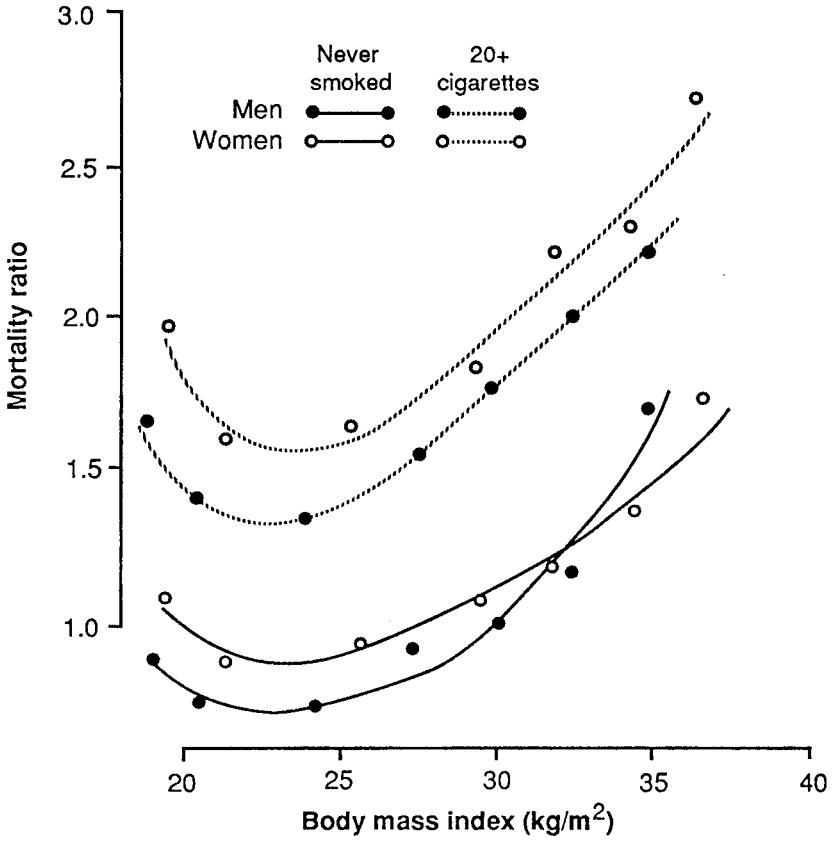

Figure 17.2

Effect of smoking on mortality rates in obesity

and mortality, while still a problem in the elderly, is less so than in younger life.[24]

GENES VERSUS ENVIRONMENT

It is now quite clear, notably from two important studies of Stunkard and colleagues, the Danish adoption study (Figure 17.3[25]), and an American twin study,[26] that obesity has an important familial, presumably genetic, basis.

It remains a possibility that the intra-uterine environment, as a product of maternal non-genetically based factors, contributes to obesity later in life. It is of interest that in a study of infants born to overweight mothers, reduced energy expenditure, particularly by way of physical activity, was a factor in rapid weight gain during the first year of life.[27] It has been shown further, in south-western American Indians, that a low rate of energy expenditure may contribute to the aggregation of obesity in families.[28]

The various studies which demonstrate familial bases for obesity should not be interpreted as a case for little likelihood of prevention through environ-

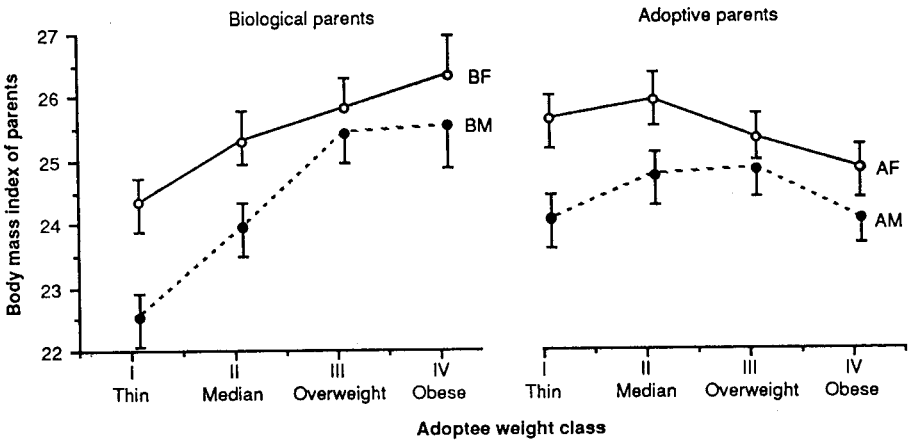

Figure 17.3

Relationships between degree of fatness in adoptees and either biological or adoptive parents

mental manipulation. Indeed, the expression of a number of genetically based diseases depends very much on food intake and physical activity; polygenic hypercholesterolaemia is an example. Even specific enzymic defects can be circumvented by dietary manipulation; phenylketonuria is one of the best examples. Further, although we are beginning to understand the bases of familial obesity, as mediated by reduced energy expenditure, the mechanisms are by no means clear. They may not necessarily reside in altered efficiency of energy-producing metabolic pathways. They may still have to do with factors determining spontaneous movement, for example.

It will also be necessary to learn more about the genetic factors in abdominal obesity. Body fat distribution is thought to be under genetic control.[29]

ENERGY BALANCE

Life Expectancy and Energy Balance

There has been a great deal of advocacy for reduced energy intakes as a way of reducing cancer rates and, possibly, improving life expectancy, on the base of experiment with rodents. These studies differ notably from the human experience; caged animals do not have the same option to strike their energy balance by way of an increase in energy expenditure through physical activity. The interaction between food intake and social activity is also increasingly understood.[30]

Obesity reflects periods of energy imbalance, with intake having exceeded expenditure, but this does not mean that obese people, as a group, eat more than lean people. Individual obese people may eat more than individual lean people, and vice versa. There also may be merit in obese people reducing their energy intake to deal with the problem of obesity. At the time seen, obese

people may be in a steady state as far as energy balance is concern. There are few studies which have looked at both energy intake and energy expenditure in relation to the development of body fatness.

One is the Zutphen Study in the Netherlands.[31] In this study, the greater the physical activity per kilogram body weight, the less the degree of obesity. An unexpected inverse relationship, of the difference between energy intake and expenditure, to body fatness may be attributable to enhanced thermogenesis in lean men and to an underestimate of energy intake in the obese. Nevertheless, in the Zutphen Study where men were followed prospectively for ten years, those with the higher energy intake, and a higher intake for kilogram body weight, lived longer.[32] The order of energy intake which distinguished survivors from those who died prematurely was of the order of 300 calories or 1200 kJ/day.

Other prospective studies also demonstrated that increasing energy intake decreases risk of coronary disease (Table 17.4[33]).

Table 17.4 Energy intake and mortality rates

Study	Kind of mortality rate influenced	Reference*
1 London	coronary	Morris and others
2 Zutphen Men	coronary & total	Kromhout and others
3 Boston-Ireland Study	coronary	Kushi and others
4 Western Swedish Women	coronary	Lapidus and others

* See Reference 33

The relationship of coronary incidence of Swedish women over twelve years in relation to quintiles of energy intake is shown in Figure 17.4.[34]

At least two prospective studies indicate that energy expenditure increments in and equivalent to the increases in energy intake of the above studies (about 300 to 500 calories or 1300 to 2100 kilojoules per day)[35] are related to increased life expectancy, although possibly not an increase in achievable life span,[36] (Figure 17.5[37]).

Food intake and energy balance

There are some characteristics of food which appear to encourage intake.[38] These include variety, palatability and novelty.[39] Schiffman has suggested that a quest for sufficient flavour is important in the determination of the amount of food eaten.[40] Satiety may also depend on the physical properties and bulk of food.[41]

There is confusion about the role of carbohydrate in the development of obesity. It is possible that there is a subgroup of obese individuals who crave carbohydrate.[42] However, sucrose carbohydrate-rich foods are often also fatty foods, examples being sweet biscuits, pastries and icecream. Rats have been shown to prefer, over-eat and become obese on sweet-fat foods.[43] Such foods may be preferred by obese people[44] and hedonistic responses to food in humans can be shown dependent on sugar-fat interactions.[45] Among simple

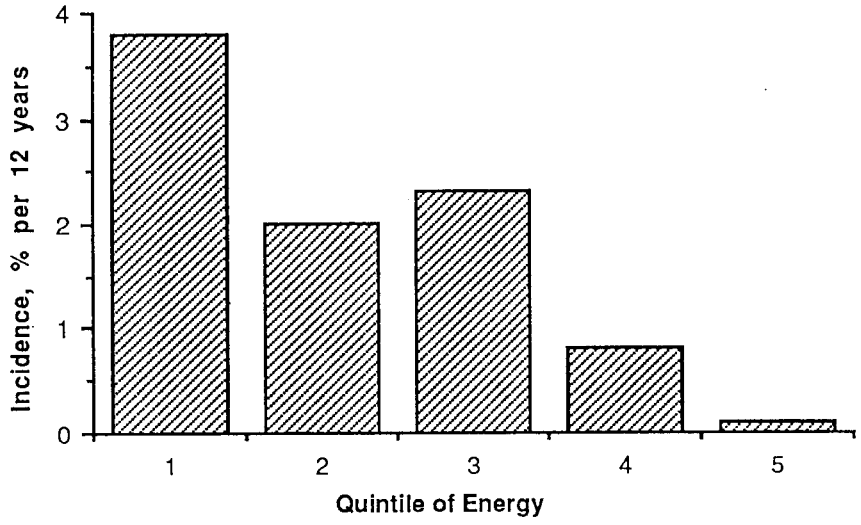

Figure 17.4

Age-standardised incidence (%) of myocardial infarction (MI) over twelve years by quintiles of intake of energy in Swedish women

sugars consumed in liquid form, sucrose has more effect on the development of body fatness.[46]

Fat intake predisposes to the development of obesity. The cross-sectional studies in women[47] and men[48] indicate that high intakes of fat are associated with obesity. A short term study in twenty-four women with three different levels of energy from fat (15 to 20%, 30 to 35% or 45 to 50%) showed that subjects spontaneously consumed an 11 per cent energy deficit on a low fat diet and a 15 per cent surfeit on a high fat diet with corresponding changes in body weight.[49]

A particular problem with studies which examine weight loss with different dietary regimes is that not all such loss is of body fat.[50] Initial weight loss may be dominantly water associated with glycogen. Relatively greater reductions in lean body mass than in body fat can occur.[51] Total energy or carbohydrate restriction is particularly prone to produce these effects.

Energy expenditure and energy balance

In primary school children[52] and in adults,[53] regular exercise allows a reduction in body fatness and maintenance at lower levels. The study of Wood[54] was in middle-aged men followed for two years in a running programme; there were associated increases in energy intake by about 400 calories or 1600 kJ per day (Figure 17.6[55]). Pi-Sunyer has also shown that, sedentary women progressively increase energy expenditure to a greater extent than their energy intake as the level of exercise on a treadmill increases.[56]

The mechanisms for favourable effects of exercise on body fatness may be several. It has been controversial as to how biologically significant increases in *resting metabolic rate* are *after exercise*,[57] but if lean body mass increases as a

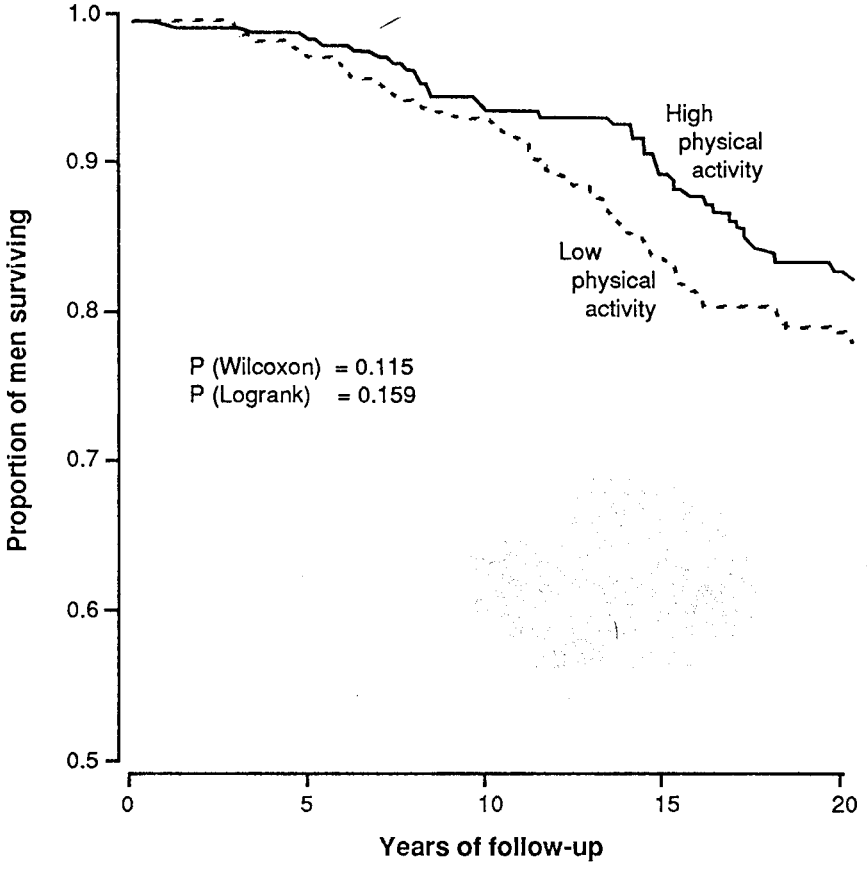

Figure 17.5

Crude twenty year survival curves for coronary heart disease death of a cohort of 636 men aged 40 to 64 at baseline, according to physical activity at baseline

result of exercise than resting metabolic rate should increase.[58] Exercise may not compensate sufficiently for the reduction in resting energy expenditure (REE) associated with energy restriction.[59] Further, account needs to be taken of how energy expenditure is expressed with changing body composition, whether in terms of total body weight or fat-free mass.[60]

It would appear that prolonged mild exercise such as walking frequently, is more acceptable and, for this reason, leads to greater overall energy expenditure than aerobic exercise in lean and post-obese subjects;[61] changes in resting metabolic rate may not be important.

As long as the same *distance* is *traversed*, an overweight individual will expend more energy than a normal weight person, leading to a measure of self-correction of over-fatness. *With exercise*, *food intake* may also be more appropriately set.[62] What may be even more important is the greater *sense of body shape and form at the time of exercise*; this would constitute a feedback signal as

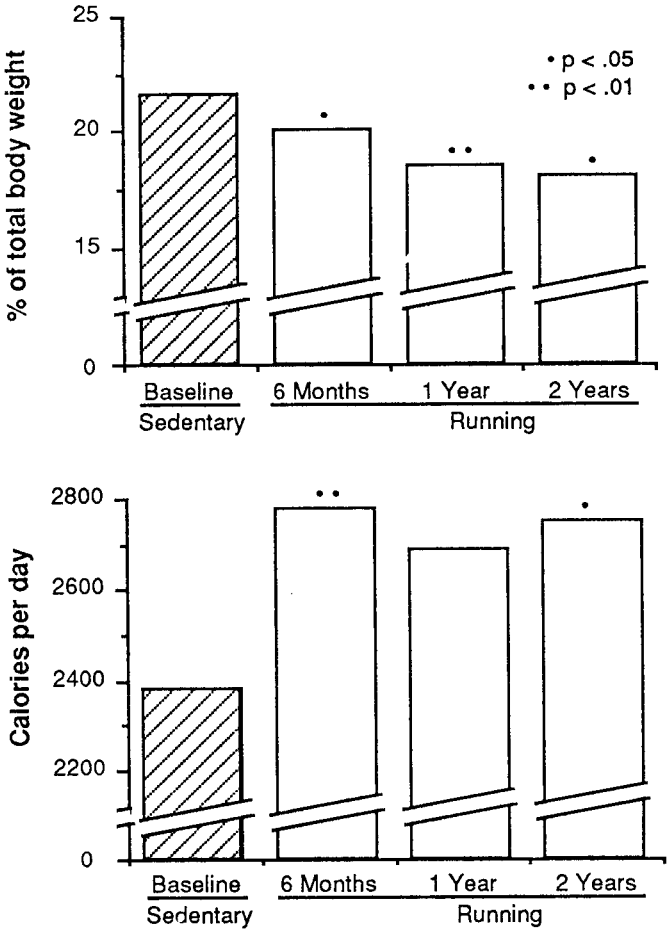

Figure 17.6

Effects of a running programme in middle-aged men

obtained by Simpson and others[63] who have used a waist cord in the previously obese in order to maintain weight loss.[64]

Efficiency of energy utilisation

As mentioned in respect of energy balance, there may be circumstances in which the efficiency of energy utilisation alters. Some intriguing work by Henry and Emery indicates that certain foods or components, may alter efficiency energy utilisation.[65] They have shown that the use of chilli (red pepper, capsicum annuum), together with mustard, in healthy subjects increases metabolic rate significantly more than an unspiced meal and that this is sustained for at least 180 minutes after the meal (Figure 17.7[66]). It has been suggested that capsicum in chilli may be responsible for this effect.

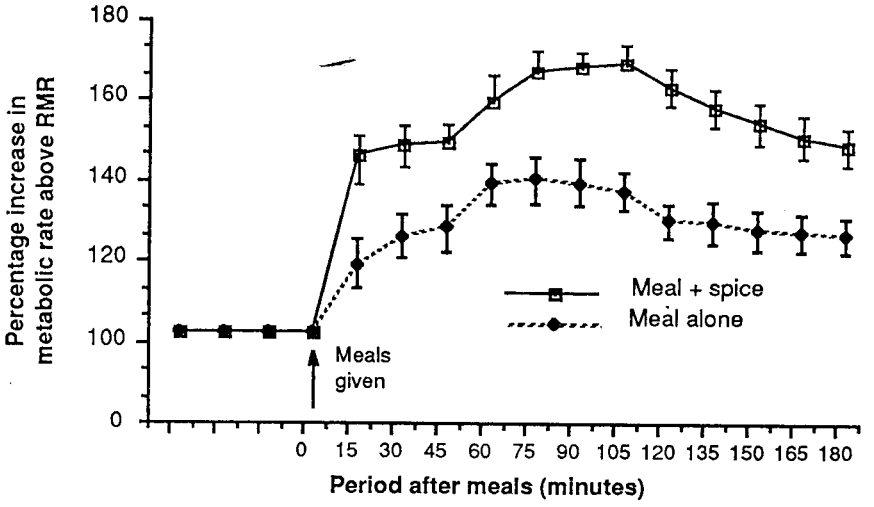

Figure 17.7

Effects of spicing food on metabolic rate

Behaviour and energy balance

Sustained improvement in energy balance appears to require behavioural change.[67] Important characteristics of behavioural therapy, according to Stunkard, are

a. The assumption that all behaviour, normal and abnormal, is acquired and maintained according to definable principle.
b. People are best described by their behaviour – what they think, feel, and do in specific situations – not by dispositional tendencies such as hostility and insecurity.
c. The attempt to specify treatment measures as precisely as possible and to evaluate outcomes as objectively as possible. Behaviour therapists have been in the forefront of efforts to introduce treatment manuals of ever greater specificity and to evaluate outcomes in the patient's environment.
d. The individualization of treatment.
e. The goals of treatment are set by negotiation between the patient and therapist, and they are renegotiated at periodic intervals.
f. Efforts are made to provide continuing and critical assessment of treatment throughout its course.

The elements of behavioural therapy of obesity are:

(i) self-monitoring – description of the behaviour to be controlled
(ii) control of the stimuli that precede eating
(iii) development of techniques to control the act of eating
(iv) reinforcement of the prescribed behaviours
(v) cognitive restructuring
(vi) diet
(vii) exercise.

RISKS ASSOCIATED WITH PREVENTION

Intense interest about obesity among young women is associated with perceived overweight when it does not exist.[68] Growth impairment has been observed with dietary restriction for obesity.[69] Clinically, micro-nutrient deficiencies are occasionally encountered in those who diet for obesity. Various eating disorders may emerge. The social and psychological consequences of obesity must be balanced against the risks arising from prevention, but in general the former will outweigh the latter.[70]

RECOMMENDATIONS

In the light of current evidence, it would seem reasonable to advance recommendations to reduce the prevalence of obesity, while not compromising other lifestyle-health relationships

- consider fatness, rather than weight, and the distribution of fatness in the assessment of obesity
- be regularly, and moderately, physically active (about 1200 kJ above sedentary on most days)
- avoid fatty foods of ruminant animal origin
- do not smoke to avoid obesity
- create a food environment conducive to healthy choices (food variety tempered with low energy density)
- remember overall health.

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