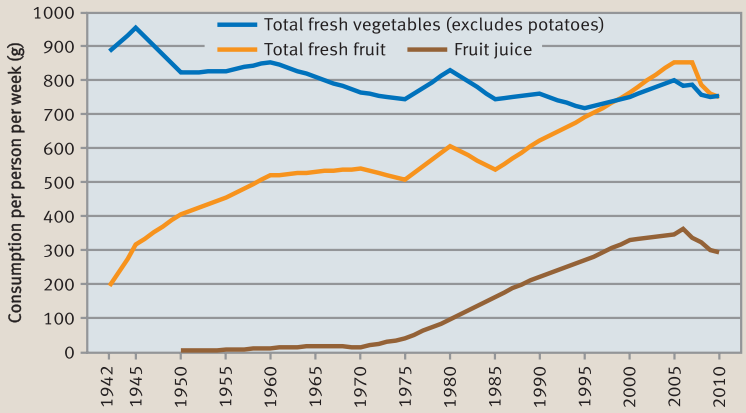

Figure 3.4 Consumption of fresh fruit and vegetables in adults aged 16 and over, United Kingdom 1942 to 2010

Source: *Coronary Heart Disease Statistics: A Compendium of Health Statistics, 2012* Edition, © British Heart Foundation 2012

over 5 g per day for children aged 7 to 14 (British Medical Association 2003a), with BHF 2012 data showing a decline since 1975 from an adult average of 7g per day to a 2010 average of 6.3 g – still above recommendations. While it is perhaps difficult to establish the unique health benefits of a reduced-salt diet when examining individuals engaged in more general dietary change behaviour, the BMA guidelines raise awareness of the need to monitor salt intake from early childhood onwards.

WHAT DO YOU THINK?

Do you add before you taste? Why? Do you have any idea how much you consume a day?

Obesity

Even though obesity is not itself a behaviour, it is contributed to mainly by a combination of poor diet and a lack of exercise (Haslam and James 2005), both health behaviours which are the theme of this and the following chapter. Global concerns about the increasing prevalence of obesity, to epidemic proportions (WHO 2000), and its impact on morbidity (including disability and a range of diseases) and mortality justify our inclusion of this condition here.

How is obesity defined?

Obesity is generally measured in terms of an individual's body mass index (BMI), which is calculated as a person's weight in kilograms divided by their height in metres squared (weight/height^2). An individual is considered to be:

- 'normal weight' if their BMI is between 20 and 24.9;
- mildly obese or 'overweight' (grade 1) if their BMI is between 25 and 29.9;
- moderate or clinically obese (grade 2) if their BMI falls between 30 and 39.9;
- severely obese (grade 3) if their BMI is 40 or greater.

BMI does not, however, take age, gender or body frame/muscle build into consideration (although BMI cut-offs are based on being 20 per cent above the height–weight chart standards for a person of 'medium' frame), and so the index should only be used as a guide in context with these other factors. As well as considering BMI, it has become clear that waist circumference, ratio of waist to hip size, and fat deposited around the abdomen (often referred to as being 'apple-shaped') further increase the implications of overweight and obesity for heart attack in both men (Smith et al. 2005) and women (Iribarren et al. 2006), for type 2 diabetes and all-case mortality in women (Hu 2003) and for some forms of cancer (Williams and Hord 2005).

Negative health consequences of obesity

As noted earlier, being underweight is the largest global cause of mortality, yet a growing number of people, predominantly in Western or developed countries, are at risk from the opposite problem – obesity. Obesity is a major risk factor in a range of physical illnesses, including, for example, hypertension, heart disease, type 2 diabetes, osteoarthritis, respiratory problems, lower back pain, and some forms of cancer.

The relative risk of disease appears to increase proportionately in relation to the percentage overweight a person is, although evidence as to this linear relationship remains mixed. A recent meta-analysis published in the *Journal of the American Medical Association* (Flegal et al. 2013), concluded from review of 97 study datasets involving over 2.8 million individuals, that there was a raised risk (calculating Hazard Ratios (HR) relative to normal weight) of death for grade 2 and grade 3 obesity (combined HR 1.29), but not for those classed as overweight (HR 0.94) or grade 1 obese (HR 0.95). In fact, being just overweight was associated with lower risk of mortality than 'normal' weight. Although based only on BMI and not weight distribution, these are important findings, leading to some academic debate about potential health benefits to being a bit overweight, certainly compared to being underweight. In contrast, the longitudinal Framingham Heart Study shows a relationship between obesity and mortality which appears over two to three decades. In their data, being overweight confers slightly more risk than 'normal' weight. In both studies, however, a J-shaped curve exists (e.g. as in Figure 3.5) reminds us also of the risk of being underweight.

Apart from physical health problems, obesity is also implicated in psychological ill health including low self-esteem and social isolation, possibly arising from the experience of stigmatising behaviour (British Medical Association 2003a; Strauss 2000, Ogden and Clementi 2011). Being overweight as a child has been associated with poorer health-related quality of life (Williams et al. 2005) and even earlier mortality (Bjørge et al. 2008).

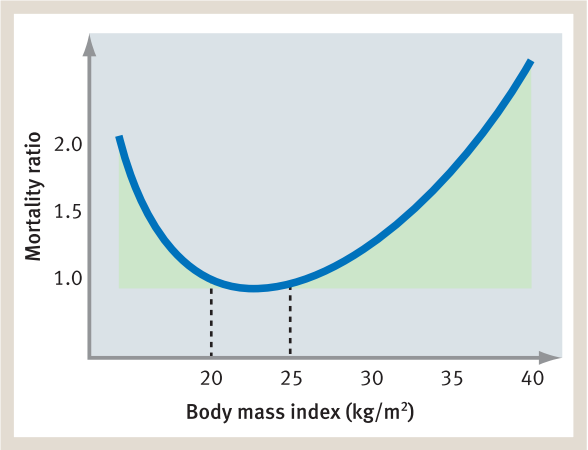

Figure 3.5 The relationship between body mass index and mortality at 23-year follow-up (Framingham heart study)

Source: Wilson et al. (2002).

Prevalence of obesity

In 1999, The European Commission estimated that 31 per cent of the EU adult population was overweight, with a further 10 per cent reaching weights defined as clinically obese. More recently, however, the proportions of adults defined as obese in England were as high as 26 per cent, among both adult men and women, with middle aged having the highest prevalence (BHF 2012). The World Health Organization collation of epidemiological data provides evidence of a three-fold increase in obesity rates in parts of North America, Australasia and China, and within the UK over the past three decades. Sadly this increase was predicted in the 1980s (Department of Health 1995). In England the prevalence of obesity in adult men has risen from 14 per cent to 26 per cent and for women over the same time period from 17 per cent to 26 per cent.

Alarmingly, excess body weight has been identified as the most common child disorder in Europe (International Obesity Taskforce and European Association for the Study of Obesity 2002) and within the English data reception class (aged 3–4) prevalence of obesity was at 9.4 per cent, rising to 19 per cent in year 6 (aged 10–11) (Department of Health Cross-Government Obesity Unit, 2011 and Lifestyle Statistics/ National Child Measurement Programme 2010/11). These data reflect the previous 9 per cent increase in the prevalence of overweight children aged between 2 and 10 years reported between 1995 and 2005 (Health Survey for England 2008). Obesity affects all age groups; however, in younger populations the particular concerns are about the early implications of obesity for psychological health

and psychosocial development, whereas with ageing, as with middle-aged and older adults, the effects on physical health begin to be seen.

Lower social class has been related to increased obesity for young females, but not for males, and as obese children tend to grow up to be obese adults (Magarey et al. 2003), interventions need to start early. To be successful, interventions need to first understand the factors associated with the development of obesity, and to do this researchers have considered the influences on food choice, intake and, crucially, overeating behaviour. These factors fall within social learning theory, e.g. the powerful influence of significant others' behaviour or communications – peers, siblings, parents, the media – or within theories of associative learning where food choice and eating behaviours are associated with the receiving of intrinsic or extrinsic rewards or **reinforcers**, such as the pleasure attached to eating with family and friends, or the perceived stress reduction gained from 'comfort eating'. As well as considering these developmental theories which focus on social learning and modelling, health psychologists have also applied cognitive theories to behaviour change with regards to overeating (see Chapter 5 ☛). It is the case that many normal-weight women wish to weigh less, thus buying in to the idea of 'thinner is better' which is normalised in much media coverage (Wardle and Johnson 2002). In spite of this, we still face an obesity epidemic and those who are overweight face being judged against this perceived 'ideal'.

Obesity has a strong association with disease, but in considering the influences on it below you will see that it is not solely due to overeating.

What causes obesity?

A simple explanation of obesity is that it is a condition that results from an energy intake that grossly exceeds the energy output (Pinel 2003). However, early twin studies and studies of adopted children (who showed weight relationship with biological rather than adoptive mothers, e.g. Price and Gottesman 1991; Meyer and Stunkard 1993) proposed some genetic explanations to obesity, which are generally one of three types:

- Obese individuals are born with a greater number of fat cells: evidence of this is limited. For example, the number of fat cells in a person of average weight and in many mildly obese individuals is typically 25–35 million. The number of cells is dramatically increased in a severely obese person, implying the formation of new fat cells.
- Obese individuals inherit lower metabolic rates and thus they burn calories more slowly and therefore they should require fewer calories to survive; however, if they don't know this and eat 'typically' they gain weight. However, research evidence shows that obese people do not consistently have lower metabolic rates than comparable thin persons, thus this common explanation seems unfounded.
- Obese individuals may have deficiencies in a hormone responsible for appetite regulation or control, or lack of control: more potential as a contributing factor.

This last explanation has received attention since the 1950s, when a gene mutation was identified in some laboratory mice that had become highly obese (Coleman 1979). Subsequent cloning of this mutated gene found that it was only expressed in fat cells and that it encoded a protein hormone called leptin (Zhang et al. 1994). Leptin is produced by fatty (adipose) tissue and is one of several signals to the hypothalamus of the central nervous system that help to regulate weight. Low leptin levels suggest low fat stores which then prompts a signal to the organism to eat, to re-establish fatty stores needed for energy (see Chapter 8 ☛). However, research has not found similar genetic mutation in all obese humans and increasing leptin by means of injection has not consistently reduced eating behaviour or body fat in the obese. More recently, exciting research showing the effects of common FTO (fat mass and obesity associated) gene variants on BMI, weight, waist circumference and body fat (e.g. Frayling et al. 2007) have been brought into the limelight by preliminary evidence that genetic susceptibility to obesity could be reduced or even negated by means of vigorous physical activity (Rampersaud et al. 2008).

reinforcers

factors that reward or provide a positive response following a particular behaviour or set of behaviours (positive reinforcer); or enable the removal or avoidance of an undesired state or response (negative reinforcer).

Another avenue of research has identified that serotonin, a neurotransmitter (see Chapter 8 ☛), is directly involved in producing satiety (the condition where hunger is no longer felt). Early animal experiments investigating the effects on hunger of administering a serotonin **agonist** have had their findings confirmed in humans, where the introduction of serotonin agonists into the body induced satiety, and reduced the frequency and quantity of food intake and body weight (Halford and Blundell 2000).

Such lines of research hold promise for future intervention; however, it is likely that such explanations are insufficient. Given the recent upsurge in obesity in developed countries, obesity is more plausibly attributed to an interaction between physiological and environmental factors such as sedentary lifestyle and behaviour patterns. People of all ages increasingly pass their time indoors, and there is evidence that activities such as watching television or computing can even reduce a person's metabolic rate, so that their bodies burn up existing calories more slowly. Lack of physical activity in combination with overeating or eating the wrong food types are associated with obesity, and it is unclear which is the primary causal factor.

People eat for different reasons and it may be that the obese differ from the non-obese in this regard. For many, eating carries positive incentives, such as the intrinsic rewards of taste enjoyment (sensory eating), and extrinsic rewards such as the pleasure of social eating (Pinel et al. 2000). Eating styles exist (van Strien et al. 1986; and see WHAT DO YOU THINK? below) whereby some people eat when they catch sight of food or food cues (external eating), or simply when their body signals hunger (internal eating); others eat when they are bored, irritated or stressed (emotional eating). There is some evidence that obese people and those who overeat are more external cue responsive and less led by internal hunger cues, and perhaps show more emotional eating (Snoek et al. 2007; van Strien et al. 2007; O'Connor et al. 2008). When eating when stressed, it seems that it is not only the amount of food that increases, but also the type of food selected, often that with high sugar or fat content (Oliver et al. 2000), and furthermore that the type of eating behaviour may differ depending on the nature of the stressor (O'Connor et al. 2008).

Effective interventions which aim to make what has been described as our 'obesogenic' environments (BMA 2003a) and behaviour more healthy need therefore to address a complexity of factors. Similarly, interventions to increase physical activity, the other major contributor to reduced obesity, are also high on the public health agenda (see Chapters 4, 6 and 7 ☛).

agonist
a drug that simulates the effects of neurotransmitters, such as the serotonin agonist fluoxetine, which induces satiety (reduces hunger).

WHAT DO YOU THINK?

What style of eating do you have? Are you likely to eat when something looks or smells good even if you have recently eaten and don't actually feel hungry? ('external/sensory eating') Are you easily swayed by others simply eating in front of you? ('external eating') Can you exercise restraint over what and when you eat? For example, if you think you have eaten more than usual over the weekend, do you try to eat less the next day? ('restraint eating'). What about your mood: does that make a difference to when and what you eat? Some people eat less when depressed, others eat more and seek out certain food types – usually sweet or fatty foods. If you're angry, frustrated or impatient, do you snack or nibble more? ('emotional eating').

Styles of eating reported by those with obesity have been 'matched' to different obesity treatments. If you are interested in reading further about this, use the references above and also Google the DEBQ (the Dutch Eating Behaviours Questionaire), a screening tool used in many studies of eating styles and which has been shown to have good construct validity and internal reliability.

A final thought on obesity

A word of caution is drawn from the BMA report (2003a) referred to previously: we as individuals, and as a society, must be careful not to over-focus on the weight of individual children – while obesity is on the increase, so too is extreme dietary behaviour and eating disorders; several recent studies point to increasing body dissatisfaction among children and adolescents, particularly females (e.g. Schur et al. 2000; O'Connor et al. 2008). Body dissatisfaction can lead to dietary restraint which can potentially have adverse physical and psychological consequences, including eating disorders (e.g. Stice et al. 2002).