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Diet, nutrition and the prevention of excess weight gain and obesity

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Abstract

Objective: To review the evidence on the diet and nutrition causes of obesity and to recommend strategies to reduce obesity prevalence.

Design: The evidence for potential aetiological factors and strategies to reduce obesity prevalence was reviewed, and recommendations for public health action, population nutrition goals and further research were made.

Results: Protective factors against obesity were considered to be: regular physical activity (convincing); a high intake of dietary non-starch polysaccharides (NSP)/fibre (convincing); supportive home and school environments for children (probable); and breastfeeding (probable). Risk factors for obesity were considered to be sedentary lifestyles (convincing); a high intake of energy-dense, micronutrient-poor foods (convincing); heavy marketing of energy-dense foods and fast food outlets (probable); sugar-sweetened soft drinks and fruit juices (probable); adverse social and economic conditions—developed countries, especially in women (probable).

A broad range of strategies were recommended to reduce obesity prevalence including: influencing the food supply to make healthy choices easier; reducing the marketing of energy dense foods and beverages to children; influencing urban environments and transport systems to promote physical activity; developing community-wide programmes in multiple settings; increased communications about healthy eating and physical activity; and improved health services to promote breastfeeding and manage currently overweight or obese people.

Conclusions: The increasing prevalence of obesity is a major health threat in both low- and high income countries. Comprehensive programmes will be needed to turn the epidemic around.

Keywords
Public health
Overweight
Obesity
Evidence-base

This review paper has been structured to provide an overview of the likely aetiological factors in the development of weight gain and obesity, to propose related population nutrient goals and content areas for food-based dietary guidelines, and to evaluate some of the potential food and diet related intervention strategies that might help to attenuate and eventually reverse this global epidemic. The process involved Medline searches on relevant topics determined by the authors and the participants in the Joint WHO/FAO Expert Consultation on diet, nutrition and the prevention of chronic diseases (Geneva, 28 January–1 February 2002). Recent reviews and key papers were sought, but this did not involve a full systematic review on each topic.

The level of evidence that a dietary factor could be involved in the promotion of or protection against the development of obesity was assigned on the basis of the evidence review and the weighting of this evidence by the authors and Expert Consultation members. The evidence judgments were based on the framework and definitions used by the World Cancer Research Fund and American Institute for Cancer Research in their review on diet and cancer[1]. The evidence in that report was rated as convincing, probable, possible or insufficient for a positive, a negative or no relationship between the variable and cancer. However, because their outcome of interest was cancer, the framework mainly centred on epidemiological studies. In the current review, randomised clinical trials were given the highest ranking with consistent results from several trials constituting convincing evidence. This is particularly important in the relationship between diet and obesity because of the major methodological problems of dietary underreporting. Obese people tend to underreport more than lean people and the underreporting may be the greatest for high fat and high carbohydrate foods[2,3]. Another difficulty

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arose in rating evidence in relation to some of the potential environmental causes of weight gain. For environmental factors, more associated evidence and expert opinion had to prevail because of the absence of direct studies or trials in the area[4].

It is important to note that this review on obesity has not covered the energy expenditure side of the energy balance equation in any depth. Physical activity is at least as important as energy intake in the genesis of weight gain and obesity and there are likely to be many interactions between the two sides of the equation in terms of aetiology and prevention. The role of physical inactivity in the development of obesity has been well described[5] and a recent report from the US Center for Disease Control and Prevention summarises the evidence base for a variety of interventions to increase physical activity at the population level[6]. Also, a thorough review of weight control and physical activity has recently been conducted by the WHO International Agency for Research on Cancer and was also used as a basis for recommendations on physical activity[7].

Current global situation and trends

Overview

The prevalence of obesity is increasing throughout the world's population. But the distribution varies greatly between and within countries. In the US, over the past 30 years, the prevalence of obesity rose from about 12–20% of the population from 1978 to 1990[8]. The UK has experienced an increase in the prevalence of obesity from 7% in 1980 to 16% in 1995[8]. Other countries, such as The Netherlands, have experienced much smaller increases from a low baseline of about 5% in the 1980s to about 8% in 1997[9]. In Asia, the prevalence of obesity has rapidly increased. In the last 8 years the proportion of Chinese men with a body mass index (BMI) $>25\,kg/m^2$ has tripled from 4 to 15% of the population and the proportion in women has doubled from 10 to 20%[10]. Pacific populations have some of the world's highest prevalence rates of obesity. The proportion of men and women with a BMI $>30\,kg/m^2$ in Nauru was 77% in 1994[11] and for Pacific people living in New Zealand in the early 1990s the prevalence rates were about 65–70%[12].

The obesity epidemic moves through a population in a reasonably consistent pattern over time and this is reflected in the different patterns in low- and high income countries. In low income countries, obesity is more common in people of higher socioeconomic status and in those living in urban communities. It is often first apparent among middle-aged women. In more affluent countries, it is associated with lower socioeconomic status, especially in women, and rural communities[13,14]. The sex differences are less marked in affluent countries and obesity is often common amongst adolescents and younger children.

Brazil is an example of a country with well-documented changes in obesity prevalence as it undergoes rapid nutrition transition. There has been a rapid increase in obesity where the prevalence among urban men with high incomes is about 10%, but still only 1% in rural areas. Women in all regions are generally more obese than men and the prevalence for those on low income is still increasing. However, the rate of obesity among women with high income is becoming stable or even declining[15].

The standard definitions of overweight (BMI $\geq 25\,kg/m^2$) and obesity ($\geq 30\,kg/m^2$) have been mainly derived from populations of European descent[8]. However, in populations with large body frames, such as Polynesians, higher cut-off points have been used[16]. In populations with smaller body frames, such as Chinese populations, lower cut-off points have been proposed[17] and studies are being undertaken to evaluate appropriate cut-off points for a variety of Asian populations[18].

Body fat distribution (often assessed by the waist circumference or the waist:hip ratio) is an important independent predictor of morbidity[19,20]. Although this review focuses on weight gain and the development of overweight and obesity, it is acknowledged that increases in abdominal fatness (particularly, intra-abdominal fat) pose a greater risk to health than increases in fatness around the hips and limbs. In general, the causes of weight gain and abdominal weight gain are the same and it is the characteristics of the individuals (such as sex, age, menopausal status) that influence the distribution of the fat that is gained.

The nutrition transition

The increasing westernisation, urbanisation and mechanisation occurring in most countries around the world is associated with changes in the diet towards one of high fat, high energy-dense foods and a sedentary lifestyle[8,21]. This shift is also associated with the current rapid changes in childhood and adult obesity. Even in many low income countries, obesity is now rapidly increasing, and often coexists in the same population with chronic undernutrition[21]. Life expectancy has increased due to advancement in nutrition, hygiene and the control of infectious disease. Infectious diseases and nutrient deficiency diseases are, therefore, being replaced in developing countries by new threats to the health of populations like obesity, cardiovascular disease and diabetes[8].

A sharp decline in cost of vegetable oils and sugar means that they are now in direct competition with cereals as the cheapest food ingredients in the world[22]. This has caused a reduction in the proportion of the diet that is derived from grain and grain products[21] and has greatly increased world average energy consumption, although this increase is not distributed evenly throughout the world's population[22].

As populations become more urban and incomes rise, diets high in sugar, fat and animal products replace more traditional diets that were high in complex carbohydrates and fibre[21,22]. Ethnic cuisine and unique traditional food habits are being replaced by westernised fast foods, soft drinks and increased meat consumption[22]. Homogenisation and westernisation of the global diet has increased the energy density[22] and this is particularly a problem for the poor in all countries who are at risk of both obesity and micronutrient deficiencies[14].

Health consequences of obesity

Mortality rates increase with BMI and they are greatly increased above a BMI of 30 kg/m^2 [23]. For example, a study in US women estimated that among people with a BMI > 29 kg/m^2, 53% of all deaths could be directly related to their obesity[24].

As obesity has increased over the last 30 years, the prevalence of type 2 diabetes has increased dramatically. The global numbers of people with diabetes (mainly type 2) are predicted to rise by almost 50% in 10 years—151 million in 2000 to 221 million in 2010[25]. The most potent predictor for the risk of diabetes, apart from age, is the BMI[23]. Even at a BMI of 25 kg/m^2 the risk of type 2 diabetes is significantly higher compared to BMI of less than 22 kg/m^2, but at BMI over 30 kg/m^2, the relative risks are enormous[26]. Type 2 diabetes is becoming increasingly prevalent among children as obesity increases in those age groups. This was first reported among the Pima Indians in 1979 where 1% of the 15–24-year-olds had diabetes (almost all type 2 diabetes)[27]. Now in many populations around the world, a substantial proportion of the teenagers with diabetes have the obesity-associated type 2 variety[28]. Asian populations appear to develop diabetes at a lower BMI than other populations[29].

A high BMI is associated with higher blood pressure and risk of hypertension, higher total cholesterol, LDL-cholesterol and triglyceride levels and lower HDL-cholesterol levels. The overall risk of coronary heart disease and stroke, therefore, increases substantially with weight gain and obesity[23].

Gall bladder disease and the incidence of clinically symptomatic gallstones are positively related to BMI[23]. There is evidence to suggest increased cancer risk as BMI increases, such as colorectal cancer in men, cancer of the endometrium and biliary passage in women, and breast cancer in post-menopausal women[8,23]. Obese people are also at increased risk of gout, sleep apnoea, obstetric and surgical complications[23].

Health care costs of obesity

The direct health care costs of obesity in the US have been estimated to account for 5.7% of total health care expenditure in 1995. Comparable figures are somewhat lower than this for other western countries such as France (2%), Australia (2%)[30] and New Zealand (2.5%)[31]. These figures underestimated the full direct costs of weight-associated disease because they estimated the costs for the population with BMI > 30 kg/m^2 and omitted any burden of lesser forms of overweight (BMI 25–30 kg/m^2). A Dutch study suggests the costs attributed to BMI 25–30 kg/m^2 are three times the cost of BMI > 30 kg/m^2 [32]. The direct costs of obesity are predominantly from diabetes, cardiovascular disease and hypertension. Indirect costs, which are far greater than direct costs, include workdays lost, physician visits, disability pensions and premature mortality which all increase as BMI increases[33]. Intangible costs (impaired quality of life) have not been estimated, but given the social and psychological consequences of obesity, they are likely to be enormous.

Potential aetiological factors in relation to obesity in populations

The format for identifying potential nutritional causes of obesity at a population level is based on the Epidemiological Triad[34] where the 'hosts' are the general population, the 'vectors' are the foods and nutrients and the 'environment' includes the physical, economic, policy and socio-cultural factors external to the individual. Issues were selected based on their relevance to approaches to reducing the burden of obesity at a population level. The evidence summary for identified issues is shown in Table1.

Host issues

There are a variety of behaviours and other host factors that have a potential effect on a population's level of obesity. These are, of course, closely linked to the vectors and the environments and in many cases the issues merge and overlap. Issues related to social aspects of eating are not covered.

Snacking/eating frequency

While there is no one definition of snacking, it is probably best to consider the content of snack foods and the increased eating frequency that snacking promotes as separate issues[35]. There is evidence from the US that snacking prevalence (i.e. occasions of snacking) is increasing, the energy density of snack foods is increasing and the contribution to total energy is increasing[36]. Snacks contribute to about 20–25% of total energy intake in countries like the US and UK[35]. However, there is little evidence that a higher frequency of eating *per se* is a potential cause of obesity. Cross-sectional studies tend to show a negative relationship or no relationship between meal frequency and BMI[37]. Low eating frequency may, of course, be a response to obesity rather than a cause. Experimental studies have found mixed results on the degree of caloric compensation that people make at meal time in response to a prior snack with some studies

Table 1 Evidence table for factors that might promote or protect against overweight and weight gain

Evidence	Decreases risk	No relationship	Increases risk
Convincing	Regular physical activity High dietary NSP/fibre intake		Sedentary lifestyles High intake of energy dense foods*
Probable	Home and school environments that support healthy food choices for children Breastfeeding		Heavy marketing of energy dense foods* and fast food outlets Adverse social and economic conditions (developed countries, especially for women) High sugar drinks
Possible	Low GIycemic Index foods	Protein content of the diet	Large portion sizes High proportion of food prepared outside the home (western countries) 'Rigid restraint/periodic disinhibition' eating patterns
Insufficient	Increased eating frequency		Alcohol

* Energy dense foods are high in fat and/or sugar; energy dilute foods are high in NSP/fibre and water such as fruit, legumes, vegetables and whole grain cereals.
Strength of evidence: The totality of the evidence was taken into account. The World Cancer Research Fund schema was taken as the starting point but was modified in the following manner: RCTs were given prominence as the highest ranking study design (RCTs not a major source of cancer evidence); associated evidence was also taken into account in relation to environmental determinants (direct trials were usually not available or possible).

showing more complete compensation among lean people[37]. There is insufficient evidence to support an effect of a higher frequency of eating on obesity or weight gain. If anything, it is protective against weight gain. The high energy density of common snack foods, however, may do the opposite and promote weight gain (see below).

Restrained eating, dieting and binge eating patterns
While a degree of selective or restrained eating is probably needed to prevent obesity in an environment of plenty, some individuals (dieters and non-dieters) score highly on the Restraint Scale and paradoxically may also exhibit periods of disinhibited eating[38]. Such individuals appear to be at risk of dieting–overeating cycles. The concepts used to define these constructs and the instruments used to measure them continue to evolve, but the studies would suggest that a 'flexible restraint' eating pattern is associated with a lower risk of weight gain whereas a 'rigid restraint/periodic disinhibition' pattern is associated with a greater risk of weight gain[39]. Binge eating disorder[40] and night eating syndrome[41] would be examples of the latter pattern. Binge eating disorders are significantly more common in obesity in cross-sectional studies. The relationships between these dietary patterns and weight gain or obesity is complex with both cause and effect relationships likely.

Eating out
In western countries, the frequency of eating food prepared outside the home is increasing and this is most apparent and best documented in the US. In 1970, 26% of the food dollar in the US was spent on food prepared outside the home. By 1995, it had climbed to 39% and is projected to rise to 53% by 2010[42,43]. This shift towards an increase in the frequency of eating meals and snacks away from home and the proportion of food budget spent on away from home foods[42,44,45] has coincided with the increasing prevalence of obesity.

In the US, food prepared away from home is higher in total energy, total fat, saturated fat, cholesterol and sodium, but contains less fibre and calcium and is overall of poorer nutritional quality than at-home food. Also, the fat content of at-home food has fallen considerably from 41% of total energy in 1977 to 31.5%, but there has been no change in the fat content of food prepared away from home (37.6%)[43].

These food composition differences and the increasing portion sizes, are likely contributors to the rising prevalence of obesity in the US[44]. Those who eat out more, on average, have a higher BMI than those who eat more at home[46]. The evidence implicating the increasing use of food prepared outside the home as a risk for obesity is largely limited to the US but this may be extrapolated to other western countries. It is unknown whether a high frequency of eating out is associated with obesity or weight gain in other populations, for example, in Asian countries, where eating outside the home may not be a risk for weight gain.

Breastfeeding
Breastfeeding has been suggested as a potential protective factor against weight gain in childhood[47] and this is important because overweight children and adolescents are at risk of becoming overweight adults[48]. A review by Butte[49] examined 18 studies (6 retrospective, 10 prospective, 1 cohort, 1 case–control) published up to 1999 with a total of nearly 20,000 subjects. There was a wide time span (1945–1999) and the definitions of breastfeeding and obesity and the length of follow up were all highly variable. Two of the studies found a

positive association between breastfeeding and later obesity and four found a negative relationship (i.e. an apparent protective effect of breastfeeding). The remainder found no differences. The largest study ($n = 9357$ children aged 5–6) found a prevalence of obesity among breastfed children of 2.8% compared to 4.5% in never breastfed children and there was an apparent dose response in relation to the duration of breastfeeding[50]. A similar study of 3731 6-year-old British children, however, found no such relationships[51].

Since that review, two further studies have been published. A US cohort study of over 15,000 boys and girls aged 9–14 years reported a significant (about 20%) reduction in the risk of becoming overweight associated with only or mostly breastfeeding in the first 6 months of life after adjusting for measured confounders[52]. The second study was a US cross-sectional survey of 2685 3–5-year-olds[53]. The risk reductions for breastfeeding were of a similar magnitude to the previous study but the lower power of the second study meant that the associations were not statistically significant. A significant reduction in risk was found with breastfeeding for being 'at risk of overweight' (between 85th and 94th percentiles for BMI).

The influence of confounding factors is a major problem in these studies and controlling for them was only attempted in some of the studies. The BMI of the parents (especially the mother) is a strong determinant for childhood obesity[49,52] and overweight mothers tend to breastfeed less and for a shorter duration[52]. There have been marked changes in formula composition and infant feeding practices over the decades and the early studies may be of limited relevance to the current day recommendations[49].

The current evidence was judged to show a probable protective effect of breastfeeding against childhood obesity. The prevention of unhealthy weight gain should, therefore, be added to all the other health reasons for promoting breastfeeding and complementary feeding.

Early nutrition

Birth weight is a crude indicator of intrauterine nutrition. A systematic review of predictors of obesity by Parsons *et al.* found that studies reported a consistent and positive relationship between birth weight and BMI (or risk of overweight) as a child or as an adult[54]. It is possible that low birth weights may also be associated with high adult BMI (i.e. that the relationship is a J-shaped curve rather than linear and positive). However, very low birth weight is a much weaker predictor of high adult BMI than high birth weight[54].

Maternal and childhood undernutrition are common in low income countries and childhood stunting is often used as a marker for this. A later exposure to more western-style diets and lifestyles (such as through migration to urban areas and/or improved economic conditions) may promote an excessive increase in body fatness or abdominal fatness. Popkin *et al.* studied 3–9-year-old children in cross-sectional studies in four countries (China, Russia, South Africa and Brazil) and found that stunted children (low height-for-age z-score) were more likely than non-stunted children to be overweight (high weight-for-height z-score) with relative risks between 1.7 and 7.8[55]. On the other hand, a cohort of children measured at age 3 in Guatemala and followed into adulthood showed that childhood stunting was associated with a low BMI and low percent body fat in men but no such relationships were seen in women[56]. Only when BMI or percent body were adjusted for, did an association between severe stunting and high waist:hip ratio become evident.

The hypothesis that intrauterine and/or early childhood undernutrition leads to adult obesity or abdominal obesity is an important one that links with the other relationships between early undernutrition and adult diseases such as hypertension and diabetes[57]. This could pose a major problem for countries undergoing the economic and nutrition transition[21]. However, the relationships are clearly complex and the available data were judged insufficient to be able to make a single summary statement in the evidence table.

Vector issues

Percent fat, percent carbohydrate and energy density

Background: Most of the debate about the fat and carbohydrate content of the diet in relation to obesity centres on the effects of altering the reciprocal proportions of carbohydrate and fat in the diet on energy density, total energy intake, body weight and lipoprotein profiles. The debate[58–60] has become vigorous and, at times, muddled because several issues are usually debated at the same time. Also, the epidemiological evidence comes from different types of studies (ecological, cross-sectional and prospective) which suffer from multiple potential sources of bias, the instruments used to measure dietary intake are blunt, and there is substantial obesity-related under-reporting of energy and fat intake[61]. Even the clinical trials use a wide variety of different dietary manipulations, some are isocaloric and some are *ad libitum* and few are long term. The evidence is reviewed in several parts to try to bring some clarity to the debate. Firstly, the effects of reciprocal differences or alterations in percent fat and percent carbohydrate on body weight will be examined in different study designs: epidemiological studies; controlled trials of high percent fat or high percent carbohydrate diets under conditions of (a) fixed total energy intakes, (b) covert manipulations with *ad libitum* total intakes or (c) overt manipulations with *ad libitum* total intakes. Secondly, the high energy density of high fat diets will be examined as the potential mechanism to explain their propensity to promote weight gain.

Percent fat in the diet—epidemiological studies: The epidemiological evidence presents mixed results on the relationship between the percent fat (or percent

carbohydrate) in the diet and obesity or weight gain. Ecological studies between populations tend to show a positive relationship between fat and obesity, especially if populations with low fat intakes are included[60,61], but negative relationships are also seen[62]. Similarly, studies in the same population over time tend to show positive relationships between obesity and dietary fat intake in populations undergoing nutrition transition but a negative relationship in many westernised populations[61,62]. Cross-sectional and prospective studies also show mixed results[62]. In light of the methodological drawbacks of these types of studies and the mixed results they have produced[61], controlled trials are needed to address the question.

Percent fat in the diet—fixed total energy trials: According to Reaven[63] the simplest way to answer the question about the impact of fat and carbohydrate in the diet on body weight 'is to focus on studies that vary in macronutrient composition, but are equal in energy'. Studies that have done this[63–65] have indeed found that 'clamping' total energy produces similar weight changes irrespective of the macronutrient composition. The rationale for many of these studies was to assess the impact of macronutrient changes independent of total energy intake. They were not to emulate the real world where total intake is *ad libitum*. The conclusion from the fixed energy studies is that if a high percent fat diet promotes weight gain, the mechanism appears to be mediated by promoting a higher total energy intake.

Percent fat in the diet—ad libitum *trials, covert manipulations:* Several trials have covertly manipulated the fat and carbohydrate proportions of equally palatable diets while allowing study participants to eat *ad libitum* total intakes[66–73]. Most of the studies were short term with the longest being 11 weeks[73]. These trials consistently show a progressive rise in total energy intake and body weight on the higher percent fat diets and the opposite on the lower percent fat diets. The amount (weight) of food eaten is similar on both types of diet. These covert manipulation studies are central to the debate on dietary fat and weight gain because they demonstrate that, other things being equal, the physiological–behavioural consequence of a high percent fat diet is a slow weight gain through the 'passive overconsumption' of total energy.

Percent fat in the diet—ad libitum *trials, overt manipulation:* Longer term trials of high and low percent fat diets have generally used educational strategies to get participants to select reduced fat food options and compared them with standard or higher fat diets. The diet is unrestricted in total amount (weight) and replacement of lost energy from fat is not specifically replaced by carbohydrate. It is important to note that, unlike the covert manipulations, it is difficult to blind such studies and, therefore, psychosocial effects, personal preferences and other effects not directly related to physiology can confound the results[74]. A recent meta-analysis of 16 *ad libitum* dietary trials (19 interventions) of at least 2 months duration showed that reduced fat diets consistently result in a reduced total energy intake and reduced weight[75]. Interestingly, weight loss was not the primary goal in more than half the studies. A reduction in the proportion of fat in the diet by 10% points corresponded with a reduction of about 1 MJ of total energy per day. The effects of such a dietary change on body weight have been estimated to be in the range of 2.6–3.2 kg, although greater weight loss is seen in more overweight individuals[75]. The weight loss was not associated with the duration of the intervention but it was larger in overweight subjects compared to normal weight subjects. It is important to note that an absolute reduction in dietary fat (g/d) does not elicit a compensatory increase in absolute dietary carbohydrate intake (g/d), although the fat:carbohydrate ratio, of course, decreases[76].

An interesting study attempting to replicate realistic food choices randomised normal weight and overweight participants into two groups who selected either full fat or reduced fat foods from small, realistic 'supermarkets' in the study centres[77]. The free access to higher fat products resulted in a significant increase in energy intake (0.9 MJ/d) and body weight (0.7 kg) over 6 months compared to the reduced fat group.

Reducing the fat content of the diet consistently produces modest reductions in body weight but one could argue that instructions to individuals to reduce other macronutrients in the diet or to restrict the intake of certain high volume foods (such as staple carbohydrates) would also result in weight loss. Indeed, there are a myriad of popular diets with a wide variety of food and drink restrictions and all have their champions who have lost weight. It is obvious that any such restrictions that result in a reduction in total energy intake will produce weight loss. The rationale for promoting a reduction in the fat content of the diet to prevent weight gain or promote weight loss is that it is concordant with the body's physiological–behavioural mechanisms regulating food intake as evidenced by the covert manipulation studies.

Another potential criticism of promoting a reduced fat content of the diet is that the *ad libitum* weight loss studies show a modest effect (a few kilograms) with a tendency to return towards the previous weight after the intervention period[76]. This rebound is common to all dietary interventions and there are a number of potential explanations for this. They include: a reduction in compliance to the diet, perhaps due to an environment that is unsupportive of healthy food choices; overeating of foods known to be low in fat and; physiological adaptations that attenuate the impact of negative energy balance on weight loss[78].

Extremely low fat, high carbohydrate diets are also very effective for weight loss[79,80] but it must be stressed that large reductions in total fat intake would be unattainable at

a population level. Average changes in the order of 2–3 kg may seem small for individuals but they are important on a population level in the context of obesity prevention. A shift of one unit of BMI in the overall distribution in the population is associated with a 5% point change in the prevalence of obesity[8].

Percent fat in the diet—mechanisms that promote weight gain: Why does a high percent fat diet tend to result in a passive overconsumption of total energy and thus promote weight gain? Potential mechanisms are through satiety, energy density, palatability and/or metabolic responses.

Foods high in fat are less satiating than foods high in carbohydrates[81,82]. When isocaloric amounts of foods are fed, a high satiety score is associated with a high volume of the food which in turn is related to a high complex carbohydrate content[81]. Is satiety related to the food volume (weight) *per se* or is it dependent on the different metabolic processes that fat and carbohydrate undergo after digestion? Pure fat (9 kcal/g) has more than twice the energy density of pure carbohydrate (4 kcal/g) or pure protein (4 kcal/g). These differences are accentuated when one considers real foods rather than nutrients because high carbohydrate foods, such as vegetables and cereals, also tend to include water and fibre which further dilute energy density whereas many high fat foods, such as oils, butter and margarine, have little water or fibre.

Several carefully controlled studies have manipulated the fat and carbohydrate content, energy density, and volume independently of each other to further explore these relationships. If energy density and palatability are kept constant, no difference in energy intake occurs in diets with varying fat and carbohydrate content[83]. Conversely, variations in energy density at constant fat:carbohydrate ratios influences total energy intake[84]. This implies that under *ad libitum* conditions, it is the high energy density of fatty foods that results in a weak satiating effect for the energy eaten and, therefore, promotes passive overconsumption[85,86]. In the real world, the fat content of foods or dietary intakes are closely related to their energy density, so that the general statement that high dietary fat intakes are likely to promote weight gain still holds. However, three caveats to this statement need to be made. The first is that some food products such as snack bars and breakfast cereals have been manufactured to be low in fat, but the addition of large amounts of sugars into the products means that they contain about the same amount of energy per 100 g as their original full-fat counterparts[87]. The second is that the water incorporated into foods appears to have greater effects on promoting satiety and reducing subsequent intake than water incorporated into beverages[88]. This would mean that until further research claries the relationship between energy density, satiety and subsequent energy intake across foods and beverages, these latter two categories should be considered separately. The third caveat is that diets that are very high in energy dilute foods (such as vegetables, fruits and whole grain cereals) and have a significant addition of fat (such as oil) may achieve a high percent of energy as fat without being very energy dense.

Prior to the elegant studies teasing out the impact of energy density and dietary fat (above), a 'glycogenostatic model' for energy balance had been proposed by Flatt as a metabolic explanation of the differential effects of high fat and high carbohydrate diets in animals and humans. This model was built on the experimental evidence from feeding trials, the largely separate metabolic pathways for carbohydrate and fat (nutrient partitioning), the minor conversion of carbohydrate into fat (*de novo* lipogenesis is a minor pathway in humans except under unusual conditions of massive carbohydrate overfeeding[90]), the lack of acute fat oxidation response to increased fat intake[85,91] and the limited capacity of glycogen stores. While each of these building blocks of the model remain valid, studies that have grossly manipulated glycogen stores over 1–2 days have found no effect on energy intake[92,93].

Another potential metabolic mechanism by which dietary fat might promote weight gain is that it has a lower thermic effect (energetic cost of processing) than carbohydrate[94] but this is likely to be a minor factor except under conditions of significant overfeeding[91].

Fats also carry many aromatic compounds that add flavour to foods and, therefore, high fat foods may be overconsumed, in part, because they are highly palatable.

Percent fat in the diet—secular changes in diet and obesity: It is apparently paradoxical that in some countries the percent fat in the diet has decreased but obesity has increased—indeed this has been dubbed 'The American paradox'[95] because that is where it is most obvious. Accurate measurement of population macronutrient intakes is problematic because of serious (and probably increasing) underreporting in dietary surveys and the many assumptions incorporated into measuring food supply. However, it does appear that dietary carbohydrate intake has risen in absolute and relative terms, dietary fat has changed little in absolute terms and decreased in relative terms and that total energy intake has increased overall[96]. These trends coupled with continued reductions in physical activity would explain the apparent paradox. Messages about reducing fat in the diet appear to have been used interchangeably with increasing carbohydrate and this may have contributed to overconsumption of carbohydrates and total energy which then promotes the storage of dietary fat as body fat. This would also be accentuated by the marketing of high sugar, high energy dense foods as 'low fat' implying (falsely) that they are neutral or helpful for preventing weight gain[87].

Percent fat in the diet—effects on lipoproteins: This topic is covered in the chapter on cardiovascular diseases, but there is an interaction between dietary composition and

weight change on lipoprotein levels. Many studies have manipulated the macronutrient content of short term diets under isocaloric weight stable conditions (such as reducing saturated fat and replacing the energy with carbohydrate or other types of fat). In many[58] but not all[97] such studies, the high carbohydrate diet is associated with increased triglycerides and decreased HDL-cholesterol (especially if predominantly simple carbohydrates are used).

The weight loss effect of a reduced-fat diet, *ad libitum* diet, however, appears to compensate for these potentially detrimental effects. Schaefer *et al.* directly compared the effects of shifting subjects from a high fat diet (35% of energy) to a low fat diet (15% of energy) under isocaloric (5–6 weeks) and *ad libitum* (10–12 weeks) conditions[98]. In order to achieve energy equivalence in the isocaloric part of the study, the weight and volume of the food consumed on the lower fat diet had to be increased by 30%. Under weight-maintenance conditions, on the low fat diet there was a significant reduction total, LDL- and HDL-cholesterol and an increase in total:HDL cholesterol ratio and plasma triglyceride concentrations. At the end of the *ad libitum* diet, subjects had lost an average of 3.6 kg and achieved greater reductions in total and LDL-cholesterol compared to the low fat isocaloric diet. The total:HDL cholesterol ratio and triglyceride levels were no different at the end of the *ad libitum* period compared to baseline.

This and other studies that assess the interaction between macronutrient composition and weight change on blood lipids[77,99,100] suggest that the effects of short term, isocaloric manipulations under metabolic ward conditions on lipids cannot be extrapolated to long term, *ad libitum* conditions in free-living individuals.

Summary of percent fat and obesity: At a macronutrient level, there is no evidence that energy from fat is more fattening than the same amount of energy from carbohydrate or protein. At a dietary level, there is still debate about the effects of diet composition on unhealthy weight gain, and more research is needed in this area. However, it was considered that the overall evidence from the randomised controlled trials was convincing that a high intake of energy-dense foods (which are often also micronutrient poor) promotes unhealthy weight gain. The short term, isocaloric substitution studies were considered far less relevant to free living individuals than the longer term, *ad libitum* studies. These latter studies show a highly consistent effect of a high fat content on promoting weight gain. The covert manipulations of fat content show that the effect is a physiological–behavioural one and is not dependent on conscious reductions in food eaten. The main mechanism for this appears to be that a diet high in fat has a weak impact on satiety because of its high energy density and this leads to a passive overconsumption of total energy. The high palatability of high fat foods and the relatively weak metabolic autoregulation in the face of a high fat diet are also likely contributors. While most high fat diets tend to be energy dense diets and thus weight-promoting diets, important caveats were noted. For example, many processed low fat foods were quite energy-dense and could promote weight gain if eaten in large amounts and conversely vegetable-based foods were quite energy dilute even with significant added fat and could protect against weight gain.

Carbohydrate type (sugar, glycemic index (GI) and non-starch polysaccharide (NSP))

The definitions of carbohydrates are often confusing. Sugars are predominantly monosaccharides and disaccharides. The term 'free sugars' has been defined in relation to the sugars that promote dental caries and refers to all mono and disaccharides added by the manufacturer, cook or consumer plus sugars naturally present in fruit juice, honey and syrups. Polysaccharides are either starch or NSP, the latter having considerable commonality with the term 'dietary fibre' which is still in common parlance and was the term used in many of the studies reviewed. Sugars, GI and NSP/fibre are considered in turn, although of course there is significant overlap between these factors within foods.

Sugar in foods: There is a reciprocal relationship between the percent fat and percent carbohydrate in the diet because these two nutrients generally contribute over 80% of total energy. Therefore, the previous section on percent fat could also be stated as: diets with a high carbohydrate content provide protection against weight gain. However, if the diet is high in sugar, does the same association apply? Large population studies have demonstrated that those who have high total energy intakes tend to have a high total sugar intake[101–106] although in relative terms, a reciprocal relationship is also seen between the percent fat and percent sugar in the diet[106]. Studies relating sugar intake to BMI consistently show an inverse relation between sugar intake as a percent of energy and BMI or obesity prevalence[106].

It is possible that the negative relationship between sucrose consumption and BMI is affected by confounding factors. For example, more active people need extra energy and this could be provided by sugar. Selective underreporting of high sugar foods and drinks by overweight/obese people is another possible confounder[107]. The high sugar content of some products with reduced fat claims may falsely imply that the products are low in energy as well.

Simple sugars have hedonistic value. Sweetening increases the palatability of many foods and it has been suggested that sweetness may lead to overconsumption[108]. However, there appears to be a limit to the hedonistic response to sweetened foods[109]. Palatability of foods is also increased by fat and therefore processed foods containing both high sugar and fat content may lead to weight gain[110]. Overall, the mixed results, especially amongst the few available trials, does not allow a

judgment to be made about the sugar content of food and obesity.

Studies have compared high fat diets with low fat diets that are high in either sugar or starch. Raben *et al.* found that similar amounts of energy were consumed on the high fat and high sucrose diets but there was a lower energy intake and weight loss with the high starch diets[111]. Saris *et al.* found a relative weight loss of 1.7 kg in the high sugar diet and 2.6 kg in the high starch diet compared to the high fat diet (both statistically significant) but the differences between carbohydrate types was not significant[100].

Sugar in drinks: The energy density of drinks such as regular soda drinks is low because of the high water content but physiologically the energy density of fluids and foods may have not have comparable effects on satiety and *ad libitum* food consumption[88,112]. It, therefore, seems prudent to consider the impact of drinks that contribute a significant amount to total energy intake (such as high sugar soda drinks) separately from foods.

In a cross-over study, Tordoff and Alleva[113] compared the consumption of soda (1150 g/d for 3 weeks) which had been sweetened with either a high fructose corn syrup or aspartame on body weight. The high fructose soda condition increased total energy intake by 335 kcal/d and resulted in a significant mean weight gain of 0.66 kg compared to the aspartame soda condition where total energy intake decreased by 179 kcal/d and weight decreased non-significantly by 0.17 kg.

From a US national survey, Harnack *et al.*[114] found that children ingesting nine or more ounces of soft drink per day consumed nearly 200 kcal/d more than those who did not drink soft drinks. In a longitudinal study in the US, Ludwig *et al.*[115] found a high intake of sugar drinks predicted the development of obesity over 19 months in 12-year-old children. They estimated that an increase of one can of soda per day increased the risk of obesity 1.6 times. This association was not seen with diet soda drinks.

Some fruit drinks and cordial drinks can also be high in sugar and may promote weight gain if drunk in large quantities but this has been less extensively studied than soda drinks.

Overall, the evidence that high sugar drinks promote weight gain is consistent and moderately strong, but is of most relevance in those populations with a high intake (such as children in many countries).

Glycemic index: A further mechanism by which carbohydrates may influence energy intake and body weight is by their GI. Different carbohydrate foods increase blood glucose and serum insulin to varying extents even when the same amount of carbohydrate is eaten. The different changes in glucose and/or insulin may have subsequent effects on food intake or the promotion of overweight and obesity[116], with lower GI diets producing greater satiety[117,118]. In addition to the effects of carbohydrates on satiety, there is a suggestion that low GI diets may provoke greater increases in cholecystokinin and fullness post-meal (satiation)[118,119].

Ludwig *et al.*[116] demonstrated that voluntary food intake was greater after high GI meals were consumed compared to medium and low GI meals. They found that rapid absorption of glucose altered hormonal and metabolic functions and promoted excessive food intake after the ingestion of a high GI meal. Agus *et al.*[120] found during a randomised cross-over trial that when the acute (9 days) effects of energy restricted diets of high and low glycemic loads were studied in overweight young men, the high glycemic load diet produced a greater decline in metabolic rate, more negative nitrogen balance and greater voluntary food intake.

On energy restrained diets, a 12 week cross-over trial by Slabber *et al.*[121] demonstrated that a low GI diet produced lower insulin levels and a greater weight loss than corresponding high GI diets. Spieth *et al.*[122] found that after 4 months of intervention, low GI diets may be more effective than reduced-fat diets in treating childhood obesity.

Low GI diets may influence fuel storage by promoting fat oxidation instead of carbohydrate oxidation[118], whereas raised insulin levels in response to high GI diets inhibit lipolysis and encourage fat storage[116,118], limiting available fuels and encouraging overeating[116].

Overall, the current evidence suggests a possible influence of GI on body weight and composition, but long term trials with changes in body weight as an outcome are needed before more definitive statements can be made[123].

Non-starch polysaccharide: A high intake of dietary NSP/fibre is generally, but not always associated with a lower BMI in epidemiological studies[124,125], but these studies are highly susceptible to measurement errors and confounding factors. Two recent reviews of trials of high versus low dietary NSP/fibre showed that the majority of studies supported a beneficial effect of NSP/fibre. Pereira and Ludwig[124] reported that 16 out of 27 trials reported beneficial subjective effects (satiety ratings) for high NSP/fibre meals or diets and 12 out of 19 showed beneficial objective effects (measured energy intake, gastric emptying, hormonal response or weight change). The review by Howarth *et al.* examined the impact of dietary NSP/fibre on satiety ratings, energy intake and weight change[125]. For studies of ≤ 2 days ($n = 20$) and > 2 days ($n = 18$), dietary NSP/fibre as a supplement or in foods either increased satiety or reduced hunger in the vast majority ($n = 27$ studies). The high NSP/fibre condition also resulted in a decrease in energy intake of about 10% ($n = 23$ studies). Studies of greater than 4 weeks duration with either a fixed energy intake ($n = 11$) or *ad libitum* intake ($n = 11$) showed consistent reductions in body weight with the high NSP/fibre condition. The mean weight loss for the fixed intake studies was 1.3 kg over 2.9 months (20 g/d) and for the

adlibitum studies was 1.9 kg over 3.8 months (24 g/d). In general, there were no differences between soluble, insoluble, or mixed fibre or between fibre as a supplement and within foods. Obese or overweight people tended to lose more weight than lean individuals (2.4 versus 0.8 kg).

A variety of mechanisms have been postulated to explain the effects of dietary NSP/fibre on energy balance and these include intrinsic effects of the NSP/fibre (such as on energy density and palatability), hormonal effects (such as gastric emptying and post-prandial glycemia and insulinemia), and colonic effects (such as fermentation to short chain fatty acids and effects on satiety)[124–126].

Overall, the evidence is convincing that a high dietary NSP/fibre intake helps to protect against weight gain.

Protein

The range of mean protein intakes across populations and across time is relatively small (10–15% of total energy)[127] and this limits the scope for influencing protein intake as a population measure to combat obesity. Nevertheless, protein is generally agreed to be the most satiating of macronutrients[128], particularly among people with a low habitual protein intake[129] and may influence body weight under *ad libitum*, reduced fat conditions[130]. Increasing protein intake may be beneficial for some individuals for weight control but the role of protein content of the diet at a population level is probably not an important determinant of obesity prevalence.

Alcohol

Alcohol is an energy dense nutrient (7 kcal/g) and because of its place at the top of the oxidative hierarchy[131], its potential for sparing fat oxidation and promoting fat storage is significant[132]. However, some metabolic studies show that isocaloric substitution of alcohol for food energy results in weight loss while the addition of alcohol does not promote weight gain[133]. There is a similar paradox seen in epidemiological studies. Dietary intake surveys tend to show that energy from alcohol is additive to food energy intake such that total energy intake is higher with a higher alcohol consumption[134]. However, the relationships between reported alcohol intake and BMI show a mixed pattern. One review of the epidemiological evidence, listed 25 studies showing a positive association, 18 showing a negative association and 11 showing no relationship[135]. For women, there was often a negative relationship[134] or possibly U-shaped relationship[131]. For men, the relationship tends to be slightly positive[134] or non-existent[131].

In an earlier review of 27 studies[136], seven showed a negative relationship between adiposity and alcohol intake, seven showed a positive relationship, nine showed different associations for women and men, and eight showed no relationship. Emery *et al.* reviewed the epidemiological studies linking a high alcohol intake with abdominal fat distribution (high waist circumference or waist:hip ratio)[137]. They concluded that the evidence for a relationship was moderate for men and suggestive for women.

The potential for confounding by concurrent lifestyle and socioeconomic factors is substantial, as is the tendency to underreport alcohol intake. Other factors also confound the relationships between alcohol and obesity: alcohol–macronutrient interactions; the possibility that obese people have reduced their alcohol consumption because of their obesity; metabolism through pathways with different energetic returns (e.g. alcohol dehydrogenase versus microsomal ethanol oxidizing system); and the direct toxic effects of alcohol[135]. Overall, the epidemiological evidence is mixed and probably highly confounded. Randomised controlled trials on the issue are unlikely to be conducted. There is currently insufficient evidence to support a general role for alcohol in the development of obesity.

Portion size

The portion size in pre-packaged, ready-to-eat and restaurant foods is increasing in the US and elsewhere, building on the consumers' desire for 'value for money'. In recent years the number of restaurants offering 'supersize' options on their menu has rapidly risen, and other food items, especially snack foods, have increased package weight[42]. The increasing size of packaging indicates lower unit cost and encourages use of more product than small package size[42,138]. These trends are occurring in many western countries but are less well documented than they are in the US.

'Supersized' portions potentially lead to increased energy intakes at the time and over the day and, therefore, could be a significant contributor to obesity, particularly in populations with a high use of meals prepared outside the home. Many people cannot accurately estimate portion size, and this leads to an underestimation of intake[42,138]. The energy compensation later in the day after a high energy meal is incomplete in many individuals[139]. Very few studies have examined the impact of portion size on overall energy consumption. One of these has shown that portion size promotes a higher total intake and that this seems to occur in adults and in 5 year olds, but not in 3 year olds[139]. The age at which the external cues (such as portion size) begin to influence intake is, therefore, appears to be between 3 and 5 years.

Overall, there is strong ecological evidence of a concurrent increase in portion sizing and obesity in countries such as the US. The proposition that large portion sizes promote overconsumption is logical and likely but the empirical studies, while supportive, are very few in number.

Environmental issues

The increasing 'obesogenicity' of the environments external to individuals is likely to be the major driving

force for the increasing obesity epidemic[34]. The environments in which people live are complex and their individual and combined elements have a marked effect on people's behaviours and dietary intakes. Individuals interact in a variety of micro-environments or settings such as schools, workplaces, homes, restaurants and fast food outlets[140]. These in turn are influenced by the broader macro-environments or sectors such as the food industry, all levels of government, and society's attitudes and beliefs. Much of the evidence of the impact of environments on dietary intake and obesity comes from cross-sectional associations and some intervention studies, although it is generally very difficult to tease out the impacts of specific environmental elements. The same statements also apply to environments that promote physical inactivity. The car-oriented design of built environments (coupled with the heavy promotion and affordability of cars and petrol), the increasing use of machines to replace occupational physical work, the increasing availability of energy-saving machines for every day tasks, and the expanding opportunities for passive recreation and entertainment are some of the dominating forces in influencing behaviours towards more inactive lifestyles[42].

Socioeconomic circumstances

While socioeconomic status (SES) is a characteristic of an individual (often measured by personal income or educational attainment), its underlying determinants are closely linked to the wider environment, especially to social, economic, employment and education policies. The relationship between SES and obesity is complex. The patterns are more exaggerated in women compared to men and children and generally show that in low income countries obesity is more prevalent in high SES individuals and in affluent countries, it is more prevalent in low SES individuals[141]. The change in obesity prevalence patterns can be seen in some countries that have monitored obesity prevalence rates over a period of economic transition[142]. It seems that in developed countries, the relationship may be bi-directional (i.e. low SES promotes obesity and obesity promotes low SES) as well as both obesity and low SES being independently influenced by other common factors such as intelligence[141]. The mechanisms by which high SES in developed countries provides some protection against obesity have not been well characterized and are likely to be multiple, including behaviours such as restrained eating practices and increased levels of recreational activity, living in less obesogenic environments with greater opportunities for healthy eating and physical activity, and a greater capacity to manipulate their micro-environments to suit their needs. People living in low SES circumstances may be more at the mercy of the increasingly obesogenic environment and end up taking the default choices on offer. Poorer neighbourhoods tend to have fewer recreation amenities[143], be less safe, and have a higher concentration of fast food outlets[144].

Overall, there is consistent support for the concept that, in affluent countries, a low SES is a risk factor for obesity in women and part of that effect is likely to be related to environments that are relatively deprived of healthier food choices and opportunities for physical activity.

Schools and other educational settings

Schools are key setting for influencing children and indeed, in a review of environment-based interventions to reduce energy intake or energy density[145], 24 out of the 75 identified studies were school-based. Overall, these interventions appeared to influence some of the behaviours in relation to food intake but only one showed an effect on obesity prevalence (in girls but not boys)[146].

Another likely obesogenic element in schools (particularly in the US) is the increasing number of soft drink vending machines in the schools and contracts the schools sign to achieve a required volume of sales[42]. One study has shown that a high consumption of high sugar soft drinks predicts increased weight gain[115].

The elements that contribute to the overall school food environment are: school food and nutrition policies (including the types of foods and drinks available and promoted at school through the school food service or vending machines); training opportunities and resources for teachers and food service staff; guidelines for offering healthy food and drink choices; promotion of healthy options in food brought from home; the curriculum content on food and nutrition; and the overall school ethos or culture on food and nutrition. Collectively, they probably influence dietary intake and, coupled with the physical activity environment, probably affect obesity prevalence but definitive evidence is still lacking around which elements are most important.

Home environment

The home and family environment is undoubtedly the most important setting in relation to shaping children's eating and physical activity behaviours, but, surprisingly, very little is known about the specific home influences. A recent review by Campbell and Crawford, however, has highlighted a number of elements in the home environment that are likely to be influential[147]. The availability, accessibility and exposure to a range of fruits and vegetables in the home was correlated with consumption[148], and at least two studies showed that repeated exposure to new foods seemed to reduce the propensity for young children to reject them[149,150]. Some aspects of the mother's nutrition knowledge, attitudes and behaviours are also significant predictors of a child's consumption of fruits, vegetables and confectionery[151]. A 1993 meta-analysis of five studies showed only a weak correlation between the food preferences of parent–child pairs[152] but the studies had significant design problems.

A more recent, better designed study found significant correlations between a toddler's food preferences and those of its family members with the most limiting factor being that a food had never been offered to a toddler, possibly because the mother did not like the food[153].

The review also noted that the continued trend for eating more food prepared outside the home has reduced the exposure of children to cooking skills learned in the home. In addition, there was some evidence that role modelling by peers and parents is another potentially important influence over children's eating patterns. Some experimental evidence suggests that using foods as a reward or restricting their availability increases the preference for those foods and that a high degree of parental control of a child's food intake correlated with a lower ability of the child to self regulate energy intake[147].

In summary, there was some empirical evidence and compelling logic that a wide range of factors in the home environment influenced food intake, although the caveat is that most of the studies involved white, middle class, US children of normal weight. As a setting, however, it is difficult to influence because of the sheer numbers and heterogeneity of homes and the limited options for access (with television advertising campaigns being the most effective but very expensive access option).

Food marketing sector

Fast food restaurants and energy-dense foods and drinks are among the most advertised products on television[42,154] and children are often the targeted market. The fat, sugar and energy content of foods advertised to children is very high compared to their daily needs and most of the foods advertised fall into the 'eat least' or 'eat occasionally' sections of the recommended dietary guidelines[155]. Many studies have documented that the overwhelmingly dominant messages that are directed at children, particularly through the powerful medium of television advertising, are the antithesis of what is recommended for a healthful diet[42,155–157].

The food industry (mainly fast food restaurants and manufacturers of high fat or high sugar foods and drinks) spends huge sums on mass media advertising, mainly through television advertisements. In 1997, they spent 11 billion US$ in the US alone[42]. The impact of this high volume of advertising on directing food choices to the products being advertised has undoubtedly been closely researched by the companies concerned but very little of this market research data is publicly available[156]. The high volume of advertising for energy dense foods and beverages is undoubtedly fuelling the increasing consumption of these products[158].

The prevalence of overweight and obesity is higher among children who watch more television[159], and the increased energy intakes of these children[42,160] may be partly responsible. Advertised products are more often requested for purchase and consumed by children[157,159]. Brand recognition not only encourages children to request products more often, but also targets those with discretionary spending money[156]. Children's behaviour has been shown to reflect television advertising patterns even when they know what they should be eating[157]. Young children under the age of about 6–8 cannot distinguish regular programmes from advertisements[161], nor do they understand the persuasive intent of commercials and overweight children with low self-esteem are more susceptible to commercials that promote consumption of foods for personal enhancement[159].

Overall, it is probable that the heavy advertising of fast foods and energy dense foods and drinks increases the consumption of those products. The evidence to support this rested on a wide variety of publicly available (often indirect evidence) studies and the huge advertising investment in influencing food choice behaviours which was based on more direct but proprietary evidence.

Other environments

There are a wide variety of settings in which people gather and that involve food and eating. Most of the evidence for their impact on eating patterns comes from cross-sectional surveys or intervention studies. Restaurants[162], work-places[163,164], supermarkets[165] and a variety of other settings probably each provide modest influences on dietary intakes and obesity but good evidence is either limited or non-existent[145].

Potential strategies to reduce obesity

A number of potential strategies to reduce obesity prevalence are described below and summarised in Table 2. The content of the potential intervention is outlined, along with a summary of the evidence of effectiveness and the implications for governments, civil society, the private sector and the international agencies. This is not an exhaustive list of potential interventions and indeed each intervention activity would need to be specific to the locality and culture of the intervention population. Strategies to increase physical activity levels would be a key part of any overall approach to obesity prevention, however, these are not considered in detail in this review of diet and nutrition approaches. A review of the evidence support for some strategies to increase physical activity has been published by the Center for Disease Control in the US[6].

Fiscal food policies

Food prices have a marked influence on food buying behaviour and consequently nutrient intakes[166]. Governments have price policy instruments at their disposal (principally subsidies and taxes) to influence buying patterns. In the case of cardiovascular disease, Marshall[167] has estimated that a tax (17.5% value added

Table 2 Potential nutrition intervention strategies to reduce overweight/obesity prevalence

Potential nutrition intervention strategies	Content	Evidence support	Recommendations
Fiscal policies to influence consumption patterns	Re-aligning fiscal policies that influence the food supply to ensure that the population has access to a safe, nutritious, affordable food; fiscal policies to discourage the intake of high fat/sugar foods with low essential nutrient density	Good evidence that food prices, availability and access influence buying patterns	*Gov, IC:* Conduct health impact assessments of fiscal policies that affect the food supply; modify such policies to improve health *CS:* Advocacy for policies to improve food supply *PS:* Support policies to improve food supply
Levies for use in nutrition promotion	Small levies on certain high volume foods/drinks (high fat and/or sugar)	Small taxes on high volume products raise substantial revenue (tobacco levy model for health promotion)	*Gov:* Consider levies to support nutrition promotion programmes
Nutrition information panels	Appropriate labelling of nutrient content of food on the package; communication strategies to educate consumers on the use of the panels	Widely used (especially by women, higher income people and those with health conditions); probably influences food formulations	*Gov:* Mandatory labelling laws; funding communications strategies to consumers (especially lower income people) *IC, PS, CS:* Support NIPs
Food claims	Regulatory framework to minimise misleading food, health, and nutrition claims	Food claims influence food choices	*Gov:* Implement and monitor a regulatory framework; allow a 'low fat' claim only with equivalent reduced energy density *IC, CS:* Support food claims framework *PS:* Develop reduced fat foods that have equivalent reductions in energy density
Nutrition signposting	Endorsed logos from respected organisations showing products meet nutrition standards	Signposts used by high proportion of the public; influences food formulations	*CS:* Consider the establishment of signposting programmes *Gov, PS:* Support programmes
Minimize marketing of energy dense foods to young children	Bans on advertising on television to young children; bans on marketing energy dense foods or drinks in school	Strong evidence on the high fat/sugar content of food ads; strong evidence that ads influence food purchases; high investment in TV ads	*Gov:* Legislation and broadcasting policies to ban television ads to young children; education sector policies to ban food/drink marketing in schools *CS, IC, PS:* Support ad bans
Community-wide programmes	Multiple settings—education, mass media and environmental interventions	Modest effects at best of previous interventions (mainly educational)	*Gov:* Fund research in 'demonstration communities' to build the evidence on interventions (including environmental) to improve eating patterns
Schools and other educational settings	Food policies/guidelines/standards; influencing food sold or brought to school; nutrition/cooking skills in curriculum	Good evidence for influencing some dietary patterns, usually insufficient to influence BMI	*Gov:* Policies/guidelines/standards on school food; maintenance of physical education; nutrition in curriculum *PS, IC, CS:* Support healthy school food programmes
Work place interventions	Food policy, healthy food available and promoted	Modest effect at best on some dietary patterns	*PS* support for health promotion programmes in workplaces
Breastfeeding	Education and BF support services, health professional training, BF-friendly environments, BF monitoring programmes	Good evidence that programmes increase BF rates, good evidence that BF improves many health outcomes but evidence for prevention of obesity is mixed	*Gov:* High priority for funding BF programmes, training, and monitoring systems; enforce Code on breastfeeding and breastmilk substitutes *PS, IC, CS:* Support BF and monitor code
Healthy eating guidelines and messages	Communication strategies around key food-based messages for the population	Raises awareness and knowledge; may influence behaviour in higher SES groups; necessary but not sufficient	*Gov:* Support for the development, updating, dissemination, promotion of food-based dietary guidelines; link with other strategies *CS, PS:* Support and promote FBDG

Table 2. *Continued*

Potential nutrition intervention strategies	Content	Evidence support	Recommendations
Individual advice on prevention of weight gain and management of overweight	Health consultations involve management of diet and physical activity behaviours	Some evidence of the influence of physicians/ health professionals on dietary and physical activity behaviours—less evidence of long term effectiveness	*Gov:* Support for training programmes and support for health professionals
Transport infrastructure and policies that promotes active transport*	Public transport, cycle paths, footpaths/walking paths	Strong evidence that costs and availability of car versus active transport options influences transport mode choice	*Gov:* Develop policies and strategies to shift transport mode from car to active transport options
Urban design that promotes physical activity	Mixed land use; medium/high density housing; transport corridors and nodes; connectivity of street design; recreation space and facilities	Ecological evidence on various aspects of urban design on transport mode choice	*Gov:* Develop policies and strategies to create and 'retro-fit' urban built environments to make them conducive to active transport and active recreation
Community-wide physical activity campaigns	Large-scale, high intensity campaigns with high visibility. Includes media, support groups, community events, risk factor screening and education, and creation of walking tracks	Ten studies reviewed and these interventions were strongly recommended by CDC	*Gov, PS, CS:* Funding and support for multi-component campaigns
Point of decision prompts to encourage stair use	Motivational signs placed close to elevators and escalators encouraging use of nearby stairs	Six studies reviewed and this intervention was recommended by CDC	*Gov:* Review building codes to allow freer access and availability of stairs. *PS, CS:* Support for local initiatives in buildings.
Individual adapted health behaviour change programmes	Programmes tailored to the individual and delivered in group settings, or by mail, telephone or directed media	Eighteen studies reviewed and these interventions strongly recommended by CDC	*Gov, CS, PS:* Funding and support for individually tailored physical activity programmes
School-based physical education	Curricula and policies to increase moderate and vigorous activity	Thirteen studies reviewed and this intervention was strongly recommended by CDC	*Gov:* School policies on physical education and physical activity participation
Social support interventions in community settings	Building and strengthening social networks to support physical activity (e.g. buddy systems, walking groups, workplace-based groups)	Nine studies reviewed and this intervention was strongly recommended by CDC	*PS, CS:* Enable and support the strengthening of social networks to encourage physical activity
Enhanced access to places for physical activity with informational outreach activities	Increasing access and reducing barriers to places for physical activity	Twelve studies reviewed and this intervention was strongly recommended by CDC	*Gov:* Policies and funding support to enhance access to places for physical activity. Monitoring access. *PS. CS:* Support improved access

Gov, national government; CS, civil society; IC, international community; PS, private sector.
* Active transport includes walking, cycling and public transport.

tax) on the main sources of saturated fat in the diet would prevent between 900 and 1000 deaths a year in the UK by reducing the demand for those foods. Such proposals are usually contentious[168] and governments are generally loath to enact such policies.

Another fiscal concept is the application of a small tax (too small to affect sales) on high volume foods of low nutritional value such as soft drinks, confectionery and snack foods. Such taxes currently apply in several jurisdictions in the US and Canada[169] although none of this revenue is used for health gains (such as funding nutrition programmes or subsidising fruits and vegetables) but that possibility remains.

Nutrition information panels

Full nutrition information panels on foods have been or are being introduced by regulation in many countries. Do nutrition panels influence the food choices made by consumers and/or the food formulations made by manufacturers towards foods with a lower fat or energy content? Most of the available information comes from cross-sectional surveys from the US where nutrition panels have been mandatory on packaged foods since 1994[170].

In the US, about two thirds of people report using nutrition panels[42] and this appears to significantly influence food choices. One survey reported that nutrition panels had changed food choices for 56% of respondents and in another survey, nutrition panels had stimulated some food selections and averted other selections in 22 and 34% of respondents, respectively[171]. A higher use of nutrition panels was associated with female sex[172], higher educational attainment[170] greater nutrition knowledge and awareness[173] and a stronger belief in the importance of diet in the prevention or management of disease[170].

People with some existing conditions such as obesity, hypertension or high cholesterol are more likely to use nutrition panels. Obese people are more likely to refer to grams of fat or calorie information than normal-weight people[170], and those who reported being on a low fat diet were almost ten times more likely to read the label[42,170]. The difference in percent energy from dietary fat between label readers and non-label readers has been variously estimated as 13%[174], 9% (unadjusted) and 5% (adjusted for demographic, psychosocial and behavioural variables)[170]. The relationship between label-reading and fruits and vegetables consumption is less clear[170,175].

On balance, mandated nutrition information panels appear to (1) facilitate the food choices of those who are trying to reduce their fat intake, (2) influence the food choices of a large proportion of the population, (3) have greater impact among women, higher educated people and those with established beliefs and knowledge about diet–disease relationships. The impact of mandatory nutrition panels on the formulation and reformulation of manufactured foods may also be significant but it is not well documented. The magnitude of the potential impact and wide reach of mandatory nutrition information panels argue for this to be a key strategy to improve the nutrition status of populations. However, it needs to be complemented by other strategies that will influence the food choices of low income and less educated consumers[173].

Nutrition 'signposting' programmes

Nutrition 'signposts' are signals (such as logos) at point of choice which indicate to the consumer that a food meets certain nutrition standards. An example is the 'Pick the Tick' symbol programme run by the National Heart Foundations in Australia and New Zealand[176]. While such signpost systems and endorsements have attracted some criticism[177], they do make identifying healthier food choices simpler for consumers[176], and are frequently used by shoppers when choosing products[176,177]. In addition, the nutrition criteria for the products serve as '*de facto*' standards for product formulation and many manufacturers will formulate or reformulate products to meet those standards[177]. Energy density criteria are needed for low fat products.

Nutrition claims

Nutrition claims are regulated in most countries because of the potential for misleading information to be promoted. In the US, over the last 10 years, 20–37% of new products carry a nutrition claim with over half those claims in recent years being for reduced or low fat[171]. The claims clearly provide information about some aspect of the content of the food but for some restrained eaters, 'low fat' or 'low calorie' claims can become an unconscious message to eat more of the product or accompanying foods[178]. In some manufactured products, the fat content has been reduced so that a low fat claim can be made, but the energy density remains high[87] negating the potential benefit for reducing weight gain.

Overall, nutrition claims are an important influence on the food choices of consumers and the formulation of food by manufacturers. There can be some negative consequences when the messages or signals are misleading or are taken in the wrong way. The regulations for nutrition claims need to ensure that products with claims for 'low or reduced fat' also have comparable reductions in energy density so that low fat, high energy foods are excluded from making the claim.

Reducing food marketing directed at children

Reducing the huge volume of marketing of high fat/sugar foods and drinks and fast food restaurants to young children, particularly through the powerful medium of television advertisements, is a potential strategy that is advocated by a number of health and community-based organisations[161,179]. It is, of course, a highly contentious strategy. The advertising and food industries oppose regulations to limit advertising to children and focus on nutrition education and physical activity strategies[180].

The evidence to show that banning television advertising to young children influences behaviours is not available because there is very little experience in this strategy. Television advertising to children under 12 years has not been permitted in Sweden since commercial television began over a decade ago. Norway, Austria, Ireland, Australia, Greece and Denmark also have some restrictions on television advertising to young children.

The implementation of this strategy will, therefore, rest on a strong rationale and the political will to reduce the use of young children as a target for promoting foods that are not recommended in the dietary guidelines[181]. The whole area is very complex and any regulations would not be able to protect children from all aspects of marketing of fast food restaurants and high fat/sugar foods and drinks and nor would advertising bans solve childhood obesity. However, there is currently a substantial imbalance in the volume and sophistication of messages aimed at children that promote energy dense, micronutrient-poor products versus healthier options.

A strong argument could be made for banning all television advertisements aimed at young children because the advertisements depend on 'pester power' and seek to undermine the parents' attempts at providing healthy food choices for their children. The fact that children would still be seeing some television advertisements during adult programming times or through other marketing strategies such as billboards and print media does not negate the rationale for controls on the use of the most persuasive medium (television) aimed at the most vulnerable and gullible of audiences (young children).

Public debates over balancing the competing rights of companies versus parents and policy analyses of the potential impacts of advertisement bans would be important first steps in such a controversial strategy.

Community-wide interventions

There have been at least seventeen community-based interventions to improve dietary patterns and related risk factors such as BMI or serum cholesterol levels[145]. Many of the trials involved sizeable communities (over 1,00,000 people) and long periods (some over 10 years) and most were in North America and Europe. Most of these studies, however, did not find any significant change in dietary behaviours or risk factors. In those studies with statistically significant changes, the magnitude of the change was only modest. The favourable results for reducing cardiovascular risk from the North Karelia project were impressive especially in the early years of the project, but reducing the population's mean BMI was not one of the main targets for intervention and this remained unaffected by the project[182]. The Minnesota Heart Health programme was a large randomised field trial that failed to influence cardiovascular risk or obesity in the intervention populations over a 6-year period[183]. Many of the interventions had strong educational, mass media or mass screening components and few involved more environmental or policy initiatives.

At the moment there is insufficient evidence to support the widespread use of community-based interventions to reduce obesity. The approach, however, remains attractive for further research because it has the potential to influence a large number of people in a variety of settings. A greater focus on environmental changes and limiting the interventions to 'demonstration areas' may help to build the evidence in this area.

Schools and other educational institutions

Schools, pre-schools and after-school care settings are well placed to influence the food environment and learning opportunities around nutrition as well as being a potential vehicle to influence parents and the wider community. A 1997 review of school-based cardiovascular disease prevention studies that involved classroom health education identified 16 such studies, of which 10 were from the US[184]. While the average percent of positive outcomes across the trials was 31% (effect ratio), for adiposity it was only 16%.

The largest study (CATCH) involved over 5000 children in 56 intervention schools and 40 control schools over 3 school years[185]. The interventions were school food service modifications, enhanced physical education and increased classroom health curricula in 28 schools and the above components plus family education in another 28 schools. While the interventions significantly decreased fat in the school lunches and increased vigorous activity, no differences in body size were seen between the intervention and control schools. A more recent, smaller study (10 schools) from the UK used a variety of school-based interventions, most of which were successfully implemented[186]. Despite these promising changes, there was no difference in body size changes between intervention and control schools[187].

The Planet Health study was a multi-dimensional intervention study that involved nearly 1300 children in 10 schools and it did show a decrease in obesity prevalence (in girls only). The intervention was an educational one focused on decreasing television viewing, decreasing high fat food, increasing fruit and vegetable intake and increasing physical activity[146].

In a review of environmental interventions to reduce energy and fat intake in educational settings, Hider found 24 studies, 21 of which were in primary schools[145]. Changes in food sales were noted in some studies[188], but dietary behaviour was unaltered in most cases.

Overall, it appears that school-related behaviours can be influenced by school-based programmes but these are not usually of sufficient magnitude to influence changes in obesity prevalence. This may not be surprising when one considers that the vast majority of children's waking hours over a year are spent outside school. A reduction in television viewing seems to be a key strategy for

school-based education intervention[189]. Further intervention research is needed in school-based programmes with potentially greater effectiveness achieved by integrating school programmes into community programmes and by focusing on broader environmental and community changes[145,184].

Workplaces

A review of studies of workplace-based interventions on aspects of dietary intake, found 16 studies, 10 of which were from the US and seven were randomised trials[145]. They used a mixture of educational and environmental strategies, mainly centred on the workplace canteen. The results were mixed with some showing no impact of the programme and others showing significant but modest benefits in dietary intake and food sales indicators. A sustained impact on reducing BMI has been difficult to achieve, although reduced serum cholesterol levels have been achieved in some studies[190].

While workplaces remain an important setting to reach adult men, the impact of intervention programmes have been modest at best.

Breastfeeding

In a systematic review of breastfeeding promotion programmes, Fairbank *et al.*[191] found a positive effect of the programmes on increasing the initiation of breastfeeding in most (41 of 48) studies reviewed, with a statistically significant difference being demonstrated in just under half of the intervention groups (20 of 48) when compared to control groups. No interventions reported statistically significant negative effects. The provision of literature alone did not appear to be successful in increasing breastfeeding initiation[192], however, group or regular one-on-one education not only increased the initiation but also duration of breastfeeding[193], especially among women who planned to bottlefeed. More success was reported for immigrant women when culture-specific education was used[194]. From these studies, it has been demonstrated that breastfeeding initiation and duration rates can be increased when information is delivered in small, informal, culture-specific groups[191].

Promotion of healthy eating guidelines and messages

The mass media campaigns and other communication strategies are used in many countries to educate the public about healthy eating choices. In the US, the 'Healthy Eating Pyramid', the '5-a-day for better health' campaign and National Cholesterol Education Programme[42] are well known examples.

Such campaigns and promotions can have a significant impact on awareness, attitudes, knowledge, and intention to change[195] but behavioural change is not usually influenced. An exception may be if the message is highly specific and achievable. An example of such a campaign targeted a behaviour change from using high fat to low fat milk. Low fat milk sales increased by about 50% (from 29% of total sales to 46%) and this was maintained 6 months after the campaign[196].

The messages appear to be more widely heard and acted upon by people from higher socioeconomic backgrounds and with a higher educational attainment[197]. Surveys have shown that although compliance with recommendations has risen since the 1980s, and many people had heard some messages, there was still a considerable number of people who were not familiar with the dietary guidelines[197,198]. Obesity is increasing at a time when healthy eating messages have never been more prevalent.

Overall, the promotion of healthier eating choices appears to be a necessary strategy for improving dietary intake, but is unlikely to be sufficient for most people to change behaviours. It may be an important motivating force for higher income people (who tend to live in less obesogenic environments) but for low income groups it is generally not sufficient to effect significant behaviour changes.

Treatment

Substantial proportions of the population in many parts of the world are already overweight or obese, and therefore, efforts to reduce obesity prevalence must not only focus on the prevention of obesity in those who have not yet become obese (primary prevention), but also on prevention of further weight gain and promotion of weight loss in those who are already obese before they develop the complications of obesity (secondary prevention). In contrast to the primary prevention of obesity, where only limited evidence exists to support many of the potential interventions, substantial data exist on efficacious approaches to treatment. The principal setting for weight loss is likely to be in physician offices or other health care settings.

Approaches to weight loss and weight maintenance have been summarised in various evidence-based reports such as the NIH clinical guidelines on the identification, evaluation and treatment of overweight and obesity in adults[199]. In general, treatments using diet, physical activity and medications are 'efficacious' (i.e. they promote weight loss in clinical trial settings with careful patient selection, close monitoring and high adherence rates in the short term). Studies with long term follow up, however, are disappointing as patients return towards their original weights. What is, therefore, lacking are positive 'effectiveness' studies whereby the efficacious interventions are translated into practice in settings where medical care is routinely delivered. Because primary care settings are where most patients are seen and where most physicians work, demonstrably effective obesity treatment strategies are essential. Gastroplastic surgery is virtually

the only intervention with long term proven effectiveness and cost effectiveness[200].

It is evident that there are a number of major barriers to the effective management of obesity within most health systems. It is beyond the scope of this report to detail these, but they include a lack of effective interventions in primary care settings, the lack of self-efficacy on the part of physicians for managing obesity, the dearth of trained health professionals to oversee and provide these therapies[201], and in most countries, the financial reimbursement systems that mitigate against best practice management of obesity. The paradox is that, while there is little investment in financing obesity management, there are huge downstream costs associated with the management of its complications.

Physical activity

Potential strategies for promoting physical activity are also shown in the table for completeness. Most of these come from the US Center for Disease Control and Prevention review of the effectiveness of interventions to increase physical activity[6].

Population goals and dietary guidelines

Population monitoring programmes for obesity and certain nutrients are a vital part of a comprehensive strategy to reduce obesity. The classification categories of BMI and waist circumference are those defined in the previous WHO report on obesity[8] although the debate continues about appropriate cut off points for some non-European ethnic groups[17]. A population goal for median BMI for adults is between 21 and 23 kg/m^2 because this reflects an optimum distribution where the population proportions of underweight and overweight are minimised. For adults, individual goals should be to remain in the healthy weight range (BMI 18.5–25 kg/m^2) and to gain less than 5 kg over adult life.

The relevant population nutrient goals relate to dietary fat, free sugars and dietary NSP/fibre. The mean total fat intake of a population is an indicator of energy density and it should be less than 30% of energy. Similarly, a mean free sugar intake of less than 10% of energy would also reflect a low mean energy density of foods and drinks. The free sugar population goal is particularly important for children. Population groups that are very physically active and have a diets that are high in vegetables, legumes, fruits and whole grain cereals may sustain a total fat intake of up to 35% without the risk of unhealthy weight gain. Achieving these population goals will require, among other things, the development of food-based dietary guidelines that are relevant and specific for each country. Guidelines on the preparation and use of food-based dietary guidelines are published elsewhere[202]. The physical activity levels needed to avoid unhealthy weight gain in adult life are unknown but may in the order of an hour per day of moderate intensity activity on most days of the week[7].

Conclusions

Obesity is arguably the biggest challenge among the epidemics facing the world because it is on the rise in low- and high income countries, no country has a track record in terms of attenuating and reversing the epidemic, and it has several major downstream health consequences in terms of diabetes, cardiovascular diseases, some cancers and arthritis that are very common and expensive to treat.

The Epidemiological Triad is helpful in identifying potential food and nutrition drivers of the epidemic and strategies for interventions. The main food-related vectors that promote the passive overconsumption of total energy are: energy dense foods (principally related their fat content but sometimes their carbohydrate content), high energy drinks, and large portion sizes. The environmental factors tend to be multiple in each of the settings in which food is consumed and include physical, economic, policy and socio-cultural dimensions. There is an urgent need to focus attention on measuring these environmental influences, assessing their impacts on energy intake and testing interventions designed to make them less obesogenic. Much more research is needed in these area and some recommendations for priorities in future research are shown in Table 3.

A variety of potential interventions and their implications have also been outlined. Overall, the level of evidence for population-based interventions is weak either because they have been tried and shown to have a modest impact (such as dietary guidelines and workplace interventions) or they have not been tried and evaluated (such as fiscal food policies and banning

Table 3 Research recommendations

- Build evidence of the impact of environmental/educational interventions in a variety of settings such as schools and other educational settings, workplaces, restaurants/cafeterias/cafés and other settings and institutions with catered food.
- Build the evidence of the impact of food labelling interventions (nutrition information panels, food claims, signposting programmes) on consumer choice, food formulation and dietary patterns.
- Improve the methods for measuring body composition, dietary intake, physical activity in populations.
- Develop and validate indicators for environmental determinants of obesity and weight gain.
- Maintain and enhance systems for monitoring trends in overweight/obesity, nutrition and physical activity and their environmental determinants.
- Conduct the body composition studies and prospective studies needed to define equivalent (equivalent body composition/equivalent disease risk) BMI values across different ethnic groups and define ethnic-specific cut-off points.
- Conduct studies to define the mechanisms by which low SES promotes overweight and obesity.
- Conduct trials on the impact of carbohydrate type (glycemic index) on body weight.

television advertisements to young children). In either case, the need to continue to develop and evaluate population-based interventions (especially environmental interventions) is paramount. A failure to act in a substantive way will undoubtedly result in continued massive increases in obesity and its complications—the burden of which will become unbearable for most countries.

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References

1 World Cancer Research Fund. *Food, Nutrition, and the Prevention of Cancer: A Global Perspective*. Washington, DC: American Institute for Cancer Research, 1997.
2 Black AE, Goldberg GR, Jebb SA, Livingstone MBE, Cole T, Prentice AM. Critical evaluation of energy intake data using fundamental principles of energy physiology: 2. Evaluating the results of published surveys. *European Journal of Clinical Nutrition* 1991; **45**: 583–99.
3 Heitmann BL, Lissner L. Dietary underreporting by obese individuals—is it specific or non-specific? *British Medical Journal* 1995; **311**(7011): 986–9.
4 Briss PA, Zaza S, Pappaioanou M, Fielding J, Wright-De Aguero L, Truman BI, *et al*. Developing an evidence-based Guide to Community Preventive Services—methods. The Task Force on Community Preventive Services. *American Journal of Preventive Medicine* 2000; **18**(1): 35–43.
5 DiPietro L. Physical activity, body weight, and adiposity: an epidemiologic perspective. *Exercise and Sport Sciences Review* 1995; **23**: 275–303.
6 Centers for Disease Control and Prevention. *Increasing Physical Activity. A Report on Recommendations of the Task Force on Community Preventive Services*. Atlanta GA.: Centers for Disease Control and Prevention, 2001.
7 WHO. Weight control and physical activity. In: Bianchini F, ed. *IARC Handbooks of Cancer Prevention*. Lyon, France: IARC Press, 2002.
8 *Obesity: Preventing and Managing the Global Epidemic. Report of a WHO Consultation*. WHO Technical Report Series No. 894. Geneva, Switzerland: WHO, 2000.
9 Seidell JC. Obesity: a growing problem. *Acta Paediatrica* Supplement 1999; **88**(428): 46–50.
10 Bell AC, Ge K, Popkin BM. Weight gain and its predictors in Chinese adults. *International Journal of Obesity and Related Metabolic Disorders* 2001; **25**(7): 1079–86.
11 Hodge AM, Dowse GK, Zimmet PZ. Obesity in Pacific populations. *Pacific Health Dialog* 1996; **3**(1): 77–86.
12 Bell AC, Swinburn BA, Simmons D, Wang W, Amosa H, Gatland B. Heart disease and diabetes risk factors in Pacific Islands communities and relationships with measures of body fat. *New Zealand Medical Journal* 2001; **114**: 208–13.
13 Seidell JC, Rissanen A, *Prevalence of Obesity in Adults: The Global Epidemic*. In: Bray GA, Bouchard C, eds. Handbook of Obesity, 2004 (In press).
14 Pena M, Bacallao J, ed. *Obesity and Poverty: A New Public Health Challenge*. Washington, DC: Pan American Health Organization (PAHO), 2000.
15 Monteiro CA, Mondini L, de Souza AL, Popkin BM. The nutrition transition in Brazil. *European Journal of Clinical Nutrition* 1995; **49**(2): 105–13.
16 Swinburn BA, Ley SJ, Carmichael HE, Plank LD. Body size and composition in Polynesians. *International Journal of Obesity and Related Metabolic Disorders* 1999; **23**(11): 1178–83.
17 WHO Regional Office for the Western Pacific. *The Asia-Pacific Perspective: Redefining Obesity and its Treatment*. Manila, Philippines: World Health Organization, 2000.
18 Deurenberg P, Deurenberg-Yap M, Guricci S. Asians are different from Caucasians and from each other in their body mass index/body fat per cent relationship. *Obesity Reviews* 2002; **3**(3): 141–6.
19 Sjostrom L. Impacts of body weight, body composition, and adipose tissue distribution on morbidity and mortality. In: Stunkard AJ, Wadden TA, eds. *Obesity: Theory and Therapy*. 2nd ed; New York: Raven Press, 1992.
20 McKeigue PM. Metabolic consequences of obesity and body fat pattern: lessons from migrant studies. In: Shetty PS, McPherson K, eds. *The Origins and Consequences of Obesity*. Chichester: John Wiley & Sons, 1996.
21 Popkin BM. The nutrition transition and obesity in the developing world. *Journal of Nutrition* 2001; **131**(3): 871S–3S.
22 Drewnowski A. Nutrition transition and global dietary trends. *Nutrition* 2000; **16**(7–8): 486–7.
23 Jung RT. Obesity as a disease. *British Medical Bulletin* 1997; **53**(2): 307–21.
24 Manson JE, Willett WC, Stampfer MJ, Colditz GA, Hunter DJ, Hankinson SE, *et al*. Body weight and mortality among women. *New England Journal of Medicine* 1995; **333**(11): 677–85.
25 Zimmet P, Alberti KG, Shaw J. Global and societal implications of the diabetes epidemic. *Nature* 2001; **414**(6865): 782–7.
26 Colditz GA, Willett WC, Rotnitzky A, Manson JE. Weight gain as a risk factor for clinical diabetes mellitus in women. *Annals of Internal Medicine* 1995; **122**1 April: 481–6.
27 Savage PJ, Bennett PH, Senter RG, Miller M. High prevalence of diabetes in young Pima Indians: evidence of phenotypic variation in a genetically isolated population. *Diabetes* 1979; **28**(10): 937–42.
28 Rosenbloom AL, Joe JR, Young RS, Winter WE. Emerging epidemic of type 2 diabetes in youth. *Diabetes Care* 1999; **22**(2): 345–54.
29 Cockram CS. The epidemiology of diabetes mellitus in the Asia-Pacific region. *Hong Kong Medical Journal* 2000; **6**(1): 43–52.
30 Segal L, Carter R, Zimmet P. The cost of obesity: the Australian perspective. *Pharmacoeconomics* 1994; **5**(Suppl.): 45–52.
31 Swinburn B, Ashton T, Gillespie J, Cox B, Menon A, Simmons D, *et al*. Health care costs of obesity in New Zealand. *International Journal of Obesity and Related Metabolic Disorders* 1997; **21**(10): 891–6.
32 Seidell JC. The impact of obesity on health status: some implications for health care costs. *International Journal*

of Obesity and Related Metabolic Disorders 1995; **19**(Suppl. 6): S13–6.
33 Wolf AM, Colditz GA. Social and economic effects of body weight in the United States. *American Journal of Clinical Nutrition* 1996; **63**(3 Suppl): 466S–9S.
34 Egger G, Swinburn B. An "ecological" approach to the obesity pandemic. *British Medical Journal* 1997; **315**(7106): 477–80.
35 Green SM, Burley VJ. The effects of snacking on energy intake and body weight. *Nutrition Bulletin BNF* 1996; **21**: 103–7.
36 Zizza C, Siega-Riz AM, Popkin BM. Significant increase in young adults' snacking between 1977–1978 and 1994–1996 represents a cause for concern! *Preventive Medicine* 2001; **32**(4): 303–10.
37 Drummond S, Crombie N, Kirk T. A critique of the effects of snacking on body weight status. *European Journal of Clinical Nutrition* 1996; **50**(12): 779–83.
38 Mela DJ. Determinants of food choice: relationships with obesity and weight control. *Obesity Research* 2001; **9**(Suppl. 5): S249–55.
39 Smith CF, Williamson DA, Bray GA, Ryan DH. Flexible vs. rigid dieting strategies: relationship with adverse behavioural outcomes. *Appetite* 1999; **32**(3): 295–305.
40 Yanovski SZ, Sebring NG. Recorded food intake of obese women with binge eating disorder before and after weight loss. *International Journal of Eating Disorders* 1994; **15**(2): 135–50.
41 Schenck CH, Mahowald MW. Review of nocturnal sleep-related eating disorders. *International Journal of Eating Disorders* 1994; **15**(4): 343–56.
42 French SA, Story M, Jeffery RW. Environmental influences on eating and physical activity. *Annual Reviews of Public Health* 2001; **22**: 309–35.
43 Lin B-H, Frazao E, Guthrie J. *Away-From-Home Foods Increasingly Important to Quality of American Diet*. Jan. Report No.: Agriculture Information Bulletin No. 749. Washington, DC: Economic Research Service, US Dept of Agriculture, 1999.
44 McCrory MA, Fuss PJ, Hays NP, Vinken AG, Greenberg AS, Roberts SB. Overeating in America: association between restaurant food consumption and body fatness in healthy adult men and women ages 19 to 80. *Obesity Research* 1999; **7**(6): 564–71.
45 Jeffery RW, French SA. Epidemic obesity in the United States: are fast foods and television viewing contributing? *American Journal of Public Health* 1998; **88**(2): 277–80.
46 Binkley JK, Eales J, Jekanowski M. The relation between dietary change and rising US obesity. *International Journal of Obesity and Related Metabolic Disorders* 2000; **24**(8): 1032–9.
47 Dietz WH. Breastfeeding may help prevent childhood overweight. *Journal of the American Medical Association* 2001; **285**(19): 2506–7.
48 Stark O, Atkins E, Wolff OH, Douglas JW. Longitudinal study of obesity in the National Survey of Health and Development. *British Medical Journal (Clinical Research Education)* 1981; **283**(6283): 13–7.
49 Butte NF. The role of breastfeeding in obesity. *Pediatric Clinics of North America* 2001; **48**(1): 189–98.
50 von Kries R, Koletzko B, Sauerwald T, von Mutius E, Barnert D, Grunert V, *et al.* Breast feeding and obesity: cross sectional study. *British Medical Journal* 1999; **319**(7203): 147–50.
51 Wadsworth M, Marshall S, Hardy R, Paul A. Breast feeding and obesity. Relation may be accounted for by social factors. *British Medical Journal* 1999; **319**(7224): 1576.
52 Gillman MW, Rifas-Shiman SL, Camargo CA Jr., Berkey CS, Frazier AL, Rockett HR, *et al.* Risk of overweight among adolescents who were breastfed as infants. *Journal of the American Medical Association* 2001; **285**(19): 2461–7.
53 Hediger ML, Overpeck MD, Kuczmarski RJ, Ruan WJ. Association between infant breastfeeding and overweight in young children. *Journal of the American Medical Association* 2001; **285**(19): 2453–60.
54 Parsons TJ, Power C, Logan S, Summerbell CD. Childhood predictors of adult obesity: a systematic review. *International Journal of Obesity and Related Metabolic Disorders* 1999; **23**(Suppl. 8): S1–107.
55 Popkin BM, Richards MK, Montiero CA. Stunting is associated with overweight in children of four nations that are undergoing the nutrition transition. *Journal of Nutrition* 1996; **126**(12): 3009–16.
56 Schroeder DG, Martorell R, Flores R. Infant and child growth and fatness and fat distribution in Guatemalan adults. *American Journal of Epidemiology* 1999; **149**(2): 177–85.
57 Godfrey KM, Barker DJ. Fetal nutrition and adult disease. *American Journal of Clinical Nutrition* 2000; **71**(Suppl. 5): 1344S–52S.
58 Katan MB, Grundy SM, Willett WC. Should a low-fat, high-carbohydrate diet be recommended for everyone? Beyond low-fat diets. *New England Journal of Medicine* 1997; **337**(8): 563–6 Discussion 566–7.
59 Willett WC. Dietary fat and obesity: an unconvincing relation. *American Journal of Clinical Nutrition* 1998; **68**(6): 1149–50.
60 Bray GA, Popkin BM. Dietary fat intake does affect obesity! *American Journal of Clinical Nutrition* 1998; **68**(6): 1157–73.
61 Seidell JC. Dietary fat and obesity: an epidemiologic perspective. *American Journal of Clinical Nutrition* 1998; **67**(Suppl. 3): 546S–50S.
62 Lissner L, Heitmann BL. Dietary fat and obesity: evidence from epidemiology. *European Journal of Clinical Nutrition* 1995; **49**(2): 79–90.
63 Reaven GM. Do high carbohydrate diets prevent the development or attenuate the manifestations (or both) of syndrome X? A viewpoint strongly against. *Current Opinion in Lipidology* 1997; **8**(1): 23–7.
64 Golay A, Eigenheer C, Morel Y, Kujawski P, Lehmann T, de Tonnac N. Weight-loss with low or high carbohydrate diet? *International Journal of Obesity and Related Metabolic Disorders* 1996; **20**(12): 1067–72.
65 Garg A, Bantle JP, Henry RR, Coulston AM, Griver KA, Raatz SK, *et al.* Effects of varying carbohydrate content of diet in patients with non-insulin-dependent diabetes mellitus. *Journal of the American Medical Association* 1994; **271**(18): 1421–8.
66 Duncan KH, Bacon JA, Weinsier RL. The effects of high and low energy density diets on satiety, energy intake, and eating time of obese and nonobese subjects. *American Journal of Clinical Nutrition* 1983; **37**(5): 763–7.
67 Tremblay A, Lavallee N, Almeras N, Allard L, Despres JP, Bouchard C. Nutritional determinants of the increase in energy intake associated with a high-fat diet. *American Journal of Clinical Nutrition* 1991; **53**(5): 1134–7.
68 Stubbs RJ, Ritz P, Coward WA, Prentice AM. Covert manipulation of the ratio of dietary fat to carbohydrate and energy density: effect on food intake and energy balance in free-living men eating *ad libitum*. *American Journal of Clinical Nutrition* 1995; **62**(2): 330–7.
69 Stubbs RJ, Harbron CG, Murgatroyd PR, Prentice AM. Covert manipulation of dietary fat and energy density: effect on substrate flux and food intake in men eating *ad libitum*. *American Journal of Clinical Nutrition* 1995; **62**(2): 316–29.
70 Tremblay A, Plourde G, Despres JP, Bouchard C. Impact of dietary fat content and fat oxidation on energy intake in

humans. *American Journal of Clinical Nutrition* 1989; **49**(5): 799–805.
71 Thomas CD, Peters JC, Reed GW, Abumrad NN, Sun M, Hill JO. Nutrient balance and energy expenditure during *ad libitum* feeding of high-fat and high-carbohydrate diets in humans. *American Journal of Clinical Nutrition* 1992; **55**(5): 934–42.
72 Lissner L, Levitsky DA, Strupp BJ, Kalkwarf HJ, Roe DA. Dietary fat and the regulation of energy intake in human subjects. *American Journal of Clinical Nutrition* 1987; **46**(6): 886–92.
73 Kendall A, Levitsky DA, Strupp BJ, Lissner L. Weight loss on a low-fat diet: consequence of the imprecision of the control of food intake in humans. *American Journal of Clinical Nutrition* 1991; **53**(5): 1124–9.
74 Willett WC. Dietary fat plays a major role in obesity: no. *Obesity Reviews* 2002; **3**(2): 59–68.
75 Astrup A, Grunwald GK, Melanson EL, Saris WH, Hill JO. The role of low-fat diets in body weight control: a meta-analysis of *ad libitum* dietary intervention studies. *International Journal of Obesity and Related Metabolic Disorders* 2000; **24**(12): 1545–52.
76 Swinburn BA, Metcalf PA, Ley SJ. Long term (5 year) effects of a reduced-fat diet intervention in individuals with glucose intolerance. *Diabetes Care* 2001; **24**: 619–24.
77 Weststrate JA, van het Hof KH, van den Berg H, Velthuis-te-Wierik EJ, de Graaf C, Zimmermanns NJ, *et al*. A comparison of the effect of free access to reduced fat products or their full fat equivalents on food intake, body weight, blood lipids and fat-soluble antioxidants levels and haemostasis variables. *European Journal of Clinical Nutrition* 1998; **52**(6): 389–95.
78 Ravussin E, Swinburn BA. Metabolic predictors of obesity: cross-sectional versus longitudinal data. *International Journal of Obesity and Related Metabolic Disorders* 1993; **17**(Suppl. 3): S28–S31 Discussion S41–2.
79 Ornish D, Brown SE, Scherwitz LW, Billings JH, Armstrong WT, Ports TA, *et al*. Lifestyle changes and heart disease. *Lancet* 1990; **336**(8717): 741–2.
80 Shintani TT, Hughes CK, Beckham S, O'Connor HK. Obesity and cardiovascular risk intervention through the *ad libitum* feeding of traditional Hawaiian diet. *American Journal of Clinical Nutrition* 1991; **53**: 1647s–51s.
81 Holt S, Miller JC, Petocz P, Farmakalidis E. A satiety index of common foods. *European Journal of Clinical Nutrition* 1995; **49**: 675–90.
82 Blundell JE. Food intake and body weight regulation. In: Bouchard C, Bray GA, eds. *Regulation Body Weight: Biological and Behavioural Mechanisms*. Chichester/New York/Brisbane/Toronto/Singapore: John Wiley & Sons, 1996.
83 Stubbs RJ, Harbron CG, Prentice AM. Covert manipulation of the dietary fat to carbohydrate ratio of isoenergetically dense diets: effect on food intake in feeding men *ad libitum*. *International Journal of Obesity and Related Metabolic Disorders* 1996; **20**(7): 651–60.
84 Rolls BJ, Bell EA, Castellanos VH, Chow M, Pelkman CL, Thorwart ML. Energy density but not fat content of foods affected energy intake in lean and obese women. *American Journal of Clinical Nutrition* 1999; **69**(5): 863–71.
85 Prentice AM. Manipulation of dietary fat and energy density and subsequent effects on substrate flux and food intake. *American Journal of Clinical Nutrition* 1998; **67**(3 Suppl.): 535S–41S.
86 Yao M, Roberts SB. Dietary energy density and weight regulation. *Nutrition Reviews* 2001; **59**(8 Pt 1): 247–58.
87 Seidell JC. Prevention of obesity: the role of the food industry. *Nutrition, Metabolism, and Cardiovascular Diseases* 1999; **9**(1): 45–50.
88 Rolls BJ, Bell EA, Thorwart ML. Water incorporated into a food but not served with a food decreases energy intake in lean women. *American Journal of Clinical Nutrition* 1999; **70**(4): 448–55.
89 Flatt JP. The difference in the storage capacities for carbohydrate and for fat, and its implications in the regulation of body weight. *Annals of the New York Academy of Sciences* 1987; **499**: 104–23.
90 Hellerstein MK. *De novo* lipogenesis in humans: metabolic and regulatory aspects. *European Journal of Clinical Nutrition* 1999; **53**(Suppl. 1): S53–65.
91 Horton TJ, Drougas H, Brachey A, Reed GW, Peters JC, Hill JO. Fat and carbohydrate overfeeding in humans: different effects on energy storage. *American Journal of Clinical Nutrition* 1995; **62**(1): 19–29.
92 Stubbs RJ, Murgatroyd PR, Goldberg GR, Prentice AM. Carbohydrate balance and the regulation of day-to-day food intake in humans. *American Journal of Clinical Nutrition* 1993; **57**(6): 897–903.
93 Shetty PS, Prentice AM, Goldberg GR, Murgatroyd PR, McKenna AP, Stubbs RJ, *et al*. Alterations in fuel selection and voluntary food intake in response to isoenergetic manipulation of glycogen stores in humans. *American Journal of Clinical Nutrition* 1994; **60**(4): 534–43.
94 Schwartz M, Schultz Y, Piolino V, Schneider H, Febler JP. Thermogenesis in obese women: effect of fructose vs. glucose added to a meal. *American Journal of Physiology* 1992; **262**: 394–401.
95 Heini AF, Weinsier RL. Divergent trends in obesity and fat intake patterns: the American paradox. *American Journal of Medicine* 1997; **102**(3): 259–64.
96 Harnack LJ, Jeffery RW, Boutelle KN. Temporal trends in energy intake in the United States: an ecologic perspective. *American Journal of Clinical Nutrition* 2000; **71**(6): 1478–84.
97 Howard BV, Abbott WG, Swinburn BA. Evaluation of metabolic effects of substitution of complex carbohydrates for saturated fat in individuals with obesity and NIDDM. *Diabetes Care* 1991; **14**(9): 786–95.
98 Schaeffer EJ, Lichtenstein AH, Lamon-Fava S, McNamara JR, Schaefer MM, Rasmussen HM, *et al*. Body weight and low-density lipoprotein cholesterol changes after consumption of a low-fat *ad libitum* diet. *Journal of the American Medical Association* 1995; **274**: 1450–5.
99 Leenen R, van der Kooy K, Meyboom S, Seidell JC, Deurenberg P, Weststrate JA. Relative effects of weight loss and dietary fat modification on serum lipid levels in the dietary treatment of obesity. *Journal of Lipid Research* 1993; **34**(12): 2183–91.
100 Saris WH, Astrup A, Prentice AM, Zunft HJ, Formiguera X, Verboeket-van de Venne WP, *et al*. Randomized controlled trial of changes in dietary carbohydrate/fat ratio and simple vs complex carbohydrates on body weight and blood lipids: the CARMEN study. The Carbohydrate Ratio Management in European National diets. *International Journal of Obesity and Related Metabolic Disorders* 2000; **24**(10): 1310–8.
101 Williams PT. Viewpoint. Sugar: is there a need for a dietary guideline in Australia? *Australian Journal of Nutrition and Dietetics* 2000; **58**: 26–31.
102 Gibson SA. Consumption and sources of sugar in the diets of British school children. Are high sugar diets inferior? *Journal of Human Nutrition and Dietetics* 1993; **6**: 355–71.
103 Lewis CJ, Park YK, Dexter PB, Yetley EA. Nutrient intakes and body weights of persons consuming high and moderate levels of added sugars. *Journal of the American Dietetic Association* 1992; **92**(6): 708–13.
104 'Dietary sugars and human disease': conclusions and recommendations. *British Dental Journal* 1990; **168**(2): 46.
105 Naismith DJ, Nelson M, Burley B, Gattenby S. Does a high-sugar diet promote overweight in children and lead to

nutrient deficiencies? *Journal of Human Nutrition and Dietetics* 1995; **8**: 249–54.
106 Hill JO, Prentice AM. Sugar and body weight regulation. *American Journal of Clinical Nutrition* 1995; **62**(Suppl. 1): 264S–73S Discussion 273S–74S.
107 Bellisle F, Rolland-Cachera MF. How sugar-containing drinks might increase adiposity in children. *Lancet* 2001; **357**(9255): 490–1.
108 Drewnowski A, Kurth C, Holden-Wiltse J, Saari J. Food preferences in human obesity: carbohydrates versus fats. *Appetite* 1992; **18**(3): 207–21.
109 Rolls BJ, Hetherington M. The role of variety in eating and body weight regulation. In: Sheperd R, ed. *Handbook of the Psychophysiology of Human Eating*. Sussex, England: John Wiley & Sons, 1989.
110 Anderson GH. Sugars, sweetness, and food intake. *American Journal of Clinical Nutrition* 1995; **62**(Suppl.): 195s–202s.
111 Raben A, Macdonald I, Astrup A. Replacement of dietary fat by sucrose or starch: effects on 14 d *ad libitum* energy intake, energy expenditure and body weight in formerly obese and never-obese subjects. *International Journal of Obesity and Related Metabolic Disorders* 1997; **21**(10): 846–59.
112 Mattes RD. Dietary compensation by humans for supplemental energy provided as ethanol or carbohydrate in fluids. *Physiology & Behavior* 1996; **59**(1): 179–87.
113 Tordoff MG, Alleva AM. Effect of drinking soda sweetened with aspartame or high-fructose corn syrup on food intake and body weight. *American Journal of Clinical Nutrition* 1990; **51**(6): 963–9.
114 Harnack L, Stang J, Story M. Soft drink consumption among US children and adolescents: nutritional consequences. *Journal of the American Dietetic Association* 1999; **99**(4): 436–41.
115 Ludwig DS, Peterson KE, Gortmaker SL. Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis. *Lancet* 2001; **357**(9255): 505–8.
116 Ludwig DS, Majzoub JA, Al-Zahrani A, Dallal GE, Blanco I, Roberts SB. High glycemic index foods, overeating, and obesity. *Pediatrics* 1999; **103**(3): E26.
117 Ludwig DS. Dietary glycemic index and obesity. *Journal of Nutrition* 2000; **130**(2S Suppl.): 280S–3S.
118 Brand-Miller J, Holt SHA, Pawlak DB, McMillan J. Glycemic index and obesity. *American Journal of Clinical Nutrition* 2002; **76**: 281S–5S.
119 Holt S, Brand J, Soveny C, Hansky J. Relationship of satiety to postprandial glycemic, insulin and cholecystokinin responses. *Appetite* 1992; **18**(2): 129–41.
120 Agus MS, Swain JF, Larson CL, Eckert EA, Ludwig DS. Dietary composition and physiologic adaptations to energy restriction. *American Journal of Clinical Nutrition* 2000; **71**: 901–7.
121 Slabber M, Barnard HC, Kuyl JM, Dannhauser A, Schall R. Effects of a low-insulin-response, energy-restricted diet on weight loss and plasma insulin concentrations in hyperinsulinemic obese females. *American Journal of Clinical Nutrition* 1994; **60**(1): 48–53.
122 Spieth LE, Harnish JD, Lenders CM, Raezer LB, Pereira MA, Hangen SJ, *et al*. A low-glycemic index diet in the treatment of pediatric obesity. *Archives of Pediatrics & Adolescent Medicine* 2000; **154**(9): 947–51.
123 Roberts SB. High-glycemic index foods, hunger, and obesity: is there a connection? *Nutrition Reviews* 2000; **58**(6): 163–9.
124 Pereira MA, Ludwig DS. Dietary fiber and body-weight regulation. Observations and mechanisms. *Pediatric Clinics of North America* 2001; **48**(4): 969–80.
125 Howarth NC, Saltzman E, Roberts SB. Dietary fiber and weight regulation. *Nutrition Reviews* 2001; **59**(5): 129–39.
126 Burton-Freeman B. Dietary fiber and energy regulation. *Journal of Nutrition* 2000; **130**(2S Suppl.): 272S–5S.
127 Gurr MI. Diet, nutrition and the prevention of chronic diseases (WHO, 1990). *European Journal of Clinical Nutrition* 1991; **45**(12): 619–23.
128 Latner JD, Schwartz M. The effects of a high-carbohydrate, high-protein or balanced lunch upon later food intake and hunger ratings. *Appetite* 1999; **33**(1): 119–28.
129 Long SJ, Jeffcoat AR, Millward DJ. Effect of habitual dietary-protein intake on appetite and satiety. *Appetite* 2000; **35**(1): 79–88.
130 Astrup A, Raben A. Carbohydrate and obesity. *International Journal of Obesity and Related Metabolic Disorders* 1995; **19**(Suppl. 5): S27–37.
131 Prentice AM. Alcohol and obesity. *International Journal of Obesity and Related Metabolic Disorders* 1995; **19**(Suppl 5): S44–50.
132 Sonko BJ, Prentice AM, Murgatroyd PR, Goldberg GR, van de Ven ML, Coward WA. Effect of alcohol on postmeal fat storage. *American Journal of Clinical Nutrition* 1994; **59**(3): 619–25.
133 Lands WE. Alcohol and energy intake. *American Journal of Clinical Nutrition* 1995; **62**(5 Suppl.): 1101S–6S.
134 Rose D, Murphy SP, Hudes M, Viteri FE. Food energy remains constant with increasing alcohol intake. *Journal of the American Dietetic Association* 1995; **95**(6): 698–700.
135 Suter PM, Hasler E, Vetter W. Effects of alcohol on energy metabolism and body weight regulation: is alcohol a risk factor for obesity? *Nutrition Reviews* 1997; **55**(5): 157–71.
136 Hellerstedt WL, Jeffery RW, Murray DM. The association between alcohol intake and adiposity in the general population. *American Journal of Epidemiology* 1990; **132**(4): 594–611.
137 Emery EM, Schmid TL, Kahn HS, Filozof PP. A review of the association between abdominal fat distribution, health outcome measures, and modifiable risk factors. *American Journal of Health Promotion* 1993; **7**(5): 342–53.
138 Wansink B. Can package size accelerate usage volume? *Journal of Marketing* 1996; **60**(3): 1–22.
139 Rolls BJ, Engell D, Birch LL. Serving portion size influences 5-year-old but not 3-year-old children's food intakes. *Journal of the American Dietetic Association* 2000; **100**(2): 232–4.
140 Swinburn B, Egger G, Raza F. Dissecting obesogenic environments: the development and application of a framework for identifying and prioritizing environmental interventions for obesity. *Preventive Medicine* 1999; **29**(6 Pt 1): 563–70.
141 Stunkard AJ. Socioeconomic status and obesity. In: Cadwick DJ, Cardew G, eds. *The Origins and Consequences of Obesity*. Chichester: Wiley, 1996, 174–93.
142 Monteiro CA, Conde WL, Popkin BM. Independent effects of income and education on the risk of obesity in the Brazilian adult population. *Journal of Nutrition* 2001; **131**(3): 881S–6S.
143 Subramania SV, Kawachi I, Kennedy BP. Does the state you live in make a difference? Multilevel analysis of self-rated health in the US. *Social Science & Medicine* 2001; **53**(1): 9–19.
144 Reidpath DD, Burns C, Garrard J, Mahoney M, Townsend M. An ecological study of the relationship between socioeconomic status and obesogenic environments. *Health and Place* 2002; **8**(2): 141–5.
145 Hider P. Environmental interventions to reduce energy intake or density. A critical appraisal of the literature. *New Zealand Health Technology Assessment Report* 2001; **4**(2).
146 Gortmaker SL, Peterson K, Wiecha J, Sobol AM, Dixit S, Fox MK, *et al*. Reducing obesity via a school-based

interdisciplinary intervention among youth: Planet Health. *Archives of Pediatrics & Adolescent Medicine* 1999; **153**(4): 409–18.
147 Campbell K, Crawford D. Family food environments as determinants of preschool-aged children's eating behaviours: implications for obesity prevention policy: a review. *Australian Journal of Nutrition and Dietetics* 2001; **58**(1): 19–25.
148 Hearn MD, Baranowski T, Baranowski J. Environmental influences on dietary behaviour among children: availability and accessibility of fruits and vegetables enable consumption. *Journal of Health Education* 1998; **29**(1): 26–32.
149 Sullivan SA, Birch L. Pass the sugar, pass the salt: experience dictates preference. *Developmental Psychology* 1990; **26**(546): 551.
150 Sullivan SA, Birch LL. Infant dietary experience and acceptance of solid foods. *Pediatrics* 1994; **93**(2): 271–7.
151 Gibson EL, Wardle J, Watts CJ. Fruit and vegetable consumption, nutritional knowledge and beliefs in mothers and children. *Appetite* 1998; **31**(2): 205–28.
152 Borah-Giddens J, Falciglia GA. A meta-analysis of the relationship in food preferences between parents and children. *Journal of Nutrition Education* 1993; **25**: 102–7.
153 Skinner J, Carruth BR, Moran J, Houck K, Schmidhammer J, Reed A, *et al*. Toddler's food preferences: concordance with family member's preferences. *Journal of Nutrition Education* 1998; **30**: 17–22.
154 Endicott RC. Ad Age Datacenter–46th Annual Report: 10 Leading National Advertisers. Advertising Age. http://www.adage.com (verified September 2001).
155 Hill JM, Radimer KL. A content analysis of food advertisements in television for Australian children. *Australian Journal of Nutrition and Dietetics* 1997; **54**(4): 174–81.
156 Nestle M, Wing R, Birch L, DiSogra L, Drewnowski A, Middleton S, *et al*. Behavioral and social influences on food choice. *Nutrition Reviews* 1998; **56**(5 Pt 2): S50–S64 Discussion S64–74.
157 Hammond KM, Wyllie A, Casswell S. The extent and nature of televised food advertising to New Zealand children and adolescents. *Australian and New Zealand Journal of Public Health* 1999; **23**(1): 49–55.
158 Feldwick P. Food advertising and the nation's diet: what are the real issues? *Business Strategy Reviews* 1995; **6**(1): 61–9.
159 Lewis MK, Hill AJ. Food advertising on British children's television: a content analysis and experimental study with nine-year olds. *International Journal of Obesity and Related Metabolic Disorders* 1998; **22**(3): 206–14.
160 Taras HL, Sallis JF, Patterson TL, Nader PR, Nelson JA. Television's influence on children's diet and physical activity. *Journal of Developmental and Behavioral Pediatrics* 1989; **10**(4): 176–80.
161 Children, adolescents, and advertising. Committee on Communications, American Academy of Pediatrics. *Pediatrics* 1995; **95**(2): 295–7.
162 Colby JJ, Elder JP, Peterson G, Knisley PM, Carleton RA. Promoting the selection of healthy food through menu item description in a family-style restaurant. *American Journal of Preventive Medicine* 1987; **3**(3): 171–7.
163 Chu C, Driscoll T, Dwyer S. The health-promoting workplace: an integrative perspective. *Australian and New Zealand Journal of Public Health* 1997; **21**(4): 377–85.
164 Schmitz M, Fielding J. Point-of-choice nutritional labeling—evaluation in a worksite cafeteria. *Journal of Nutrition Education* 1986; **18**(1 Suppl.): 65s–8s.
165 Winett RA, Wagner JL, Moore JF, Walker WB, Hite LA, Leahy M, *et al*. An experimental evaluation of a prototype public access nutrition information system for supermarkets. *Health Psychology* 1991; **10**(1): 75–8.
166 Guo X, Popkin BM, Mroz TA, Zhai F. Food price policy can favorably alter macronutrient intake in China. *Journal of Nutrition* 1999; **129**(5): 994–1001.
167 Marshall T. Exploring a fiscal food policy: the case of diet and ischaemic heart disease. *British Medical Journal* 2000; **320**(7230): 301–5.
168 O'Rourke A. VAT and fat. Will sales tax influence consumption? *British Medical Journal* 2000; **320**(7247): 1469 Discussion 1470.
169 Jacobson MF, Brownell KD. Small taxes on soft drinks and snack foods to promote health. *American Journal of Public Health* 2000; **90**(6): 854–7.
170 Neuhouser ML, Kristal AR, Patterson RE. Use of food nutrition labels is associated with lower fat intake. *Journal of the American Dietetic Association* 1999; **99**(1): 45–53.
171 Weimer J. Accelerating the trend toward healthy eating. *America's Eating Habits: Changes and Consequences*. Washington, DC: USDA/Econ. Res. Serv., 1999, 385–401.
172 Smith CS, Taylor JG, Stephen AM. Use of food labels and beliefs about diet–disease relationships among university students. *Public Health Nutrition* 1999; **3**(2): 175–82.
173 Wang G, Fletcher SM, Carley DH. Consumer utilization of food labeling as a source of nutrition information. *Journal of Consumer Affairs* 1995; **29**(2): 368.
174 Zarkin GA, Dean N, Mauskopf JA, Williams R. Potential health benefits of nutrition label changes. *American Journal of Public Health* 1993; **83**(5): 717–24.
175 Kreuter MW, Brennan LK, Scharff DP, Lukwago SN. Do nutrition label readers eat healthier diets? Behavioral correlates of adults' use of food labels. *American Journal of Preventive Medicine* 1997; **13**(4): 277–83.
176 Noakes M, Crawford D. The National Heart Foundation's "Pick the Tick" program. Consumer awareness, attitudes and interpretation. *Food Australia* 1991; **43**(6): 266–62.
177 Young LR, Swinburn B. The impact of the Pick the Tick food information programme on the salt content of food in New Zealand. *Health Promotion International* 2002; **17**(1): 13–9.
178 Miller DL, Castellanos VH, Shide DJ, Peters JC, Rolls BJ. Effect of fat-free potato chips with and without nutrition labels on fat and energy intakes. *American Journal of Clinical Nutrition* 1998; **68**(2): 282–90.
179 Sustain: The Alliance for Better Food and Farming. *TV Dinners – What's being served up by the advertisers?* 2001. London. ISBN 1903060168.
180 O'Brien D. *The gathering storm: Marketing food to children*. American Association of Advertising Agencies. Bulletin 6228, 3 March 2003.
181 *Television food advertising during children's viewing times*. Policy statement of the Public Health Association of Australia. http://www.phaa.net.av/policy (verified October 2002).
182 Pietinen P, Nissinen A, Vartiainen E, Tuomilehto A, Uusitalo U, Ketola A, *et al*. Dietary changes in the North Karelia Project (1972–1982). *Preventive Medicine* 1988; **17**(2): 183–93.
183 Luepker RV, Murray DM, Jacobs DR Jr., Mittelmark MB, Bracht N, Carlaw R, *et al*. Community education for cardiovascular disease prevention: risk factor changes in the Minnesota Heart Health Program. *American Journal of Public Health* 1994; **84**(9): 1383–93.
184 Resnicow K, Robinson TN. School-based cardiovascular disease prevention studies: review and synthesis. *Annals of Epidemiology* 1997; S**7**: S14–S31.
185 Luepker RV, Perry CL, McKinlay SM, Nader PR, Parcel GS, Stone EJ, *et al*. Outcomes of a field trial to improve children's dietary patterns and physical activity: The child and adolescent trial for cardiovascular health. CATCH collaborative group. *Journal of the American Medical Association* 1996; **275**(10): 768–76.

186 Sahota P, Rudolf MC, Dixey R, Hill AJ, Barth JH, Cade J. Evaluation of implementation and effect of primary school based intervention to reduce risk factors for obesity. *British Medical Journal* 2001; **323**(7320): 1027.
187 Sahota P, Rudolf MC, Dixey R, Hill AJ, Barth JH, Cade J. Randomised controlled trial of primary school based intervention to reduce risk factors for obesity. *British Medical Journal* 2001; **323**(7320): 1029–32.
188 Carter M, Swinburn B. Measuring the impact of a school food programme on food sales in New Zealand. *Health Promotion International* 1999; **14**(4): 307–16.
189 Robinson TN. Reducing children's television viewing to prevent obesity: a randomized controlled trial. *Journal of the American Medical Association* 1999; **282**(16): 1561–7.
190 Nicholson C, Shrapnel B, Bauman A, Booth M, Burt S. A controlled trial evaluation of a worksite nutrition education program. *Australian Journal of Nutrition and Dietetics* 1993; **50**(1): 4–8.
191 Fairbank L, O'Meara S, Renfrew MJ, Woolridge M, Sowden AJ, Lister-Sharp D. A systematic review to evaluate the effectiveness of interventions to promote the initiation of breastfeeding. *Health Technology Assessment* 2000; **4**(25): 1–171.
192 Gilmore M, O'Driscoll D, Murphy H. A pilot survey of an attempt to promote breastfeeding. *Irish Journal of Medical Sciences* 1979; **148**(9–10): 272–5.
193 Kistin N, Benton D, Rao S, Sullivan M. Breast-feeding rates among black urban low-income women: effect of prenatal education. *Pediatrics* 1990; **86**(5): 741–6.
194 Rossiter JC. The effect of a culture-specific education program to promote breastfeeding among Vietnamese women in Sydney. *International Journal of Nursing Studies* 1994; **31**(4): 369–79.
195 van Wechem SN, Brug J, van Assema P, Kistemaker C, Riedstra M, Lowik MR. Fat Watch: a nationwide campaign in The Netherlands to reduce fat intake—effect evaluation. *Nutrition and Health* 1998; **12**(2): 119–30.
196 Reger B, Wootan MG, Booth-Butterfield S. Using mass media to promote healthy eating: a community-based demonstration project. *Preventive Medicine* 1999; **29**(5): 414–21.
197 Turrell G. Compliance with the Australian Dietary Guidelines in the early 1990's: have population-based health promotion programs been effective? *Nutrition and Health* 1997; **11**(4): 271–88.
198 Geiger CJ. Communicating dietary guidelines for Americans: room for improvement. *Journal of the American Dietetic Association* 2001; **101**(7): 793–7.
199 *Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults*. National Heart, Lung, and Blood Institute, National Institutes of Health Publication 98–4083. 1998.
200 Sjostrom L, Narbro K, Sjostrom D. Costs and benefits when treating obesity. *International Journal of Obesity and Related Metabolic Disorders* 1995; **19**(Suppl. 6): S9–12.
201 Galuska DA, Will JC, Serdula MK, Ford ES. Are health care professionals advising obese patients to lose weight? *Journal of the American Medical Association* 1999; **282**(16): 1576–8.
202 *Preparation and Use of Food-based Dietary Guidelines. Report of a Joint FAO/WHO Consultation*. WHO Technical Report Series No. 880. Geneva, Switzerland: World Health Organisation, 1996.