Proceedings of the Nutrition Society (2005), **64**, 217–227 DOI:10.1079/PNS2005429

The Summer Meeting of the Nutrition Society jointly with the Association for the Study of Obesity was held at Trinity College, Dublin, Republic of Ireland on 5–8 July 2004

Plenary Lecture

Dietary strategies for the prevention of obesity

Susan A. Jebb
MRC Human Nutrition Research, Elsie Widdowson Laboratory, Fulbourn Road, Cambridge CB1 9NL, UK

The rising tide of obesity has led to a resurgence of interest in dietary strategies to prevent excess weight gain. Data from controlled intervention studies is sparse, but nonetheless evidence from other diverse sources has identified a number of specific dietary factors and aspects of eating behaviour that either promote or protect against obesity. The present paper reviews the evidence in relation to energy density, the macronutrient composition of the diet, including the nature of carbohydrate foods and sugar-rich drinks, portion size and snacking habits. It concludes that there is now sufficient evidence to develop clear dietary guidelines to prevent weight gain that are largely consistent with those for the prevention of CVD and cancer. However, coordinated action across multiple stakeholders is also required if these guidelines are to be translated into sustained changes in eating habits.

Obesity: Diet: Energy density: Soft drinks: Portion size

Obesity is in the spotlight as never before. The prevalence of obesity has trebled in the last 20 years and the burden of obesity-related morbidity is increasing (National Audit Office, 2001). One of the few incontrovertible facts is that obesity is the result of long-term energy imbalance. Analysis of the trends in relation to obesity suggests that in economically-developed countries the majority of the population are less active than they should be for good health, while simultaneously eating more than they need. The need to develop preventative action, especially focused on children, is now well accepted, but at present very few countries have developed integrated national strategies for the prevention of obesity. In general, there is widespread acceptance of the need to encourage the population to become more physically active. However, the precise dietary factors contributing to excess energy intake and/or those most amenable to modification to reduce the risk of obesity remain a subject of lively discussion and debate. This situation, in part, reflects the complexity of dietary habits, the difficulties of conducting highly-controlled dietary intervention studies and the lag time between exposure and the development of overt obesity. Nonetheless, taking a holistic view of the scientific literature and adopting a precautionary approach to public health gives clear pointers towards appropriate dietary strategies for the prevention of obesity. The present paper will review the evidence in relation to a number of specific dietary components and food habits as part of an overall strategy for the prevention of obesity.

An evolutionary perspective

Many animals can precisely regulate their body weight to maintain a healthy body size and shape over many years. However, spontaneous obesity develops in rodents and primates in an environment that facilitates positive energy balance (Hansen, 2001). This outcome may be a consequence of imposed sedentariness, e.g. animals kept in captivity, or offered *ad libitum* access to very-energy-dense foods, often described as 'cafeteria-feeding' (Rothwell & Stock, 1979). In different strains of rat voluntary energy intake may increase by 30–60% and, while diet-induced thermogenesis also increases, it is not of sufficient magnitude to prevent weight gain (Rothwell *et al*. 1982).

Abbreviation: GI, glycaemic index.
Corresponding author: Dr Susan Jebb, fax +44 1223 437515, email susan.jebb@mrc-hnr.cam.ac.uk

Man, too, has an innate homeostatic regulation system. Over many years scientists have identified numerous components of the appetite regulation system, including signals from the gastrointestinal tract, circulating metabolites, neural pathways and longer-term regulatory systems based on feedback loops from adipose tissue (Schwartz *et al*. 2000). However, a key feature of this appetite control system appears to be an asymmetry in the control of hunger and satiety (Prentice & Jebb, 2004).

Man is very responsive to the sensation of hunger and readily overeats, but in contrast the satiety signals are relatively weak and easily over ridden. It has been argued that this characteristic is a relic of evolutionary history, during which repeated bouts of energy deprivation have left their mark on human metabolism and modern man is poorly equipped to handle the 21st century in which a wide range of highly-palatable and affordable food is available on a permanent basis (Prentice, 1997). However, few commentators believe that it is possible to turn the clock back. Instead, there is a need to identify specific dietary factors that may undermine appetite control. These factors form the scientific basis from which to develop policy options to tackle obesity at a population level and to direct the development of practical coping strategies to enable individuals to modify their microenvironment and/or their individual lifestyle choices.

Dietary surveys provide a snapshot of eating habits and over time can be used to examine trends in food habits and eating behaviour. Unfortunately, it has proved remarkably difficult to match this information to changes in the prevalence of obesity. This situation results from the complexity of dietary habits, changes over time, the lag phase in the development of obesity and the importance of physical activity. In addition, there is the fundamental problem of under-reporting of food intake. Since under-reporting was first described in the 1980s it has been recognised to be endemic in most dietary surveys (Black *et al*. 1991). Under-reporting is an individual characteristic that is relatively reproducible and independent of the method of dietary assessment (Livingstone & Black, 2003). It combines aspects of under-eating, perhaps because of illness or intentional dieting, modifications in behaviour as a consequence of the observer and finally mis-reporting of the food actually eaten, either in terms of the quality or quantity of items consumed. These factors all tend towards non-random bias and it is apparent that measured energy intakes in most dietary surveys are physiologically implausible and cannot reflect habitual consumption.

The extent of under-reporting is frequently assessed using the 'Goldberg cut-off' (Goldberg *et al*. 1991). However, this cut-off represents a very conservative estimate and although it has high sensitivity, it has low specificity. The nature of the cut-off means that only those individuals who have a relatively low energy expenditure are accurately classified as under-reporters, leaving large proportions of under-reporters with higher energy needs who are not identified. This problem may be overcome to some extent by the use of individualised estimates of energy needs, although the validity of this approach hinges on the accuracy of the assessment of energy expenditure,

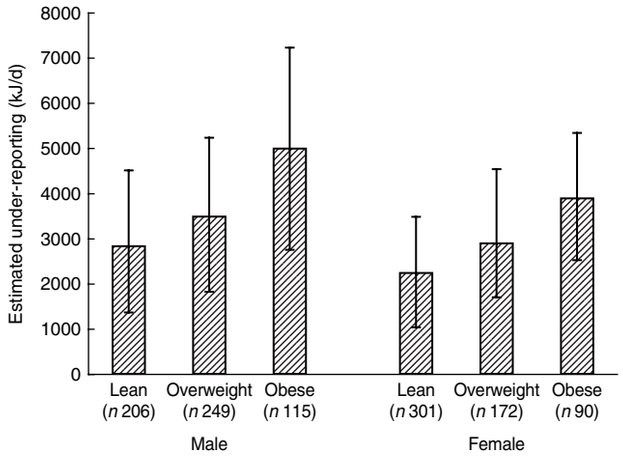

Fig. 1. Estimated under-reporting (based on individual estimates of energy expenditure) by weight status in the British National Diet and Nutrition Survey 16–64 years (Rennie *et al*. 2004). Values are means with the median and interquartile range represented by vertical bars.

especially physical activity. Using this individual approach an analysis of the British National Diet and Nutrition Survey suggests that measured energy intakes are on average 25% below that expected (Rennie *et al*. 2004, 2005). More importantly, under-reporting is not evenly distributed across the population, being more common in individuals who are overweight or obese (Fig. 1). However, some obese individuals report accurately while some lean individuals under-report their intakes. It is extraordinarily difficult to model the precise characteristics of under-reporters and to produce any appropriate adjustments in dietary surveys. This situation confounds the possibility of testing diet–disease hypotheses, either by obscuring true relationships or introducing artefactual associations.

Thus, epidemiological associations alone provide insufficient foundation for nutrition policy. Recent initiatives, notably the World Health Organization/Food and Agriculture Organization (2003) report on the prevention of chronic diseases, have pulled together evidence from epidemiology, experimental and intervention studies to develop a dietary strategy for the prevention of obesity (Table 1).

Dietary fat and energy density

The economic transition is often characterised by an increase in the proportion of fat in the diet, and in ecological analyses there is a positive association between the proportion of energy from fat and the prevalence of overweight and obesity (Bray & Popkin, 1998). Within populations, the relationship between dietary fat and obesity is much less consistent (Willett, 1998). However, experimental studies have demonstrated a clear and reproducible mechanism by which fat may be associated with an increased risk of obesity (Poppitt & Prentice, 1996). Highly-controlled studies in which energy intake has been reliably measured by direct observation within

https://doi.org/10.1079/PNS2005429 Published online by Cambridge University Press

Table 1. Dietary factors that may affect the risk of obesity (World Health Organization/Food and Agriculture Organization, 2003)

Evidence	Decreases risk	No relationship	Increases risk
Convincing	Regular physical activity High dietary NSP or fibre intake		Sedentary lifestyles High intake of energy-dense foods
Probable	Home and school environments that support healthy food choices for children Breast-feeding		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-glycemic-index foods	Protein content of the diet	Large portion sizes High proportion of food prepared outside the home (Western countries) 'Rigid restraint and/or periodic disinhibition' eating patterns
Insufficient	Increased eating frequency		Alcohol

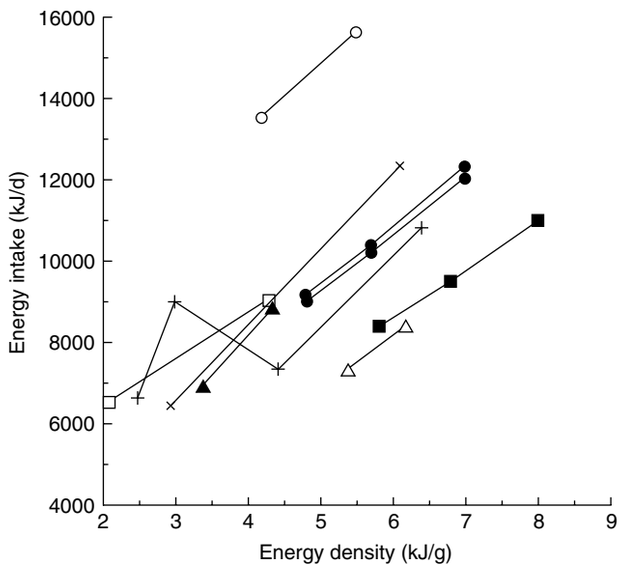

Fig. 2. Relationship between energy density of diets and voluntary energy intake from nine individual studies. (From Poppitt & Prentice, 1996; reprinted with permission.)

whole-body calorimeters have established that increases in the proportion of fat in the diet are associated with increased energy intake, a phenomenon described as high-fat hyperphagia (Stubbs *et al*. 1995). Over time this phenomenon is associated with positive fat balance. Conversely, low-fat diets, even when consumed *ad libitum*, are associated with negative fat balance, at least in the short term.

Yet, in the context of larger community-based intervention studies the relationship between fat, energy intake and body weight is much less clear. A meta-analysis of a number of intervention studies involving *ad libitum* low-fat diets has shown a small, yet significant ($P<0{\cdot}0001$), decrease in body weight in the first 3–6 months, but this effect may not be sustained in the longer term (Astrup *et al*. 2000). In the free-living situation the impact of low-fat diets appears much smaller than found in controlled experimental studies because first, compliance to the dietary prescription may wane in these longer-term interventions and second, it is recognised that choosing low-fat foods alone is not sufficient to prevent over-consumption of other nutrients, especially carbohydrate. Indeed, many low-fat products on the market today have an energy content that is comparable to the high-fat equivalent.

More detailed consideration of high-fat hyperphagia suggests that energy density, rather than fat, may be the primary driver of over-consumption. In studies in which dietary fat was manipulated while maintaining constant energy density the high-fat hyperphagia can be abolished (Stubbs *et al*. 1996). Fig. 2 shows that foods with a high energy density are associated with passive over-consumption, a process whereby the volume of food consumed is not appropriately down regulated to match the increased energy density of the food. The importance of energy density as a determinant of food intake is gradually becoming recognised in relation to nutrition policy. In the UK schemes to label foods on the basis of energy-density criteria are under consideration.

In practical terms foods with a high energy density tend to be high in fat, high in added sugars, low in fruit and vegetables and have a low water content. These characteristics exemplify many of the items offered in fast-food outlets. An analysis of the energy density of fast food across the entire menu range, as available in summer 2002, demonstrates that the energy density is 60% higher than that observed across the British diet as whole (Prentice & Jebb, 2003). The links between fast food and obesity provide an interesting example of the way in which data from diverse sources can be drawn together to build the evidence base for nutrition policy. These sources include secular trends in the growth of fast foods and obesity, ecological studies showing disproportionate numbers of fast-food outlets in areas characterised by low socio-economic status (Reidpath *et al*. 2002) and an experimental study showing that in obese adolescents energy intake is higher on those days on which fast food is consumed than on non-fast-food days, although there is no difference in lean individuals (Ebbeling *et al*. 2004).

https://doi.org/10.1079/PNS2005429 Published online by Cambridge University Press

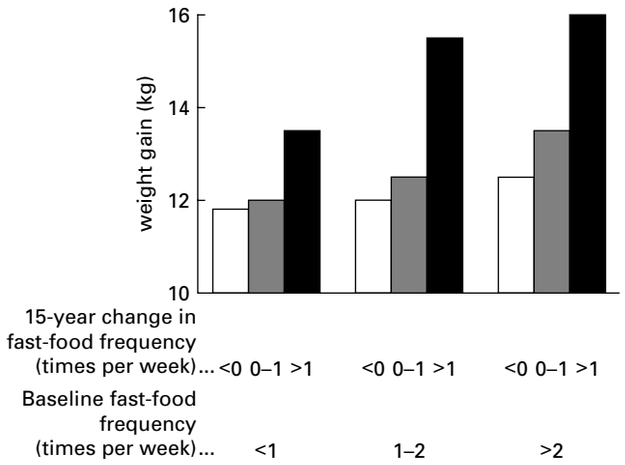

Fig. 3. Association between frequency of fast-food consumption at baseline, 15-year change in fast-food frequency (times per week; <0 (i.e. decrease in fast-food frequency), □; 0–1, ▩; >1, ■) and 15-year change in body weight in 3031 adults aged 18–30 years. (Data from Pereira *et al.* 2005.)

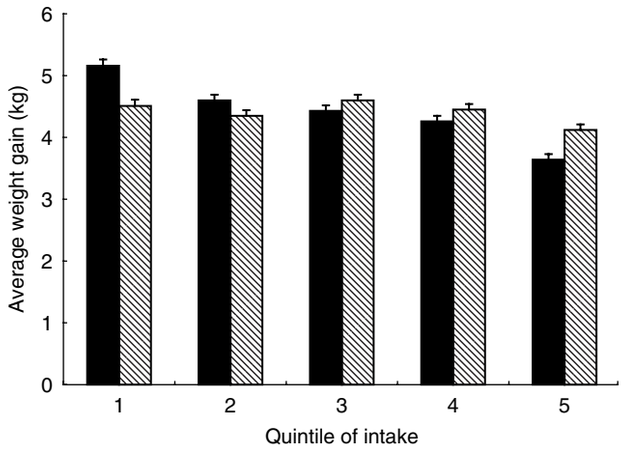

Fig. 4. Type of carbohydrate consumed and weight gain (adjusted for age, changes in exercise, snacking, hormone-replacement therapy and other variables) in the Nurses' Health Study during a 12-year follow-up. Values are means with their standard errors represented by vertical bars. ■, fibre ($P<0{\cdot}0001$ for trend); ▨, wholegrain ($P < 0{\cdot}0001$) for trend.

Finally, a prospective analysis has shown that the frequency of fast-food consumption at baseline and the increases over time are associated with increases in body weight over a 15-year period (Pereira *et al.* 2005; Fig. 3).

To date there has been little work on the use of the low-energy-density message in the treatment of established obesity (Ello-Martin, 2004), but the present overview of the evidence provides a rational basis to recommend low-energy-dense diets for the prevention of weight gain.

Dietary carbohydrates

Throughout the 1990s there was an emphasis on the role of dietary fat and energy density in the aetiology of obesity. Diets high in fat usually contain a relatively low proportion of carbohydrate and, perhaps as a consequence, carbohydrate has received relatively little attention. However, in recent years there has been growing interest in the nature of the carbohydrate source. Research in this area is complicated by the diversity of classification systems for dietary carbohydrates, which may be identified on the basis of their fibre content, proportion of wholegrain, non-milk extrinsic sugars or added sugar content. While it is clear that there are important associations between the type of carbohydrate consumed and the risk of metabolic diseases, there has been less work that has examined the relationship with body weight. However, recent epidemiological analyses have suggested that those individuals who consume the most fibre (Liu *et al.* 2003) or wholegrain foods (Koh-Banerjee *et al.* 2004) show an attenuated pattern of weight gain over time (Fig. 4). Intervention studies to increase fibre intake tend towards demonstrating modest weight loss, at least in the short term; however when the dietary change is achieved through food rather than supplements the data is not wholly consistent (Howarth *et al.* 2001). To date there have been no large-scale intervention studies to examine the impact of increasing wholegrain food consumption on body weight when delivered as a single intervention.

The concept of glycaemic index (GI) and/or glycaemic load is also receiving increasing attention, since it potentially offers a classification system based on a physiological function (postprandial glycaemia). There are a plethora of studies that have considered the impact of GI on health. Most of the studies have been conducted in patients with type 2 diabetes or impaired glucose intolerance, for whom there is good evidence of metabolic benefits (Anderson *et al.* 2004). However, the impact on body weight is much less clear. Most studies of the impact of GI on weight are small, short term and often poorly controlled. A systematic review of the most rigorously controlled trials has concluded that there is no evidence of beneficial effects on weight loss (Raben, 2002). However, more naturalistic studies, based on dietary advice to consume a low-GI diet, are more positive (Bouche *et al.* 2002), although the reduction in body weight may be attributable to broader changes in dietary habits than the change in specific GI. Most low-GI diets include increases in the proportion of fruit and vegetables in the diet, with an emphasis on wholegrain and unrefined foods, and discourage consumption of refined carbohydrate in the form of cakes, biscuits, confectionery and soft drinks. Together this approach results in a diet with a lower energy density, and this factor may in itself drive weight loss or help to prevent weight gain.

Research is underway to clarify the mechanistic basis of the putative effects of GI on appetite control. It is hypothesised that low-GI diets may curb hunger and increase satiety as a result of delays in gastric emptying and relatively slow absorption of glucose into the bloodstream, avoiding the peaks and troughs in blood glucose levels (and frequently insulin levels) characteristic of the consumption of high-GI foods (Brand-Miller *et al.* 2002). Recent animal data show marked changes in nutrient

https://doi.org/10.1079/PNS2005429 Published online by Cambridge University Press

partitioning and body-weight gain with low-GI diets, and suggest that the positive effects may be mediated through the suppression of counter-regulatory hormones secreted in response to periods of rebound hypoglycaemia following high-GI meals (Pawlak *et al*. 2004). To date there is good evidence for improvements in metabolic risk and little reason to imagine low-GI diets have detrimental effects on body weight. However, hard evidence for a positive benefit in weight control, attributable specifically to GI, is still lacking. The challenge for policy makers is to understand when it is appropriate to incorporate the emerging science into policy, whether reductions in the GI can be achieved at a population level and, if appropriate, how to communicate this complex concept to consumers.

In contrast, the public health recommendations to increase fruit and vegetable consumption are a core component of most nutrition strategies, reflecting the known association with reduced risks of heart disease and some cancers. There is an outstanding question of whether this option also aids weight control. The high water content, high fibre and low energy density of fruits and vegetables would all tend to be associated with increased satiety and hence a decreased risk of obesity. In a 12-year follow-up of the Nurses Health Study (He *et al*. 2004) women in the highest quintile for increased consumption of fruit and vegetables were found to have a 24% lower risk of becoming obese than those with the greatest decrease in fruit and vegetable consumption. Experimental studies involving the addition of fruits and vegetables to the diet tend to show short-term reductions in energy intake, but when it is the only intervention the reduction is rarely sustained in the longer term (Rolls *et al*. 2004). More complex dietary interventions in which increases in fruit and vegetables are promoted alongside reductions in dietary fat have been more successful. For example, in 1000 patients at high risk of coronary artery disease a 3 kg greater reduction in body weight has been found in those patients following an Indo-Mediterranean diet (rich in wholegrains, fruits, vegetables and nuts) compared with those on a low-fat Step 1 US National Cholesterol Education Program diet over a 2-year period (Singh *et al*. 2002). However, it is difficult to dissect the importance of the increase in fruit and vegetable consumption from the broader changes in dietary habits.

Unlike the preceding research that has sought to identify sources of carbohydrate that offer some protection against obesity, there is growing interest in the possibility that sugar may be a specific risk factor for weight gain. Studies of total sugar intake have generally failed to show positive associations with disease outcomes and the evidence in relation to body weight favours a protective effect, probably as a result of the reciprocal relationship with dietary fat (Hill & Prentice, 1995). However, research is now turning to the source of sugar in the diet rather than total intake, with an increasing focus on the detrimental effects of sugar-rich soft drinks.

Sugar-rich soft drinks (excluding fruit juice) represent the largest contributor to sugar intakes in young individuals, accounting for 25% of the non-milk extrinsic sugars intake (Gregory & Low, 2000), and a substantial percentage of non-milk extrinsic sugars in adults (16; Henderson *et al*. 2002). Fruit juices have a similar sugar content to those of many soda-type drinks but in practice represent a small proportion of total sugar intake from beverages. However, it is notable that one of the possible reasons why studies of fruits and vegetables alone tend not to be effective may be because the increase in fruit intake is frequently achieved through the use of fruit juices rather than whole fruits. When consumed as a beverage these foods may not yield the same satiating properties as the intact food item (Rolls *et al*. 2004).

Although there is a lack of direct evidence linking sugar-rich beverages to obesity, there is a growing body of indirect evidence that now points strongly towards this source as a target for strategies to improve public health. Ecological analyses have linked the increasing consumption of high-fructose maize syrup, predominantly in soft drinks, to the increased prevalence of obesity in the USA (Bray *et al*. 2004). A longitudinal study in 12-year-old children has suggested that each serving of soft drinks increases the risk of becoming obese by 1·6-fold over a 19-month period (Ludwig *et al*. 2001), and in adults weight gain has been found to be greatest in women who increase their consumption of sugar-sweetened soft drinks from one serving or less per week to one serving or more per d over a 4-year period and smallest in those who decrease their intake (Schulze *et al*. 2004). These studies are limited by the self-reported nature of the consumption data. Stronger evidence has been provided from direct observation at a summer camp in the USA, which has shown that children who consume the largest quantities of sweetened beverages have a markedly higher energy intake than those who do not (Mrdjenovic & Levitsky, 2003). It was also found that there is a trend towards an association with increases in body weight, suggesting that the energy is not utilised in physical activity, although over the 4–8-week study period the change in body weight was not significant.

The link between sugar-rich beverages and positive energy balance may be the result of the low satiating properties of beverages. There is an inverse association between the viscosity of foods (Mattes & Rothacker, 2001) and subsequent hunger. Studies using a preload paradigm have demonstrated much poorer energy compensation for energy consumed as beverages compared with foods (DiMeglio & Mattes, 2000; Fig. 5). Short-term experimental studies in which subjects were given a sugar-rich drink, an artificially-sweetened variety or water have shown that energy intake at a subsequent meal is not significantly different for any of the treatment conditions, and hence net energy intake is greater in those subjects who had the sugar-rich drink (Tordoff & Alleva, 1990).

There are two longer-term studies that have examined the integrated effect on body weight. In the first study, a 10-week intervention study, overweight subjects were randomised to receive foods containing sugar or artificially-sweetened alternatives. Of the intervention foods >80% were consumed as beverages. Marked differences in body weight between the two groups were found at 4 weeks, which continued to increase over 10 weeks, resulting in a 2·6 kg difference between groups (Raben *et al*. 2002). In the second study an increased prevalence of obesity was reported in schools over a 1-year period,

https://doi.org/10.1079/PNS2005429 Published online by Cambridge University Press

Fig. 5. Effect of solid (jelly beans) *v.* liquid (soda) carbohydrate on *ad libitum* energy intake over 4 weeks. (⊠), Baseline; (■) after 4 weeks. Values are means with their standard errors represented by vertical bars. (From DiMeglio & Mattes, 2000; reprinted with permission from Macmillan Publishers Ltd.)

which was found to be attenuated by the provision of advice to decrease soft drink consumption (James *et al*. 2004), but the design and statistical analysis of this study has been criticised (French *et al*. 2004) and further work in this area is required. However, the accumulating evidence has stimulated important discussions at a policy level, not least since soft drink consumption is potentially amenable to intervention by substitution with artificially-sweetened varieties or water.

Protein

Protein usually contributes about one-sixth of the energy content of the diet and has remained relatively constant over time. However, current interest in high-protein low-carbohydrate diets as a strategy for weight loss has fuelled interest in the wider role of protein in body-weight regulation (Butler, 2004). Although a high-protein diet is associated with increases in dietary-induced thermogenesis and modest reductions in digestible energy, there is little evidence that increases in protein intake, within an achievable range, contribute substantially to differences in energy losses through these routes (Prentice, 1995). Instead, there is growing acceptance that the negative energy balance achieved with a high-protein diet is primarily driven by reductions in energy intake (Astrup *et al*. 2004).

There is some evidence from single-meal or single-day studies that high protein intakes may be associated with increased satiety (Poppitt *et al*. 1998). Simpson and colleagues (Simpson & Raubenheimer, 2005) have described an elegant model to account for the effect of changes in the protein:carbohydrate value of the diet on spontaneous energy intake, which is consistent with food intake control in some animal studies. The satiating properties of protein appear to be independent of energy density and re-emphasise the complexity of the appetite control system.

However, as with other macronutrients, the negative energy balance is poorly sustained in community-based trials. Studies of weight loss on high-protein diets initially tend to show greater reductions in body weight than with low-fat regimens. However, studies of 1-year duration have found no significant difference between the two dietary approaches (Foster *et al*. 2003; Stern *et al*. 2004). This outcome may reflect decreases in compliance, adaptation of the appetite control system or the impact of the strong environmental determinants that can override innate regulatory systems. A more integrated approach has combined a low-fat diet with a modest increase in protein, achieved by lean meat and low-fat dairy products, to give a macronutrient intake (protein, fat and carbohydrate respectively) of 30, 30 and 40% *v*. 12, 30 and 58% *v*. habitual dietary composition (Skov *et al*. 1999). Both low-fat groups were found to lose weight, with greater losses in the high-protein group, over the first 6 months. Subsequently, both groups were found to regain weight, although a greater proportion of subjects in the high-protein group were reported to be able to maintain clinically-important weight losses of >10%. However, to date there has been no long-term consideration of the potential for diets with an increased proportion of protein to prevent weight gain, and further research on broader health outcomes is required before high-protein diets can be recommended as part of a public health strategy for the prevention of weight gain.

The nature of the protein may also be important, perhaps reflecting differences in the proportion of specific amino acids (e.g. branched-chain amino acids) or other components of protein-rich foods. For example, it has been suggested that a dairy-rich diet may prevent weight gain. Data from the US National Health and Nutrition Examination Survey III suggests that the odds ratio of being obese in the highest quartile of fatness decreases significantly ($P<0{\cdot}0009$) with increasing Ca intake (Zemel *et al*. 2000; Fig. 6). Multiple mechanisms have been proposed that are based on work with animals and human subjects. In the transgenic obesity-prone agouti mouse weight gain is attenuated by increases in dietary Ca, with increased faecal fatty acid excretion and increases in thermogenesis, possibly mediated through decreases in 1,25-dihydroxycholecalciferol, an inhibitor of uncoupling protein 2 expression (Shi *et al*. 2002). Effects on nutrient partitioning, with putative feedback loops to appetite, have also been proposed (Shi *et al*. 2001) but the precise mechanism remains unclear (Harris, 2005).

However, meta-analyses based on the results of Ca intervention studies re-analysed to examine effects on body weight or composition have been inconsistent (Heaney *et al*. 2002; Barr, 2003). To date few studies have been set up specifically to address weight-related issues. In one of the most positive studies thirty-two subjects were randomised to one of three dietary groups receiving: a standard hypoenergetic diet containing 400–500 mg Ca/d plus placebo supplement; a similar diet with a supplement of 800 mg Ca/d; a dairy-rich hypoenergetic diet providing 1200–1300 mg Ca/d plus placebo supplement (Zemel *et al*. 2004). Subjects in these three groups were reported to have lost 6·4, 8·6 and 10·9% of

https://doi.org/10.1079/PNS2005429 Published online by Cambridge University Press

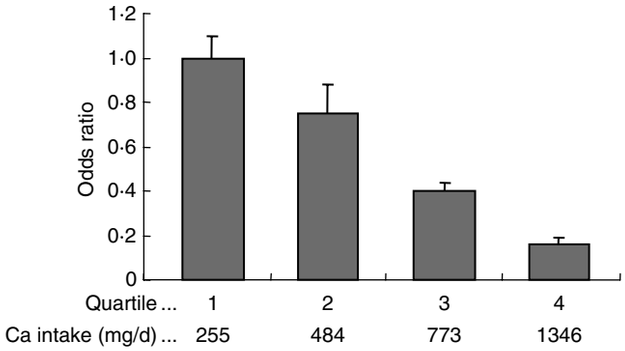

Fig. 6. Odds ratio of being in the highest body-fat quartile in relation to quartiles of calcium intake. Values are means with their standard errors represented by vertical bars. The odds ratio of being obese in the highest quartile of fatness decreased significantly with increasing calcium intake ($P<0·0009$). (Data from US National Health and Nutrition Examination Survey III; Zemel *et al.* 2000.)

their initial body weight respectively. However a recent, more comprehensive trial over 1 year has failed to find any differences in body weight or fat mass during the intervention period (Gunther *et al*. 2005).

Eating behaviour

It is evident that there is a paucity of well-controlled long-term community-based trials to investigate the effect of macronutrient manipulations on weight loss. However, to date there is little evidence to suggest that weight loss is mediated by metabolic effects. Instead, the key issue is compliance to the dietary prescription. This position has been reinforced by a recent study of four commercial diet-plans of varying macronutrient content that has demonstrated that adherence is the strongest predictor of success (Dansinger *et al*. 2005). Studies of preventative dietary strategies are even rarer, requiring larger numbers of subjects and longer-term follow-up. While not disregarding the potential for benefit, in terms of public health guidelines, it is also important to consider broader aspects of eating behaviour.

Portion size

Food of any composition, if consumed in excess, will lead to weight gain. One of the most noticeable changes in the diet in recent years has been the increase in portion size, especially in relation to soft drinks and energy-dense foods such as savoury snacks and confectionery (Nielsen & Popkin, 2003). There is now good experimental evidence to indicate that increases in portion size favour excess consumption (Rolls, 2003). Studies in young children have shown that while appetite control is initially good and 3-year-old children appear insensitive to the effects of portion size, this innate regulation is rapidly lost and for children ≥5 years of age energy intake tends to increase when presented with larger portions (Rolls *et al*. 2000). In adults the studies of the impact of varying portion size

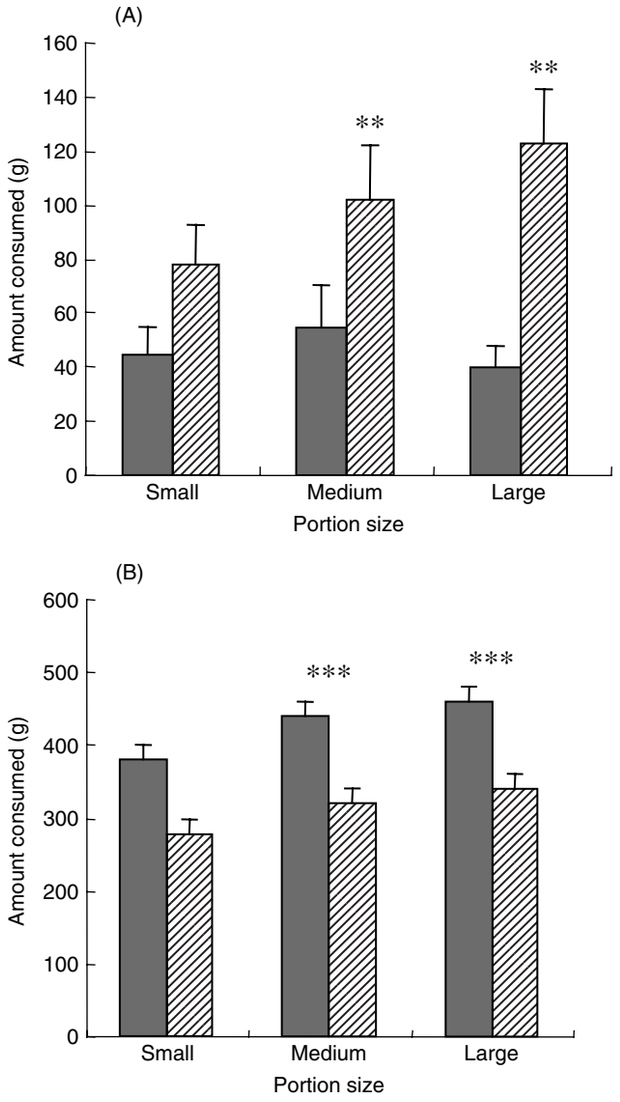

Fig. 7. *Ad libitum* energy intake when offered small, medium or large portions for (A) 3-year-old (■) and 5-year-old (▨) children and for (B) men (■) and women (▨).Values are means with their standard errors represented by vertical bars. Mean values were significantly different from those for the small portion size: $^{**}P>0·02$, $^{***}P>0·001$. (Data from Rolls *et al.* 2000, 2002.)

have shown that post-meal satiety and energy intake at the subsequent eating episode are unaffected, implying that larger portions will be associated with greater energy intake (Rolls *et al*. 2002; Fig. 7). In a more naturalistic study restaurant patrons choosing larger portions of the main course were observed to consume 43% more energy than those choosing the regular size portion and to consume 25% more energy in the meal as a whole, suggesting inadequate energy compensation in other components of the meal (Diliberti *et al*. 2004).

Although this experimental data is not wholly conclusive, it is paradoxical that at a time when levels of physical activity are so low there is a trend towards increasingly large portions. There is a real challenge to persuade

https://doi.org/10.1079/PNS2005429 Published online by Cambridge University Press

Table 2. Changes in snacking habits in USA; data from National Food Consumption Survey (1977–8; adjusted for sex, age and puberty) and Continuing Survey of Food Intake (1989–91 and 1994–6; adjusted for sex, age and education)
(Mean values with their standard errors)

	1977–8		1989–91		1994–6	
	Mean	SE	Mean	SE	Mean	SE
Snacking occasions	1·70	0·04	1·69	0·07	1·92**	0·07
Energy intake per occasion:						
kJ	1030	34·5	1105	65·5	1305**	64·2
kcal	247	8·28	265	15·7	313**	15·4
Energy intake/g snack food:						
kJ	4·38	0·13	5·43	0·46	5·50**	0·29
kcal	1·05	0·03	1·30	0·11	1·32**	0·07

Mean values were significantly different from those for 1977–8: **$P<0·01$.

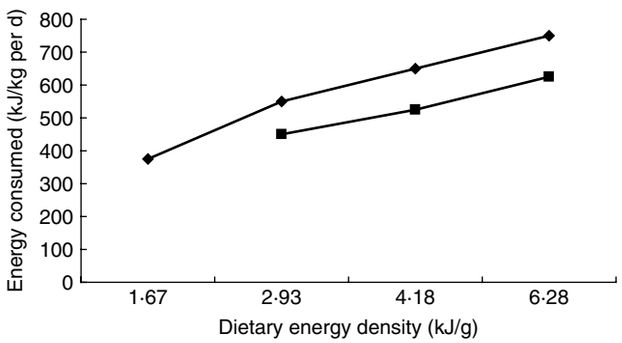

Fig. 8. Effect of meal frequency (three meals per d (■) or four meals per d (◆)) and dietary energy density on energy intake during re-feeding programmes for malnourished children. (From Brown *et al.* 1995.)

consumers to accept smaller portions while larger sizes continue to offer greater value for money.

Snacking

It is not only the quality and quantity of food that has changed in recent years. Profound changes in lifestyle have led to a situation in which the traditional 'three-meal-a-day' eating pattern has been replaced by more frequent eating episodes of variable size commonly, but loosely, described as snacking. Large-scale survey data, despite all its imperfections, show that over time there has been an increase in eating frequency and that this pattern of eating is associated with an increase in the energy consumed on each eating occasion, presumably reflecting larger portion sizes, and an increase in the energy density of items consumed, reflecting the change in diet quality (Zizza *et al.* 2001; Table 2). The impact of this change on body weight is difficult to assess because of the complexity of the nature and type of snacks consumed.

Dietary surveys frequently show an inverse association between eating frequency and body weight, but this relationship is thought to reflect greater levels of under-reporting by obese subjects than their lean counterparts (Bellisle *et al.* 1997). Studies conducted under highly-controlled experimental conditions suggest that the effects on energy expenditure are similar when isoenergetic quantities of food are consumed either in a small number of large meals or multiple smaller snacks (Van de Venne-Verboeket & Westerterp, 1991; Van de Venne-Verboeket *et al.* 1993). However, there are clear beneficial effects of frequent feeding on lipid and glucose metabolism (Farshchi *et al.* 2005).

These laboratory studies, however, have found it difficult to replicate the social and cultural factors that impact on food choice and snacking habits in the free-living situation. Observational studies are beginning to address the issue, but results are conflicting and heavily confounded by multiple other influences on body weight and fatness, not least interactions with physical activity habits (Francis *et al.* 2003; Phillips *et al.* 2004). For example, analysis of dietary data from the British National Diet and Nutrition Survey (4–18 years) has shown that the most-sedentary individuals have higher intakes of savoury snacks (Rennie & Jebb, 2004).

Set in a wider context, the rise in eating frequency suggests potential cause for concern in relation to body weight. Studies in malnourished children in developing countries have shown that increases in meal frequency, and also in energy density, are a very effective method for increasing energy intake during re-feeding programmes (Brown *et al.* 1995; Fig. 8). It seems ironic that the strategy developed to re-feed the world's most malnourished children has become the norm in many households in the developed world. The threat this poses to body weight needs to be seriously evaluated.

Diet and obesity: whose responsibility?

In analysing the dietary factors associated with an increased risk of weight gain, the present review has not considered the multitude of factors that underpin eating behaviour. These factors include numerous influences relating to the period *in utero* (Oken & Gillman, 2003), early feeding practices such as breast-feeding *v.* bottle feeding (Von Kries *et al.* 1999), weaning practices (Birch, 1999), the later family food environment (Campbell & Crawford, 2001) and indeed wider societal factors (Reidpath *et al.* 2002), which have been considered elsewhere. However, looking across the breadth of the scientific literature there is now a clear framework for reducing the

https://doi.org/10.1079/PNS2005429 Published online by Cambridge University Press

risk of obesity that is consistent with dietary strategies for the prevention of many chronic diseases, including CVD and cancer (Swinburn *et al*. 2004). This framework builds upon and refines previous dietary recommendations. However, it is increasingly recognised that individual nutritional education needs to be balanced with wider environmental changes that support and facilitate healthier choices. This shift in emphasis, even at a political level, has been highlighted by the House of Commons Health Select Committee (2004) Obesity Report, which noted 'While we recognise that individuals have a key role in determining their own health and lifestyles ... it is critical that obesity is tackled first and foremost at a societal rather than an individual level'.

There is now sufficient evidence to take clear and decisive steps to develop dietary strategies for the prevention of obesity. In the UK this issue has been taken up by the government under the auspices of the White Paper Choosing Health (Department of Health, 2004), and there is hope that there may now be more concerted and coordinated action by government to take a lead in this area and to hold other stakeholders accountable for their actions. Nutrition scientists alone cannot tackle the dietary issues that underpin obesity. However, they have a valuable role in providing the evidence base for rational interventions and evaluating the impact of dietary change on body weight and the risk of obesity.

References

Anderson JW, Randles KM, Kendall CW & Jenkins DJ (2004) Carbohydrate and fibre recommendations for individuals with diabetes: a quantitative assessment and meta-analysis of the evidence. *Journal of the American College of Nutrition* **23**, 5–17.

Astrup A, Grunwald GK, Melanson EL, Saris WH & Hill JO (2000) The role of low-fat diets in body weight control: a meta-analysis of ad libitum dietary intervention studies. *International Journal of Obesity* **24**, 1545–1552.

Astrup A, Larsen TM & Harper A (2004) Atkins and other low-carbohydrate diets: hoax or an effective tool for weight loss. *Lancet* **364**, 897–899.

Barr S (2003) Increased dairy product or calcium intake: Is body weight or composition affected in humans? *Journal of Nutrition* **133**, 245S–248S.

Bellisle F, McDevitt R & Prentice AM (1997) Meal frequency and energy balance. *British Journal of Nutrition* **77**, S57–S70.

Birch LL (1999) Development of food preferences. *Annual Review of Nutrition* **19**, 41–62.

Black AE, Goldberg GR, Jebb SA, Livingstone MBE & Prentice AM (1991) Critical evaluation of energy intake data using fundamental principles of energy physiology. 2. Evaluating the results of dietary surveys. *European Journal of Clinical Nutrition* **45**, 583–599.

Bouche C, Rizkalla SW, Luo J, Vidal H, Veronese A, Pacher N, Fouquet C, Lang V & Slama G (2002) Five-week, low-glycemic index diet decreases total fat mass and improves plasma lipid profile in moderately overweight non-diabetic men. *Diabetes Care* **25**, 822–828.

Brand-Miller J, Holt SHA, Pawlak DB & McMillan J (2002) Glycemic index and obesity. *American Journal of Clinical Nutrition* **76**, 281S–285S.

Bray GA, Nielsen SJ & Popkin BM (2004) Consumption of high-fructose corn syrup in beverages may play a role in the epidemic of obesity. *American Journal of Clinical Nutrition* **79**, 537–543.

Bray GA & Popkin BA (1998) Dietary fat intake does affect obesity! *American Journal of Clinical Nutrition* **68**, 1157–1173.

Brown KH, Sanchez-Grinan M, Perez F, Peerson JM, Ganoza L & Stern JS (1995) Effects of dietary energy density and feeding frequency on total daily energy intakes of recovering malnourished children. *American Journal of Clinical Nutrition* **62**, 13–18.

Butler D (2004) Slim pickings. *Nature* **428**, 252–254.

Campbell K & Crawford D (2001). 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* **58**, 19–25.

Dansinger ML, Gleason JA, Griffith JL, Selker HP & Schaefer EJ (2005) Comparison of the Atkins, Ornish, Weight Watchers, and Zone diets for weight loss and heart disease risk reduction. *Journal of the American Medical Association* **293**, 43–53.

Department of Health (2004) Choosing health: making choices easier. http://www.whig.org.uk/groups/doh/choosing-health-wp/

Diliberti N, Bordi PL, Conklin MT, Roe LS & Rolls BJ (2004) Increased portion size leads to increased energy intake in a restaurant meal. *Obesity Research* **12**, 562–568.

DiMeglio DP & Mattes RD (2000) Liquid verses solid carbohydrate: effects on food intake and body weight. *International Journal of Obesity and Related Metabolic Disorders* **24**, 794–800.

Ebbeling CB, Sinclair KB, Pereira MA, Garcia-Lago E, Feldman HA & Ludwig DS (2004) Compensation for energy intake from fast food among overweight and lean adolescents. *Journal of the American Medical Association* **291**, 2828–2833.

Ello-Martin JA (2004) A diet reduced in energy density results in greater weight loss than a diet reduced in fat. *Obesity Research*, 12S.

Farshchi HR, Taylor MA & Macdonald IA (2005) Beneficial metabolic effects of regular meal frequency on dietary thermogenesis, insulin sensitivity and fasting lipid profiles in healthy obese women. *American Journal of Clinical Nutrition* **81**, 16–24.

Foster GD, Wyatt HR, Hill JO, McGuckin BG, Brill C, Mohammed S, Szapary PO, Rader DJ, Edman JS & Klein S (2003) A randomized trial of a low-carbohydrate diet for obesity. *New England Journal of Medicine* **348**, 2082–2090.

Francis LA, Lee Y & Birch LL (2003) Parental weight status and girls' television viewing, snacking and body mass indexes. *Obesity Research* **11**, 143–151.

French SA, Hannan PJ & Story M (2004) School soft drink intervention study. *British Medical Journal* **14**, E7315–E7466.

Goldberg GR, Black AE, Jebb SA, Cole TJ, Murgatroyd PR, Coward WA & Prentice AM (1991) Critical evaluation of energy intake data using fundamental principles of energy physiology. *European Journal of Clinical Nutrition* **45**, 569–581.

Gregory J & Low S (2000) *National Diet and Nutrition Survey of Young People Aged 4–18. Years*. Vol. 1: *Report of the Diet and Nutrition Survey*. London: H. M. Stationery Office.

Gunther CW, Legowski PA, Lyle RM, McCabe GP, Eagan MS, Peacock M & Teegarden D (2005) Dairy products do not lead to alterations in body weight or fat mass in young women in a 1-y intervention. *American Journal of Clinical Nutrition* **81**, 751–756.

Hansen BC (2001) *Causes of Obesity and Consequences of Obesity Prevention in Non-Human Primates and Other Animal Models*. Chichester, West Sussex: John Wiley & Sons Ltd.

Harris RBS (2005) Dairy protein, calcium and body weight – the need for a mechanism. *International Journal of Obesity* **29**, 388–390.

He K, Hu FB, Colditz GA, Manson JE, Willett WC & Liu S (2004) Changes in intake of fruits and vegetables in relation to risk of obesity and weight gain among middle-aged women. *International Journal of Obesity* **28**, 1569–1574.
Heaney RP, Davies M & Barger-Lux J (2002) Calcium and weight: Clinical studies. *Journal of the American College of Nutrition* **21**, 152S–155S.
Henderson L, Gregory J, Irving K & Swan G (2002) *The National Diet and Nutrition Survey: Adults Aged 19 to 64 Years. Energy, Protein, Carbohydrate, Fat and Alcohol Intake*. London: H. M. Stationery Office.
Hill JO & Prentice AM (1995) Sugar and body weight regulation. *American Journal of Clinical Nutrition* **62**, Suppl., 264S–274S.
House of Commons Health Select Committee (2004) *Obesity*. Vol. 1: *Report Together with Formal Minutes. Third Report of Session 2003–4*. London: The Stationery Office.
Howarth NC, Saltzman E & Roberts SB (2001) Dietary fiber and weight regulation. *Nutrition Reviews* **59**, 129–139.
James J, Thomas P, Cavan D & Kerr D (2004) Preventing childhood obesity by reducing consumption of carbonated drinks: cluster randomised controlled trial. *British Medical Journal* **328**, 1237.
Koh-Banerjee P, Franz M, Sampson L, Liu S, Jacobs DR, Spiegelman D, Willett W & Rimm E (2004) Changes in whole-grain, bran and cereal fiber consumption in relation to 8-y weight gain among men. *American Journal of Clinical Nutrition* **80**, 1237–1245.
Liu S, Willett WC, Manson JE, Hu FB, Rosner B & Colditz GA (2003) Relation between changes in intakes of dietary fiber and grain products and changes in weight and development of obesity among middle-aged women. *American Journal of Clinical Nutrition* **78**, 920–927.
Livingstone MBE & Black AE (2003) Markers of the validity of reported energy intake. *Journal of Nutrition* **133**, 895S–920S.
Ludwig DS, Peterson KE & Gortmaker SL (2001) Relation between consumption of sugar-sweetened drinks and childhood obesity. A prospective, observational analysis. *Lancet* **357**, 505–508.
Mattes RD & Rothacker D (2001) Beverage viscosity is inversely related to postprandial hunger in humans. *Physiology and Behaviour* **74**, 551–557.
Mrdjenovic G & Levitsky DA (2003) Nutritional and energetic consequences of sweetened drink consumption in 6- to 13-year-old children. *Journal of Pediatrics* **142**, 604–610.
National Audit Office (2001) *Tackling Obesity in England*, pp. 1–66. London: National Audit Office.
Nielsen SJ & Popkin BM (2003) Patterns and trends in food portion sizes 1977–1998. *Journal of the American Medical Association* **289**, 450–453.
Oken G & Gillman MW (2003) Fetal origins of obesity. *Obesity Research* **11**, 496–506.
Pawlak DB, Kushner JA & Ludwig DS (2004) Effects of dietary glycaemic index on adiposity, glucose homoeostasis, and plasma lipids in animals. *Lancet* **364**, 778–785.
Pereira MA, Kartashov AI, Ebbeling CB, Van Horn L, Slattery ML, Jacobs DR & Ludwig DS (2005) Fast-food habits, weight gain, and insulin resistance (the CARDIA study): 15-year prospective analysis. *Lancet* **365**, 36–42.
Phillips SM, Bandini LG, Naumova EN, Cyr H, Colclough S, Dietz WH & Must A (2004) Energy-dense snack food intake in adolescence: Longitudinal relationship to weight and fatness. *Obesity Research* **12**, 461–472.
Poppitt S & Prentice AM (1996) Energy density and its role in the control of food intake: Evidence from metabolic and community studies. *Appetite* **26**, 153–174.
Poppitt SD, McCormack D & Buffenstein R (1998) Short-term effects of macronutrient preloads on appetite and energy intake in lean women. *Physiology and Behaviour* **64**, 279–285.
Prentice AM (1995) Are all calories equal? In *Weight Control: The Current Perspective*, pp. 8–13 [R Cottrell, editor]. London: Chapman & Hall.
Prentice AM (1997) Obesity – the inevitable penalty of civilisation? *British Medical Bulletin* **53**, 229–237.
Prentice AM & Jebb SA (2003) Fast foods, energy density and obesity – A possible mechanistic link. *Obesity Reviews* **4**, 187–194.
Prentice AM & Jebb SA (2004) Energy intake/physical activity interactions in the homeostasis of body weight regulation. *Nutrition Reviews* **62**, S98–S104.
Raben A (2002) Should obese patients be counselled to follow a low-glycaemic index diet? No. *Obesity Reviews* **3**, 245–256.
Raben A, Vasilaras TH, Moller AC & Astrup A (2002) Sucrose compared with artificial sweeteners: different effects on ad libitum food intake and body weight after 10 weeks of supplementation in overweight subjects. *American Journal of Clinical Nutrition* **76**, 721–729.
Reidpath DD, Burns C, Garrard J, Mahoney M & Townsend M (2002) An ecological study of the relationship between social and environmental determinants of obesity. *Health and Place* **8**, 141–145.
Rennie K & Jebb SA (2004) Sedentary lifestyles are associated with being overweight and consumption of savoury snacks in young people (4–18 years). *Proceedings of the Nutrition Society* (In the Press).
Rennie KL, Coward WA, Wright A & Jebb SA (2004) Estimating under-reporting of energy intake in a UK national survey. *Obesity Research* **12**, A219.
Rennie KL, Jebb SA, Wright A & Coward WA (2005) Secular trends in under-reporting of energy intake in the National Diet and Nutrition Survey of young people. *British Journal of Nutrition* **93**, 241–247.
Rolls BJ (2003) The supersizing of America. *Nutrition Today* **38**, 42–53.
Rolls BJ, Ello-Martin JA & Carlton-Tohill B (2004) What can intervention studies tell us about the relationship between fruit and vegetable consumption and weight management. *Nutrition Reviews* **62**, 1–17.
Rolls BJ, Engell D & Birch LL (2000) Serving portion size influences 5-year-old but not 3-year-old children's food intakes. *Journal of the American Dietetic Association* **100**, 232–234.
Rolls BJ, Morris EL & Roe LS (2002) Portion size of food affects energy intake in normal-weight and overweight men and women. *American Journal of Clinical Nutrition* **76**, 1207–1213.
Rothwell N & Stock M (1979) A role for brown adipose tissue in diet-induced thermogenesis. *Nature* **281**, 31–35.
Rothwell NJ, Saville ME & Stock MJ (1982) Effects of feeding a cafeteria diet on energy balance and diet-induced thermogenesis in four strains of rat. *Journal of Nutrition* **112**, 1515–1524.
Schulze MB, Manson JE, Ludwig DS, Colditz GA, Stampfer MJ, Willett WC & Hu FB (2004) Sugar-sweetened beverages, weight gain, and incidence of type 2 diabetes in young and middle-aged women. *Journal of the American Medical Association* **292**, 927–934.
Schwartz MW, Woods SC, Porte D, Seeley RJ & Baskin DG (2000) Central nervous system control of food intake. *Nature* **404**, 661–671.
Shi H, DiRenzo D & Zemel MB (2001) Effects of dietary calcium on adipocyte lipid metabolism and body weight regulation in energy-restricted aP2-agouti transgenic mice. *FASEB Journal* **15**, A598.

Shi H, Norman AW, Okamura WH, Sen A & Zemel MB (2002) 1 alpha-25-dihydroxyvitamin D3 inhibits uncoupling protein 2 expression in human adipocytes. *FASEB Journal* **16**, 1808–1810.

Simpson SJ & Raubenheimer D (2005) Obesity: The protein leverage hypothesis. *Obesity Reviews* (In the Press).

Singh RB, Dubnov G, Niaz MA, Ghosh S, Singh R, Rastogi SS, Manor O, Pella D & Berry EM (2002) Effect of an Indo-Mediterranean diet on progression of coronary artery disease in high risk patients (Indo-Mediterranean Diet Heart Study, a randomised single-blind trial). *Lancet* **360**, 1455–1461.

Skov AR, Toubro S, Ronn B, Holm L & Astrup A (1999) Randomised trial on protein vs carbohydrate in ad libitum fat reduced diet for the treatment of obesity. *International Journal of Obesity* **23**, 528–536.

Stern L, Nayyar I, Seshadri P, Chicano KL, Daily DA, McCrory J, Williams M, Gracely EJ & Samaha FF (2004) The effects of low-carbohydrate versus conventional weight loss diets in severely obese adults: one-year follow-up of a randomised trial. *Annals of Internal Medicine* **140**, 778–785.

Stubbs RJ, Harbron CJ, Murgatroyd PR & Prentice AM (1995) 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* **62**, 316–329.

Stubbs RJ, Harbron CG & Prentice AM (1996) 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* **20**, 651–660.

Swinburn BA, Caterson I, Seidell JC & James WP (2004) Diet, nutrition and the prevention of excess weight gain and obesity. *Public Health Nutrition* **7**, 123–146.

Tordoff MG & Alleva AM (1990) Effect of drinking soda sweetened with aspartame or high fructose corn syrup on food intake and body weight. *American Journal of Clinical Nutrition* **51**, 963–969.

Van de Venne-Verboeket WPHG & Westerterp KR (1991) Influence of feeding frequency on nutrient utilisation in man: consequences for energy metabolism. *European Journal of Clinical Nutrition* **45**, 161–169.

Van de Venne-Verboeket WPHG, Westerterp KR & Kester ADM (1993) Effect of the pattern of food intake on human energy metabolism. *British Journal of Nutrition* **70**, 103–115.

Von Kries R, Koletzko B, Sauerwald T, von Mutius E, Barnert D, Grunert V & Von Voss H (1999) Breast feeding and obesity: cross sectional study. *British Medical Journal* **319**, 147–150.

World Health Organization/Food and Agriculture Organization (2003) *Diet, Nutrition and the Prevention of Chronic Diseases*. *WHO Technical Report Series* no. 916. Geneva: WHO.

Willett WC (1998) Is dietary fat a major determinant of body fat? *American Journal of Clinical Nutrition* **67**, Suppl., 556S–562S.

Zemel MB, Shi H, Greer B, Direnzo D & Zemel PC (2000) Regulation of adiposity by dietary calcium. *FASEB Journal* **14**, 1132–1138.

Zemel MB, Thompson W, Milstead A, Morris K & Campbell P (2004) Calcium and dairy acceleration of weight and fat loss during energy restriction in obese adults. *Obesity Research* **12**, 582–590.

Zizza C, Siega-Riz AM & Popkin BM (2001) Significant increase in young adults' snacking between 1977–1978 and 1994–1996 represents a cause for concern! *Preventative Medicine* **32**, 303–310.