

Plenary Lecture

Dietary strategies for the prevention of obesity

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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.

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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,

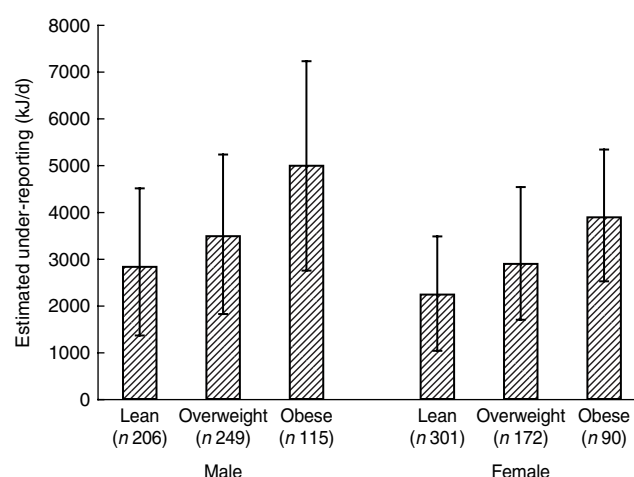


Fig. 1. Estimated under-reporting (based on individual estimates of energy expenditure) by weight status in the British National Diet and Nutrition Survey 16–64 years (Rennie *et al.* 2004). Values are means with the median and interquartile range represented by vertical bars.

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.

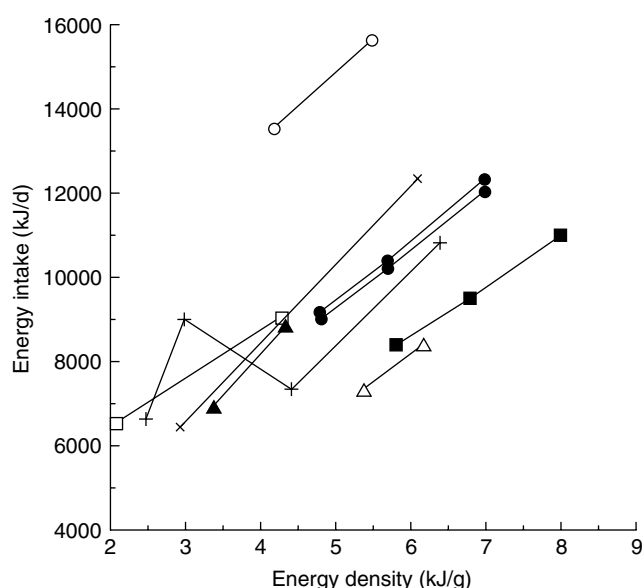
Thus, epidemiological associations alone provide insufficient foundation for nutrition policy. Recent initiatives, notably the World Health Organization/Food and Agriculture Organization (2003) report on the prevention of chronic diseases, have pulled together evidence from epidemiology, experimental 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

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 ($P < 0.0001$), 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).

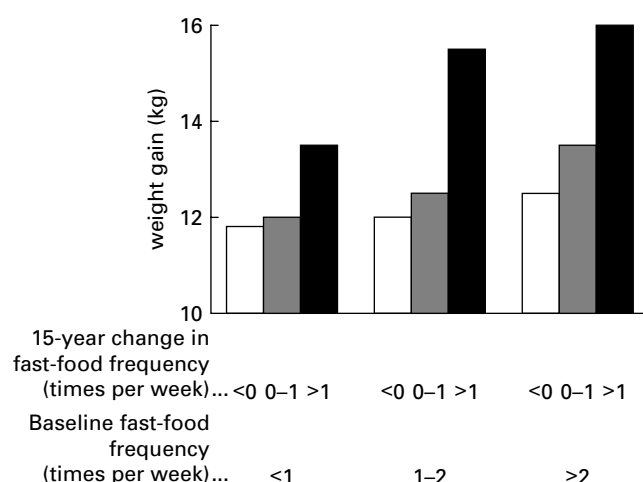


Fig. 3. Association between frequency of fast-food consumption at baseline, 15-year change in fast-food frequency (times per week; <0 (i.e. decrease in fast-food frequency), □; 0–1, ■; >1, ■) and 15-year change in body weight in 3031 adults aged 18–30 years. (Data from Pereira *et al.* 2005.)

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

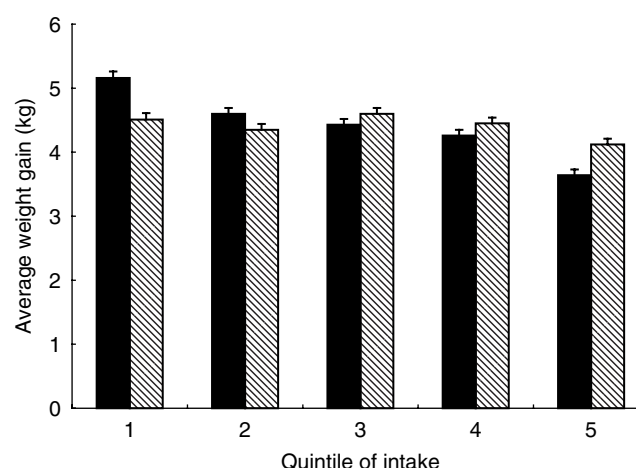


Fig. 4. Type of carbohydrate consumed and weight gain (adjusted for age, changes in exercise, snacking, hormone-replacement therapy and other variables) in the Nurses' Health Study during a 12-year follow-up. Values are means with their standard errors represented by vertical bars. ■, fibre ($P < 0.0001$ for trend); ▨, wholegrain ($P < 0.0001$ for trend).

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

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,

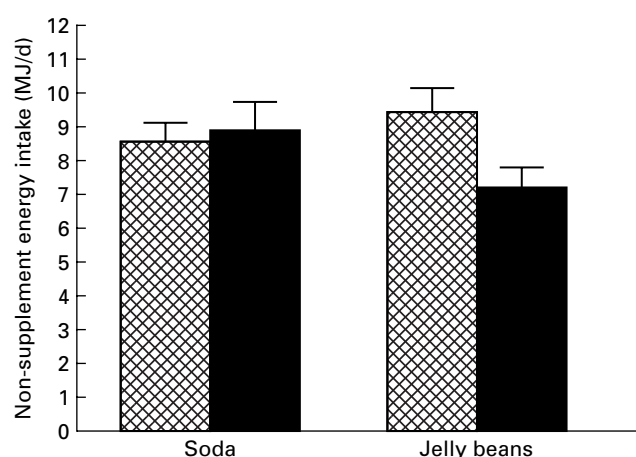


Fig. 5. Effect of solid (jelly beans) v. liquid (soda) carbohydrate on *ad libitum* energy intake over 4 weeks. (▨), Baseline; (■) after 4 weeks. Values are means with their standard errors represented by vertical bars. (From DiMeglio & Mattes, 2000; reprinted with permission from Macmillan Publishers 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 ($P < 0.0009$) 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-dihydroxy-cholecalciferol, 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

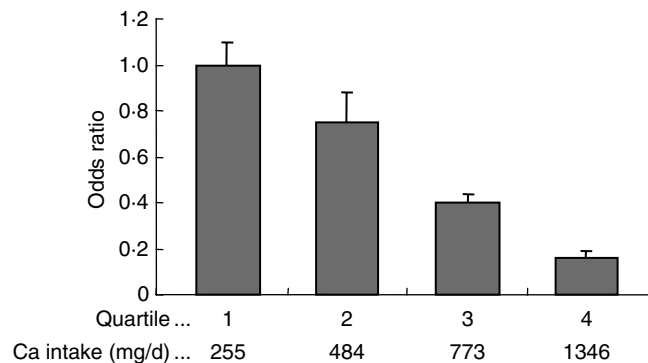


Fig. 6. Odds ratio of being in the highest body-fat quartile in relation to quartiles of calcium intake. Values are means with their standard errors represented by vertical bars. The odds ratio of being obese in the highest quartile of fatness decreased significantly with increasing calcium intake ($P < 0.0009$). (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

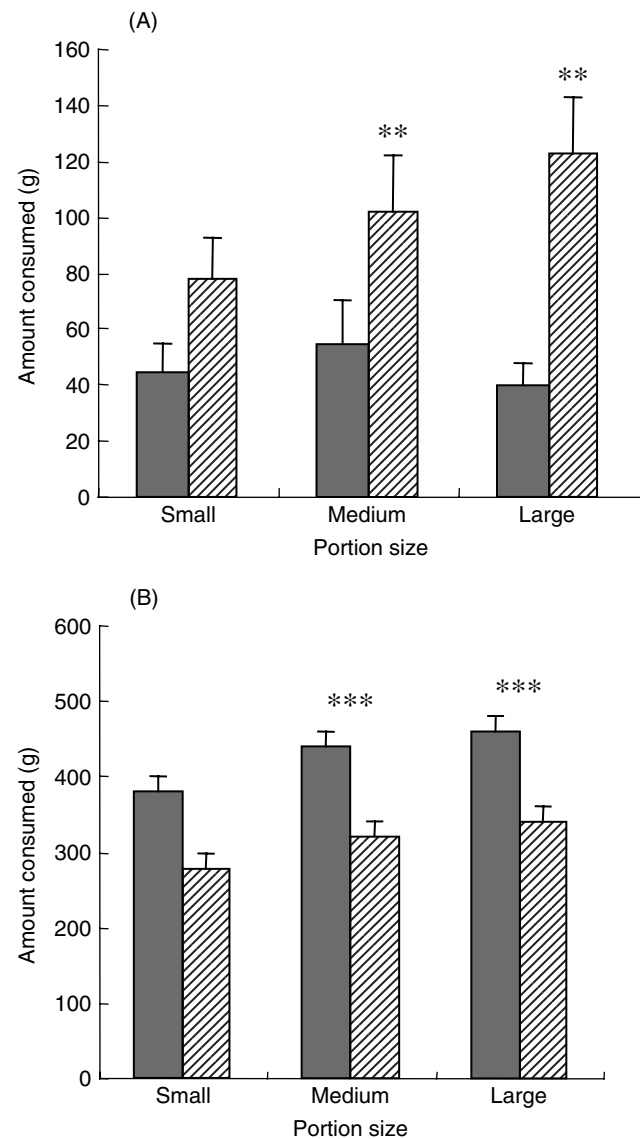


Fig. 7. Ad libitum energy intake when offered small, medium or large portions for (A) 3-year-old (■) and 5-year-old (▨) children and for (B) men (■) and women (▨). Values are means with their standard errors represented by vertical bars. Mean values were significantly different from those for the small 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

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: ** $P < 0.01$.

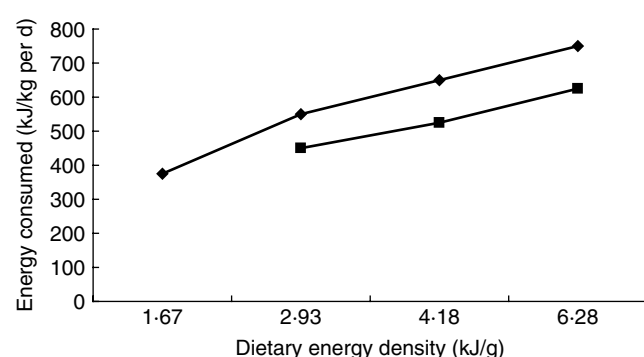


Fig. 8. Effect of meal frequency (three meals per d (■) or four meals per d (◆)) and dietary energy density on energy intake during re-feeding programmes for malnourished children. (From Brown *et al.* 1995.)

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

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.

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