Despite widespread interest in body-weight control, the prevalence of obesity continues to rise worldwide. Current public health advice for obesity prevention is clearly failing. The present paper examines the appropriateness of current public health advice for body-weight control, i.e. to reduce consumption of fatty foods, to reduce consumption of sugar and to avoid snacking between meals. An increase in carbohydrate : fat ratio should improve body-weight control, as high-carbohydrate low-fat diets are less likely to lead to overeating, and if overeating does occur, less of the excess energy is likely to be stored as fat. However, it is suggested that for the long-term prevention of weight gain, advice to increase consumption of carbohydrate-rich foods may be more effective than advice which focuses on reducing consumption of fatty food. Moreover, in view of the inverse relationship between fat and sugar intakes, sugar may have a positive role to play in body-weight control in facilitating an increase in carbohydrate : fat ratio. Snacking for most individuals appears not to adversely affect body-weight control, and for some it may improve control. This situation may exist because frequent eating helps appetite control, thus preventing overeating at meals, and as snacks overall tend to be higher in carbohydrate and lower in fat than meals, frequent eating may be a strategy for increasing carbohydrate : fat ratio. It is also suggested that eating ‘little and often’ may be a more compatible pattern of eating for a physically-active lifestyle than eating large meals. Perhaps the most appropriate advice on food intake that would work synergistically with concurrent advice to increase physical activity is to eat more carbohydrate, and to eat frequently.

Body-weight control: Macronutrients: Eating frequency

The prevalence of overweight and obesity is rising sharply in many countries throughout the world, despite public health efforts to prevent obesity and a huge level of consumer interest in slimming worldwide. Current approaches to obesity prevention are clearly failing. Either inappropriate advice is being given to the public, or the public are unwilling or unable to follow current advice.

The energy balance equation \( \Delta \text{body energy} = \text{energy intake} - \text{energy expenditure} \) shows that an accumulation of body fat can only occur when there is a sustained positive energy balance, i.e. when energy intake exceeds energy expenditure for a prolonged period. In theory the solution is simple; to achieve body-weight stability in the population we need to identify and implement public health advice which will be effective in enabling the population to match energy intake with energy expenditure. The present paper considers the ‘energy intake’ component of the energy balance equation, and makes suggestions about public health advice on food intake for improved body-weight control.

Public health messages for body-weight control

Present public health advice for body-weight control includes advice to:

- reduce consumption of fatty foods;
- reduce consumption of sugar-containing foods;
- avoid snacking between meals;
- increase physical activity.

**Corresponding author:** T. R. Kirk, fax +44 (0)131 317 3528, email t.kirk@mail.qmced.ac.uk
The appropriateness of each of the three dietary advice messages will be examined. In doing so the compatibility of the dietary advice with concurrent advice to increase levels of physical activity in the population will be considered.

Reducing consumption of fatty foods

There is considerable evidence to support the hypothesis that an increase in carbohydrate : fat ratio should help body-weight control. There are two strands of physiological evidence. First, an increase in carbohydrate : fat is thought to have a satiety-inducing effect, so that overeating is less likely to occur, possibly because carbohydrate exerts a more powerful metabolic effect than fat on satiety, through feedback mechanisms such as glucostatic mechanisms (Mayer, 1955; Van Itallie, 1990) or mechanisms sensitive to the rate of hepatic glucose oxidation (Friedman, 1991). However, current thinking is that an increase in carbohydrate : fat induces satiety because it is associated with a reduction in the energy density of the diet, so that for a given energy intake a larger volume, or weight, of food has to be eaten. Thus, as it is thought that individuals eat to a constant volume or weight of food (Poppitt & Prentice, 1998), at least in the short and medium terms, passive over-consumption is less likely on a diet of low energy density. The second strand of physiological evidence is that if overfeeding does occur on a diet with a high carbohydrate : fat value, slightly less of the excess energy is likely to be stored as fat, because carbohydrate overfeeding, particularly in the first 2 weeks, substantially increases the rate of carbohydrate oxidation, but only slightly suppresses fat oxidation. Thus, net storage of energy is small. Fat overfeeding, on the other hand, has little effect on substrate oxidation, so that net energy storage is greater (Horton et al. 1995). Moreover, there is strong evidence that de novo lipogenesis is quantitatively an insignificant pathway in human subjects under normal Western diet conditions (Acheson et al. 1988; Hellerstein, 1999). De novo lipogenesis is only invoked when carbohydrate overfeeding is massive (Acheson et al. 1988). Furthermore, when de novo lipogenesis does occur, it is an energetically inefficient process, whereas the conversion of dietary fat to body fat is highly energetically efficient (Flatt, 1978).

Empirical evidence from dietary intervention trials shows that an increase in carbohydrate : fat ratio achieved by low-fat intervention is effective in producing spontaneous weight loss (for example, see Yu-Poth et al. 1999; Astrup et al. 2000). Astrup et al. (2000) conducted a meta-analysis of the results from sixteen controlled low-fat ad libitum diets of 2–12 months duration involving 1728 subjects of both sexes. This analysis showed a spontaneous weight loss of 2.5 kg (95 % CI 1.5, 3.5, P < 0.001) more in the intervention group than in control groups. Moreover, the magnitude of the weight loss was greater as pretreatment weight increased, and was proportional to the magnitude of the reduction in dietary fat.

There may be justification for refining the ‘carbohydrate : fat’ ratio hypothesis. Preliminary evidence suggests that certain population subgroups are particularly prone to high-fat low-carbohydrate-induced obesity, i.e. the physically inactive (Lissner et al. 1997) and individuals with a genetic predisposition to obesity (Heitmann et al. 1995), possibly because they have a reduced ability to oxidize fat (Astrup et al. 1996, 1997). Furthermore, it is possible that obesity is induced more by episodic bouts of fat overfeeding, rather than by sustained exposure to a high-fat diet. Notwithstanding these refinements to the hypothesis, the evidence strongly suggests that an increase in dietary carbohydrate : fat ratio, as a public health measure, should help in the prevention of obesity at the population level.

Reducing consumption of fatty foods or increasing consumption of carbohydrate-rich foods?

In most low-fat dietary intervention trials reviewed by the present author (for example, see Schlundt et al. 1993; Shah et al. 1994; Jeffery et al. 1995; Lyon et al. 1995) the low-fat intervention produced an energy deficit which caused spontaneous weight loss. This situation occurred because absolute fat intake decreased substantially and there was no accompanying increase in absolute carbohydrate intake. Indeed, absolute carbohydrate intake tended to remain at approximately baseline level, and in some studies actually decreased. The lack of concomitant increase in absolute carbohydrate intake caused the energy deficit, which produced the spontaneous weight loss. Thus, there is good evidence to suggest that a reduction in fat intake without a corresponding increase in absolute carbohydrate intake is effective in producing spontaneous weight loss in the short to medium term. Thus, advice which focuses on reducing consumption of fatty foods may be appropriate for producing initial weight loss. However, we suggest that advice that produces a reduction in consumption of fatty foods without an absolute increase in consumption of carbohydrate-rich foods is unlikely to be effective in the long term amongst free-living individuals. There are three reasons. First, consumers are likely to develop resistance to prolonged exposure to negative advice, and, in the long term, are more likely to be motivated by positive advice (Hochbaum, 1981; Neilson & Larson-Brown, 1990). Second, individuals in free-living situations find advice to reduce consumption of foods high in fat particularly difficult to implement in practice (Mela, 1994; Van Assema et al. 1999). Third, low-fat diets in which absolute carbohydrate intake is not increased are likely to provide only a low weight (low volume) of food. Diets that do not provide a reasonable quantity of food are unlikely to be satisfying for many individuals, and are unlikely to be adopted in the context of lifelong eating habits. Long-term weight control requires a dietary pattern that allows a reasonable quantity of food.

We have tested an alternative approach to increasing carbohydrate : fat ratio. Our hypothesis was that positive intervention to increase consumption of carbohydrate-rich foods, without advice to reduce consumption of fatty foods, will result in a passive decrease in intake of fat, with an increase in absolute carbohydrate intake and no increase in energy intake. Three intervention studies have been conducted.

In the first study (Kirk et al. 1997) sixty young adult women of normal body weight or slightly overweight (mean BMI 23 (range 20–30) kg/m²) were matched and randomly allocated to either an intervention or control group. The...
intervention group were required to increase their consumption of breakfast cereals (unsweetened) by 60 g/d (approximately two bowls) with semi-skimmed milk for a period of 12 weeks. No other dietary advice or instructions were given. The control group were asked to maintain their habitual diet.

In the intervention group the percentage energy from fat decreased by 5·4 (from 35 to 29), with a corresponding increase of 5·1 in the percentage energy from starch (from 26 to 31; Table 1). These changes were found at 4 weeks and were sustained at 12 weeks. There were no changes in energy intake, indicating isoenergetic replacement of fat by starch. No similar dietary changes occurred in the control group. Dietary fat reduction was achieved because breakfast cereals replaced fatty foods in the diet. There were significant decreases in consumption of spreading fats, as breakfast cereals replaced ‘buttered’ bread and toast, and significant decreases in consumption of cakes, biscuits and confectionery as breakfast cereals replaced these as daytime snacks.

The second study (Crombie & Kirk, 1999) set out to test the same hypothesis in a potentially more difficult target group, overweight middle-aged men. Sixty-three middle-aged men (mean BMI 29.4 (range 27–32) kg/m²) were matched and randomly allocated to either one of two intervention groups or a control group. Intervention group 1 were required to increase their consumption of breakfast cereals (mixture of unsweetened and sweetened) by 90 g/d (approximately three bowls) with semi-skimmed milk for 20 weeks. Group 2 increased consumption of semi-skimmed milk only (300 ml/d), and the control group were asked to maintain their habitual diet. Assessments were made at 6, 12 and 20 weeks. Table 2 shows results for energy and macronutrient intakes at 20 weeks.

In intervention group 1, the breakfast-cereal group, the percentage energy from fat decreased by 5·1 (from 40 to 34), with a concomitant increase of 5·9 in the percentage energy from carbohydrate (from 40 to 47), i.e. isoenergetic replacement of dietary fat by carbohydrate had occurred. There were no significant changes in dietary macronutrient intakes in either the milk intervention group, or the control group. Thus, dietary fat reduction was achieved and sustained in both these studies without subjects making a conscious effort to do so. Note that, unlike the low-fat trials previously referred to, in these trials absolute carbohydrate intakes increased substantially.

Body-weight control was not mentioned to participants in either of these studies. However, in the first study there was a trend towards weight loss in the intervention group (−1.7 kg for intervention group v. control group), although this decrease failed to reach statistical significance. Table 3, however, shows that in the second study there was a statistically significant increase in mean body weight in the control group (+1.8 kg, \( P < 0.005 \)), a non-significant mean weight gain in the semi-skimmed-milk group (+0.9 kg), but no weight change in the breakfast-cereal group (+0.2 kg). The results for body weight in sedentary subjects were interesting. Sedentary subjects in both the control and the semi-skimmed-milk groups showed significant increases in weight (+1.95 kg, \( P < 0.05 \) and +3.3 kg, \( P < 0.005 \) respectively), but again there was no significant weight change in the breakfast-cereal group (−0.4 kg). This study provides some evidence that an increase in the consumption of high-carbohydrate foods, such as breakfast cereals, may be an effective approach for the prevention of weight gain.

The third study was a pilot study to investigate the effectiveness of a high-carbohydrate diet on weight maintenance following an initial period of weight loss. A sample of twenty-nine overweight or moderately obese men and women (mean BMI 30 (range 26–36) kg/m²) were asked to replace one main meal daily with a meal of breakfast cereal and semi-skimmed milk for a 2-week period. A significant mean weight loss of 2·0 kg (\( P < 0.001 \))

### Table 1. Changes in fat and starch intakes following increased consumption of breakfast cereals (by 60 g/d) in normal-weight young females (Kirk et al. 1997)

<table>
<thead>
<tr>
<th>Treatment group…</th>
<th>Intervention (n 26)</th>
<th>Control (n 22)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Week 4</td>
<td>Week 12</td>
</tr>
<tr>
<td>Energy (MJ/d)</td>
<td>+0·05</td>
<td>+0·10</td>
</tr>
</tbody>
</table>
| Percentage energy from:
| Starch           | +5·1***             | +4·7***       | −0·1                | +0·1          |
| Fat              | −5·4***             | −5·5***       | +1·1                | −1·4          |
| Protein          | +0·8*               | +0·6*         | +0·3                | −0·4          |

Mean values were significantly different from those for the control group (independent \( t \) test): * \( P < 0.05 \), *** \( P < 0.001 \).

### Table 2. Changes in fat and total carbohydrate intakes following increased consumption of breakfast cereals in overweight middle-aged men (Crombie & Kirk, 1999)

<table>
<thead>
<tr>
<th>Treatment group†…</th>
<th>Intervention 1</th>
<th>Intervention 2</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>21</td>
<td>21</td>
<td>21</td>
</tr>
<tr>
<td>Energy (MJ/d)</td>
<td>+0·20</td>
<td>−0·57</td>
<td>−0·61</td>
</tr>
</tbody>
</table>
| Percentage energy from:
| CHO               | +5·9*          | +0·6           | +1·5    |
| Fat               | −5·1***        | −0·8           | +0·8    |
| Protein           | +0·7           | −0·1           | +0·3    |

CHO, carbohydrate. Means values were significantly different from those for intervention group 2 and the control group (independent \( t \) test): * \( P < 0.05 \), *** \( P < 0.001 \).

† Intervention group 1 increased their cereal consumption by 90 g/d with semi-skimmed milk for 20 weeks. Intervention group 2 increased consumption of semi-skimmed milk only (300 ml/d). Controls maintained their habitual diet.

### Table 3. Changes in body weight (kg) at 20 weeks following increased consumption of breakfast cereals in overweight middle-aged men (Crombie & Kirk, 1999)

<table>
<thead>
<tr>
<th>Treatment group†…</th>
<th>Intervention 1</th>
<th>Intervention 2</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>21</td>
<td>21</td>
<td>21</td>
</tr>
<tr>
<td>All</td>
<td>+0·20</td>
<td>+0·9</td>
<td>+1·8*</td>
</tr>
<tr>
<td>Inactive</td>
<td>−0·4</td>
<td>+1·95*</td>
<td>+3·3***</td>
</tr>
<tr>
<td>n</td>
<td>9</td>
<td>10</td>
<td>8</td>
</tr>
</tbody>
</table>

Mean values were significantly different from those at baseline: * \( P < 0.05 \), *** \( P < 0.001 \).

† Intervention group 1 increased their cereal consumption by 90 g/d with semi-skimmed milk for 20 weeks. Intervention group 2 increased consumption of semi-skimmed milk only (300 ml/d). Controls maintained their habitual diet.
was achieved. For the next 4 weeks subjects were advised to increase their consumption of carbohydrate-rich foods, such as pasta, bread, rice and breakfast cereals, without energy restriction. At the end of this 4-week period, the initial weight loss had been maintained and mean body weight remained significantly lower (P < 0·001) than baseline. This study gives preliminary evidence that high-carbohydrate diets may be effective in maintenance of weight following initial weight loss. We are presently conducting further studies to investigate the effectiveness of health promotion strategies aimed at promoting increased consumption of carbohydrate-rich foods in general in achieving dietary fat reduction and improved body-weight control.

To conclude this section, the evidence suggests the following. Advice to reduce consumption of fatty foods may be appropriate for producing weight loss in the short to medium term, but is unlikely to be effective in the long term. Advice to increase consumption of carbohydrate-rich foods, on the other hand, may be more appropriate for the prevention of weight gain and for weight maintenance following weight loss, and is more likely to be effective in the long term. As long-term management of the obesity problem at the public health level must ultimately focus on obesity prevention rather than obesity treatment (James, 1995; World Health Organization, 1998), carbohydrate promotion should perhaps become the dominant approach.

Reducing consumption of sugar-containing foods

The role of sugar in body-weight control is controversial. It has been suggested that sweetness may stimulate eating (Geiselman & Norm, 1982; Blass, 1991), and sugar avoidance is commonly advised for weight loss. However, the actual evidence is sparse. Indeed, sugar may have a positive role to play in body-weight control for two reasons. First, sugar may facilitate an increase in carbohydrate : fat through the ‘sugar–fat see-saw’ effect. Numerous observational studies have shown an inverse relationship between percentage energy from fat and percentage energy from sugar, but not between percentage energy from fat and percentage energy from starch (for example, see Gibney, 1990; Bolton-Smith & Woodwood, 1994). In other words, there is a strong ‘sugar–fat see-saw’ but only a weak ‘starch–fat see-saw’. Thus, maintenance of sugar intake should help to depress fat intake. Conversely, a reduction in sugar intake may bring about a concomitant passive increase in starch. Thus, maintenance of sugar intake may reduce this dissatisfaction and improve compliance. For these reasons it should be easier to sustain a high carbohydrate : fat value when sugar consumption is not restricted.

To investigate the role of sugar in compliance to high-carbohydrate low-fat diets a study was carried out to compare compliance with advice to reduce both dietary fat and sugar, and compliance with advice to reduce fat whilst maintaining sugar intake (Drummond & Kirk, 1999). Ninety-three normal to moderately-overweight Scottish men were matched and randomly allocated to one of three groups. Group A was given advice to reduce both dietary fat and sugar and to increase starch intake. Group B was given advice to reduce dietary fat only and increase starch whilst allowing ad libitum sugar intake. Group C was asked to maintain their normal eating habits. Compliance was monitored at 6 weeks and 6 months.

Table 4 shows that group A (the low-fat low-sugar group) appeared to achieve significant reductions in both the percentage energy from fat and the percentage energy from sugar at 6 weeks. However, at 26 weeks, although percentage from fat remained significantly lower than at baseline, the earlier reduction in percentage energy from sugar was not sustained. The carbohydrate : fat value (calculated as percentage energy from carbohydrate : percentage energy from fat) appeared to increase significantly over baseline (20 % increase, P < 0·05) at 6 weeks, with a further slight increase (25 % increase, P < 0·05) at 6 months. The latter increase was achieved as the result of the restoration of sugar intake after 6 weeks. Group B (the low-fat ad libitum sugar group) also achieved a significant reduction in percentage energy from fat, at both 6 weeks and 26 weeks (both P < 0·05), whilst maintaining their intake of sugar. Note that this group achieved a substantial increase in carbohydrate : fat ratio, which at 6 months was significantly greater than that for both of the other groups (40 % increase, P < 0·005).

The results for energy intake and body weight in the low-fat sugar-restricted group (Table 5) are physiologically impossible. This group appeared to be in substantial energy deficit throughout the study. If this reported energy deficit had been valid, the group would have experienced a mean weight loss of about 8 kg over 6 months; however, this group showed no significant change in body weight. This finding suggests that the group were under-reporting or under-eating during the recording periods, perhaps in an attempt to eat, or appear to eat, a diet in line with the advice.

<table>
<thead>
<tr>
<th>Treatment group</th>
<th>Percentage energy as fat</th>
<th>Percentage energy as sugar</th>
<th>Change in CHO : fat (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n 6 weeks 26 weeks</td>
<td>6 weeks 26 weeks</td>
<td>6 weeks 26 weeks</td>
</tr>
<tr>
<td>Low-fat sugar-restricted</td>
<td>24 -4·8* -5·4*</td>
<td>-2·7* -1·8</td>
<td>+20* +25*</td>
</tr>
<tr>
<td>Low-fat sugar ad libitum</td>
<td>25 -8·0* -8·0*</td>
<td>+1·7 +0·8</td>
<td>+39*** +40***†</td>
</tr>
<tr>
<td>Control</td>
<td>25 -2·8 +1·4</td>
<td>+0·8 -1·0</td>
<td>-1 -6</td>
</tr>
</tbody>
</table>

Mean values were significantly different from that at baseline: * P < 0·05, ** P < 0·001. Mean value was significantly different from those of both the low-fat sugar-restricted group and the control group: † P < 0·005.
given. Thus, it is unlikely that the actual reductions in fat and sugar consumption were as great as the values shown in Table 4, i.e. the actual compliance was poorer. By contrast group B, the low-fat ad libitum-sugar group did experience a significant mean weight loss by 6 weeks, and most of this weight loss was maintained at 26 weeks. Maintenance of weight loss may well have been helped by good compliance and the high carbohydrate : fat ratio achieved in this group. This study provides evidence, first, that simultaneous reductions in dietary fat and sugar may be difficult to sustain in free-living subjects and, second, that sugar may have a positive role to play in body-weight control by improving compliance with high-carbohydrate low-fat diets. If this finding is valid, public health advice to reduce consumption of sugar-containing foods may not be appropriate for body-weight control, and may indeed be counter-productive.

Reducing snacking between meals

Avoidance of snacks and energy-containing drinks between meals is standard advice for body-weight control. Indeed, guidelines for obesity prevention in Scotland state: ‘Snacking and the loss of a formalized meal pattern reduce energy intake and body-weight changes (Drummond & Kirk, 1999). As under-reporting or under-eating in dietary surveys is more common in overweight subjects (Prentice et al. 1986; Black et al. 1993) and is possibly biased towards snacks (Livingstone et al. 1990; Heitmann & Lissner, 1995; Poppitt et al. 1995), inclusion of under-reports may create a biased negative association, or mask an actual positive association, between eating frequency and body weight. Furthermore, rather than being a cause of overweight, infrequent eating in overweight individuals may be a consequence of obesity due to post hoc reductions in eating frequency following weight gain in subjects attempting to lose weight or prevent further weight gain (Summerbell et al. 1996; PT Rodgers, personal communication). However, Table 6 includes four recent studies in which attempts were made to exclude under-reporters using cut-offs based on energy intake/BMR (Goldberg et al. 1991). Of these studies, two found an inverse relationship (Whybrow & Kirk, 1997; Drummond et al. 1998), and two studies found no relationship (Summerbell et al. 1996; Crawley & Summerbell, 1997).

To obtain more robust data, future observational studies need to identify dietary under-reporters and exclude these from the analysis, use standardized criteria for defining eating frequency so that results from different studies can be compared and, if possible, they should include physical activity assessments. Whilst caution is needed in interpreting the results of some observational studies, the evidence is adequate to hypothesize that frequent eating does not adversely affect body-weight status, and in some population subgroups may improve body-weight control. However, this hypothesis needs to be tested experimentally.

### Table 5. Compliance following advice to normal to moderately-overweight Scottish men to reduce dietary fat with and without sugar restriction; energy intake and body-weight changes (Drummond & Kirk, 1999)

<table>
<thead>
<tr>
<th>Treatment group</th>
<th>n</th>
<th>6 weeks</th>
<th></th>
<th>26 weeks</th>
<th></th>
<th>6 weeks</th>
<th></th>
<th>26 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low-fat sugar-restricted</td>
<td>24</td>
<td>0·30</td>
<td>0·5</td>
<td>0·31</td>
<td>0·5</td>
<td>0·6</td>
<td>−1·31*</td>
<td>0·5</td>
</tr>
<tr>
<td>Low-fat sugar ad libitum</td>
<td>25</td>
<td>−0·31</td>
<td>−0·6</td>
<td>−1·5</td>
<td>−1·78*</td>
<td>−0·77</td>
<td>−1·5***</td>
<td>−1·2**</td>
</tr>
<tr>
<td>Control</td>
<td>25</td>
<td>0·2</td>
<td>0</td>
<td>0·30</td>
<td>+0·2</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>

Mean values were significantly different from those at baseline: *P* < 0·05, ***P* < 0·001.

### Table 6. Observational studies examining the relationship between eating frequency and body-weight status

<table>
<thead>
<tr>
<th>Study</th>
<th>Relationship</th>
<th>Under-reporters excluded?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fáby et al. (1964)</td>
<td>-ve</td>
<td>✓</td>
</tr>
<tr>
<td>Metzner et al. (1977)</td>
<td>-ve</td>
<td>✓</td>
</tr>
<tr>
<td>Kant et al. (1995)</td>
<td>-ve</td>
<td>✕</td>
</tr>
<tr>
<td>Edelstein et al. (1992)</td>
<td>-ve</td>
<td>✓</td>
</tr>
<tr>
<td>Drummond et al. (1998)</td>
<td>-ve</td>
<td>✓</td>
</tr>
<tr>
<td>Whybrow &amp; Kirk (1997)</td>
<td>-ve</td>
<td>✓</td>
</tr>
<tr>
<td>Charzewskas et al. (1981)</td>
<td>None</td>
<td>✕</td>
</tr>
<tr>
<td>Dreon et al. (1988)</td>
<td>None</td>
<td>✕</td>
</tr>
<tr>
<td>Basdevant et al. (1993)</td>
<td>None</td>
<td>✕</td>
</tr>
<tr>
<td>Ruxton et al. (1996)</td>
<td>None</td>
<td>✓</td>
</tr>
<tr>
<td>Summerbell et al. (1996)</td>
<td>None</td>
<td>✓</td>
</tr>
<tr>
<td>Crawley &amp; Summerbell (1997)</td>
<td>None</td>
<td>✓</td>
</tr>
</tbody>
</table>

-ve, negative; ✕, under-reporters not excluded; ✓, under-reporters excluded.
The work of Booth (1988) is frequently cited as experimental evidence that snacking leads to over-consumption. Based on evidence from short-term laboratory-based studies, Booth (1988) concluded that snacks or high-energy drinks taken more than 1 h before meals fail to exert a satiety effect, so that energy compensation at the next meal does not occur, i.e. the energy from the snacks is consumed additionally. Whilst this conclusion may be valid for the short-term experimental situation, results from these studies have been extrapolated to predict that in the real-life situation sustained snacking will lead to chronic over-consumption and thus weight gain. However, it could be that in more natural situations individuals may compensate for sustained snacking through cognitive or physiological adaptation. Until recently, very little work has been done to examine the long-term effect of snacking in free-living subjects. I suggest that it is this type of study, rather than short-term laboratory-based studies, that should provide the basis for public health advice on body-weight control.

To investigate the effects of sustained eating on energy compensation in free-living subjects we have carried out two studies. In the first, a pilot study (Yates et al., 1997), ten lean young men who were habitual infrequent snackers were required to consume two high-carbohydrate high-fat snacks, each providing 0.88 MJ, one before lunch and the other before dinner. For the first 2 weeks five subjects ate the snack 0.5 h before meals, and the remainder ate the snack 1.5 h before meals. After a 1-week ‘washout period’ the regimes were reversed and the intervention continued for a further 2 weeks.

The two daily snacks did not displace existing eating occasions, as the mean eating frequency increased from 3.95 to 5.85 eating occasions daily. At the end of the snacking intervention partial energy compensation had occurred in both snacking groups. Energy compensation was achieved by subjects reducing the energy consumed at main meals (Fig. 1). In this study more complete compensation occurred at the evening meal than at lunch, possibly because lunch was eaten in the workplace, either purchased from the canteen or brought from home as pre-packed lunches, and with pre-determined portion sizes there was less scope for adjusting the size of the meal.

Interestingly, and contrary to what may be predicted from the Booth (1988) hypothesis, greater compensation (approximately 70% overall) occurred on the 1.5 h snacking regimen than on the 0.5 h snacking regimen (50% overall). This pilot study provides preliminary evidence that snacking intervention in the medium term resulted in partial energy compensation.

The second study set out to examine the effect on energy compensation and body weight of long-term snacking intervention (Kirk & Cursiter, 1999). Sixty-six physically-active adult males of normal body weight were allocated to either an intervention or control group. The intervention group was required to consume one high-carbohydrate moderately-high-fat snack (chocolate confectionery) containing 0.88 MJ in the afternoon on 6 d per week for 6 months. The control group was asked to maintain their normal diet. Table 7 displays body-weight data. If energy compensation had not occurred, i.e. the energy provided by the intervention snacks was consumed in addition to habitual energy intake, the intervention group would have experienced a mean weight gain of approximately 4–5 kg more than the control group. However, after 6 months of snacking intervention, there was no significant increase in body weight in the intervention group and no difference in weight change between intervention and control groups.

Subjects in the intervention group did not gain weight because complete energy compensation developed as snacking intervention progressed. Energy compensation occurred as a result of two separate responses which were equal in magnitude; first, the experimental snacking episode partially displaced existing snacking episodes, and second, subjects reduced mean energy consumed at main meals. These studies provide evidence that when snacking is sustained subjects may adapt to the increased snacking load by reducing energy consumed at other eating occasions so that weight gain does not occur.

**How could frequent eating help body-weight control?**

There are four possible physiological advantages associated with frequent eating that may improve body-weight control. First, frequent eating may help to control hunger and improve the accuracy of energy compensation. Burley et al. (1993) have shown that spreading energy intake over the day into five eating occasions (three meals and two snacks) rather than three meals resulted in a flatter profile of hunger throughout the day, so that hunger was less likely to build up before main meals, thus helping to prevent gorging at meals. Westerterp-Plantenga et al. (1994) have also shown that frequent eaters were better able to compensate for energy deficits and excesses by adjusting the size of subsequent meals. I suggest that one practical explanation for why frequent eaters may be more effective energy compensators than infrequent eaters is that they have the option of adjusting both snacking frequency and meal size in response to energy deficits or excesses, whereas infrequent eaters are more limited to adjusting meal size.

Second, frequent eating may help to increase dietary carbohydrate : fat ratio. A number of studies have shown...
that snacks and high-energy drinks, overall, tend to be higher in carbohydrate and lower in fat than main meals (Basdevant et al. 1993; Summerbell et al. 1995; Drummond et al. 1996). Thus, promotion of snacking may be one strategy to enable an increase in carbohydrate : fat of the diet.

Third, frequent eating is likely to shift the temporal distribution of energy intake away from the latter towards the earlier part of the day. Some studies have suggested that obese individuals tend to consume a high proportion of their daily energy intake in the evening (Beaudoin & Mayer, 1953; Machinot et al. 1975; Baekel et al. 1983; Bellisle et al. 1988; Fricker et al. 1990), as they tend to skip breakfast and daytime snacks and eat large evening meals. Furthermore, it has been suggested that energy consumed in the later half of the day may be more readily stored as fat than an isoenergetic amount consumed earlier. Thus, infrequent eating concentrated in the evening may be involved in weight gain (Fricker et al. 1990). This factor may be related to circadian changes in insulin secretion; insulin levels tending to be higher in the latter part of the day (Apfelbaum et al. 1972), resulting in greater uptake of fatty acids by adipocytes (Eckel & Yost, 1987).

Fourth, a pattern of eating ‘little and often’ may be more compatible with a physically-active lifestyle than a pattern of eating two or three large meals per d. There is evidence that athletes achieve a relatively high energy intake by eating frequently (Lindeman, 1990; Butterworth et al. 1994; Kirsch & von Ameln, 1998). There is less evidence for other groups, as few published studies investigating eating frequency have included estimates of physical activity. However, in a recent analysis of 1836 men and women aged 25–64 years from Glasgow, Scotland, a positive correlation between eating frequency and physical activity level was found (TR Kirk, F Jack and C Bolton-Smith, unpublished results). Thus, it is possible that in response to the high energy expenditure associated with a physically-active lifestyle the general population increase their energy intake by increasing their eating frequency rather than by eating larger meals. This strategy could avoid the gastric discomfort and lethargic mood which often follows the eating of infrequent large meals, and which reduces the motivation to exercise. If this supposition is correct, then current population advice to reduce snacking may be working against current population advice to adopt a physically-active lifestyle.

Whilst the balance of evidence suggests that snacking per se does not adversely affect, and sometimes may benefit, body-weight control, further research is clearly needed in order to provide information for food-product development initiatives and health promotion strategies. First, research is needed to investigate the effects on energy compensation of varying the snack composition and snacking pattern, e.g. by varying macronutrient composition, energy density and energy content of snacks, and by varying the timing of snack ingestion relative to meals. Second, research is needed to elucidate the interactions between eating frequency and physical activity. Third, research is needed to establish whether different population subgroups are likely to respond differently to changes in eating frequency, e.g. in relation to age, sex, body-weight status and level of dietary restraint. Preliminary evidence suggests that males are better compensators than females (Drummond et al., 1998), the young better than the old, the lean better than the overweight and the dietary unrestrained better than the restrained.

It may be the case that for some individuals (the minority) avoidance of snacking may be appropriate advice for body-weight control. Perhaps, for example, overweight middle-aged women with high dietary restraint scores are more likely to show poor compensation in response to increased snacking. For some individuals, however, a change from a habitual frequent-eating pattern to infrequent eating may adversely affect body-weight control by impairing ability for energy compensation and by reducing the likelihood of achieving a physically-active lifestyle. Moreover, it is possible that advice to restrict snacking could trigger dietary restrained behaviour in some individuals who previously showed healthy appetite control. The conclusion is that for most individuals advice to avoid snacking may not be appropriate for body-weight control, and indeed for some individuals may be counter-productive.

### Conclusions: public health advice for body-weight control

Our conclusions regarding the appropriateness of public health advice on food intake for body-weight control are:

1. For initial weight loss, advice focusing on reducing consumption of fatty foods may be appropriate; however, this advice is unlikely to result in dietary change which is sustainable in the long term;

2. To prevent weight gain, advice focusing on increasing consumption of carbohydrate-rich foods may be more appropriate; moreover, this advice is more likely to result in dietary change which is sustainable;

3. To achieve good compliance to high-carbohydrate low-fat diets, advice to reduce consumption of sugar-
containing foods may not be appropriate and may be counter-productive;

(4) to achieve good appetite control, advice to reduce snacking between meals may not be appropriate for most individuals, and for some individuals may be counter-productive.

What then is the most appropriate public health advice for body-weight control? We suggest that advice messages should be simple, positive and few in number. We consider that advice to increase the level of physical activity in the population is of paramount importance. Advice on food intake should, therefore, work synergistically with advice to adopt a physically-active lifestyle. It is thought that a high carbohydrate intake is beneficial for physical activity, and it is suggested that a ‘little and often’ eating pattern may help to sustain a physical-activity lifestyle. Thus, the most appropriate advice messages are perhaps threefold: increase physical activity; eat more carbohydrate; eat frequently.

References


© Nutrition Society 2000