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2nd Plenary Session on 'Body-weight balance and regulation'

Role of dietary carbohydrate and frequent eating in body-weight control

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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 longterm 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 (Δ body energy = energy intake – 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.

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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 bodyweight 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, 1996; Bell et al. 1998; Rolls et al. 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 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 freeliving 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 ($-1.7 \,\mathrm{kg}$ for intervention group ν . 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 ($+1.8 \,\mathrm{kg}$, P < 0.005), a non-significant mean weight gain in the semi-skimmed-milk group ($+0.9 \,\mathrm{kg}$), but no weight change in the breakfast-cereal group ($+0.2 \,\mathrm{kg}$). 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 ($+1.95 \,\mathrm{kg}$, P < 0.05 and $+3.3 \,\mathrm{kg}$, 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 $2.0 \, \text{kg}$ (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)

Treatment	Intervent	ion (<i>n</i> 26)	Control (n 22)		
group	Week 4	Week 12	Week 4	Week 12	
Energy (MJ/d) Percentage energy from:	+0.05	+0-10	+0.43	+0.26	
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 middleaged men (Crombie & Kirk, 1999)

Treatment group† n	Intervention 1	Intervention 2	Control
	21	21	21
Energy (MJ/d) Percentage energy from:	+0.20	-0.57	-0.61
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.

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

Treatment group†	Intervention 1	Intervention 2	Control	
All	+0.20	+0.9	+1.8*	
n	21	21	21	
Inactive	-0.4	+1.95*	+3.3***	
n	9	10	8	

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 semiskimmed milk for 20 weeks. Intervention group 2 increased consumption of semi-skimmed milk only (300 ml/d). Controls maintained their habitual diet.

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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 fat intake (Naismith & Rhodes, 1995). Second, sugar may improve the palatability of low-fat diets enabling better long-term compliance. Attempts to follow weight-reducing

diets often fail as individuals lose the discipline required to avoid eating palatable foods they enjoy. Allowing 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 4. Compliance following advice to normal to moderately-overweight Scottish men to reduce dietary fat with and without sugar restriction; changes in percentage energy from fat and sugar (Drummond & Kirk, 1999)

		Percentage energy as fat		Percentage energy as sugar		Change in CHO: fat (%)	
Treatment group	n	6 weeks	26 weeks	6 weeks	26 weeks	6 weeks	26 weeks
Low-fat sugar-restricted	24	-4.8*	-5.4*	-2.7*	-1.8	+20*	+25*
Low-fat sugar ad libitum	25	-8.0*	-8.0*	+1.7	+0.8	+39***	+40***†
Control	25	-2.8	+1.4	+0.8	-1.0	-1	-6

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.

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)

Treatment group		Energ	gy (MJ)	Body weight (kg)		
	n	6 weeks	26 weeks	6 weeks	26 weeks	
Low-fat sugar-restricted	24	−1·78*	-1.31*	-0.6	-0.5	
Low-fat sugar <i>ad libitum</i>	25	−1. 5	-0.77	−1·5***	-1.2***	
Control	25	-0.31	-0.30	+0.2	0	

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

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 bodyweight 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 the conscious recognition of foods eaten. This appreciation is needed by people anxious to limit their intake. Snacking may also bypass short-term satiety signals' (Scottish Intercollegiate Guidelines Network, 1996). These guidelines assume that snacking leads to over-consumption and thus contribute to weight gain. However, the evidence for a role of frequent eating (the terms 'eating frequency' and 'snacking frequency' are used in the present paper synonymously; evidence suggests that when individuals increase their eating frequency they do so by increasing snacking frequency whilst maintaining the number of meals per d; Whybrow & Kirk, 1997) in the cause of obesity is equivocal. Evidence comes from observational studies and experimental studies.

Observational studies that have examined the relationship between frequency of eating and body weight have either failed to find a significant relationship, or have found an inverse relationship. No studies, to our knowledge, have found a positive relationship. In the twelve studies reviewed for the present paper, six studies found an inverse relationship, and six papers found no relationship between eating frequency and body-weight status (Table 6).

Some of these studies, particularly the earlier ones, have been criticized on the grounds that dietary under-reporting and *post hoc* changes in eating frequency following weight gain were not taken into account in interpreting the results (Lichtman *et al.* 1992; Summerbell *et al.* 1996; Bellisle

et al. 1997). 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 6. Observational studies examining the relationship between eating frequency and body-weight status

Study	Relationship	Under-reporters excluded?
Fábry <i>et al.</i> (1964)	-ve	×
Metzner et al. (1977)	-ve	X
Kant et al. (1995)	-ve	X
Edelstein et al. (1992)	-ve	X
Drummond et al. (1998)	-ve	✓
Whybrow & Kirk (1997)	-ve	✓
Charzewska et al. (1981)	None	X
Dreon et al. (1988)	None	X
Basdevant et al. (1993)	None	X
Ruxton et al. (1996)	None	X
Summerbell et al. (1996)	None	✓
Crawley & Summerbell (1997)	None	✓

-ve, negative; X, 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 using preload designs, 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 overconsumption 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 snacking 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 regimens 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

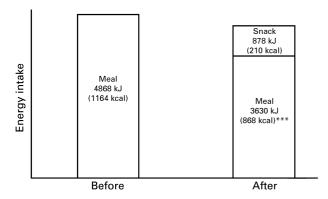


Fig. 1. Adjustment of energy intake at evening meals following 2 weeks of snacking. A 878 kJ (210 kcal) snack was given before main meals (lunch and evening meal) for 14 d, either 30 or 90 min before the meal. Mean value was significantly different from that for energy intake at the meal before the 2-week period of snacking: ***P < 0.001. (From Yates *et al.* 1997.)

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 $1.5\,\mathrm{h}$ snacking regimen than on the $0.5\,\mathrm{h}$ 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 moderatelyhigh-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

Table 7. Body-weight (kg) changes in sixty-six physically-active adult males of normal body weight following sustained snacking intervention (Kirk & Cursiter, 1999)

(Mean values and standard deviations)

Baseline		3 months		6 months			
Mean	SD	Mean	SD	Mean	SD	Change at 6 months	
78·5	11·2	79·2	11·4 8·5	79.0 76.6	11·2	+0·5 +0·5	
		78.5 11.2	78.5 11.2 79.2	78.5 11.2 79.2 11.4	78.5 11.2 79.2 11.4 79.0	78.5 11.2 79.2 11.4 79.0 11.2	

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; Baeke 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 counterproductive.

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

- Acheson KJ, Schutz Y, Bessard T, Anantharaman K, Flatt JP & Jequier E (1988) Glycogen storage capacity and de novo lipogenesis during massive carbohydrate overfeeding in man. *American Journal of Clinical Nutrition* **48**, 240–247.
- Apfelbaum AS, Reinberg A, Assan R & Lacatis D (1972) Hormonal and metabolic circadian rhythms before and during a low-protein diet. *Israel Journal of Medical Science* **8**, 867–873.
- Astrup A, Buemann B, Toubro S & Raben A (1996) Defects in substrate oxidation involved in the predisposition to obesity. *Proceedings of the Nutrition Society* **55**, 817–828.
- Astrup A, Raben A, Buemann B & Toubro S (1997) Fat metabolism in the predisposition to obesity. *Annals of the New York Academy of Sciences* **827**, 417–430.
- Astrup A, Ryan L, Grundwald G, Storgaard M, Saris W, Melanson E & Hill WO (2000) The role of dietary fat in body fatness: evidence from a preliminary meta-analysis of *ad libitum* low-fat dietary intervention studies. *British Journal of Nutrition* (In the Press).
- Baecke JA, van Staveren WA & Burema J (1983) Food consumption, habitual physical activity, and body fatness in young Dutch adults. *American Journal of Clinical Nutrition* 37, 278–286.
- Basdevant A, Craplet C & Gut-Grand B (1993) Snacking patterns in obese French women. *Appetite* **21**, 17–23.
- Beaudoin R & Mayer J (1953) Food intake of obese and non-obese women. *Journal of the American Dietetic Association* **29**, 29–33.
- Bell EA, Castellanos VH, Pelkman CL, Thorwart ML & Rolls BJ (1998) Energy density of foods affects energy intake in normal-weight women. *American Journal of Clinical Nutrition* 67, 412–420.
- Bellisle F, McDevitt R & Prentice AM (1997) Meal frequency and energy balance. *British Journal of Nutrition* **77**, S57–S70.
- Bellisle F, Rolland-Cachera MF, Deheeger M & Guilloud-Bataille M (1988) Obesity and food intake in children: evidence for a role of metabolic and/or behavioral daily rhythms. *Appetite* 11, 111–118.
- Black AE, Prentice AM, Goldberg GR, Jebb SA, Bingham SA, Livingstone MBE & Coward WA (1993) Measurements of total energy expenditure provide insights into the validity of dietary measurements of energy intake. *Journal of the American Dietetic Association* **93**, 572–578.
- Blass EM (1991) Suckling: Opiod and non-opiod processes in mother infant bonding. In *Chemical Senses*. vol. 4, *Appetite and*

- *Nutrition*, pp. 283–302 [MI Friedman, MG Tordoff and MR Kare, editors]. New York: Marcel Dekker Inc.
- Bolton-Smith C & Woodward M (1994) Dietary composition and fat to sugar ratios in relation to obesity. *International Journal of Obesity* **18**, 820–828.
- Booth DA (1988) Mechanisms from models actual effects from real life: The zero-calorie drink-break option. *Appetite* **11**, 94–102.
- Burley VJ, Cotton JR, Weststrate JA & Blundell JE (1993) Effect on appetite of replacing natural fat with sucrose polyester in meals or snacks across one whole day. In *Obesity in Europe*, pp. 213–219 [H Ditschuneit, FA Gries, H Hauner, V Schusdziarra and JG Wechsler, editors]. London: John Libbey & Co.
- Butterworth DE, Nieman DC, Butler JV & Herring JL (1994) Food intake patterns of marathon runners. *International Journal of Sport Nutrition* **4**, 1–7.
- Charzewska J, Kulesza W, Brzezinska J & Chwojnowska Z (1981) Relationship between obesity or overweight development and the frequency of meals, their distribution during the day and consumption of atherogenic food products. *Zywienie Czlowieka* 8, 217–227.
- Crawley H & Summerbell CD (1997) Feeding frequency and BMI among teenagers aged 16–17 years. *International Journal of Obesity* **21**, 159–161.
- Crombie N & Kirk TR (1999) Prevention of weight gain and blood cholesterol reduction after consumption of a high carbohydrate food in men. *International Journal of Obesity* **23**, Suppl. 5, 657.
- Dreon DM, Frey-Hewitt B, Ellsworth N, Williams PT, Terry RB & Wood PD (1988) Dietary fat: carbohydrate ratio and obesity in middle-aged men. *American Journal of Clinical Nutrition* **47**, 995–1000.
- Drummond S, Crombie NE, Cursiter MC & Kirk TR (1998) Evidence that eating frequency is inversely related to body weight status in male, but not female, non-obese adults reporting valid dietary intakes. *International Journal of Obesity* 22, 105-122
- Drummond S, Crombie NE & Kirk TR (1996) A critique of the effects of snacking on weight status. *European Journal of Clinical Nutrition* **50**, 779–783.
- Drummond S & Kirk TR (1999) Assessment of advice to reduce dietary fat and non-milk extrinsic sugar in adult males in a free-living population. *Public Health Nutrition* **2**, 187–197.
- Eckel RH & Yost TJ (1987) Weight reduction increases adipose tissue lipoprotein lipase responsiveness in obese women. *Journal of Clinical Investigation* **80**, 992–997.
- Edelstein SL, Barrett-Connor EL, Wingard DL & Cohn BA (1992) Increased meal frequency associated with decreased cholesterol concentrations; Rancho Bernardo, CA, 1984–1987. *American Journal of Clinical Nutrition* **55**, 664–669.
- Fábry P, Fodor J, Hejl Z, Braun T & Zvolankova K (1964) The frequency of meals. Its relation to overweight, hypercholesterolaemia, and decreased glucose-tolerance. *Lancet* ii, 614–615.
- Flatt JP (1978) The biochemistry of energy expenditure. In *Recent Advances in Obesity Research*, vol. 2, pp. 211–228 [GD Bray, editor]. London: Newman Publishing.
- Fricker J, Giroux S, Fumeron F & Apfelbaum M (1990) Circadian rhythm of energy intake and corpulence status in adults. *International Journal of Obesity* **14**, 387–393.
- Friedman MI (1991) Metabolic control of caloric intake. In *Chemical Senses*. vol. 4, *Appetite and Nutrition*, pp. 19–38 [MI Friedman, MG Tordoff and MR Kare, editors]. New York: Marcel Dekker Inc.
- Geiselman PJ & Norm D (1982) Sugar infusion can enhance feeding. *Science* **218**, 490–491.
- Gibney MJ (1990) Dietary guidelines: a critical appraisal. *Journal of Human Nutrition and Dietetics* **6**, 13–22.

- Goldberg GG, Black AE, Jebb SA, Cole PR, Murgatroyd PR, Coward WA & Prentice AM (1991) Critical evaluation of energy intake data using fundamental principles of energy physiology: 1, derivation of cut-off limits to identify underrecording. European Journal of Clinical Nutrition 45, 569–581.
- Heitmann BL & Lissner L (1995) Dietary underreporting by obese individuals—is it specific or non-specific? *British Medical Journal* **311**, 986–989.
- Heitmann BL, Lissner L, Srensen TI & Bengtsson C (1995) Dietary fat intake and weight gain in women genetically predisposed for obesity. *American Journal of Clinical Nutrition* **61**, 1213–1217.
- Hellerstein MK (1999) De novo lipogenesis in humans: metabolic and regulatory aspects. *Europeam Journal of Clinical Nutrition* **53**, Suppl. 1, 553–565.
- Hochbaum GM (1981) Strategies and their rationale for changing people's eating habits. *Journal of Nutrition Education* **13**, Suppl. 1, S59–S65.
- Horton TJ, Drougas H, Brachey A, Reed GW, Peters JC & Hill JO (1995) Fat and carbohydrate overfeeding in humans: different effects on energy storage. *American Journal of Clinical Nutrition* **62**, 19–29.
- James WPT (1995) Public health approach to the problem of obesity. *International Journal of Obesity* 19, S37–S45.
- Jeffery RW, Hellerstedt WL, French SA & Baxter JE (1995) A randomized trial of counseling for fat restriction versus calorie restriction in the treatment of obesity. *International Journal of Obesity* 19, 132–137.
- Kant AK, Schatzkin A, Graubard BI & Ballard Barbash R (1995)
 Frequency of eating occasions and weight change in the NHANES I Epidemiological Follow-up Study. *International Journal of Obesity and Related Metabolic Disorders* 9, 468–474.
- Kirk TR, Burkill S & Cursiter MC (1997) Dietary fat reduction achieved by increasing consumption of a starchy food an intervention study. *European Journal of Clinical Nutrition* **51**, 455–461.
- Kirk TR & Cursiter MC (1999) Long-term snacking intervention did not lead to weight gain in free-living man. *Scandinavian Journal of Nutrition* **2**, Suppl. 34, 3–17.
- Kirsch KA & von Ameln H (1998) Feeding patterns of endurance athletes. *European Journal of Applied Physiology* **47**, 197–208.
- Lichtman SW, Pisarka K, Berman ER, Pestone M, Dowling H, Offenbacher E, Weisel H, Heshka S & Matthews DE (1992) Discrepancy between self-reported and actual caloric increase and exercise in obese subjects. *New England Journal of Medicine* **327**, 1393–1398.
- Lindeman AK (1990) Eating and training habits of triathletes: A balancing act. *Journal of the American Dietetic Association* 90, 993–995.
- Lissner L, Heitmann BL & Bengtsson C (1997) Low-fat diets may prevent weight gain in sedentary women: prospective observations from the population study of women in Gothenburg, Sweden. *Obesity Research* **5**, 43–48.
- Livingstone MBE, Prentice AM, Strain JJ, Coward WA, Black AE, Barker ME, McKenna PG & Whitehead RG (1990) Accuracy of weighed dietary records in studies of diet and health. *British Medical Journal* 300, 708–712.
- Lyon XH, Di Vetta V, Milon H, Jequier E & Schutz Y (1995) Compliance to dietary advice directed towards increasing the carbohydrate to fat ratio of the everyday diet. *International Journal of Obesity* **19**, 260–269.
- Machinot S, Mimouni M & Lestradet H (1975) L'alimentation spontanée de l'enfant obese au moment de la premiere consultation (Spontaneous diet of the obese child at the time of first consultation). *Cahiers de Nutrition et de Dietetique* 1, 45–46.

- Mayer J (1955) Regulation of energy intake and the body weight. Annals of the New York Academy of Sciences 63, 15–43.
- Mela DJ (1994) The intake and acceptance of fat in the diet. *Nutrition and Food Science* **3**, 19–21.
- Metzner HL, Lamphiear DE, Wheeler NC & Larkin FA (1977) The relationship between frequency of eating and adiposity in adult men and women in the Tecumseh Community Health Study. *American Journal of Clinical Nutrition* 30, 712–715
- Naismith DJ & Rhodes C (1995) Adjustment in energy intake following covert removal of sugar from the diet. *Journal of Human Nutrition and Dietetics* **8**, 167–175.
- Neilson J & Larson-Brown LD (1990) College student's perception of nutrition messages: How motivating are they? *Journal of Nutrition Education* **22**, 30–34.
- Poppitt SD & 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, Swann D, Black AE & Prentice AM (1995) Is underreporting of energy intake in obese women macronutrient specific? Covert measurements in a metabolic facility. *International Journal of Obesity* **29**, Suppl. 2, 29.
- Prentice AM, Black AE, Coward WA, Davies HL, Goldberg GR, Murgatroyd PR, Ashford J, Sawyer M & Whitehead RG (1986) High levels of energy expenditure in obese women. *British Medical Journal* **292**, 983–987.
- Rolls BJ, Castellanos VH, Halford JC, Kilara A, Panyam D, Pelkman CL, Smith GP & Thorwart ML (1998) Volume of food consumed affects satiety in men. *American Journal of Clinical Nutrition* **67**, 1170–1177.
- Ruxton CHS, Kirk TR & Belton NR (1996) The contribution of specific dietary patterns to energy and nutrient intakes in 7–8year-old Scottish schoolchildren. III. Snacking habits. *Journal of Human Nutrition and Dietetics* 9, 23–31.
- Schlundt DG, Hill JO, Pope-Cordle J, Arnold D, Virts KL & Katahn M (1993) Randomized evaluation of a low fat ad libitum carbohydrate diet for weight reduction. *International Journal of Obesity* **17**, 623–629.
- Scottish Intercollegiate Guidelines Network (1996) *Obesity in Scotland. Integrating Prevention with Weight Management.* Edinburgh: Scottish Intercollegiate Guidelines Network.
- Shah M, McGovern P, French S & Baxter J (1994) Comparison of a low-fat, ad libitum complex-carbohydrate diet with a low-energy diet in moderately obese women. *American Journal* of Clinical Nutrition **59**, 980–984.
- Summerbell CD, Moody RC, Shanks J, Stock MJ & Geissler C (1995) Sources of energy from meals versus snacks in 220 people in four age groups. *European Journal of Clinical Nutrition* **49**, 33–41.
- Summerbell CD, Moody RC, Shanks J, Stock MJ & Geissler C (1996) Relationship between feeding pattern and body mass index in 220 free-living people in four age groups. *European Journal of Clinical Nutrition* **50**, 513–519.
- Van Assema P, Kempers B, Brug J & Glanz K (1999) An exploratory study into Dutch consumers' experiences with implementing recommendations to reduce fat intake. *Journal of Human Nutrition and Dietetics* **12**, 103–112.
- Van Itallie TB (1990) The Glucostatic theory 1953–1988: Roots and branches. *International Journal of Obesity* **14**, 1–10.
- Westerterp-Plantenga MS, Wijckmans-Duysens NA & ten Hoor F (1994) Food intake in the daily environment after energyreduced lunch, related to habitual meal frequency. Appetite 22, 173–182.
- Whybrow S & Kirk TR (1997) Nutrient intakes and snacking frequency in female students. *Journal of Human Nutrition and Dietetics* **10**, 237–244.

World Health Organization (1998) Obesity. Preventing and Managing the Global Epidemic. Geneva: WHO.

Yates H, Crombie NE & Kirk TR (1997) Evidence of energy intake compensation at meals after snacking intervention – a pilot study. *International Journal of Obesity* **21**, S113.

Yu-Poth S, Zhao G, Etherton T, Naglak M, Jonnalagadda S & Kris-Etherton PM (1999) Effects of the National Cholesterol Education Program's step I and step II dietary intervention programs on cardiovascular disease factors: A meta-analysis. American Journal of Clinical Nutrition 69, 632–646.

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