



Evidence-based Reduction of Obesity: Identification of a Subculture's Least Fattening Eating Patterns

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Public health nutritional interventions, like clinical treatments in medicine or psychology, should be based on direct evidence of their efficacy relative to prior existing modalities. Yet the contribution of applied human nutrition and the psychology of eating to research into ways of slowing the rise of obesity has been limited to the intake of energy nutrients and investigator-prejudged questions about appetite and food choices. In contrast, it is feasible to get members of the public to describe their patterns of eating in their own words. Self-assessed current frequency of each common potentially less or more fattening eating pattern can then be used to associate weight change with changes in habit frequency. This design has been used to identify the long-term least fattening eating patterns retrospectively and prospectively in several studies in England and Germany. Without using assessments of usual nutrient intake, these studies demonstrate that the least fattening patterns over 1–2 or more years are drinks between meals with no energy intake, cutting back on fat in all foods and replacing as much sitting and riding as feasible each day with walking or cycling. An hypothesis to be tested by this method is that the only known automatic mechanism for eating less dessert is the learnt effect of boosting the readily digested carbohydrate content of high-energy main courses. Such evidence-based advice as well as self-therapy for emotional eating and body-shape distress should be the front line of defence against unhealthy weight gain and re-gain.

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EFFICACY OF INTERVENTIONS TO REDUCE OBESITY

No established intervention has been shown by controlled evaluation to be generally successful at reducing obesity in adults. Dietary advice, behaviour modification for eating and exercise, drug treatment and combinations of such individual therapies, all fail to prevent group average regain of lost weight to the original or control levels or above within a year or so after the end of professional involvement. Mass educational interventions and consumer product labelling and regulation for eating, drinking and physical activity have similarly been found when properly evaluated to have no long-term impact on obesity, with the exception of parent-involved promotion of an active lifestyle in school children (Epstein *et al.*, 1994): the impact in adulthood is as yet unknown.

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Substantial amounts of weight may be lost on average across groups during clinical or public health interventions but the group mean weight always begins to increase when professional involvement ceases. When some clinical treatment is prolonged, weight still generally increases by a year after the end of the maintenance phase. These facts invalidate the usual criterion of successful outcome—a lower weight at 12 months than before intervention or than in a control group: such a time-point is on a rising curve and so provides no evidence that the intervention reduced the risks to health from the fat content of the body. Indeed, residual weight loss at merely 12 months is consistent with greater obesity ultimately than originally or in the control group: the intervention could in fact be pathogenic.

That is, the only sound evidence for reduction in obesity (health-risking fatness) from group data is zero average weight gain between two time points after the end of intervention (and no drop-outs either). There appears to be only one report of weight-control related education and therapy with such an outcome (Lewis *et al.*, 1992). That intervention included the long-established cognitive therapies for the large minority suffering from emotional eating and shape distress, also applied in this context by Rosen *et al.* (1995). Another key element, however, applies to all weight control and is the concern of the present paper: this was a briefing on the research results identifying the specific habits of eating and movement which are least and most fattening, as described by ordinary people from the same population (Blair *et al.*, 1989; Booth, 1989, 1994, 1996). The unprecedented outcome of continued weight loss on average and by nearly all individuals in the group in Lewis *et al.*'s (1992) study awaits a fuller evaluation: the follow-up was limited to 6 months after the end of professional intervention; also there were no inter-individually controlled comparisons by path analysis or other standard methods in applied social psychology (Blair *et al.*, 1989).

Therefore the educational, preventive and therapeutic measures currently being advocated in order to slow the rise of obesity in industrialized regions of the world lack a sound scientific basis. The reason for this extraordinary state of affairs for a part of the medical and social sciences is that obesity research and the sciences of nutrition and exercise have neglected the immediate practical implications of the elementary thermodynamics of energy exchange between the human body and the environment (Booth, 1978, 1980; Garrow, 1974, 1981). The only way to avoid an unhealthy excess of fat on the body is for the individual to ingest no more energy than he or she expends on average over the months and decades of adulthood. This basic fact of “fat physics” applies to initial fattening, such as when a person becomes more sedentary without changing their eating habits effectively, or to the regain of weight after a course of dietetic, psychological and/or pharmaceutical treatment or responding to public health education and regulation (Gill, 1997), again because nobody knows which of their own permanently sustainable food choices is least fattening.

It is therefore pointless to focus dietary advice on weight reduction on patients who seem to be motivated to contemplate changing their behaviour towards food. This strategy may be helpful in getting individuals to stop smoking (Borland *et al.*, 1994) but nobody can stop eating or even cut out a lot of common foods for the rest of life. It is no use wanting to lose weight if nobody can tell you what are the feasible less fattening eating patterns which are feasible in your subculture for the rest of the period of your life when your energy expenditure is at or below your current level. No controlled trial of dietetic treatment procedures has shown them

to be effective at reducing weight in the long term and so there is no evidence that advice on food choices to accord with dietary guidelines based on nutrient epidemiology are of help in obesity. Indeed, the conviction that one can follow the prescribed diet is unproductive even of short-term weight loss. Rather, what predicts weight loss maintained for at least a year is the belief that one knows feasible ways of changing eating and movement habits permanently in order to lose weight and to keep it off (Blair *et al.*, 1989)—weight control self-efficacy, not eating control self-efficacy.

There is no sense either in teaching new patterns of eating based on generic principles of behavioural therapy (e.g. stimulus control) when there is no evidence for long-term efficacy of such modifications of those particular aspects of eating or movement behaviour (Booth, 1978, 1980). Indeed, the combination of diet formula or foods and behaviour modification is known to be ineffective at keeping weight off at all after 5 years (Wadden *et al.*, 1989). Hence, these large investments in psychotherapists' time have no impact on the disease risks from prolonged overweight.

Treatment of obesity with drugs, hormones or food additives, whether or not in combination with dietary advice and psychotherapy, will always be vitiated by the same fallacy. Even if a safe intake-suppressing and/or metabolism-accelerating drug were taken for the rest of life, the user would still have to balance the energy intake and expenditure in their remaining habits of eating, drinking and movement. Yet the lifelong feasible less-fattening patterns in a patient's culture and lifestyle have yet to be identified by evidence for efficacy in weight-loss maintenance. Therefore, however well we understand the normal neural mechanisms of energy balance regulation through which leptin and other agents must be working (Booth, 1978; Campfield, 1996), we will not be able to prevent people who move less without eating as much less from (re-)gaining weight during prolonged drug therapy or after somatic gene engineering until we can identify and support less fattening dietary and activity patterns against the more fattening ones in each subculture that differs from others.

IDENTIFICATION OF EATING PATTERNS THAT KEEP WEIGHT OFF

A number of studies have tried to identify the food choices which promote maintenance of weight loss, using generic questions posed in the investigators' own terms, recall of the foods consumed in the previous 24 h or of the frequencies with which foods have been eaten recently, or records of weights or household measures of foods as they are eaten. However, such methods cannot identify eating patterns as the eaters construe them. This is crucial not just for subsequent communication of advice but also for identification of eating patterns as they are organized within the individual's own mind. It is these cognitive structures which determine the usually ingested combinations of nutrient (food recipes and menus), independently of biases from recording or recall.

The Zero-calorie Drink Break

The importance of not imposing the investigator's categories was brought home to us by the first study to relate a dietary health behaviour questionnaire to weight loss history (Booth, 1994; Lewis & Booth, 1986). When we asked English dieters about

what we described as “cutting back on fat intake”, there was evidence of association of that intentional pattern with weight loss maintained since the last episode of dieting. So our method was sensitive to the greater energy efficiency of fat deposition from dietary fat relative to dietary carbohydrate. Yet “cutting back on snacks” was not associated with weight loss or maintenance. A “snack” however is a very ambiguous term in England: it can mean anything eaten between mealtimes (which was our main interest), a small complex meal eaten at any time of day, or the eating of “snack foods” at mealtimes or between meals, at or away from a drink occasion.

It was therefore surprising recently to see the provision of snackfoods for 25% of daily energy intake called “snacking” in England. “Snacking” may have a more consistent meaning in California, since it was the only retrospective response associated with poor maintenance of weight loss at 2 years after dietary treatment by Kayman *et al.* (1990).

The word(-part) “snack” was therefore avoided in the first study using questions based on culturally specific descriptions of eating and movement habits. The hypothesis had been developed from research on satiety mechanisms (Booth, 1978) that regular ingestion of even small amounts of any energy nutrient in or with drinks between meals would be especially fattening (Booth, 1988). “Cutting back on calories between meals” was the practice prospectively most strongly associated with maintenance of weight loss for 12 months or more (Blair *et al.*, 1989; Booth, 1996). Avoiding “sweet things” was also one of the least fattening habits, whose frequency was correlated with that of cutting calories between meals, presumably because many such calorific drinks and nibbles taste sweet. This result has since been extended to German culture in a longer-term but retrospective study (Westenhöfer *et al.*, 1997). Both studies also confirmed the strongly fattening effects of foods which are thought of as containing fat; since this was between meals as well as at meals, no difference in satiety effects is likely to be involved (cp. Dibsall *et al.*, 1996).

Fast-digested First Courses

However, the 27 habit self-characterizations selected for the Blair *et al.* (1989) study did not differentiate between early and later parts of meals. A second hypothesis (Booth, 1988) that therefore remains to be tested by this method concerns a learnt effect of rapidly digested components of main-course recipes on amounts of dessert selected from the same menu—namely, the associative conditioning of lack of appetite to concurrent gastric distension and dessert type. This is the only known mechanism for non-deliberate reduction in the amount of food at a meal (Booth *et al.*, 1976, 1982, 1994). Boosting the readily digested carbohydrate content of main courses trains the appetite to want less of whatever dessert is on such a meal menu.

Nearly all research on influences on meal size is reported and interpreted in terms of the meal’s energy content. Yet psychologists showed 25 years ago that meal sizes are controlled by sensed volume (Booth, 1972; Pudal, 1976; Wooley, 1972). Thus, contrary to the conclusion that fat is a weak satiator, high energy-density foods cause active overconsumption of energy by means of the meal-volume control mechanisms, as Poppitt and Prentice (1996) have now concluded. However, from the above mechanism the prediction is that this fattening effect comes from high-energy desserts and could be countered by high energy density in an earlier course, so long as it were rapidly digested enough to produce a slight oversatiation immediately after the dessert (Booth *et al.*, 1982).

Our current research on less fattening habits therefore focuses on self-characterizations that distinguish between recipes in earlier and later stages of meals.

IMPLEMENTING EVIDENCE-BASED PREVENTION OF UNHEALTHY WEIGHT GAIN

After almost a decade, this evidence-based approach to obesity-preventing eating and movement patterns still has not been put out to the necessary individually controlled trials of efficacy. Instead, the research community holds to paradoxical positions founded on self-stultifying professional traditions. For example, evaluations of implementations are not considered unless they compare randomized groups. Yet these group trials have been shown to be insensitive at best, or even invalid, for interventions that depend on behaviour, like changes in eating and activity patterns, or have conscious effects such as food choices, visceral sensations and other people's reactions.

Another example is the recent symposium on temporal patterning of eating in relation to obesity-related diseases where it was incorrectly stated that there was no evidence for the proposal that calorific drink breaks between meals are particularly fattening. The symposium followed the tradition of considering only intake records, despite the fact that these are distorted by the extra attention paid to choices of foods and amounts, probably more than recall of spontaneous eating patterns. It is contradictory to require evidence on weight-reducing eating patterns to include dietary assessment of nutrient intake when the doubly labelled water technique has abundantly confirmed the original evidence that overweight people underreport energy intake (Garrow, 1974).

The total daily nutrient intakes and average eating frequencies extracted from standard dietary assessment result from long successions of richly appreciated food choices. Therefore it is scientifically far more realistic to let people say what they are doing with foods at different moments, to measure the cognitive biases in their recall of episodes and frequencies, to relate such self-characterized behavioural reports to weight change and then to inform the populace of these research findings in the terms that they can apply, as overweight people are adequately motivated to do. The biopsychological mechanisms by which self-described eating patterns have their effects on the development and prevention of obesity can be understood by undertaking more basic research to relate the sociopsychological conceptualizations of the everyday food choices during and between meals to the accumulation over successive days of the episodes of energy and nutrient ingestion (e.g. Dibsall *et al.*, 1996).

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