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Short communication

Satiety. No way to slim

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ABSTRACT

This short overview considers a prospect that claims to boost satiety are used to prescribe or sell materials to dieters that do not slow their daily rate of energy intake, thereby worsening their problems with body weight and even perhaps increasing the prevalence of obesity. Implying that a drug or a food contributes to weight control by providing extra satiety is a mistake in two ways. First, the notion of a hormone analogue or a food constituent having a specifiable satiating power is scientifically incoherent. Secondly, a slimming satiety is a particular pattern of eating and drinking, in which substances have no fixed roles. Such a dietary custom has to be shown to produce a larger step decrease in weight with the medication or food product than without it. Suppression of food intake at a usual time for eating does not imply reduction in the eater's total intake of energy in a calendar period and hence lower weight while the material is still used within that eating pattern. It is the maintained pattern of behaviour that slims and prevents regain, not a satiety-augmenting substance. Regulators should not allow incomprehension of the basic science of energy balance to be exploited by advocacy of a food or medication for "satiety" believed by consumers to be a means of avoiding unhealthy fatness.

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The issue: satiety as a slimming claim

Vigorous attempts are being made to obtain regulatory approval for products claiming to help reduction of weight by satiating appetite for food. There are increasing numbers of reviews, books and grants on satiety as an aid to slimming.[1] Yet, on scientific principle, such a general implication can never be true.

Definitions and measurements of satiety are beside the point. Two widely neglected facts are fundamental to the control of body weight.

The first is that average rates, not cumulative amounts, of energy intake and expenditure determine how much weight is lost or gained. This is the physics of fatness, an inescapable fact of the thermodynamics of energy balance.

The second fact is that any alteration in those rates of energy exchange produces a change in weight that comes to an asymptote while the altered rate of energy intake and/or expenditure persists. As the fat content of the body changes, lean mass changes in the same direction and hence also the rate of use of energy to keep those tissues working (Garrow, 1974).

It follows that any dietary way to slim must lower the rate of intake of energy for the weeks needed for a step reduction in body fat content. In addition, that way of losing weight must be maintained (or replaced by an equally effective means) in order to avoid regaining the initial fatness. Less food eaten or greater fullness rated over a test period does not in itself slow the daily rate of intake of energy. Even repeated observation of an acute suppression of intake does nothing to show a smaller total amount of energy intake over the period of the study. Feeling fuller after every meal is no guarantee of lower daily energy intake. It follows that no augmentation of satiety by a medically prescribed or commercially marketed material can be relied on by itself to reduce obesity or to prevent overweight for the years required to reduce the risks to health.

The science of support to effective weight-controlling food choices

Weight-controlling satiety therefore is not an effect of any sort of medication, food group or food product. Rather, satiety that slims is eating less often in ways that fatten by raising the average daily rate of intake of energy. That is, a 'slimming satiety' is an habitual pattern of eating and drinking that reduces weight when its frequency increases and is maintained throughout life at that new frequency (Blair, Booth, Lewis, & Wainwright, 1989; Booth,

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[1] For several reasons, research publications criticised here are not cited. They are readily identifiable by their authors and by informed readers (including the reviewers of this paper). It would be counterproductive to give such unhelpful papers further publicity. Much of the material appears in the 'grey' literature of presentations at conferences, chapters in books and other writing not subjected to expert peer review. Criticisms of evidence offered in support of a slimming claim have evoked threats of legal action, even though expert testimony in court against such claims has consistently turned cases against companies (Garrow, 2010).

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1980, 1996). Conversely, a 'fattening hunger' is an eating custom that increases weight when it occurs more often (Booth, 1996).

At least two clear examples of fattening hunger have been identified (Booth, Blair, Lewis, & Baek, 2004). One is a habit of selecting an option with a higher proportion of energy as fat, whether at a meal or between meals. The other is the frequent choice between meals of a small amount of food or a drink containing any source of energy, including starch, sugar or alcohol (Blair et al., 1989; Coakley, Rimm, Colditz, Kawachi, & Willett, 1998; Kayman, Bruvold, & Stern, 1990; Westenhoefer, von Falck, Stellfeldt, & Fintelmann, 2003). Nevertheless, high-fat choices might help to reduce snacking. A modicum of fat (and/or protein) within a meal may slow the rise of bodily hunger before and at the next meal at a conventional interval of 4–7 h (Booth, Chase, & Campbell, 1970). That rise in hunger might have resulted in the ingestion of energy between meals or an enlarged subsequent meal.

Generalising, an important slimming satiety could be a culturally recognised pattern of choices of foods and drinks at a meal that helps to prevent any intake of energy before the next meal, without increasing the energy content of either meal (Booth, 1988b). One might choose a quickly prepared and satisfying food for breakfast that contains a lot more protein than do cereal and toast. Also, slowly digested protein in lunches or evening meals might help to stop afternoon cake or eating just before bedtime. The same effect would be achieved if the hormonal signals generated by dietary protein towards the end of absorption of a meal were augmented by a long used food constituent or a safe new medication.

A widely suspected fattening hunger is habitual choice of more courses or larger portions at meals. The crucial issue though is not whether they are proportionately less filling. The only question is if the settled habit produces a insufficiently compensatory reduction in rate of energy intake measured as a step increase in weight.

Words and numbers versus realities

Research into these weight-controlling choices among foods and drinks has been gravely weakened by longstanding failures to measure the basic social, somatic and sensory mechanisms by which eating inhibits eating—the sating of appetite for food (Booth, 2008). These mistakes are rooted in systematic misuse of test-meal intakes and ratings of appetite (Booth, 2009). The result is misunderstandings about 'satiety' among experts in industry, medicine and academia and the funders and regulators they advise.

A prime example of the confusion created by such errors was the conclusion that only carbohydrate is strongly satiating, whereas fat and protein are weak satiators at best. The truth is that protein and indeed modest amounts of unemulsified fat are strongly satiating, and in ways that can be important for control of weight, while the role of carbohydrate-induced satiety must be minimal, for the following reasons.

Starch is often the most abundant energy-nutrient in a meal. Its digestion stimulates glucoreceptors, glucoregulatory hormones and glucose utilisation for the first hour or so after the meal (depending on the amount of carbohydrate). In contrast, amino acids from digestion of protein are sequestered in muscle, and the fat within solid foods is slowly digested and then circulates in chylomicrons, until the rate of absorption of glucose declines, leading to oxidation of alanine and glutamine from muscle and of fatty acids from the circulating fat.

Hence intake tests or appetite ratings within an hour or two of a meal can easily show reliable effects of its contents of carbohydrate but not of protein or fat (while all three are reflected in blood hormones and metabolites). However, such satiety (or blood chemistry) is irrelevant to slimming because food is seldom eaten so soon after a meal. Similarly, products that slow early digestion are unlikely to reduce daily rates of energy intake because such moderate delaying of assimilation does not affect the next meal.

Early research on the satiation of eating was built on recognition of this critically timed series of mechanisms activated by the consumption of food. Great damage has been done by using the term 'satiety' to label an imagined timeless property of constituents of foods, regardless of context of their eating in the culture, the body and interactions with others.

This unscientific notion also suffers from a severe statistical problem. The longer that a measure is made after an experimental manipulation, the more variable will be the mean value observed. Therefore reliable effects are harder to see with later intake tests or appetite ratings. In addition, tests near the next usual mealtime will be more constrained by habit and so additionally insensitive to effects of any prior manipulation. Nevertheless, large numerical effects (some also statistically reliable) have been seen at mealtime tests 3 or 4 h after intake of disguised variations in protein and/or fat (e.g., Booth et al., 1970; Cotton, Burley, Weststrate, & Blundell, 1994; Dibsdall, Wainwright, Read, & Booth, 1996; French, Wainwright, Booth, & Hamilton, 1992; Sepple & Read, 1990). These late effects on satiety, i.e. slowing of the rise in hunger, are the ones most relevant to slimming, both by helping to prevent snacking and also by moderating the size of the next main meal. It has indeed been shown that, at the same reduction of energy intake, sufficient protein in low-carbohydrate diets is crucial to the better compliance than seen with low-fat diets (Skov, Toubro, Ronn, Holm, & Astrup, 1999).

The concept of a biological marker for satiety also is fundamentally flawed by its neglect of the mechanisms of satiety. Merely correlating the areas under curves for blood glucose and ratings of fullness does not show how digestion products contribute to choices of when and what to eat, let alone any relation of those choices to obesity. The levels of gut hormones or of metabolites such as glucose or fatty acids in the blood cannot measure satiety because, even if a substance makes a contribution to normal inhibition of appetite, its physiological effects signalled to the brain and suppressing intake need to be tracked until they cease in order to measure the impact of the substance on rate of energy intake. There is not even a specifiable proportion of satiety at one moment under given conditions that is attributable to a particular mechanism. Arbitrarily timed 'satiety' tests provide no scientifically interpretable data.

In short, satiety can never be a property of a substance, whether in a food, in the blood, in a medication or in the brain. Hence it is impossible to design a valid physical test or marker for the satiety effect of a food or a drug. Indeed, satiety is irrelevant. The only way to identify a contribution of a substance to weight loss is to measure those mechanisms by which it influences customary patterns of uses of foods and beverages, so altering the rate of energy intake. Revival of this "psychobiological long haul" is vital to complement that "psychosocial short-cut" in its application to evidence-based public health policies, clinical treatments and the formulation and marketing of foods and drinks (Booth, 1988a, 1988b).

Failures to regulate implied slimming claims

The absence of such a scientifically sound basis for food policy has not stopped commerce and public health putting out product claims and educational messages that mislead about weight control. Indeed, regulators have even promoted such activities.

Slims "as part of a calorie-controlled diet"

British government regulations required any advertisement of a product that might be used in efforts to slim to include wording to

the effect that the product "can help weight control only as part of a calorie-controlled diet." This was naïvely conceived as a disclaimer. The regulation is exploited to make claims to slim without even mentioning the concept. Merely the brand logo and pictures identifying the product are shown in a TV commercial that includes a brief flash of "... calorie-controlled diet." Supermarkets can be relied on to display the brand alongside other products for dieters. That is, a product that does not claim to help slimming is allowed to imply it can be effective!

Worse, the required statement itself is doubly deceptive because it is a tautology that posits the impossible. If the energy in the diet, with or without the product, were actually controlled, then weight necessarily would be controlled as well (given no less physical activity). Yet even the best procedures of dietary assessment or research measurements of energy expenditure cannot estimate accurately an individual's usual energy intake. So an ordinary eater has no hope of calculating total daily calories.

The dieter might select meals by use of a handbook that assigns points to each dish for energy content, particularly of fat, perhaps discounted for potential suppression of hunger by protein, fibre or water. That is not calorie control. At best, it is support for 'flexible dieting' (Westenhoefer et al., 2003) – personal exploration of sustainable choices for their effects on weight loss and maintenance.

The "low-fat" debacle

On medical advice, food regulations were introduced in the 1980s that allowed reductions in the traditional fat contents of food products to be labelled as "low fat" or "reduced fat." Such labels are misunderstood by consumers to mean "less fattening" (Table 2 in Booth, 1987; Carels, Konrad, & Harper, 2007; Oakes & Slotterback, 2001).

It was technologically impossible to replace the delicious textures and aromas that fat provides, especially the great variety after cooking with starch and/or protein, e.g., different sorts of crispness and crunchiness and of appetising smell from the Maillard reaction or oxidation of fat. Hence, the food supply industries offered low-fat versions of products that made only trivial contributions to *per capita* intake of fat, such as milk, yoghurt and spreads. Consumers' appreciation of dairy creaminess was degraded by masking the loss of its aroma and mouthfeel by fruit flavours and starch thickeners. Worse, the use of such labelling largely on milk products helped to deceive consumers into thinking that they were on a low-fat diet (Brug, Van Assema, Kok, Lenderink, & Glanz, 1994).

Apparently, regulators and the research community have not learned from that debacle and so its effects still linger (van Trijp, 2009).

"Low-sugar" nonsense

Such naivety about marketing was extended to sugar under the illusion that the amount of sugar in a food or drink creates more risk to health than starch or other sources of energy. The only danger specific to sugar is to the teeth from repeated exposure at short intervals. The idea that usual daily energy intake as sugar contributes to obesity was refuted long ago (BNF Task Force, 1987) although attempts have been made to resurrect the myth (WHO, 2003). Unlike fats, sugars are no more energy-efficient than starches in the deposition of fat.

The label "diet" product is permitted when non-nutritive sweeteners replace sugars. This not only trades on the fallacy that, by itself, lowering the sugar content of a product delivers reduction in weight (Mattes & Popkin, 2009). The label also promotes magical thinking—the supposition that any substance could reduce weight, let alone keep it off, without regard to persisting changes in patterns of eating and exercising. As a result, having a 'diet' drink can be used to excuse intake of much more energy than the sugar saved. Some consumers even expect a non-nutritive sweetener implicitly to burn off fat (Freeman & Booth, 2010; Freeman, Richardson, Kendal-Reed, & Booth, 1993).

The new prospect: slims by "increasing satiety"

The European Union now requires a health claim as phrased by the company to be supported by documentation evaluated by experts consulted by the European Food Safety Authority. The EU makes a claim illegal if the evidence is judged not to substantiate it.

Claims of short-term intensification of satiety were excluded initially because scientifically adequate tests were not proposed. Unfortunately the weaknesses in research on appetite outlined above were exploited to create an impression in some quarters that satiety is an effect that can be attributed to a particular material consumed in an undefined context within an unspecified pattern of eating and drinking. Far worse, though, some on the EU panel reviewing health claims were reported to deny the fact that consumers read a slimming effect into a satiety claim (Food Standards Agency, 2009).

Such debate within a regulatory authority should never have begun. The performance of a particular food at reducing usual daily intake is unmeasurable in principle. All depends on the usual daily pattern of food choices by the individual and the particular amounts of the product consumed with which amounts of other materials on what occasions within that pattern. The basic scientific question remains to be asked. Does a specified usage of the substance support a dietary custom that is a slimming satiety?

A drug or food could not survive randomised controlled trial for reduction or prevention of obesity, in health or in chronic disease. Indeed, no trial of an intervention on obesity has found evidence of indefinitely sustained loss of entry-group average weight following the end of intervention. The reason is obvious from trials that track an indicator of energy intake or expenditure, or even just body weight. The medication, diet, exercise regimen and/or lifestyle education do not alter activities that affect energy exchange and also are maintained. Continuing relapses in behaviour change and/or regain in weight are always evident in the last two follow-up measures. The correct conclusion therefore is that the trial provided no evidence that the intervention had any effect on long-term abdominal fatness.

Open trials also do not usually show continued weight loss after the end of therapy. One that did was unique in including evidence-based information on specific self-described habits (Blair et al., 1989) as well as cognitive-behavioural therapy for the minority who have emotional problems about bodily shape, emotional eating or temptations to eat (Lewis, Blair, & Booth, 1992). Another also encouraged self-experimentation but without communicating local evidence on what works (Westenhoefer et al., 2003).

Causing obesity by exploiting dieters

Ill founded slimming claims are liable to create more obesity. Unsuccessful dieting tends to fatten (Mann et al., 2007; Savage, Hoffman, & Birch, 2009). Evidence that greater weight gain followed replacement of meals with formula implied to be fast-slimming was presented long ago to both food manufacturing (Booth, 1988a) and public health (Booth, 1993).

Fattening by 'slimming' products arises partly because the experience convinces users of their own worthlessness (Foster, Sarwer, & Wadden, 1997), whereas in fact the product was worthless. So people give up trying to slim, and yet are easily

tricked into another cycle of yo-yo dieting on the next false promise. This temptation may come from a trial of a new medication or an educational programme. Trials are always exploitative unless they monitor the persistence of a change in any particular pattern of eating or exercise while also testing if the change induces decrease in weight (Blair et al., 1989; Knauper, Cheema, Rabiau, & Borton, 2005).

The politics of research into satiety and obesity

Research on the roles of eating and drinking in weight control suffers from the pressure put on academic research generally to become more commercially relevant or governmentally palatable. Public funders who work with manufacturers of products for dieters (drugs or foods) risk giving credibility to the false impression that increases in satiety cause loss of weight.

Commercial subsidy of public funds for academic research is not only a dubious way to spend taxpayers' money. Companies' profits from existing products are routinely wasted on decisions based on visualisations of sensory and consumer data instead of measurements of what encourages different customers to eat products. Such underinformed advice makes it harder for funding committees to recognise the combination of experimental psychology, human physiology and cultural scholarship required for effective research on weight control.

The poor state of research into medical or dietary support for weight control does the pharmaceutical and food industries no good either. As pointed out above, a satiety-augmenting medication or food constituent might have a role in changing a slimming pattern of eating and drinking in a way that is sustainable for life, thus contributing to flexible avoidance of unhealthy fatness. Development of such food or drug materials depends on identifying the customs of eating, drinking and movement that are readily feasible in the current environment and provide a commercially viable opportunity. Then the material can be designed technically for positions in the market that support specifically those customs that induce a step reduction in weight while they are maintained.

Hence food marketing, public health nutrition and clinical science need to start working together to measure local eating cultures and the effect of change in each pattern on weight over the few weeks to asymptote. Until that happens, it will not be possible to develop and recommend materials for those uses that actually do help indefinitely maintained loss of unhealthy weight.

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