



Reply to Comments

Weight is controlled by eating patterns, not by foods or drugs

Reply to comments on “Satiety. No way to slim”[☆]

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ABSTRACT

The five papers in this special section of *Appetite* seem to agree that augmentation of satiety at an unspecified delay by use of a medication or food product in an indeterminate context provides no assurance that the substance contributes to reduction of obesity. Rather, satiety that slims is a specific pattern of eating that reduces the rate of energy intake while that pattern persists. These scientific principles have major implications for research that could provide the evidence needed to regulate claims to deliver weight-controlling satiety or to reduce discomfort allied with hunger arising from attempts to reduce weight. Since satiating efficacy is an attribute of a specified pattern of eating, it cannot be the property of any substance, even one that supports such appetite-reducing behaviour. Hence the evidence required depends on identifying the eating customs that are effective in long-term control of weight, in words that enable members of the public to make their own selections among those obesity-preventative practices and to use a food or a drug in a way that supports such a dietary habit. We hope that these four comments and our more extensive reply help to clarify issues that are crucial to slowing the rise in obesity.

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Surface consensus and underlying divergences

We are most grateful to the authors of the four comments on our brief analysis of a claim that a food substance or a drug augments satiety and any implication that the material thereby aids weight loss (Booth & Nouwen, 2010). Our six papers (including this reply) illustrate the complexities of dietary reduction and prevention of obesity. Patterns of eating and drinking are highly diverse. Each pattern is subject to a strongly interactive array of social and biological influences. Research therefore needs to come to grips with the physiological and cultural processes of satiation within and between the meals, snacks and drinks comprising particular dietary practices that may have a role in the control of body weight (Booth, 1976, 1988a).

From those principles, we argued that a valid claim that a food or drug boosts satiety should include a specification of the behavioural context of effective use of the substance in the evidence communicated to users (Booth & Nouwen, 2010). We pointed out in addition that an inference that such contextualised satiety contributes to weight control is justified only if the setting

is a customary pattern of eating and drinking that has been shown to reduce weight while that habit is sustained (e.g., Blair, Booth, Lewis, & Wainwright, 1989). We are therefore delighted to read that the European Food Safety Agency (EFSA) decided against uncontextualised claims that protein augments satiety (Mela, 2011). We hope this augurs against the authorities of the European Union ever approving a satiety claim that does not specify the usage of the material within an eating pattern that has been shown to maintain a decrease in weight.

At first sight, the commentators appear to agree with our general position. When referred to explicitly, its cogency is acknowledged. Yet considerable divergences emerge when the comments turn to specific issues. This appears to be because the implications for research are much more extensive than could be explained in a Short Communication. Therefore we have asked for space to clarify the position in this reply to the comments so far.

Smeets and van der Laan (2011) provide a lucid and well referenced critique of satiety claims. We thank them for this helpful contribution because we took on the different task of calling attention to longstanding fundamental scientific theory that provides a directly evidence-based approach to tackling the public and personal problem of obesity. Socioeconomic, physiological and cognitive-behavioural programmes should engage together in developing effective nourishment of the members of the human community. Substantial improvement in citizens' lives requires fully informed collaborative action by commerce, clinical

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and public health psychology and medicine, policy development across government, and formal and informal education and entertainment. The need for such radical reform of research relating to contemporary human obesity is beginning to be acknowledged (e.g., Rowe et al., 2011), although without recognising the inadequacy of established investigative methods, research training and scientific education (Booth & Booth, 2011).¹

Such reform is of course far beyond an exchange like the present one. This reply merely indicates some developments of work cited by the commentators that would move in the direction needed. Unfortunately, the ‘best practice’ advocated by some who have commented and their colleagues (Blundell et al., 2010) does not address either of the key scientific questions that were identified in the 1970s. On the one hand, by what physiological and social mechanisms do particular patterns of consumption of foods and drinks reduce the appetite for further foods? Complementarily, what ultimate effect does a change in the frequency of such a pattern have on the energy content of the body? No intake or rating tests of appetite by themselves can elucidate the mechanisms of satiety or energy balance. The expressions of appetite and changes in weight need to be set within designs and analyses that measure the causal processes as they go on.

To start tackling the first question, the experimental design of prior ‘loading’ and subsequent ‘compensation’ of intake of any food was introduced to analyse specified sets of physiological mechanisms of satiety (Booth, Campbell, & Chase, 1970). Any rating of appetite for food (regardless of wording) can be used to measure a facilitatory or inhibitory biological or social mechanism that is operative at the moment that desire to eat is judged (Booth, Mather, & Fuller, 1982); different wordings for hunger and its sating are redundant. Interactions among these mechanisms produce each state of satiety. Since such interactions are liable to vary with individuals’ habits, an ingested substance cannot have a fixed sating efficacy. Variations in habit determine answers also to the second question, the role of intake in weight control. Reduction and maintenance of individuals’ weights depend on the rates of energy intake generated by how often they engage in each culturally identifiable eating or drinking practice. That is the theory on which we based the target paper (Booth & Nouwen, 2010).

The social nexus of the market

In their comment, Smeets and van der Laan (2011) call attention to the larger picture featuring such fallacies as “healthy” and “unhealthy” foods and food groups. They note that the products sought by people who regard themselves as dieters are marketed to women. Of course we cannot agree with their suggestion that this “bias” should be balanced out by marketing such products to men. The bias that needs to be corrected is the passivity of the food industries with regard to their science base. Biologically and socially realistic psychological investigation of the actual mechanisms of the market has been consigned to the ghetto of ‘blue sky’ research, safely out of the way of commercial operations. Instead, such findings should long have been at the forefront of the human evidence for product development, bridging between technology and marketing in a single research operation (Booth, 1988b, 1988c, 1988d; Booth & Booth, 2011).

Similarly, clinical trials of treatments for obesity have not tracked the psychosocial processes essential to the efficacy of the medication or surgical procedure and of each component of any accompanying package of advice, including each facet of diet and exercise (Booth & Booth, 2011).

For example, as Smeets and van der Laan (2011) emphasise, diet products generally are ineffective at best and may contribute at least as often to fattening as to slimming. Yet many people want to eat slim, of both genders. Hence technical development and marketing strategies should be coordinated on a unitary human research base, to deliver products that cultivate eating and drinking practices demonstrated to promote wellbeing. What independent investigations then show to work will channel greater demand into the products that can be used effectively, especially in this era of tweeted ‘word of mouth’. de Graaf (2011) and others appear to agree with us on this objective. What is lacking is an appreciation of the research designs required.

Clearly we do not dispute the contention that there is a demand to be satisfied for weight control products (Mela, 2011). Rather our view is that it is incumbent on those who earn a living supplying food to develop products and market them in ways that communicate the evidence how those who wish to reduce weight can use those foods in patterns that actually do attain that end.

It is surprising to read comments to the effect that it is justified to claim that a product helps dieters because an unidentified small proportion of the populace keep weight off who use the product long term. The assertion that the product aids slimming by boosting satiety is a generalisation. The evidence on those who do not benefit and the proportion harmed have to be considered as well (Smeets & van der Laan, 2011).

Hence we cannot agree that any credit be given to consumers’ desires for slimming aids and the commercial efforts to provide such products without positive evidence of improvement in weight control (Bellisle & Tremblay, 2011). This is demand created in part by regulation of food labelling that was based on scientifically ill founded advice (Booth & Nouwen, 2010). The issues are what the purchasers want to get out of products with lower contents of sugar and fat or more fibre and water, and how companies justify diverting profits into technological fixes that often degrade quality without delivering any substantiated value for money. If the food regulations or the results of pharmaceutical or feeding trials permit or even encourage products that may eventually prove to be risky for some, there are serious questions about developing and marketing such substances. That stricture has been reinforced repeatedly for so-called anti-obesity medications.

Consumers’ wishes or opinions have no bearing on the validity of a satiety claim. What works is entirely down to what consumers actually do with the food or drug claimed to augment some of eating’s suppression of the desire to eat some foods at some later times. The sole basis for valid inference that a substance augments satiety controlling weight is a demonstrated contribution to loss of weight from a particular pattern of use which can usually be maintained long enough to reduce the risks of degenerative diseases or to financial or interpersonal wellbeing. The effects on satiety need to be tracked in the same study as the effects on weight, and both sets of causal processes have to be monitored as they happen. No less importantly, such a study should test any hypothesis about how the substance works in that context. Drug trials may not need to do that when the tissue action is already known. Yet if an agent may affect conscious bodily states such as hunger pangs or sensations of fullness, or habitual actions like having cookies or cakes with coffee, these psychological events need to be specifically monitored in a design that tests if any of them mediate the efficacy of taking the medication or consuming the food substance.

¹ There are four verbal errors of varying seriousness in Booth and Booth (2011), for which those authors apologise. The fourth line of the second paragraph in the second column of page 214 should have read “habitual actions that have been shown to influence” (deleting “changes in weight”). On page 217, the twelfth line up from the end of column 2 should start “change is maintained” (not a plural). Page 219, column 1, paragraph 3, should have “nor” (not “or”) two lines from the end. When the term “Behavior Change” is quoted from the USA, the word ‘behavior’ should be set without the ‘u’ (page 213, column 2).

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