

## Authors' manuscript

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### 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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*[Special Section footnote]*

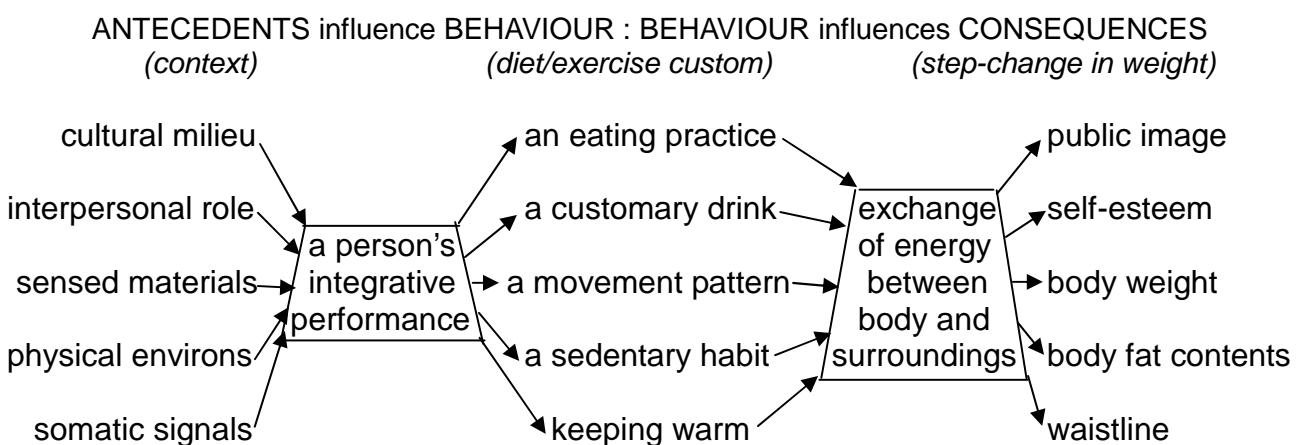
This is the reply to four comments on “Satiety -- no way to slim” (Booth & Nouwen, 2010) in a Special Section of *Appetite* edited by Jennifer O. Fisher.

### *(online) Highlights*

- The effect of a food or drug on hunger and its satiating varies among eating customs.
- Claims to suppress eating or related sensations need to show the efficacy in weight reduction that many eaters want.
- Regulation of foods or drugs needs research on the social and biological mechanisms of weight control by eating habits.

### *(online) Graphic Abstract*

#### **The A → B → C of slimming**



## 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.

### *Keywords*

satiety  
weight control  
hunger management  
eating habits  
causal analysis

## 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 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, Alexander, Almeida *et al.*, 2011), although without recognising the inadequacy of established investigative methods, research training and scientific education (Booth

1 & Booth, 2011<sup>1</sup>).

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

13 To start tackling the first question, the experimental design of prior ‘loading’ and subsequent  
14 ‘compensation’ of intake of any food was introduced to analyse specified sets of physiological  
15 mechanisms of satiety (Booth, Campbell & Chase, 1970). Any rating of appetite for food  
16 (regardless of wording) can be used to measure a facilitatory or inhibitory biological or social  
17 mechanism that is operative at the moment that desire to eat is judged (Booth, Mather & Fuller,  
18 1982); different wordings for hunger and its satiation are redundant. Interactions among these  
19 mechanisms produce each state of satiety. Since such interactions are liable to vary with individuals’  
20 habits, an ingested substance cannot have a fixed satiating efficacy. Variations in habit determine  
21 answers also to the second question, the role of intake in weight control. Reduction and  
22 maintenance of individuals’ weights depend on the rates of energy intake generated by how often  
23 they engage in each culturally identifiable eating or drinking practice. That is the theory on which  
24 we based the target paper (Booth & Nouwen, 2010).

## 25 **The social nexus of the market**

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

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

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

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

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

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

31 Consumers' wishes or opinions have no bearing on the validity of a satiety claim. What  
32 works is entirely down to what consumers actually do with the food or drug claimed to augment  
33 some of eating's suppression of the desire to eat some foods at some later times. The sole basis for  
34 valid inference that a substance augments satiety controlling weight is a demonstrated contribution  
35 to loss of weight from a particular pattern of use which can usually be maintained long enough to

1 reduce the risks of degenerative diseases or to financial or interpersonal wellbeing. The effects on  
2 satiety need to be tracked in the same study as the effects on weight, and both sets of causal  
3 processes have to be monitored as they happen. No less importantly, such a study should test any  
4 hypothesis about how the substance works in that context. Drug trials may not need to do that when  
5 the tissue action is already known. Yet if an agent may affect conscious bodily states such as hunger  
6 pangs or sensations of fullness, or habitual actions like having cookies or cakes with coffee, these  
7 psychological events need to be specifically monitored in a design that tests if any of them mediate  
8 the efficacy of taking the medication or consuming the food substance.

9 Contrary to the imputations by Mela (2011) and de Graaf (2001), we are far from arguing  
10 that food suppliers are “unscrupulous” and even “deceitful.” The industries inherited a material  
11 culture in which the traditional ways of preserving foods (by baking, adding salt or sugar and/or  
12 mixing with fat) have transmitted eating habits across the generations that demand to be fed. What  
13 our paper attacked was the medicalised response to this situation (see also Gracia-Arnaiz, 2010). It  
14 is the scientifically underinformed bases of public health nutrition policy and of food development  
15 and marketing that create counterproductive conflict (Hoek & Jones, 2011). The approach needed is  
16 neither marketing-style polling surveys nor medical-style feeding trials, nor merely better  
17 collaboration between policy advisors and hands-on researchers (Hoek & Jones, 2011; Rowe *et al.*,  
18 2011). Both commerce and health should be encompassed by a scientific framework that is realistic  
19 about the multiple systems in a person’s life, rather than reductive either to neuroscience or to  
20 phenomenology, or reliant on a “psychobiology” that is neither cognitive nor physiological.  
21 Scholarly scientific research needs a larger vision than skirmishes over salt, calories, energy density,  
22 this or that vitamin or mineral, or any other substance. The primary commitment should be to  
23 provide each citizen and all the service organisations -- commercial, voluntary and governmental -  
24 - with quantitative evidence on which are the healthiest among local eating customs and other  
25 common patterns of energy-exchanging action (Booth & Booth, 2011).<sup>2</sup>

## 26 Satiety based on learnt biosocial mechanisms

27 Bellisle and Tremblay (2011) agree that there is a great need for a return to laboratory  
28 research into normal biological and social influences on human food intake. A start was made in the  
29 early 1970s on the experimental analysis of both physiological and social processes affecting the  
30 amount eaten in a laboratory test. That work by clinical experimental psychologists in the USA and  
31 Germany was designed to provide evidence on the causation of the state of satiety that ends a meal.  
32 Unfortunately, by the early 1980s those promising beginnings were being subverted by merely  
33 weighing the foods or even estimating calories eaten as tests for concepts named with paradoxically  
34 different forms of the word ‘sating.’ In this connection it is good to see cited the French founder far  
35 earlier of laboratory research into the behavioural physiology of hunger/satiety in animals (Le

1 Magnen, 1957a; see the collection of English translations in this journal in 1999, plus for example  
2 Le Magnen & Tallon, 1966, for early observational analyses of satiety mechanisms). Bellisle herself  
3 was instrumental in extending the physiological work of Le Magnen's laboratory to human  
4 volunteers (Bellisle, Louis-Sylvestre, Demozay *et al.*, 1983).

5 The point that needs adding is that Le Magnen (1957b) showed also that influences on  
6 appetite and satiety are learned. He succeeded in reducing rats' food choices and intakes by pairing  
7 arbitrary textures, tastes, smells and colours with injection of concentrated glucose or the early  
8 appetite-suppressant, D-amphetamine. The evidence for learned control of eating is generally not  
9 well understood. Contrary to de Graaf (2011) and the authors he cites, conditioned satiety is not a  
10 response that can be measured merely by an amount eaten or a rating of appetite (Booth, 1990,  
11 2009a). As explained below, those weights or scores need have nothing to do with specific  
12 inhibitory influences over eating or with the eater's particular past experiences.

13 Satiety is a state in a process of partial and sometimes selective satiation of appetite by  
14 influences that Le Magnen proposed have been established at their current norms by personal  
15 experience recently or in the distant past. Some of the basic mechanisms of such human memory-  
16 building are shared with many other species. These elemental learning processes connect responses  
17 or stimuli that control responses with their consequences -- mechanisms known as association,  
18 reinforcement or conditioning. Conditioned satiety is some extent of inhibition of eating in the  
19 immediate presence of both particular food cues and also a relatively full digestive tract, as a result  
20 of an aversive after-effect of eating in the presence of that combination of stimuli (Booth, 1972,  
21 2009a; Booth & Davis, 1973). Hence, even if reduction in meal size is shown to have been learnt,  
22 that is not evidence that satiety has been associatively conditioned. The test for conditioned satiety  
23 is the absence of that state of inhibited eating with a different food on a full stomach and with the  
24 same food but an empty stomach. Learnt reductions in intake and rated expectations of satiety that  
25 do not meet these criteria may have nothing to do with the conditioned satiety observed repeatedly  
26 in people, monkeys and rats since the 1970s.

27 Furthermore, this satiated response to a combined external and internal conditioned stimulus  
28 has so far been demonstrated only after pairing that multimodal configuration with a transient  
29 aversive effect that appears to be unique to concentrated maltodextrin consumed on an empty  
30 stomach (Booth & Davis, 1973; Booth, Lee & McAleavy, 1976; Booth, Gibson, Toase & Freeman,  
31 1994; Booth, O'Leary, Li & Higgs, 2011). Contrary to some of the comment (de Graaf, 2011),  
32 sensory-somatic satiety has never been shown to be conditioned by high energy density. Purely  
33 sensory aversions have been conditioned by abnormal forms or routes of administration of glucose,  
34 dextrinised starch, imbalanced amino acids, poisons and unfamiliar drugs. Ordinary  
35 glucosaccharides, essential amino acids and triglycerides condition only sensory preferences and

1 increases in food intake. Hence high energy density seems more likely to train extra attraction into  
2 foods rather than teaching satiety. What role if any that maltodextrin-conditioned satiety plays in the  
3 learning of satiety in general has to be a matter for adequately designed experiments simulating  
4 common patterns of eating. Fresh bread to break the overnight fast may be digested similarly  
5 enough to concentrated maltodextrin. That could be helpful if it reduced appetite for a large fried  
6 breakfast in those who have that habit. Yet it could be counterproductive if any conditioning of  
7 satiety cuts back appetite for some high-protein food at breakfast in those who would otherwise  
8 snack during the morning, if that is a particularly fattening habit. Everything depends on the context  
9 of eating practices in which a substance is involved.

10 As de Graaf (2011) implies, these matters are critical to advice to the regulators. It can  
11 hardly be doubted that decisions on how much to eat are learnt, along with other aspects of appetite  
12 for food and drink. Yet remarkably little mechanism-identifying research has been done on the  
13 building of implicit control or explicit expectations of amounts eaten. The conditioning of satiety is  
14 vested by definition in the control of intake later in the meal. Its localisation in the size of dessert  
15 was evident in the first report for human beings of the conditioning of meal size by maltodextrin  
16 (Booth, Lee & McAleavy, 1976). The other form of food-specific satiety, acquired temporarily  
17 from immediately previous eating of a food, has still to been resolved between habituation, learnt  
18 norms for portion sizes and other mechanisms (Epstein *et al.*, 2009; Kral, 2006).

19 These considerations about learnt satiety nevertheless have their main importance in this  
20 discussion as illustrations of the centrality of causal investigation. Satiety is not a concept defined  
21 by a test. Satiety involves mechanisms, and a mechanism can only be measured via its antecedents  
22 and consequences. A satiated response in isolation shows nothing about its likely causes. Unless we  
23 investigate the mechanisms that make the processes of satiation continue after the end of a meal and  
24 then make appetite rise as expected before the next mealtime, we shall never be able to counter the  
25 challenges from an environment of abundance (Bellisle & Tremblay, 2011) or make any inroads on  
26 the psychology or biology that could support a pattern of satiety that slims (Booth, 1988a; de Graaf,  
27 2011).

## 28 **Multi-step approaches: habits or materials?**

29 Bellisle and Tremblay (2011) emphasise that many constraints on human eating have  
30 nothing to do with past survival of the species but arise from the exigencies of cultural history,  
31 lifelong personal development and interpersonal relationships. It follows that satiety cannot be  
32 entirely a “psycho-physiological mechanism.” Some inhibition of eating comes from physiological  
33 signals but food intake is also suppressed socially by recent consumption of food. Hence the satiation  
34 of appetite for food is a psycho-socio-physiological process, or more briefly a biosocial effect.

35 Clearly then, a biosocial approach to evidence-based weight control (Booth & Booth, 2011)

1 needs to begin by identifying customary activities of eating and exercise in the terms generally used  
2 within each culture. Questionnaires just about foods or food groups cannot do this (French, Jeffery  
3 & Murray, 1999).

4 The next two stages in a biosocial approach measure individuals' frequencies of carrying out  
5 these culturally identified patterns of ingestion (or of movement and stillness) and then relate a  
6 change in the frequency of each pattern to the asymptotic change in weight that follows if the habit  
7 affects energy exchange. This requires randomised  $N = 1$  experiments ('multiple baseline': Barlow,  
8 Nock & Herson, 2008), with cross-lagged correlational analyses (cp. Lawler & Suttle, 1972) of the  
9 observed changes in each habit's frequency and reported weight, separated from any changes in  
10 other energy-related activities. No mechanistic conclusions can be drawn from mere correlations of  
11 differences in intake of a substance with satiety responses and with weight changes, even in the  
12 same investigation, let alone in separate studies (de Graaf, 2011).

13 Once a causal link has been demonstrated to change in weight from change in frequency of  
14 an eating pattern recognised by the eaters, then we need to identify major influences from the social  
15 and physical environment on the lapsing from that eating habit that ends its contribution to weight  
16 loss (Booth & Booth, 2011). Finally, we also need to characterise the interactions within the  
17 individual's mind among perceived interpersonal influences and the cultural, sensed and bodily  
18 influences on lapsing from a habit change that has reduced weight (cp. Laurier & Wiggins, 2011).  
19 These cognitive diagnoses have been illustrated for choice between a diet soda and a sugar soda  
20 (Freeman & Booth, 2010), the importance of which is implied by de Graaf (2011) in referring to  
21 'liquid calories.' Another example is visual integration into choice of the amount of spread on a  
22 piece of bread and the labelled percentage of fat in the spread (Booth & Freeman, 1999; Booth,  
23 Sharpe, Freeman & Conner, 2011). Such work is indispensable for evidence on which to design  
24 foods, transport and other public environs that have been shown to affect weight via energy  
25 exchange in everyday life.

26 Given this need for stages in a biosocial approach, we were interested to see Bellisle and  
27 Tremblay (2011) advocating research in four steps. However, their steps centre on food substances,  
28 not on patterns of eating. Hence essential information is missing, as follows.

29 Bellisle and Tremblay illustrate their first step by a feeding experiment that seeks to relate  
30 consumption of milk to satiety. To understand the mechanisms of any effects, it is essential to know  
31 the circumstances in which the obese women in the cited study drank (most of) their milk, i.e. with  
32 what other foods, when in relation to meals and the overnight fast, and the next opportunity to eat  
33 and the foods available then. Also an isoenergetic control drink needs the same proportions of  
34 protein, carbohydrate and fat as the milk, in order to avoid confounding by differences between  
35 nutrients and doses in the timing of suppression of appetite for food.

1 The same information about the eaters' perceptions of their eating is required for each of the  
2 other steps of Bellisle and Tremblay (2011). In step two, evidence of some sort is needed that  
3 ghrelin caused relevant changes in intake in the people studied; otherwise both appetite ratings and  
4 ghrelin levels could be driven by lack of energy intake and consequent shifts between metabolic  
5 fuels that ghrelin counters. Step 3 is designed to provide evidence that a deficiency in calcium or  
6 vitamin D helps to increase intake of fat and total energy intake. Step 4 seems to be intended to  
7 extend the evidence from dietary supplementation to the consumption of ordinary dairy products.  
8 Whether any effect comes from the calcium in milk or some other constituent (such as fat or  
9 protein, or particular compounds) would remain an open question. More importantly, Bellisle and  
10 Tremblay (2011) agree that "context is all." Hence, for any such finding to be interpreted in  
11 mechanistic terms, or usable in the clinic or in public education, data are needed on which dairy  
12 products eaten in what proportions with what other foods at what times have what consequences for  
13 the total energy intake each week or month.

In fact, within the biological, social and psychological reality of ordinary life studied in  
Steps 3 and 4, the only practicable way to measure a change in energy intake over a given period is  
by a change in weight (Garrow, 1981). The intakes reported from feeding trials are estimated from  
records generated by ill specified and highly reactive procedures (dietary assessment). The values  
for nutrient contents of the recorded foods are averaged from poorly constructed sampling (food  
composition databases). Hence, the best available procedures for assessing an individual's usual  
intake of fat, for example, are notoriously unreliable. The resulting estimates of total energy intake  
have been shown many times to be invalid, especially for obese people (e.g. in experiments using  
doubly labelled water). The only way round these difficulties that has been proposed so far is to  
elicit the timings of the most recent occasions on which the individual has eaten in a widely known  
pattern. Also the change in a particular eating custom has to be disconfounded from any other  
changes in energy intake or expenditure. The programme of interest to Bellisle and Tremblay (2011)  
needs to identify those locally recognised dietary patterns that include milk and other calcium-rich  
foods.

## Energy nutrients and satiety

29 Mela (2011) provides a useful review of reports that do and do not find evidence that protein  
30 reduces weight. Such divergences in conclusions are entirely consistent with our basic thesis that  
31 the effect of a substance depends on the context in which it is used. If we are right, feeding tests and  
32 lifestyle intervention trials cannot be informative if they fail to track the varied uses of the protein  
33 and their effects on persisting changes on weight while they are occurring, before asymptote is  
34 reached. Unfortunately that implication is ignored. Worse, our argument is reversed and we are  
35 accused of implying that a substance can aid weight control, meaning by its use in any context. The

1 causal sequence that we were illustrating has been turned backwards, so that the effect of the eating  
2 pattern is attributed to the protein. That move is bound to generate a contradiction. Hence the self-  
3 contradiction is not ours but the result of inverting our logic. From protein contributing to reduced  
4 energy intake by preventing a snack, it does not follow that a slimming effect can be attributed to  
5 protein after all, regardless of context of use.

6 The same fallacy is generated about sugar between meals in fizzy drinks. To point out that  
7 sugar sodas might be fattening because they are consumed between meals is the reverse of stating  
8 that sugar is fattening. If readers stick with the original argument, they will find that it is fully self-  
9 consistent. For a substance to have a role in slimming, it has to be used as part of an eating custom  
10 that has been shown to change weight when the frequency of that habit is changed.

11 We gave several examples of possibilities that may merit investigation. Mela (2011) appears  
12 to agree that those speculations are worth pursuing. Yet the point is missed by the assertion that  
13 carbohydrate, fat and protein are equally satiating (Mela, 2011). No substance or category of  
14 substances has a fixed satiating power. Administering a nutrient to see if it can alter intake or ratings at  
15 a time unrelated to data on digestive processes cannot have anything to do with the mechanisms of  
16 satiety or slimming. The early mistake we cited about late-satiating high-fat food is far from a straw  
17 man: he has merely been given a scarecrow's overcoat by renaming as energy-dense food (Burley,  
18 2007). The role of low energy density in maintained loss of weight is not the effect of fibre  
19 independently of how it is used, or even of food groups such as vegetables and whole grain foods  
20 (Raynor, Van Walleghen, Bachman *et al.*, 2011). It is not the food species selected for a person's  
21 meals that could make the day's pattern into an effective slimming diet. The menus handed out by  
22 weight loss clinics need to be fitted into proven slimming patterns among the shopper's, cook's and  
23 household's habits, in a series of meals without fattening interludes or disruptions.

24 Energy density and also liquid calories (de Graaf, 2011) are broad categories of dietary  
25 substances that can be extracted from epidemiological data (Booth, 2002) and manipulated in non-  
26 mechanistic feeding experiments, roughly equivalent to low and high water content. Both constructs  
27 are crude abstractions from the biochemical and culinary diversity and dynamics that actually affect  
28 energy exchange. The issue is what each of these terms might mean culturally and physiologically.  
29 Substances can only be relevant within the context of biosocial influences on eating customs.

30 "Liquid calories" are presumably the calorific beverages that emerged historically from milk  
31 and fresh juices: how fattening they are may well depend on when they are drunk (Booth, 1988a;  
32 Chapelot, 2005). "Energy density" is the result of drying, the addition of salt or sugar or the use of  
33 storage parts of plants such as nuts and grain, that was originally necessary for food to be edible  
34 through the winter and more recently for long shelf lives in food shops. Dry savoury and sweet  
35 products now satisfy the market for portions of food and drink that can be consumed anytime and

anywhere. Hence people eat snackfoods and drink sugar sodas and coffees with cream. Yet, instead of investigating the effects of and influences on those customs, research is designed around the quasi-substances.

The biosocial theory that the first line of defence against obesity is the ‘zero-calorie drink break’ (Booth, 1988a) remains underexplored (Blair *et al.*, 1989; Kayman, Bruvold & Stern, 1990; Coakley, Rimm, Colditz *et al.*, 1998). The behaviour pattern of drinking sodas is hidden behind a defence of intense sweeteners against bulk sweeteners. Blaming obesity on the substance sugar is like blaming the rooster for the sun coming up. The danger from diet sodas is relying on them to cut fat that has been put on from dry foods eaten away from meals (Freeman, Richardson, Kendal-Reed & Booth, 1993). What people should do when faced with risk from fatness is to cut back on any sources of calories after a meal has finished. At least, that is the implication from the limited amount of causal analysis done so far on undisturbed and properly monitored dietary patterns (*op. cit.* above). The originally proposed physiological mechanism of mixing in the stomach remains to be tested (Booth, 1988a).

## Is there a physical test for satiety?

16 We are quoted correctly as arguing that the notion of a biological marker for satiety is  
17 fundamentally flawed by a neglect of mechanisms. The riposte merely restates that flaw unawares:  
18 the physical measurement is “associated” with the rating score or intake weight that is labelled  
19 “satiety” (de Graaf, 2011). Yet it is logically impossible for the chemistry of a blood sample to  
20 measure the physiological processes that produced that plasma level at that moment, the causal  
21 connections between that physiology and a specifiable choice of foods and hence the amount eaten,  
22 and the role of that intake in the whole eating pattern that reduces weight. If it were shown that a  
23 blood chemical influences satiety, that finding does not reveal a mechanism; rather, it provides a  
24 tool for investigating the mechanisms implicated.

25 For example, how does CCK reduce intake in test meals? Does it amplify the gastric  
26 distension signal as in rats (Schwartz, McHugh & Moran, 1993)? When does gastric distension  
27 inhibit eating in human beings, and in what context? Even if that were the only mechanism by  
28 which CCK influenced satiety, there could be no general relationship between CCK level in the  
29 general circulation and the amount of food eaten in unspecified circumstances.

30 In another example, by what mechanism does the blood glucose dip relate to meal requests  
31 (Campfield, Smith, Rosenbaum & Hirsch, 1996)? One suggestion is that, rather than a switch  
32 between sources of glucose causing hunger, both are driven by a central clock. If so, the transient  
33 change in rate of release of glucose into the blood has no causal role in inducing the start of a meal.

More basically, it is illogical to propose a physical index of a psychological phenomenon such as the experiencing of epigastric pangs or faintness from lack of food when no method has

been established for measuring the particular effect to be indexed. The recent draft of scientific guidance on satiety claims by the European Food Safety Authority states that evidence of changes in biochemical markers can be considered only in the context of behavioural measurement (EFSA Panel, 2011).

5 For these reasons we are far less sanguine than Bellisle and Tremblay (2011) about the  
6 possibility of any physical test for (suppression of) eating in everyday living. Even the weights of  
7 food eaten in the laboratory are uninformative unless they vary with one or more identified  
8 biological or social sources of influence on choices of mouthfuls, independently of other influences,  
9 within a simulation of a common occasion in life. Indeed, unlike plasma ghrelin levels and other  
10 proposed biochemical markers, food intake is not a genuine physical entity, despite the long  
11 tradition of weighing items rather than measuring, say, their volumes and culinary roles. The  
12 amount eaten or drunk of any particular material results from a series of transient personal  
13 integrations of influences that can change with each mouthful. As such, the causal dynamics of a  
14 state of appetite are unique to each item of food or drink and each stage in the meal, snack or drink.  
15 In other words, the physical weight or energy content of a meal is an epiphenomenon accumulated  
16 from a series of decisions of currently underinvestigated complexity or simplicity. Furthermore,  
17 eating and drinking are intentional, even when automatic or impulsive, but are subject to numerous  
18 unconscious influences, as well as to some of which we can be aware (Booth & Booth, 2011).

19 The untrustworthiness of numbers from the arbitrary tests for “satiety” as predictors of  
20 numbers from blood chemistry is acknowledged (de Graaf, 2011). As reiterated above, that follows  
21 from the diversity of the processes by which appetite is suppressed by eating. It is therefore very  
22 puzzling why the hope is expressed that starting to attend to the physiology will make it easier to  
23 find such mechanistically meaningless correlations. Indeed, we would be contradicting ourselves if  
24 we accepted the commentators’ repeated pleas for further research to validate this or that measure,  
25 because the numbers that they advocate have no scientific basis. The main aim of our paper was to  
26 return resources to research directly on the physiological and cultural processes influencing patterns  
27 of eating and drinking that have been shown to have a role in avoiding gain or regain of unhealthy  
28 fatness. That is, the design of each laboratory experiment should mimic the context of an eating  
29 pattern that alters body weight when its frequency is changed.

## The pain of hunger

31           Bellisle and Tremblay (2011) raise the important additional issue of the bodily suffering  
32 involved in some individually successful ways of becoming less obese long enough to reduce the  
33 risk of degenerative disease or to slow its progression. de Graaf (2011) goes much further by  
34 claiming that satiety helps in slimming generally by reducing epigastric pangs, although these are  
35 not the sensations of “fullness” that the verbal approach defines as satiety. The problem with such a

1 broad claim is that many of those who cry out for help with hunger are following unsustainable and  
2 even unhealthy patterns of eating. Hunger pangs are a substantial part of what Stunkard (1976)  
3 called “the pain of obesity.” They are a major liability of many of the diets that are professionally  
4 prescribed when treating obesity but with which few can comply. There is no reason to believe that  
5 temporary weight loss benefits physical health; indeed it may increase mortality, though that could  
6 be an effect of extra exercise while obese (Harrington, Gibson & Cottrell, 2009). This long  
7 recognised concern has been the main motivation for seeking to measure the roles in energy  
8 exchange of patterns of eating that are customary and therefore widely feasible. Adoption of such  
9 patterns might help to prevent the waistline from expanding without creating intolerable wanting of  
10 food (Booth, 1998).

11 If hunger pangs are so central to satiety claims, why are their causes not being investigated?  
12 There has been very little genuinely psychobiological advance since Griggs and Stunkard (1964)  
13 showed only a weak association between the epigastric pang and gastric motility using signal-  
14 detection methodology. Our work has included the physiology and the clinical psychology of the  
15 management of hunger pangs and other sensations that some people have when they want food  
16 (Booth *et al.*, 2011; Dibsdall, Wainwright, Read & Booth, 1996; Talbot, Nouwen, Gingras *et al.*,  
17 1997). The question is which foods do what when that affects either or both the subjective  
18 experience of a pang or/and its physiological cause -- if any: the pangs could mainly be culturally  
19 instigated imagination around a concept such as the walls of the stomach rubbing together (Booth,  
20 1980). The answer might come first from basic research on the phenomenology of ordinary  
21 appetite. Yet the hunger pangs in slimmers might have different causes.

22 One reason that these research issues have not been noticed may be that the common  
23 complaint about objectively wanting more food, “I’m hungry”, has been confused with the much  
24 rarer expression of the subjective experience of frequent and/or intense uncomfortable sensations in  
25 the upper abdomen, “Ouch, that was another pang of hunger” (Booth, 1976). Even some  
26 psychologists assume that people use words to “report” on a private world of contents of  
27 consciousness. As shown long ago (Wittgenstein, 1953), this introspectionist approach to mental  
28 processes is as incapable as the behaviourist approach of explaining how we use words to deal with  
29 realities such as getting food at a time we are used to eating and/or out of a belief that the body now  
30 needs energy. The self-assessment methods in current use cannot provide evidence that epigastric  
31 pangs were affected. Feelings of hunger, a desire to eat, lack of fullness and increased pleasantness  
32 of foods are merely different expressions of the one state of wanting some food; hence none of them  
33 can pick out the sensations that some people have when hungry or satiated (Booth, 1976, 2009b,c;  
34 Booth, Mather & Fuller, 1982; Booth *et al.*, 2011). The redundancy in the advocated verbal  
35 construct of satiety is exposed by a recognition that satisfying an appetite is satisfying, an activity

1 that gets what is wanted is pleasing, and lacking appetite for food or water is more comfortable than  
2 having that hunger or thirst motivation, except perhaps if plagued with guilt, jealousy or worry  
3 about the eating that sated.

4 Before ending, it should be noted that the pain suffered from restricted access to a surfeit of  
5 food is not on the same scale as the pain of the hunger that is chronic insecurity of supply of food.

## Conclusions

In summary, much of this commentary substantiates the worries that provoked our little paper (Booth & Nouwen, 2010). It exposes major deficiencies in current advice to regulators, food suppliers and pharmaceutical developers concerning scientific evidence on everyday satiety and weight control. None of that work so far measures either biological or social mechanisms that might connect a person's momentary states of satiety to the risks from body fat to health and other wellbeing. When such research is cited, no serious attention is paid to the causal processes that the work may have been designed to measure. Only such disregard for the causes and consequences of eating and drinking can sustain the illusion that weight control relates to a material substance, whether the protein content or digestion-slowing structure of a food product or a medication altering hormone action or gene expression.

17 If anything like a satiety claim is ever allowed, in our view the information on the marketed  
18 product needs to be in language that has been shown to communicate the scientifically established  
19 facts. The truth might be that a certain size of portion of the product works with adequate amounts  
20 of nutritious food to reduce any pangs of hunger that occur over a certain range of hours later but  
21 does not in itself contribute to loss of weight. What should never be permitted is anything similar to  
22 what we called a tautology that posits the impossible -- the concept of weight control by a  
23 "calorie-controlled diet." Aids to the management of hunger are similarly unworthy of trust when  
24 the objective deprivation and subjective pangs suffered are not of help in general to lifelong weight  
25 control.

26 The attitude to all this expressed in our paper was not contempt (Bellisle & Tremblay, 2011)  
27 but indignation, as Bellisle and Tremblay acknowledge earlier in their comment. In any case, what  
28 matters in a publication is not an attitude but what is stated and its impact. We are trying to bring the  
29 basic biosocial science required for slimming to the attention of as many creators and users of  
30 research as possible. Stark but unexaggerated language is one way to try. We hope that these five  
31 new papers from eight investigators having a wide range of expertise, when considered together,  
32 will provide further illumination for those whose research could help to tackle the dangers of  
33 obesity.

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## Footnotes

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 ‘behaviour’ should be set without the ‘u’ (page 213, column 2).
2. We avoid the term lifestyle in scientific discussion, especially 'lifestyle behaviors' (*sic*), because such vocabulary prevents serious thought about the biosocial realities. What needs investigating and changing are specific customary patterns of action ('Behaviour'), their causes ('Antecedents') and their effects ('Consequences'), as illustrated in the online graphic abstract for this paper. Of course, it may be necessary to use the word 'lifestyle' to help searches to connect to work in such terms.