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Learnt reduction in the size of a meal.
Measurement of the sensory-gastric inhibition [of eating] from
conditioned satiety

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Abstract

Many experiments on the role of learning in the amount eaten at a distinctive test meal have been claimed to observe “conditioned satiety.” None published from outside this author’s group has used either the necessary design of the contingencies to be learnt or the measurements that distinguish a satiating effect from other loss of interest in food. One experiment has just been published without an adequate design but giving the best evidence yet from another group for the conditioning of sensory-gastric satiety; yet the authors conclude for changes in sensory preference with no learnt gastric involvement in the meal size response. To encourage correct use of the demonstrations in rats, monkeys and people of conditioned satiety and its mechanism, this paper briefly reviews publications that attribute control of meal size to learnt satiety.

Keywords. Conditioned satiety. Conditioned preference/aversion. Internal state dependence. Configural stimuli. Maltodextrin.

New evidence for maltodextrin-conditioned human satiety

Yeomans, Gould, Leitch and Mobini (2009) provide some of the best evidence yet for aversive conditioning of a sensory-gastric configural stimulus contributing to learnt reduction in the size of an experimental meal. Their results on intake at test meals show that a smaller amount was eaten when the sensory characteristics of the food and the amount of food in the stomach towards the end of the meal both replicated the sensory-gastric combination that had been followed on previous occasions by the prompt and transient aversive signal from the wall of the duodenum generated by digestion of concentrated maltodextrin (MD) into hypertonic maltose and/or glucose (Booth & Davis, 1973; Booth, Lee & McAleavey, 1976). Only after training on concentrated MD within the larger portions (Yeomans *et al.*, 2009, Figure 1A, group HED-300g) does the ingestion of the test meal reinstate the combination of adequate fullness of the stomach and the distinctive food and thus terminate close to that portion size of 300 g, despite the strong conditioning of sensory preference by adequate amounts of MD at any concentration (the groups LED-300g and HED-150g).

Yet these authors conclude that their data show no evidence for learning of satiety. They interpret their findings as resulting from the learning only of various degrees of sensory facilitation of ingestion. How could this be?

Much confusion has arisen from failing to distinguish the effects of relative sensory aversion/preference on the amount of food consumed at a meal from those of a relative sensory-somatic satiety/hunger. The opening sentence of the Abstract of Yeomans *et al.* (2009) ignores this distinction. “The concept of learned satiety suggests that associations between the sensory quality and post-ingestive effects of foods may lead to acquired control of meal-size.” Investigators’ suggestions that satiation of acceptance (learnt or not) has a role in controlling the size of a meal make no sense unless they have excluded a general loss of acceptance of those foods. Indeed the usual test for a conditioned aversion is a decrease in the amount consumed.

By definition, the satiation of any appetite depends on prior indulgence in the desired activity. So it is logically impossible to measure a state of satiated eating at or before the start of a meal. By conceptual necessity, the state of satiety can only occur in the later stages of a meal or after it. That is, satiety is a response to stimuli distinctive to states created by eating, whether physiological (e.g. signals from digestive tract), cultural (e.g. a conventional view of what is enough), interpersonal

(e.g. attitude attributed to a companion) or sensed (e.g. habituated eating of a food, as may contribute to ‘sensory-specific satiety’).

Furthermore, appetite for food is specified by the eater’s recognition of edible material (Booth, 1994). Hence the satiating of that appetite has to be a response also to food stimuli -- an eating-inhibitory response to all foods in the case of generic satiety and to particular sensed characteristics in the case of specific satieties (Booth, 1985).

Hence, associatively learnt satiety is the result of pairing of the combination of a food cue and an eating-generated bodily or social cue with a consequence that aversively conditions classically or reinforces instrumentally (by punishing, not by negatively reinforcing: Jarvandi, Booth & Thibault, 2009). Learnt satiety must be distinguished both from a learnt sensory aversion (or avoidance, nausea etc.) and also from other consequences that are associative, such as poor conditioning of preference, or are non-associative such as eating that has been habituated to immediately preceding food.¹

Back to basics on the sizes of meals

For decades, research into mechanisms influencing the amount eaten on a particular occasion (‘meal size’) has been gravely weakened by treating intake as a measure of this or that influence. On the contrary, intake cannot measure any mechanism. The size of a meal does not provide any account of eating; it is a result of eating that requires explanation in terms of the influences on it, and each mechanism requires identification by its own measure. This was pointed out in the behavioural literature at least 50 years ago (McCleary, 1959) and has been regularly reiterated since (e.g. Booth, 1977, 1981, 1990). Some basic points now follow against mechanistic interpretations of the size of meals without any within-meal analysis.

Meal size always confounds sensory influences with somatic influences

The size of a test meal is invoked as the primary measure of ‘palatability,’ conceived as an invariant personal preference for a food, and of ‘satiety’ using the term merely as a label for an arbitrarily timed test of intake or rated fullness. Both these interpretations can’t be right. In fact, neither is. Even on the simplest theory, sensory facilitation from the food determines the starting of the meal and somatic and/or social inhibition from consumption of food determines the meal’s ending. Furthermore, it is obvious that sensory, somatic and social effects on eating all change rapidly during a meal.

Therefore, ways have to be found of measuring separately the momentary influences on the tendency to eat an available foodstuff from whatever are at that point in time the externally sensed characteristics of the material(s), the internally signalled somatic effects before and after ingestion and the social impact on amount consumed, both interpersonal (e.g., from current companions) and cultural (e.g. from dieting customs or marketers' labels or logos). Then also we need to diagnose the interactions in the mind of all the major influences of the moment (Booth & Freeman, 1993; Booth, Konle & Sharpe, 2008; Galea *et al.*, 2008).

Hence when one source of influence is varied alone, its effects interact in unmeasured ways with the other sorts of influence. Too often, the test foods are not even designed to vary independently in sensory and somatic effects at any stage.

As a result, many reports have misinterpreted learnt differences in meal size as evidence for or against conditioned satiety, when in fact all the evidence provided points to purely sensory effects, with no somatic involvement, i.e. conditioned aversions or preferences or discriminative avoidance or approach. Sometimes the observation of a conditioned preference has even been taken as evidence against conditioned satiety. Bolles *et al.* (1981) were careful not to make this mistake. However, this first study by another group of learnt effects of ingestion of starch in rats had several other flaws that have been repeated in the designing and reporting of subsequent animal and human studies, including erroneous assumptions in the report's title, even if not in its text.

Satiety not conditioned by carbohydrate density or amount

Bolles *et al.* (1981), like Warwick and Weingarten (1996) and Yeomans *et al.* (2009), presuppose that the amount or density of energy or carbohydrate conditions satiety. On the contrary, the evidence is that the conditioning consequence is an osmotic effect -- nothing to do with the metabolic destination of ingested materials (Booth & Davis, 1973; Booth *et al.*, 1976). Rapid digestion of a starch product such as maltodextrin (MD) generates a hypertonic solution that creates an aversive associative effect shortly after the training meal. This conditioner of inhibition of eating is weak, and so it can act only over a short delay. Hence the acquired reduction in appetite becomes elicited in future by the most recent combination of food stimuli and gastric distension – that at the end of the meal.

Hence the amount of MD is irrelevant to conditioning of satiety. All that matters is the concentration of MD in the first portions of the meal and that this

initially eaten material gets through to digestion before the products of digestion stop the stomach from emptying rapidly. The concentration of MD must be high enough to create hypertonicity when digested to maltose and then glucose. Booth *et al.* (1982) found 25% MD to be sufficient. In rats, concentrations as high as 50% have been used (Booth & Davis, 1973; Gibson & Booth, 2000).

Nevertheless, MD-conditioned satiety is the only nutrient-generated eating-inhibitory associative mechanism that has yet been found. Like all satiety, MD-conditioned satiety contributes to homeostasis. However, this associatively aversive effect of MD has never been proposed to be compensatory as Zandstra *et al.* (2002) assume (as also do Yeomans *et al.*, 2009). Nothing in a meal has the power to correct the size of that meal fully for an increase in its energy content though density or portion size. Furthermore most of the high energy content used by Zandstra and others (2002) was fat, and the rest was sugar, not maltodextrin.

Nutrient-conditioned preference vs texture-conditioned aversion

Bolles *et al.* (1981) indicated in the title of their paper that the acquired greater intake of the nutrient-richer food that they observed was a learnt preference. Yet, as they acknowledge in discussion, the sharp disparity in intakes was a learnt aversion to the nutrient-poorer food -- not from its failure to replete but from the aversive texture of the non-nutritive filler (Booth & Thibault, 1999).

The use of MD or longer-chain starch products rather than sugars is critical to avoiding osmotic effects in the mouth and throat. These are so aversive that they stop rats from drinking a very sweet solution of glucose or other sugars (Booth *et al.*, 1972). So long as the burn in the throat and also extreme swelling in the stomach (Booth, 1979) are prevented by the use of chains of glucose molecules as in MD and starch, all concentrations and amounts of carbohydrate condition preference to any taste or aroma with which they are associated (Booth *et al.*, 1972; Sclafani & Ackroff, 1994). Hence, the strongest tests for conditioned satiety contrast the fullness-dependent learnt loss of preference with an emptiness-dependent conditioned appetite or uncontextualised conditioned preference early in the meal or before it starts. In interpreting the learnt changes in sizes of meals that they see, Warwick and Weingarten (1996) and Davis, Smith and Singh (2000) do not allow for aversive conditioning by the actions of hypertonic sugars above or in the stomach or the appetitive conditioning by small concentrations or amounts of sugar below the stomach and so incorrectly attribute the changes in intake to conditioned satiety.

Contextualised intakes, rates and ratings can all measure satiety

The first fully satisfactory evidence for conditioned satiety was obtained in rats, by using the amounts each animal ate during very brief access to a choice between samples of a liquid food differing only in the mixture of tastes in them (Booth & Davis, 1973). The first evidence in people came from differences between distinctive foods in intake during the earlier and later parts of the meal (Booth *et al.*, 1976). This direct measure has regrettably seldom been used for any mechanistic analysis – an important exception being the investigation of glucose-conditioned flavour preference in rats (Sclafani, 2002). Brief choice is laborious to run but, if it is the only way to get decent data, why is it not used more often?

The paper defining the term ‘conditioned satiety’ (Booth, 1972) relied for its evidence on grouped rates of eating during a fixed period of access to a single test food, compared between foods differing in flavour-composition pairing. Rates at the start of access confound latency to eat with rate of eating as such. Worse, averaging intakes across eaters in the later stages of access creates an artefactual ‘rate’ from different times at which individuals stop eating.

Unfortunately, rates of eating within meals are insensitive to normal variations in sensory, somatic or social factors (in rats: Booth, 1978, page 239, Table 1; in people: Booth *et al.*, 1976). When the test meal is restricted to a single food, it seems that rates are affected only by variations in unusual ingredients, i.e. degrees of dislike of having a meal on that product.

Because brief choice intake and rate of intake in human participants were poor measures of normal influences, including learnt ones, in the mid-1970s we introduced ratings of the momentary disposition to eat. These have the major advantage of not affecting the processes that they are meant to be measuring -- at least, not as severely as intake measures do. Human beings have exquisitely precise capacity to express behavioural dispositions symbolically when given the opportunity to state their attitude in publicly agreed quantitative terms. So we constructed as wide a range as we could of wordings for the current desire to eat in order to rate the momentarily more or less sated appetite for food, either in general (e.g. ‘hungry’, ‘full’: Booth, 1976) or specifically to a food that was present or remembered (Booth *et al.*, 1982). Central to the technique was the presentation or naming of small portions of widely used foods appropriate to eat at the time of testing (as in the purely behavioural experiment of Booth *et al.*, 1976) and asking what the person would eat at that time

(Booth *et al.*, 1982; Dibsall *et al.*, 1996; O’Leary *et al.*, 2006) or, more vaguely, how pleasant it would be to eat some of each food at that time. Rated pleasantness of foods proved to be the most sensitive measure of wanting to eat or likings for foods (not necessarily accompanied by any pleasurable sensations), as influenced through either physiological or social cognition (Booth *et al.*, 1982).

Thus, to provide the conclusive contrast of conditioned appetite or preference with conditioned satiety, just as Booth and Davis (1973) used brief choice tests at the start and end of the test meals in rats, so Booth *et al.* (1982) used rated pleasantness of both the test foods before and after eating. Clearer results were obtained when the pleasantness ratings were not complicated by differences in overall intake and in the portion-size norms or habituation for particular foods (Booth & Toase, 1983; details in Booth *et al.*, 1994).

Unfortunately, the uses by other groups of brief choice intakes, rates of intake and ratings of pleasantness have not monitored any somatic component of the inhibition of eating by eating. Yet, by definition, bodily state towards the end of a meal is integral to conditioned satiety (Booth, 1972). Furthermore, in biomedical circles at least, satiety is conceived as a state that depends on signals from the body to the brain. Against this assumption, it should be noted that purely social inhibition of appetite as a result of eating is not only equally viable logically as a (learnt) mechanism of satiety but some have argued that it is often the only mechanism. An example is the ‘boundary’ theory of the ending of meals in conventional dieters (Herman & Polivy, 1983). However, the evidence so far from amounts eaten confounds potential physiological cues with the culturally informed self-control: Cecil, Francis and Read (1998) showed that social information as well as gastric and post-gastric states can influence amount eaten. The challenge is to investigate the role of learnt levels of signals from the digestive tract in the individuals’ uses of socially set thresholds of satiety.

Interpretation of indirect evidence for learnt satiety

Uncontextualised ratings of appetite

Yeomans *et al.* (2009) did not design or interpret their verbal tests for the disposition to eat in a way that measures a state of learnt satiety. Assessing current liking of a food in only one state, whether at the start of the meal (as Yeomans and colleagues chose to do) or at the end of the meal (as necessary to measure satiety),

gives a measure of relative preference/aversion without context, which therefore is irrelevant to any difference between internal states of hunger and satiety.

This strategy also conflates momentary pleasantness with the unworkable notion of a fixed liking for or palatability of a food. It is nonsense to ask how much steak is liked as dessert or icecream as a main course.

Furthermore, it is redundant to rate “hunger” and “fullness” as well as pleasantness of foods, because these are merely other words for the strength of appetite for food. The scores are found to be highly correlated whenever they have been properly analysed. In Yeomans *et al.* (2009), as in other reports presupposing that different words measure different “sensations,” intake (Figure 1A) and changes in pleasantness (Figure 3), hunger (Figure 4B) and fullness (Figure 4D) all show the same numerical pattern over the four conditions, indicating acquired averting of the flavour of the larger meal containing concentrated maltodextrin.

This neglect of state-dependency is surprising since this group showed that the sensory preference conditioned by pharmacological effects of caffeine is dependent on a state of deficit of the stimulation of receptors in neural systems that have become tolerant of caffeine through heavy use (Chambers, Mobini & Yeomans, 2007), i.e. a substance-specific appetite for caffeine, exactly the same as the conditioned appetites for protein and carbohydrate shown by comparing sensory pleasantnesses or intake preferences between depleted and repleted states (Booth *et al.*, 1994; Gibson *et al.*, 1995). Conditioned satiety is an exact parallel to conditioned appetite: when the same sensory cue is used throughout the meal, then the only cue controlling the difference in behaviour is the relative state of repletion/depletion at some site in the body.

Sensory sating

Sensory-somatic inhibition of eating by eating cannot be measured before the inhibitory eating. Nevertheless, purely sensory sating might be detectable. However, the known cases either are not associatively acquired or are specific to one type of sensory input.

A flavour can have a sickly effect, particularly an intense sweetness (Booth *et al.*, 1972; Lavin *et al.*, 2002). Yet when a person is hungry this can only kill off the hunger. Equally, it only intensifies satiety when some inhibition has developed from eating. There is no evidence that it is state dependent.

The sickliness of sweetness might be conditioned nausea, acquired by the pairing of the taste of a sugary food with some emetic or disgust after-effect. It might

be unlearned. In either case, the phenomenon is not conditioned sensory-gastric inhibition of eating.

Sensory-specific satiety may have a non-associative component (habituation) and learnt social component such as norms for the amount eaten of a particular food within a meal. In either case, this form of satiation has nothing to do with inhibition from the learnt combination of flavour and degree of gastric distension.¹

Implicitly expected satiety?

When the wordings of questions are inappropriate to the circumstances, cooperative participants make the best sense they can of the task they are set. One intriguing possibility is that the conditioned satiating pattern of pleasantness ratings before the test meal seen by Yeomans and colleagues (2009) is an implicit expression of the decline in the wanting of food that is expected by the end of the test meal.

Such food-specific and context-specific expectations of satiety (which of course have to be learnt) can be precise enough to detect physiological after-effects introduced by changing the nutrient contents of a familiar food (Dibsdall *et al.*, 1996, 1997). Answers to vague questions about the extent to which “a large amount” of unspecified foods might be eaten at an unspecified time in the future merely express current appetite and will be very insensitive to any specific factor influencing prospective consumption. On the other hand, differences in physiological or culturally conceived influences on current appetite are detected more precisely by the average “pleasantness” of eating each of several staple foods than by ratings of “hunger” or “fullness” (Booth *et al.*, 1982). Hence the pleasantness of a test food rated in any context might well have the capacity of reflecting all learnt consequences of eating it.

Indeed, looked at in that way, the ratings obtained by Yeomans *et al.* (2009) are strong evidence for implicitly expected satiety.

Experiments in the laboratory and mechanisms in everyday life

The many experimental demonstrations of classical conditioning of satiety in rats, monkeys and people using high concentrations of a rapidly digested derivative of starch must be distinguished from the general theoretical position that loss of sensory-somatic appetite through aversive conditioning contributes to the ending of many meals in human beings, alongside contributions from portion-size norms, self-presentation to companions, sensorimotor-specific habituation, boredom and perhaps

other processes (all of which remain relatively underinvestigated mechanistically: Booth, 2008).

Starch-conditioned sensory-gastric inhibition

The only physiological mechanism so far suggested for an aversively conditioning effect of starch derivatives (described above) requires the consumption of at least a small amount of food or drink containing a strong solution of maltodextrin, at a time when emptying of the stomach is not inhibited by signals from nutrient in the small intestine remaining from previous eating.

Maltodextrin is widely used as a calorific but minimally sweet thickener in foods such as meat pies. However its concentrations in such products are not nearly as high as the 25 g per 100 ml that seems to be needed in order to generate the aversively conditioning stimulus or indeed a motivationally aversive state such as discomfort, cf. Booth *et al.* (1982) and O’Leary *et al.* (2006).

The nearest approach is the crumb of freshly baked bread, which has over 40 g of readily digested starch per 100 g of aerated gel. If a small roll or slice were eaten on an empty stomach at the start of a two-course meal, it might well condition inhibition of ingestion to the combination of the sensory characteristics of the last food eaten and a degree of distension of the stomach achieved at the end of the ‘training’ meal(s) (Booth *et al.*, 2007)..

Any other learnt satiety

If satiety in life is learned in any other way, it could be via other associatively aversive consequences of normal eating. This learning must be strong enough to counter classically conditioned, instrumentally reinforced and socially learnt facilitation of eating, in the presence the specific combinations of food characteristics and repletion signals that recur towards the end of meals. On the other hand, it must not be so strong as to condition aversions, nausea, disgust or fear to foods in the presence of a relatively empty stomach early in meals or independently of somatic context altogether.

It may be that sufficiently large first courses containing substantial amounts of rapidly digested forms of macronutrients can generate these moderately aversive conditioning stimuli, or implicit or explicit expectations of other untoward consequences, such as fattening (Booth, 1987). Unfortunately it is very hard to disconfound the crucial gastric cue in conjunction with the food cue by comparing

large and small portions of a single food -- hence the difficulty in interpretation with which this short review started.

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Footnote

¹ There should be no confusion between the two phenomena called ‘sensory-specific satiety’ (SSS) and ‘conditioned satiety,’ let alone arguments that one depends on the other. By operational definition, SSS is non-associative, dependent solely on immediately prior exposure to the satiating food stimuli, while conditioned satiety is associative, dependent solely on prior contingency of consequences to cues from food and from context, that can be remembered over days and is evoked immediately the conditioned combination of sensory and somatic or social cues is reinstated, with no immediately prior exposure to that food being required. SSS can be measured by the food-variety effect on amount eaten or by the decline in the pleasantness of eating the test food as its consumption progresses, with constant rated intensity of its sensory characteristics as control for sensory adaptation.