

The highlight for November is by David Booth in the School of Psychology at the University of Birmingham in England. When reflecting on the individuals who have made impacts on the general field of taste aversion learning, I have always included Dr. Booth as one of the major players. It is interesting in this context that while much of his research has addressed conditioned taste aversions (see his careful extensions of the range of stimuli effective in serving CS and US functions), it is his work on feeding (and not necessarily its suppression) for which he is best known. For almost four decades, he has explored the associative factors involved in feeding, focusing primarily on how conditioning might increase and decrease food intake. Interestingly, this history with (and interest in) conditioning has allowed Dr. Booth to view feeding from a different perspective than those assessing the conditioned suppression of food intake. What is often viewed as an aversion may instead be satiety; consequently a conditioned suppression could reflect either process and would require additional investigation to determine which of these factors mediated the change in behavior. As he describes in his highlight, aversion learning is simply one procedure effective in modulating food intake. Over the years, he has argued quite convincingly that food regulation involves much more than the restraining influences of food aversions. Conditioned food preferences and conditioned satiety initiate and terminate feeding (and in a manner quite different than that effected by aversions). His work has been characterized by careful scrutiny of all of these multiple factors and the experimental conditions under which they are acquired and expressed. He has discussed these multiple factors not as simple elements (or atoms) that sum to control behaviour but as configural stimuli that go into multiple (horizontal and vertical) associations that ultimately control feeding. Feeding is complex; however, its understanding has been greatly increased by the work of Dr. Booth and his colleagues who have documented and evaluated its conditioned (and unconditioned) control and the manner by which this control is itself modulated by the multiple factors involved.

Conditioned taste aversions and the learnt controls of food intake

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Delay between cue and consequence

John Garcia's discovery of long-delay learning liberated my work on hunger and its satisfaction from the impossible constraint of cue-consequence intervals no greater than a second or so.

Generalist eaters could not survive if the fuels and essential building blocks in diverse foods had to be detected within seconds, or even in the few minutes of the briefest bouts of eating. The contents of a meal take an hour or two to leave the stomach (in which nutrients are not sensed). So it is hours before the brain could gather all the information about digestible carbon compounds (carbohydrate, fat and protein) and essential nitrogen (some protein amino acids) that is needed in order to select adaptively among the edible offerings of nature or of human ingenuity.

Nevertheless, the evidence then accumulating in my laboratory (plus, I should confess, what I viewed as plain commonsense) made me very doubtful of any idea that unlearnt hunger drove us and non-human omnivores to eat anything and everything, restrained only by conditioned taste aversions (CTAs). For the reasons stated next, I preferred the term 'toxiphobia' to Garcia's CTA and also proposed 'nutriphilia' as a name for the much more versatile set of long-delay learning mechanisms that accounts for normal choices and intakes of foods and calorific

drinks. This appetite for food is much more than uncontextualised aversive conditioning of reactions to gustatory stimuli by stimuli from gastrointestinal upset.

Indeed, was the reduction in intake of a fluid after pairing with a poison actually an aversion conditioned to a taste in the fluid?

Was CTA conditioned?

My group's first publication on flavours and poisons pointed out that the (still) standard design for testing acquired aversions to tastes could be confounded by non-associative effects (Pain & Booth, 1968). There should be a delay between training and testing long enough to ensure that malaise from the poison has fully worn off.

Far better, a discriminative design can be used, in which the rat is given a choice between the poison-paired taste and a control taste. If saccharin must be used to motivate sufficient fluid intake, then either a sour tastant or a bitter tastant can be mixed in, at quite low concentrations – sufficient to be discriminated but not enough to reduce saccharin intake markedly: cp. Booth & Davis, 1973; D'Mello *et al.*, 1977.

Was CTA taste?

Secondly, it is very doubtful that toxiphobia is confined to taste. The innate ingestive reflexes to the taste of saccharin ensured that rodents took in fluid in an amount that could be decreased by learning. However, many foods eaten in large amounts have no taste like sugar, and the other classic tastants are aversive (except for salt at very low concentrations in animals that are not sodium-depleted). Furthermore, many foods are distinguished not by taste but by aroma, texture, colour and shape, and indeed their attractions lie largely in distinctive combinations of those modalities with complex mixtures of tastants. Indeed, work on such learnt configuring of multisensory combinations has proved crucial to the understanding of ingestive appetite (of which more below).

Pain and Booth (1968) were primarily interested in showing that poisoning conditioned olfactory aversion or avoidance, not just gustatory. Glucose solution was used as a carrier but its sweetness was far too low for the later-discovered phenomenon of taste-potential to account for rejection of the conditioned odour. Claims that the odourant was tasted could be dismissed, as even in oil form it was tasteless on the human tongue. Gustation was conclusively excluded by a reversible loss of conditioned olfactory aversion when a nasal tube was used to bypass the olfactory mucosa (Baker & Booth, 1989).

After many experiments had demonstrated that nutritional consequences to olfactory cues induced aversions and preferences, a notion grew that nutrients conditioned olfaction whereas toxins conditioned taste. This was scotched by showing that the aversion conditioned to odour by lack of an amino acid that is essential to growing rats could be conditioned to taste as well (Booth & Simson, 1974).

Was CTA aversion?

Thirdly, it is far from obvious that the reduction of intake of poison-associated saccharin solution is the result of classical conditioning of respondents (or involuntary reactions, to use ordinary language) such as ingestive movements. If so, the saccharin is a conditioned stimulus (CS) that becomes associated with the aversive unconditioned stimulus (US) generated by lithium chloride, X-irradiation or a (strange) drug. The almost negligible intake of saccharin solution after pairing with the poisoning could also be explained as poison-reinforced (punished) instrumental escape from the drink's flavour and avoidance of the drinking tube, perhaps after the tube's contents had been sampled or with a conditioned discriminative stimulus (S^D) of vapour from the saccharin solution or of the sight or feel of the spout. Garcia's

terminology of ‘cue’ and ‘consequence’ has the advantage of equivocating between CS or S^D and US or reinforcer.

It has never been clear how much of eating and drinking is respondent and how much is operant (intentional acts, in philosopher-speak). It proved remarkably difficult to use the bodily effects of withholding food as an aversively conditioning stimulus (Simson & Booth, 1973a). Only very recently it has become evident that hunger can be effective as an instrumental negative reinforcer of discriminatively avoidant eating at the same time as the control condition involving nutrient-repletion conditions preference for the control stimulus – a difference between cued intakes in the opposite direction (Thibault & Booth, 2006; Jarvandi, Booth & Thibault, 2007).

The saccharin model of ingestion

That said, the rat’s drinking (or not) of a solution of saccharin is an excellent tool for basic and applied studies of toxiphobia. Saccharin was a problem for me though in the mid-1960s as I began to settle into research on ingestion, first with Neal Miller at Yale and then back in the UK at Sussex. Leaders in that area regarded activities evoked by the sweet taste as the prime model of appetite for food in all its aspects. Furthermore, psychologists for decades had maintained laboratory rats on an unnatural single food while doing experiments on “feeding”, meal sizes, satiety and reward with solutions or pellets of saccharin or glucose.

I had barely disentangled myself from the resulting screw-ups (e.g. Booth 1972a; Booth, Lovett & McSherry, 1972) when leading lights in research into eating by human beings began to use saccharin solutions as models for pleasure from food and sensory preferences among foods and drinks. Nonsense of this sort recurs to this day, despite decades of data to the contrary (such as Booth, Mather & Fuller, 1982; Booth, Thompson & Shahedian, 1983; Gibson & Booth, 1986, 1989, 2000) and frequent reviews (e.g., Booth, 1985; Booth, 1991; Booth & Thibault, 1999).

Saccharin is such a terrible model for normal eating and drinking because sweet materials are consumed in a congenital reflex that has nothing to do with calories (very few sources of which are sweet) or even micronutrients in ripe fruit (available from other plant materials). My view is that the taste of sugars motivates and reinforces sucking and swallowing in the infants of omnivorous mammals because it stops them spitting out mother’s milk. For these species to thrive, once the young become mobile, an innate reflex is needed to get a poisonous material out of the mouth the first time it is tasted (Booth, 1991). Gustatory receptors for the nitrogen in plant alkaloids that drive expulsion will however also detect the nitrogen in milk’s rich content of peptides vital to gut immunity and body growth. So, receptors for bitterness **were countered by selection of genes for receptors** sensitive to the oxygen in sugars, **that** drive reflexes to sweetness **which** are ingestive rather than egestive.

Sweetness even elicits linguo-facial movements independent of swallowing that rub the nipple to provoke let-down of milk and maybe even gather up drops that do not go straight in. In any case, this tongue protrusion and lip licking must have some social, i.e. communicative, role if they are a sign of emotion (pleasure) separate from ingestive motivation (appetite).

The appetite/satiety learning model of ingestion

Ironically, the raw sugar solutions on which psychologists’ work on satiety in rats had been based since the 1950s proved to have highly aversive conditioning effects on intake of solutions strongly or weakly sweetened with saccharin and/or sugars. Jacobs (1958) had observed a remarkable switch from taking nearly all glucose from a very sweet concentrated solution to taking most of their glucose from a much less

sweet dilute solution in rats given continuous access to the two solutions. I struggled to fit my initial data to Michel Cabanac's proposal of innate nutrient-specific satieties. After my talk on a long series of puzzling results in Haverford PA at the 1968 meeting of the Food and Fluid Intake series, Harry Jacobs referred me to Le Magnen's first reviews of his work in Paris since 1955 on learnt controls of food intake, inspired in part by Rsózka's (1954) evidence that 'bait shyness' in rodents depends on learning.

Le Magnen had paired odours mainly, but also tastes, textures and even colours, with the administration of agents that affect appetite and monitored intakes in single-stimulus and choice tests. Unfortunately none of the appetite-related USs in the doses he used condition preference as he had expected. Glucose (even when tubed to the stomach or injected under the skin: Booth, 1979), insulin (Lovett, Goodchild & Booth, 1968) and amphetamine (D'Mello *et al.*, 1977) all have aversive effects, as others also have shown – with the reduction in intake mediated by varied types of respondent too (see, for example: Parker, 1980; Limbeer, Hall & Parker, 2006). Only when mild doses of naturally occurring nutrients are administered does conditioning of preference occur. There is always a limit on a benefit: beyond the optimum, it becomes too much of a good thing and may even become harmful.

In addition, we have recently reexamined Le Magnen's claim that rats learn to eat more before long periods of food deprivation, as well as eating more and more immediately after deprivation on a cycle. The effect is there when lengths of pre- and post-prandial fasts are disconfounded, but it is instrumental: intake **increases** of a food cuing hunger because a depleted supply of energy or nitrogen acts as a negative reinforcer – intriguingly, in a battle with an increase in intake of the control food cuing repletion before hunger arises, because that conditions preference (White *et al.*, 2000; Thibault & Booth, 2006).

Nutrient-conditioned flavour preferences

Relative acceptances

The recommendation of discriminative paradigms to distinguish associative effects from protracted motivational change (Pain & Booth, 1968) was somewhat obscured by subsequent arguments whether or not two-stimulus tests are more "sensitive" than one-stimulus tests. The proper way to conceptualise such comparisons is as simultaneous or successive presentations of two stimuli. Although the simultaneous procedure has been called 'preference' and the separate testing 'acceptance', both results should be called relative acceptance – or indeed relative acceptance/rejection, since even increases and decreases of intake of a single sample do not by themselves distinguish decreased appetitive from increased aversive contingencies. For example, when all the behaviour being studied is basically appetitive, it is quite feasible to have aversive conditioning without conditioned aversion resulting: all that need happen is a conditioned reduction in preference.

Our analyses of the Jacobs glucose switch (Booth *et al.*, 1972) and also the loss of appetite with imbalanced amino acid mixtures (Booth & Simson, 1971, etc.) showed strong preference-conditioning effects of digested carbohydrate and protein on a great variety of tastes and smells. Aversions were found with acute deficiencies of essential amino acids but repletion conditioned preferences that were equally strong or stronger. This contrasts with attempts to 'cure' chronic deficiency in a vitamin or an essential mineral: such animals are so ill that it must be very difficult to create any appetitively associative state.

Both good sense and hard evidence point to at least some and probably most food-selective activities being based on preferences, not aversions. Yet some psychologists denied this for a long time because they were over-impressed by the weakness of

preferences induced by association with recovery from poisoning or long-term vitamin deficiency.

The fact is that rats and human beings hardly have to miss one meal for glucose or essential amino acids to increase subsequent acceptance of food having a flavor or texture presented up to an hour or more before the nutrient reaches the intestine. For amino acids, the first evidence was found in rats by Booth and Lovett (1970). Protein-conditioned preference was shown to extend to our species by Gibson, Wainwright and Booth (1996). For glucose, the first evidence was in the final experiments of Booth, Lovett and McSherry (1972), replicated by Holman (1980) and greatly elaborated by Sclafani and co-workers since. In people, carbohydrate-conditioned flavour preference was first reported (among other phenomena) by Booth, Fuller and Mather (1982) in adults and replicated in children by Birch and Deysler (1986). A clear demonstration when appropriate conditions were instated was recently achieved by Brunstrom and Mitchell (2007).

Indeed, in people at least, it might be questioned if there are any genuine (or ‘absolute’) sensory aversions for ‘real’ foods or drinks. Even the revulsion for *sauce béarnaise* created by illness afterwards, the aversion to alcohol induced by antabuse or the disgust elicited by stirring a just-used comb in a drink may not remove the attractions of the food or drink, especially if the context is changed enough.

In rats, though, it is now clear that the orosensory/gastric-specific satiety conditioned by highly concentrated maltodextrin (MD) can involve true aversion, assuming that olfactory neophobia is a genuine aversion to the novel odour. When the aversive effect of concentrated MD comes close in time to ingestion of a food odour (which thereby ceases to be novel), the paired odour is selected in a smaller amount than the novel in a later choice between odourised test foods (Gibson & Booth, 2000).

Conditioning of preferences by mixtures of nutrients

The above experiments were done with a single nutrient at a time. However most foods are mixtures of nutrients and many meals are combinations of foods. The effects of a nutrient on preference for a flavour may vary with the nutrient. How can the brain sort out from the postingestional signals which consequence to associate with which cue? There is nothing in the digested nutrients to indicate which flavour of food it came from, let alone which texture or colour.

Except for analytically impossible designs like ‘cafeteria diets’, mixed meals are very seldom fed to lab rats. Even when the diets were limited to two or three, the cues and their association with reinforcement were never considered and the rats were supposed to do “macronutrient selection” by magic, both normally and under the influence of drugs and hormones (Thibault & Booth, 1999; Booth & Thibault, 2000).

Perhaps the simplest solution would be for each nutrient in the mixed meal to condition preferences to the cues in the meal regardless. The result would be “proportional reinforcement” of acceptance of all the foods. This hypothesis has been supported (Baker & Booth, 1989).

However such a mechanism does not account for differences in roles of main course and dessert foods or indeed for the very basic fact that meals end, however ‘palatable’ the foods are.

Appetitive and aversive postingestional consequences of the same nutrient

It does not seem possible for a sequence of cues to be sorted to match a sequence of consequences. This is because learning over a delay between cue and consequence is a mechanism with a variety of speeds of decay of reinforcement with time. At short delays a reinforcer over long delay may be as effective as a reinforcer that only works over short delays.

However the picture changes if one source of reinforcement has both appetitive and aversive associative effects. Just such a property of rapid release of high concentrations of glucose in the wall of the duodenum turned out to be the mechanism of conditioning contextualised increases and decreases in preference, i.e. learnt appetites and satieties (Booth & Davis, 1973; Booth, 1985).

Thus far, all the evidence for an orosensory/gastric configural CS with a CR of reduced acceptance (i.e. a conditioned satiety) comes from pairing cues in the meal with the after-effects of ingesting maltodextrin (MD: a highly soluble product from starch, brand name Polycose^(R) in the USA). The glucose released by digestion of MD in any concentration conditions a preference in rats for whatever concentration of sweetener precedes it (Booth, Lovett & McSherry, 1972), shown to be very powerful by Tony Sclafani and colleagues (using different odours as CSs). Yet free glucose at concentrations in the stomach over about 25% conditions avoidance of or aversion to whatever sweetener concentration is paired with it (Booth *et al.*, 1972). These opposing associative effects of the same substance are readily explained, e.g. by the aversive effect being osmotic and the appetitive effect being chemospecific.

What is tougher to explain is how concentrated MD can condition increased acceptance of a flavour in the early stages of a meal and decreased acceptance of the same flavour towards the end of that meal. The answer turned out to be in the weakness and transience of the aversive consequence. Whatever the capacity for delay, the longer the time over which a consequence can act the weaker is its associative effect. Therefore only the most recent flavour can be conditioned aversively – that presented at the end of the meal. The strong preference conditioning effect of glucose from MD, on the other hand, acts on flavours from the start of the meal.

This mechanism is not enough to explain aversive conditioning at the end of the meal and appetitive conditioning at the beginning when the food has the same flavour throughout. The explanation then has to be compounding or even configuring with cues specific to the start and end of a meal. The most obvious of these is degree of stretch in the wall of the stomach, and so it proves to be in rats, monkeys and students (Gibson & Booth, 1989, 2000).

As well as training amounts eaten, concentrated MD conditions down the rated pleasantness of a flavour presented at the end of the meal. No evidence of a conditioned unpleasantness has yet been seen in grouped data from people. We do now have preliminary evidence though that concentrated MD does create the postulated visceral discomfort 10-20 minutes after ingestion on an empty stomach (O'Leary, Li, Higgs, Booth, 2006).

Roles for food aversion and avoidance

A major argument for the focus on taste was the fact that gustatory pathways converge with vagal nerves from the viscera in the nucleus of the solitary tract and Garcia was convinced that the aversive consequence was nausea. Apart from the evidence that olfactory CSs work (and olfactory afferents do not project to the NTS), the main weakness in this argument is that the visceral pathways from reverse peristalsis would have to converge on the neurons that receive afferents from gustatory receptors in the mouth; both bundles of nerves entering the NTS, even the same part, is not sufficient. So far as I know, this still has not been shown.

Indeed, many drugs that have no effect on nausea have since passed the CTA test in rats. For example, we showed that several centrally acting appetite suppressants create aversively conditioning USs and that the resulting CSs are avoidance-

sustaining conditioned reinforcers (Booth *et al.*, 1977; D'Mello *et al.*, 1977; Stolerman *et al.*, 1977).

Furthermore, the questionnaire study that has been taken to show that gastrointestinal upset in human beings is the sole cause of food aversions (Pelchat & Rozin, 1982) has been shown to be confounded by suggestive sequencing of questions. When questioning focusses first on the range of adverse symptoms that the respondent has experienced, and when the nature of a “dislike” blamed on a symptom is distinguished by the respondents between sensory aversion and fear of the food, rated dislike of a named food is as strongly associated with hangovers from consumption of alcohol as it is with GI upset (Knibb *et al.* 2000).. Moreover, the GI-associated dislike is as much fear as aversion (Knibb *et al.* 2000). Again there may be no genuine sensory aversion, only a loss of sensory preference (and perhaps dependent on perceived context).

Sensory-Somatic[-Social] Configuring

I should not give the impression that in the late 1960s I had more than a glimmering of ideas, let alone evidence, on the mechanisms of nutraphilia or learnt appetites for foods.

In particular, the contextualisation of learnt sensory selection among foods was not clear in the data until 1973 (Booth & Davis, 1973) and settled in my own mind until 1975, emboldened by the data in Revusky (1968) on thirst-dependent sensory control of fluid intake and Sam Revusky's own sharp but liberal approach to theories of learning, at a mini-conference he organised at Memorial University, where Tony Riley and Linda Parker also sampled the delights of Newfoundland (only 3 hours and 25 minutes off UK time). Not until 1983 was there (later published) evidence for appetitive and aversive conditioning of configural CSs comprised of a volume in the stomach and a strength of odour or colour in the food, in monkeys (Booth & Grinker, 1993) and in people (Booth & Toase, 1983; in full, Booth, 1994).

This theory couched in the terminology of behaviour processes is in fact also a theory of the recognition of foods and drinks as objects and of eating and drinking occasions as perceptual situations (Booth, Gibson, Toase & Freeman, 1994). An appetite (or satiety) for taking a mouthful (or not) of one of these small objects is at its optimum when the particular levels of the sensed characteristics of that material, the signalled state of the body and social appropriateness of a meal or snack, all are at the level at which facilitation of the act of eating (or its inhibition) is at a maximum – the ideal point.

Atomistic versus psychophysical learning theory

This generalisation from configural CSs to multimodal ideal points is crucially dependent on an unavoidable implication of the Jacobs glucose switch. The CS is not the taste of glucose; it is a particular level of sweetness. This point is independent of how much the learnt switch in relative acceptance is induced by aversive consequences of osmotic effects or appetitive consequences of nutritional effects. That is, the stimulus actually conditioned is at a peak of a function of strength of response against concentration of sweetener. On both sides of that level of sweetness, the learnt response declines. This is what behaviorists call a stimulus generalisation gradient and introspectionists call a [dis]similarity function. From an objective cognitive-behavioural (performance) viewpoint, the peaked psychophysical function is a vertical cut through a cone, with another cut at a right-angle being the function for recognition of the learnt context (Booth & Freeman, 1993).

This quantitative approach contrasts to the qualitative approach of current learning theory of configurational stimuli. Instead of exploiting graded intradimensional

generalisation gradients, each element in a compound stimulus is treated by contemporary learning theorists as a discrete category or ‘atom’. Hence experimental designs giving current textbook meanings to “intra-“ and “extra-“ dimensional generalisation cannot be used on the configuration of cuing from a sensed food characteristic (at a particular strength) and a degree of distension of the stomach (or of flow of energy between fat and lean tissues, perhaps signalled hormonally to the brain). Current theories of object recognition suffer from the same flaw: features of an object are considered categorically only, not as graded functions of response level on the stimulus level for each feature.

Nutritional reinforcement versus multisensory configuring

Implicit in the above is an important distinction should be emphasised between what Wickelgren (1979) calls ‘vertical’ associations between a sensory CS (or S^D) and a postingestional US (or reinforcer) versus ‘horizontal’ associations between two elements in a compound cue -- i.e., CS-US vs. CS1-CS2 associations.

Holman (1980) showed that the postingestional associative effect of glucose on odour preference can be distinguished from any associative effect of the taste of sugar, by the simple expedient of presenting sugar half an hour after the odour and relying on learning over a delay with a visceral US, instead of presenting the glucose (or it could have been saccharin) along with the odour.

Conditioned appetites and satieties come from both ‘horizontal’ and ‘vertical’ associations acting together: (oro)sensory cues and (entero)sensory cues – and indeed exteroceptive social cues - are associated together into a configural CS (with neither reinforcing the other, nor necessarily eliciting a response); this flavour-fulness[-mealtime] CS is associated with an appetitive or aversive US, conditioning motivation into the CS as Pavlov saw in his dogs. This mechanism has been elaborated in theories of incentive learning.

Zellner, Rozin *et al.* (1983) showed that the addition of sugar to a novel type of tea caused a subsequent increase in rated pleasantness of that tea. Many recent experiments have paired tastes with odours in drinks. The results are plagued with weakness in design (such as presenting many different pairings without prior elucidation of the parameters of conditioning) and importing theoretical assumptions by classifying the effects as “evaluative” conditioning. Little attention has been paid until recently to the possibility that multisensory configurations (‘horizontal’ associations - indeed, perceptual learning) gain control of the already learnt responses to one of the components. The new tea flavour that you get to like is in its sweetened version, as well as or instead of your familiar unsweetened tea. Integration of previously separate percepts has been seen among odours (Stevenson, 2001) and between odour and sweetness, with the aroma becoming rated both sweeter and more pleasant (Yeomans, Mobini *et al.*, 2006). This does not occur when mixtures of odorants are presented without a chance for learning a particular configuration (Laing *et al.*, 2002).

Similarly, when feeling full is a conditioned satiety, i.e. gastrically contextualised loss of sensory preference, the feeling full is for that food. Note that this food-specific satiety is a response to the conditioned stimuli that need not have been presented before then in the meal. This should be distinguished from the food-specific satiety that arises from immediately prior presentation, as in habituated responding (although it may be norms for portion size operating).

Envoi

It is immensely satisfying to a biosocial psychologist to find that a postdoctoral career spent mostly on trying to understand the causes and consequences of patterns

of eating has supported the ancient understanding of human life as a unity across our personal, cultural and biological aspects, while at the same time finding rich affinities with the lives of other species that share the planet.

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