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Booth, David A; Jarvandi, Soghra; Thibault, Louise

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Theoretical paper

Food after deprivation rewards the earlier eating

David A. Booth,^a Soghra Jarvandi,^b Louise Thibault^b

^aSchool of Psychology, College of Life and Environmental Sciences, University of Birmingham, Edgbaston, Birmingham, West Midlands, B15 2TT, U.K.

^bSchool of Dietetics and Human Nutrition, Macdonald Campus of McGill University, 2111 Lakeshore Road, Ste-Anne-de-Bellevue, Québec, Canada H9X 3V9.

Corresponding author: David Booth <D.A.Booth@Bham.ac.UK>

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This theoretical paper is based on research conducted in rats and planned in people presented in a dissertation for the degree of PhD at McGill University by Soghra Jarvandi, MD, now at the Division of Health Behavior Research, School of Medicine, Washington University, St Louis, MO 63108, USA. The seven experiments summarised here were conducted by her (Jarvandi, Booth & Thibault, 2007a; Jarvandi, Thibault & Booth, 2007b, 2009) and two other graduate students, Yehmin Yiin (Thibault & Booth, 2006) and Jennifer White (White, Mok, Thibault & Booth, 2001). The collaborative work in Montreal was supported by a grant to Prof. Thibault by the Natural Sciences and Engineering Council of Canada, with travel funded from infrastructure support in grants for research led by Prof. Booth from Agri-Food at the Biotechnology and Biological Sciences Research Council, U.K.

ABSTRACT

Food intake can be increased by learning to anticipate the omission of subsequent meals. We present here a new theory that such anticipatory eating depends on an associative process of instrumental reinforcement by the nutritional repletion that occurs when access to food is restored. Our evidence over the last decade from a smooth-brained omnivore has been that food after deprivation rewards intake even when those reinforced ingestive responses occur long before the physiological signals from renewed assimilation. Effects of food consumed after self-deprivation might therefore reward extra eating in human beings, through brain mechanisms that could operate outside awareness. That would have implications for efforts to reduce body weight. This food reward mechanism could be contributing to the failure of the dietary component of interventions on obesity within controlled trials of the management or prevention of disorders such as hypertension, atherosclerosis and type 2 diabetes.

Keywords

Overeating. Food reward. Delayed reinforcement. Food deprivation. Skipping meals. Restraint breakdown. Obesity-related disease.

“Highlights” (3-5 required, each with a maximum of 125 charsps)

- Intake of the food eaten before deprivation is increased by the later physiological repletion.
- This reward of eating by assimilation of nutrients is stronger when the deprivation is longer.
- Such a simple learning mechanism in animals is likely to operate unconsciously in people.
- This long-delay food reward process may be making an unnoticed contribution to obesity.

Food rewards long prior food intake: outline of this paper

Our recent research indicates that physiological signals from nutrients ingested after food deprivation increase the subsequent intake of a particular food that was eaten before that fast. Furthermore, this intake-reinforcing effect of repletion strengthens as the duration of the food deprivation increases. The evidence for such a learning mechanism comes from rats. Learning of this sort is therefore likely to be a capacity also of the human brain, operative independently of a person's expectations about the duration of the fast or plans to eat more in order to avoid hunger. Such unconscious reward could be a major factor in the difficulty of reducing intake of energy in overweight and obesity.

First below, we review the existing evidence relevant to different aspects of this new idea of food reward. Then we consider potential theoretical and empirical implications, including the design of experiments to test for such a learning mechanism in human eating.

Evidence for reward of eating responses by food

Food reinforces many sorts of response, in human beings as well as in other species (Epstein & Leedy, 2006). Yet there is remarkably little evidence that eating is itself reinforced by food. Le Magnen (1957, 1999) reported an experiment in which rats increased their intake of a distinctive food presented before 13 hours of withholding their maintenance food; there was both an absolute increase and an increase relative to intake of a different food before deprivation for 3 hours. Two subsequent attempts to increase intake before deprivation (anticipatory eating) were unsuccessful (Ackroff & Sclafani, 1995; B. Revusky, 1970). However, when the temporal parameters of the original design were closely followed (Figure 1), intake-reinforcing effects of the longer fast were replicated (White, Mok, Thibault & Booth, 2001) and the effect was robust enough for detailed investigation (Jarvandi, Booth & Thibault, 2007a; Jarvandi, Thibault &

Booth, 2007b, 2009; Thibault & Booth, 2006).

Figure 1 about here

The new interpretation argued in the present paper is that even just the 3 hours of deprivation makes re-feeding reinforce the eating before the fast but 13 hours makes the eventual re-feeding far more rewarding. This explanation of anticipatory eating is considerably simpler than that given in our previous papers, which was in terms of the avoidance of depletion.

It must be noted that the design by Le Magnen which we have followed does not coordinate the amount of reinforcement to the amount of responding. Hence our evidence does not directly show that the learnt intake is instrumental / operant, rather than reactive / respondent (Table 1). Nevertheless, the animals might extract an instrumental contingency from the schedule, especially if test food intake happens to rise before delayed re-feeding (as it well might during early adaptation to that food and schedule). Further work will be needed for a definitive demonstration that anticipatory eating is based on the response-reinforcement contingency and not the stimulus-consequence contingency of classical conditioning.

Table 1 about here

Learning over long delays

The most extraordinary aspect of the original and recent findings is the many hours between the reinforced response and the reinforcing stimulus processes. Nevertheless, some potentially relevant effects have been known for a long while.

Miller and Kessen (1952) found that orally delivered food reinforcement was attenuated within a few minutes of delay in the delivery of food after the instrumental response. A classic study showed that the appetitively reinforcing (rewarding) effects of food operate over as little as half a minute (Grice, 1948). However, such evidence comes from sessions with multiple small

reinforcers, each followed soon by more responses. The aversively reinforcing (punishing) effects of foot shock have been investigated on similar schedules.

In contrast, when a single aversive manipulation of bodily state was applied after a single bout of intake of a distinctive material, strong association to that cue (Table 1) was observed in later performance (i.e., conditioning of aversion to the cue) when the consequence had been delayed for 12 hours or more (Garcia, Ervin, Yorke & Koelling, 1967; Nachman, 1970; S. Revusky, 1968). However, the appetitive association of a sensory cue with a nutritional manipulation (conditioning of preference) was seen with a delay of merely half an hour (Holman, 1975) or up to an hour or so at the most (Sclafani & Ackroff, 1994; Simson & Booth, 1973b). Nevertheless, it should be noted that these stimulus-conditioning experiments used incomplete foods in small amounts and/or unphysiological routes and timings (Mather, Nicolaïdis & Booth, 1978). The same weakness attended the response-rewarding experiments of Miller and Kessen (1952) and the many failed attempts to reinforce eating intravenously. In contrast, in Le Magnen's and our experiments the rats were re-fed on a nutritionally balanced maintenance diet. That is, the reward that induces anticipatory eating is of the size and quality generated by natural assimilation of substantial amounts of a complete food. Hence the evidence taken as a whole does not justify scepticism about instrumental learning of extra intake from physiological reward signals after a long delay in restoring access.

A further principle of instrumental learning over long delays throws extra light on the evidence for anticipatory eating, broadening the basis for the new interpretation proposed here. The learning of increased intake before the longer fast is consistent with the long-known potentiation of food's reinforcement of other responses by deprivation. For example, lengthening the period of food deprivation from 6 hours to 12 hours in rats produced a sharp increase in the

rate of lever-press responses on a variable interval schedule of reinforcement by small amounts of food (Miller, Bailey & Stevenson, 1950). Miller and Kessen (1952) went on to show that oral action of the food was important to such reinforcement. Nevertheless, they also found that effects of food delivered directly to the stomach were sufficient to reinforce, although more weakly than with oral consumption.

The findings for both longer and shorter fasts in Le Magnen's paradigm can be explained by an associative effect of deprivation which is analogous to deprivation's motivational effect of potentiating (oral) reward, described above. The proposal is that fasting potentiates an associative effect of the actions of food after consumption, so that more reward is generated by the re-feeding at the end of a longer period of deprivation. That is to say, the postingestional effects of food after even the briefer fast are rewarding, but less so than the assimilation of food after the more prolonged fast when repeated over trials.

That increase in the ultimate amount of reinforcement with increased delay in re-feeding is distinct from any effect of that delay between response and reinforcement on the learning evident at the very next trial. The impact of reinforcement from a single occasion of re-feeding on ingestive responding to food is liable to be attenuated by operating over a longer period of time. Hence the shorter delay could produce faster learning, despite having the lower capacity for postingestional reward that can be seen after a good number of training trials. Yet this attenuation of association in a single trial by the longer delay between response and reinforcer should eventually be overcome by the greater reinforcement from refeeding that is generated after a longer delay. In short, the omission of one meal could reward extra eating immediately but the omission of more meals could induce a greater increase in intake if repeated often enough.

Clear evidence of these opposing effects of delay in reinforcement on instrumental

learning have been seen in our experiments on anticipatory eating (Jarvandi, Booth & Thibault, 2009; 2012; Thibault & Booth, 2006). The acquired increase in intake is eventually greater with the longer period before re-feeding, whereas the more rapid increase in intake is with the shorter delay between eating and reinforcement. It should be noted that this conjunction of faster learning with less capacity of reinforcement is more consonant with shorter and longer fasts affecting intake by the same mechanism than by different mechanisms (Jarvandi *et al.*, 2012).

Reward *versus* avoidance

In our previous papers on anticipatory eating, we have suggested that the rat contrasts aversive states of depletion that arise during the longer fast with their absence after the short fast. This interpretation was inspired by the observation that intake before the longer fast increased only if a substantial amount had been eaten during the preceding trial on the shorter fast (White *et al.*, 2001). We argued that sufficient intake before the shorter fast would eliminate or drastically reduce hunger, providing a sharp contrast with the hunger that developed during the longer fast. The contrast would make it easier for discriminative stimulus control of the extra eating to avoid or reduce punishment by the longer fast (Table 2).

Table 2 about here

The invocation of such a contrast between short and long fasts has been undermined by the discovery that anticipatory eating is learnt in a set of trials on the longer fast alone (Jarvandi *et al.*, 2009). Furthermore, the shorter fast in its own series of trials showed signs of an up-and-down pattern of learning anticipatory eating which has been a consistent feature of our results (from Thibault & Booth, 2006, onwards). The signs of an oscillatory learning curve were very weak with short periods of deprivation but that would not be surprising, since only one or two meals would be missed during deprivation of a rat for only 2-3 hours.

We attributed this rise, fall and rise again of the learnt extra intake to ‘self-extinction’ and re-acquisition of anticipatory eating (Jarvandi *et al.*, 2007a,b, 2009). On our original depletion avoidance account, the cycles of extinction and re-acquisition were attributed to attenuation and restoration of the punishing effects of lack of food by the rise and fall in intake before food is withheld. Our new proposal explains these cycles as loss and regain of potentiation of the positively reinforcing power of repletion (cp. Table 2).

Avoidance of an aversive effect of depletion (negative reinforcement) remains conceivable as an explanation for anticipatory eating for both lengths of fast. Its exclusion in favour of reward by the re-feeding that ends any fast requires evidence yet to be sought, such as from designs in which explicit response-reinforcement contingencies are imposed. Meanwhile we favour a rewarding effect of repletion as a simpler explanation that makes better contact with other literature.

Does the reward of intake require a distinctive food?

An additional question arises from the fact that reinforcement occurs during a series of shorter fasts alone, as well as from the longer fasts. Did the extraction of the response-reinforcement contingency from either of those series of fasts require the discriminative stimulus provided? One sign of discriminative learning was the possibility of a stimulus generalisation error in the first trial of the second series in each rat (Jarvandi *et al.*, 2009). However, even if the odour or texture given to the trial food before a particular length of fast gains some control of behaviour, the cues designed to be discriminative between lengths of fasts cannot be crucial to extra eating that appears early in a series of trials followed by a constant period of deprivation, as in that 2009 paper.

Nevertheless, the trial foods were quite distinct from the maintenance food in our

experiments on anticipatory eating (with one exception: Jarvandi *et al.*, 2007a). Generally the maintenance food was coarsely ground blocks, whereas the training-testing food was a fine powder or a liquid. Hence the movements in the reinforced intake response were different from those for maintenance intake. The rats' gnawing of maintenance food had to be shaped to licking or sucking the test food or fluid. If so, the test food was distinctive not only sensorily in odour or texture but also in motor pattern of the reinforced response.

The rewarding effect of re-feeding after a long delay may not be confined to food intake. If some other responses than eating were shown to be rewarded in rats in this paradigm, then an alternative to signals from nutritional repletion should be investigated. There is evidence that an aversive event before a reward enhances its reinforcing action by a contrast effect (Zentall, 2010). Hence, if the longer fast is more aversive, the restoration of access to maintenance food might be more rewarding. Nevertheless, until it is shown that all of the anticipatory eating seen in rats arises from such a contrast, it remains a viable proposal that physiological signals during assimilation contribute to the rewarding of the ingestive response over the long delay.

Postingestional sources of reward over long delay

These experiments provide clear evidence for parallel increases of the duration of deprivation and the strength of reward from feeding freely on maintenance food at the end of the fast. That contrasts with the limit on the rewarding effect of the delivery of a small amount of food to within at most a minute or so of responding (even when the response is marked by an extraneous event: Thomas & Lieberman, 1990). Hence food reward at two to twelve hours after the responses cannot come from the actions of the maintenance food up to the time only that it is in the mouth. Food reward over long delay must arise from actions of the restored food after it has been ingested. This also appears to be the only way of explaining the classical conditioning

of sensory preferences to protein-rich and carbohydrate-rich foods in mixed meals (Baker & Booth, 1989a). Indeed, some or all of the signals that condition sensory preference might also positively reinforce eating responses (Table 1). In fact there is already evidence that repletion of a lack of protein can reward eating over 12 hours earlier, as well as the repletion of a lack of carbohydrate (Thibault & Booth, 2006).

The question therefore arises how normal assimilative processes send signals to the brain that result in extra eating of a food when it is available subsequently (Booth, 2008). Research into this issue should not use nutritionally incomplete or poorly assimilated gastric loads or intravenous infusions. Processes that might generate reward signals cannot be measured either from doses of nutrients or biomarkers of hunger or its satiety (Booth & Nouwen, 2011). The pharmacology and anatomy of the brain circuits of this associative process tell us nothing of the generation of signals in tissues around the body. It would therefore be productive to track the physiological and biochemical processes that parallel the potentiation of long delay reward by omission of meals. Does the withholding of just one meal result in at least a little reinforcement? Is there an increase in capacity for reward from repletion that is graded with the number of meals missed? Can the omission of a single substantial meal be matched by an accumulated effect of a number of meals that are restricted in size? Does the gradient of increasing reward with greater deprivation flatten beyond a certain degree of depletion? In answering such questions, results from experiments on intake within fixed periods of access to food should be complemented by analyses of spontaneous pattern of meals under the conditions of training.

Potential signals to the brain include the reversal of glycogenolysis or gluconeogenesis after several meals have been missed in rats (Newman & Booth, 1981) and changes as digestion proceeds in the circulating levels of gastrointestinal hormones such as cholecystokinin or ghrelin

(Abizaid, Liu, Andrews *et al.*, 2006).

We already know something of the postingestional associative processes for stimulus conditioning (Table 1). The amount that an animal eats of a distinctive food is increased when that sensory cue is followed by postingestional actions of carbohydrate (Booth, Lovett & McSherry, 1972; Sclafani & Nissenbaum, 1988; Sclafani, 1995) or protein (Simson & Booth, 1973a; Baker, Booth, Duggan & Gibson, 1987). The sensed characteristics of the food(s) in a meal become better liked in proportion to the nutrition that they yield (Baker & Booth, 1989b). Glucose that gets beyond the stomach conditions preference to whatever sensory characteristics the food possesses (Ackroff & Sclafani, 1991; Booth *et al.*, 1972; Sclafani, Cardieri, Tucker *et al.*, 1993; Sclafani & Nissenbaum, 1988). These are the mechanisms by which foods acquire the incentive properties that have often been misnamed food reward (Berridge & Robinson, 2003).

A little work has begun on manipulating the nutrient contents of the rewarding food, namely the inclusion of fat in the maintenance diet (Jarvandi *et al.*, 2007b). Together with fat in the trial food (White *et al.*, 2001), these manipulations produced the least extra eating seen before the longer deprivation in the seven experiments that we have so far conducted (Jarvandi *et al.*, 2012). Those two experiments involving high-fat foods also produced the smallest effects on the eating before the briefer deprivation. However the increased intake of food in our initial experiment (White *et al.*, 2001) could have come from familiarisation. Hence the possibility of weak learning with high-fat food merits further attention. Such effects could reflect the strong influence of high-fat food on metabolism in the body and the brain (e.g., Gerozissis, 2004; Greenwood & Winocur, 2005). The effects of a high-fat diet on the brain have been connected to poor learning in other paradigms, as well as to increased obesity (Woods, D'Alessio, Tso *et al.*, 2004).

Human susceptibility to delayed food reward contingencies

Neural mechanisms with the capacity for learning processes found in our commensal omnivore, the rat, are likely to be found also in the human brain. Such a mechanism might operate automatically and unconsciously, without involving conceptually based decision making (Brunstrom, 2004; Cohen, 2008). Indeed, any unconscious rewarding of eating before missed meals could support the reasoned choice of an amount of food that minimises symptoms of lack of food before the next expected access ('hunger management' based on 'expected satiety': Dibsall, Wainwright, Read & Booth, 1997; Thibault & Booth, 2006; White *et al.*, 2001). On the other hand, an unconscious rewarding effect of food eaten after missing a meal (or limiting the sizes of meals) might be strong enough to have the adverse effects of causing extra eating after such 'dieting.'

Jarvandi (2008) proposed an experimental design to test for reinforcement of anticipatory eating by delayed repletion, independently of any management of expected periods without food. Central to the proposal is diversion of attention from the associative contingency for anticipatory eating by a plausible motivational contingency on the discriminative food, namely its satiating effects. After the experiment, participants could be asked to rate the probability of a variety of experimental hypotheses, only one of which is the instrumental contingency. The effects of viewing these hypotheses on such assessment could also be monitored. A more sensitive measure, although more heavily reliant on blinding by the declared motivational contingency, is to follow the completion of trial intake by a guess as to the deprivation period assigned to that day before it was declared. Human replication of the reward effect in rats does not require the deprivation period to be predicted: all that is needed is a more reinforcing effect on prior eating of re-feeding after longer deprivation.

Implications for human obesity and its neuroscience

The theoretical advances made in this paper on issues in the fundamental experimental psychology of food consumption may have considerable social importance. It follows from thermodynamics that an increase in body weight and fat content can arise only from a persistently greater rate of intake of energy than of its expenditure through metabolism and movement (Garrow, 1974). Hence any general mechanism that is liable to increase the amounts of food eaten is a candidate contributor to the recent and continuing increase in obesity and related diseases (WHO, 2000). The associative process by which re-feeding increases intake of food consumed before the deprivation may become central to understanding overeating, because longstanding attempts to explain obesity behaviourally are under challenge -- namely, a high palatability of foods and overeating after breaching dietary restraint.

Misnomers 'food reward' and 'palatability'

The role of food in obesity has long been attributed to a supposed property of palatability or, even more bogus, an addicting function that can be injected by food manufacturers. In fact the appeal of a particular food at any moment is highly contextualised and idiosyncratic (human data: Birch & Marlin, 1982; Booth, 1985; Pliner, 1982; rats: Boggiano, Dorsey, Thomas & Murdaugh, 2009; Weingarten, 1985). Foodstuffs vary in appeal for everyone, even within a meal, let alone among the various foods' places in the social customs and personal patterns of eating (Booth, 1991; Conner, Haddon, Pickering & Booth, 1988; Wansink, 2004). Such dependence on context is to be expected from the evidence that preferences among foods and drinks are strongly controlled by stimulus-stimulus contingencies (configuring) among the sensed features of the ingestible item, the somatic state and the social situation, and the physiological effects of the glucose and essential amino acids released by digestion (Booth, 1985; Booth, Gibson, Toase &

Freeman, 1994; Brunstrom & Fletcher, 2008). Therefore, any increase in palatability in recent decades has to have come from an increase in reinforcing capacity of these chemospecific signals to the brain. There is no evidence nor mechanistic rationale for such extra metabolic reinforcement.

Invoking ‘food reward’ to account for sensory facilitation of eating is even less appropriate than reliance on the notion of palatability or a liking for the food. First, the notion of a fixed property of the food for an individual excludes further learning. Secondly, the acceptance of a sensed food declines sharply during eating, under a variety of influences ranging from sensorimotor habituation and sensory-gastric conditioned satiety to cultural conventions on sizes of portions at a meal and the effects of interpersonal impression management (Booth, 2009). Thirdly against such use of the term ‘food reward,’ the enthusiastic acceptance of a food provides no evidence whether or not that process also has the associative effect of rewarding eating or indeed any other responses. The evidence that food has been rewarding can come only from observing changes of responding that are attributable solely to an instrumental contingency – the response being predictive of the putative reward (Balleine, 2005; Berridge & Robinson, 2003; Wise, 2006; see Tables 1 and 2).

Not distinguishing the motivational process from the associative process results in confusions about neural mechanisms of such behaviour processes and any contribution that they might make to obesity (Olszewski, Fredriksson, Olszewska *et al.*, 2009; Simmons, Martin & Barsalou, 2005; Wang, Volkow, Telang *et al.*, 2004). Obesity cannot be blamed on something magical in the brain evoked or suppressed by foods, drinks, drugs or surgery, whether called reward, appetite or disinhibition, or a lack of satiety, restraint or willpower (Booth & Nouwen 2011). Body weight is ultimately controlled by the frequency of repeated actions of consuming

satisfying food and drink, moving around and keeping warm or cool (Blair, Booth, Lewis & Wainwright, 1989; Booth & Booth, 2011). Such complex activities are served by neural activity in many regions of the brain, scattered among synapses that have been adapted to the culture (Booth, 1967; Wickelgren, 1999).

Dietary restraint

Much more realistically, some psychologists have proposed that attempts to restrict one's intake are defeated, and may even worsen obesity, by triggering overeating through ill defined processes such as instrumental reduction of aversive states or the abandonment of an intention to eat less (Herman & Polivy, 1975; Polivy & Herman, 1985). The evidence for such disinhibition of dietary restraint has been obtained within single sessions in the laboratory (Herman & Mack, 1975, and many replications).

However, the relevance of these observations to life outside the laboratory has been questioned. Such an imposed episode of overeating is of course liable to produce lower intake later, in compensation (Booth, 1972). High scores on questions using the concept of dietary restraint do not correspond to reduced daily intake (Stice, Fisher & Lowe, 2004; Stice *et al.*, 2010). Furthermore, no overeating is recorded after a diet had been breached (Tomiyaama *et al.*, 2010).

Does diet-breaching reward intake of food?

The findings in rats reviewed here raise questions about human learning processes that can be investigated in the laboratory and in the field. First, do a person's first reductions in intake lead to additional eating of all the foods that were available before each period of dieting as a result of the rewarding effect of any amount eaten after deprivation? The omission of protein from one day's breakfast is sufficient for classical conditioning of sensory preference by protein

in that day's lunch (Gibson, Wainwright & Booth, 1995). Maybe cutting back on one meal generates sufficient reward from the subsequent meal to increase intake of foods eaten previously by the associative mechanism evident in rats. Secondly, is any such extra intake incompletely compensated by eating after the period of self-deprivation, so increasing average daily intake of energy?

More specific forms of these questions could be important too. Snacking and especially food binges are liable to be followed by intentionally reduced intake. Yet this rational self-deprivation might amplify the reward of the consumption of the foods that provoked the cutback in intake (cp. Cameron, Goldfield, Finlayson *et al.*, 2010). Even an increase in eating intended to manage the hunger arising from self-restriction could be further increased by the unintended reinforcement process seen in rats, if the mechanism exists in people. Hence further work on this learning process in both rats and people would be timely.

Conclusions

It has now been established in rats, and proposed above for investigation in human beings, that food eaten after a reduction in intake rewards the eating of foods preceding that reduction. This reward effect comes from physiological signals of which people may not be aware. There may be no rationale in the culture that corresponds to the extra eating prior to missed meals that is induced by the eating which ends that fast. If that is so, this mechanism for obesity is liable to be unrecognised by the general public, or by professionals in health care, the food supply, education and research.

Such an unrecognised associative process may help to explain why educational, cognitive-behavioural, pharmaceutical and surgical interventions on obesity have so far failed to survive controlled trials of the management and prevention of obesity-related disorders such as

hypertension, atherosclerosis and type 2 diabetes. Hence closer study of a phenomenon reported over fifty years ago (Le Magnen, 1957) may have uncovered a major cause of obesity, paradoxically arising from the public and private efforts to cut back on food intake in order to reduce or avoid fattening.

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Table 1. Terminology for Edward Thorndike's instrumental learning (B.F. Skinner's operant conditioning) and Ivan Pavlov's (Type 1) classical (respondent) conditioning. For the distinction between punished and punishment-avoidant responses, see Table 2.

	Instrumental learning (reinforcement of response)	Classical conditioning (reinforcement of stimulus)
Level of response before training	R_0 : initially emitted responding	$R_0 = UR$: Unconditioned Response
Cue to the imposed contingency	S^D : Stimulus Discriminative of reinforcement	CS: to-be-Conditioned Stimulus
Trained associative contingency	Response \rightarrow Reinforcer (reinforcing stimulus)	CS \rightarrow US: Unconditioned Stimulus (reinforcer)
Response level after reward (appetitive reinforcement)	$R_0 + R$ in the presence of the S^D	$R_0 + R = CR$: conditioned appetitive response to CS
Response level after punishment (aversive reinforcement)	$R_0 + R$ (if avoidance) in the presence of the S^D	$R_0 - R = CR$: conditioned suppressive response to CS

Table 2. Currently and previously proposed associative mechanisms of appetitive and aversive reinforcement underlying reward and avoidance learning and their self-extinction in anticipatory eating. Note that this rough sketch of the variously hypothesised learning processes is not theoretically complete or precise.

Type of	Response	Subsequent	Repletion on	Test for learnt
instrumental	remembered at	depletion during	restoration of	intake
learning	reinforcement	food deprivation	access to food	
Reward	Increased intake	(No associative effect)	Appetitive reinforcement	Increase
Punishment	Any intake	Aversive reinforcement	(No associative effect)	Decrease
Avoidance	Increased intake	No aversive depletion	(No associative effect)	Increase
Extinction	Any intake	Less depletion	Less repletion	Decrease

Caption to Figure

Figure 1. The full cycle of a single trial of training and testing for anticipatory eating. Note that the horizontal array indicates only the sequence of operations and is not to scale in time on the clock. After the first trial, the period of access to maintenance food following the trial period is continuous with that before the next trial.

Figure 1

