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Learning and Memory Processes and Their Role in Eating: Implications for Limiting Food Intake in Overeaters

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Abstract Understanding the role of psychological factors involved in overeating is critical if we are to develop effective interventions to curb the rise of obesity that is associated with the modern food environment. Here we review recent experimental research on the role of cognitive processes such as learning and memory in eating behavior. From habituation to learning to associate the rewarding consequences of ingestion with food cues, we contemplate how learning about food has been influenced by the changing food environment. We also consider how learning and memory processes interact with satiety processes and how higher-level cognitive systems modulate responses to food cues. Finally, what we remember about eating episodes affects later eating. Encoding information about meals and snacks allows us to take into account recent energy intake and food enjoyment during later eating events. We suggest that interventions that encourage attentive eating might prove fruitful in helping appetite control.

Keywords Habitation · Reward · Learned satiety · Episodic memory · Remembered liking · Memory processes · Overeating · Obesity

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Introduction

Rising levels of obesity are a current major threat to global health [1]. The number of obese adults and children worldwide has been increasing steadily over the past 30 years and although there is evidence that this trend may be levelling off in some countries, the overall rates of obesity in these high income areas remain high [2, 3]. Statistics from the World Health Organization estimate that more than 1 in 10 of the world's adult population was obese in 2008. These figures are concerning because obesity is associated with an increased likelihood of the main noncommunicable diseases (NCDs) (eg, type 2 diabetes, cardiovascular disease, and cancer) [4]. The significant challenge faced by societies in dealing with the effects of obesity on individual and economic health was acknowledged in a recent high-level meeting of the United Nations General Assembly on NCDs, which urged action to address risk factors associated with NCDs such as unhealthy diet.

Increased food intake is an acknowledged driver of rising obesity rates [5]. Changes to the global food environment coincided with the start of the upward trajectory of obesity prevalence, leading to suggestions that increased availability of cheap, palatable, energy-dense foods in larger portion sizes has contributed to increased levels of obesity by promoting food intake [6]. Not only is food more available, it is also more aggressively marketed than ever before with tempting images of food ubiquitous in advertising and other media [7]. However, an important question is why increased marketing and availability of palatable food may be associated with overconsumption and weight gain in at-risk groups, while some people appear to be resistant to the obesogenic effects of a food-abundant environment. Genetic differences that result in individual variability in the psychological and physiological responses to food and cues that signal food availability are likely to strongly influence vulnerability to weight gain in an obesogenic environment. However, these genetic differences are also likely to interact with other factors associated with the modern food environment and 21st century lifestyles, such as food variety and eating on the go, to affect consumption and food choices. Understanding these factors has implications for the development of new approaches to help people control their food intake and lose weight or avoid weight gain in the first place. The aim of this review is to highlight recent advances in the understanding of the psychological processes contributing to overeating and to discuss the implications of these advances for the development of new approaches to help individuals limit food intake. The particular emphasis here is on studies of humans and the potential role of learning and memory processes.

Simple Learning: Habituation

Appetitive responses to foods decline or habituate as these foods are eaten. This can be conceptualized as a simple form of learning that limits the amount eaten during a meal or the size of a snack [8]. Habitation to one food is disrupted by tasting a different food (dishabituation); thus, greater access to a variety of foods in the modern food environment could contribute to overeating because eating more varied foods within a meal would disrupt habituation and increase meal size [9, 10]. There is also evidence of individual differences in habituation rates, such that slower habitation to food stimuli is reported in obese versus lean participants [11]. More recently, it has been reported that slow rates of habituation predict weight gain in children over 12 months [12]. Thus, people who have a slow rate of habituation might be especially vulnerable to the effects food variety, which has implications for targeted treatment strategies designed with the modern food environment in mind. Moreover, it is known that non-food stimuli can also act as dishabituating stimuli. For example, interrupting eating by watching TV, reading, or playing a computer game can disrupt habituation [13]. This suggests that avoidance of concurrent activities while eating might help with control of food intake, especially for individuals who already have slow habitation rates. Habituation to foods occurs during the course of a meal but can also occur over days. A recent report is consistent with the idea that reducing food variety across, as well as within, meals could also be a potential strategy to avoid overconsumption [14•]. Intriguingly, even thinking about eating a food may be sufficient to induce habituation and reduce consumption, as suggested by Morewedge et al. [15], who reported that repeatedly imagining eating a food led to a decrease in intake of that food. This proposition has yet to be tested fully, but it is possible that a cognitive strategy based on thinking about consumption of a food before eating could help limit intake.

The Power of Food Cues: Learning to Like and Want Foods

The mere presence of food can act as a trigger to stimulate food-seeking behavior and provoke appetite, even when satiated [16]. The motivating power of food cues (eg, the sight and taste of food) comes at least in part from learning about associations between these cues and the pleasurable consequences of eating. As a result of this learning, tasty foods acquire the ability to attract attention and elicit consumption and this process is known as incentive salience attribution [17]. Berridge et al. [18] have used the term food "wanting" to refer to the process of incentive salience attribution. In other words, "wanting" used in this context is the pull that food cues exert on behavior. Evidence suggests that brain dopamine systems are critical for food "wanting." Conversely, brain γ-aminobutyric acid (GABA)/opioid systems are thought to be important for the pleasurable or hedonic "liking" reactions to the taste of foods [18]. Both "liking" and "wanting" are usually involved in the rewarding effects of food and thus individual differences in these processes could contribute to overeating in an environment full of palatable foods. Enhanced activation in "liking" brain circuits would be expected to lead to greater hedonic reactivity to food, which would then trigger enhanced food "wanting." In support of this idea, some recent evidence suggests that genotypic variations in opioid and dopamine signaling in the brain are related to preferences for high fat and sugary foods and may contribute to binge eating [19•]. Furthermore, measures of attentional processing of food-related cues, which could be a behavioral index of enhanced "wanting," have been reported to be greater in overweight versus lean participants and positively associated with food intake and cravings [20]. Importantly, biased attentional processing of food cues has been shown to predict weight gain [21, 22•] and data from functional MRI (fMRI) scanning of adolescents at risk of obesity suggest that these biases may relate to enhanced responsiveness in brain reward circuits [23]. Others have reported that a proposed measure of "wanting," time taken to respond in a forced choice food selection task, is related to binge eating traits [24].

A question that has been hotly debated is whether responsiveness to food cues and overeating are caused by greater liking and wanting responses or by enhanced wanting dissociated from liking [25]. The idea that liking and wanting are mediated by distinct neural substrates suggests that they can be separated under certain conditions [26]. However, there are challenges associated with measurement of these constructs in people [25], which means it is probably too early to say whether distorted wanting separate from liking underlies the exaggerated responses that some people show towards food cues. The study of the causal role of reward processes in overeating and obesity is also complicated by the fact that becoming obese is likely to lead to compensatory changes in reward mechanisms [27]. Thus, differences between lean and obese participants found in some of the studies mentioned may just be a reflection of the consequences of obesity, underlining the importance of future longitudinal studies.

Motivational State and Food "Liking" and "Wanting"

Another explanation of individual differences in food cue responsiveness relates to differences in the effects of motivational state on food reward processes. Developments in understanding the brain systems involved in appetite control point to an intimate connection between processing of reward signals and metabolic state signals [28, 29]. Overeating of palatable foods would be due to a failure to downregulate "wanting" and "liking" during satiety. Castellanos et al. [30] found that attention paid toward food cues by obese participants did not change as a function of eating to satiety, whereas lean participants showed an attentional bias toward food only when hungry. These data are consistent with the idea that reactions to food stimuli are not downregulated in response to changes in nutritional state in obesity, although it should be noted that Nijs et al. [31] reported that obese people are overall more likely to direct automatically their visual attention more to the sight of foods than nonobese people, especially when hungry. Intriguingly, an fMRI study of children with a congenital form of obesity due to a lack of leptin did not show the usual downregulation of brain reward activity in the nucleus accumbens when sated; an effect that was restored upon treatment with leptin [32]. Conversely, administration of the gut peptide ghrelin to healthy volunteers increased neural activity in reward-related brain areas, suggesting that an enhanced ghrelin response when hungry could trigger eating due to enhanced hedonic and incentive food responses [33]. More work is required in this area but it is possible that individual differences in the effect of hunger and satiety on reward processing determine meal size; thus, interventions that target these processes might prove effective in weight management.

Top-Down Modulation of Responses to Food Cues

Reward-related responses to food can be modulated by metabolic signals acting on brain reward pathways as described above but they are also modulated by inhibitory control processes and expectations about foods mediated by cortical brain areas [34–36]. Recent data suggest that attention to food stimuli in the environment is modulated by the type of information held in working memory, such that holding information in working memory about food (eg, thinking about food) has a strong facilitatory effect on detection of food items in a search array [37]. This mechanism might be another reason why obese individuals who are preoccupied with thoughts of food show biased attentional processing of food-related items [20].

Another model suggests that dietary choices are influenced by the value assigned to particular foods and that this process is dependent upon on the ventromedial prefrontal cortex (vmPFC). The ability to resist immediate temptation depends upon being able to integrate both short- (eg, taste, anticipated pleasure) and long-term (eg, health) goals in the determination of value and this process is thought to depend upon activity on the dorsolateral prefrontal cortex (dlPFC). In support, an fMRI study of food choices made by dieters showed that activity in the vmPFC was correlated with both taste and health ratings in participants who exhibited self-control (choosing not to eat/liked healthy items) but in non–self-controllers, vmPFC activity was only correlated with taste ratings. Moreover, activity in the dlPFC was correlated with successful self-control [34]. This network may underlie the influence of health cues and diet reminder on self-control behavior in the face of tempting cues [38, 39•]. The presence of health cues has been reported to improve dietary choices by increasing the weighting that healthiness receives in the vmPFC value signals, an effect that is related to activity in the dlPFC [40]. Conversely the well-known breakdown of dietary restraint that is observed after consumption of a preload or stressful situation [41] may reflect the failure of top-down control from the dlPFC [42]. These data are consistent with the suggestion that individuals for whom food cues are particularly salient may find it more difficult to adhere to long-term health/weight loss goals only if they also show problems with response inhibition, which would relate to a general deficit in top-down control of behavior [43–45]. They further suggest that external cues that emphasize dieting or healthy eating and weight goals could be helpful to those wishing to control their food intake, although how these cues would translate into an effective large-scale intervention is less clear.

An alternative approach that has been suggested is that training of working memory and cognitive control may prove useful in resisting the pull of tempting food cues and enable healthful decision making. Cognitive inhibitory control is also required for restriction of substances such as alcohol and there is evidence that training on tasks that strengthen inhibitory control reduces consumption of alcohol in heavy drinkers. Houben et al. [46] asked heavy drinkers to complete computer tasks that involved practicing memory tests and measured self-reported drinking behavior before and after training. Training improved memory capacity and reduced alcohol intake for more than 1 month after the training. The reduction in drinking was related to the level of automatic impulses to consume alcohol. Similar results have also been reported recently for abusers of stimulant drugs [47]. The effects of working memory training on food intake have yet to be examined, although Houben et al. [48] found that training of inhibitory control using a stop signal task reduced consumption of chocolate.

Learning About Calories

As a result of learning about associations between food cues and the nutritive or caloric consequences of consuming those foods, people can come to anticipate the effect of consuming particular foods on the body and physiologically prepare for the arrival of nutrients in the body [48]. These cephalic responses to food comprise a range of physiological reflexes that minimize the impact of incoming nutrients on the internal environment and enhance the efficiency of nutrient utilization [49]. Experience with the post-ingestive effects of eating can also lead to learned reductions in meal size to avoid the aversive consequences of overconsumption, known as conditioned satiety [50]. It has recently been suggested that this kind of learning may be expressed through the selection of portion sizes based on conditioned expectations about the satiating effects of food [51].

An interesting proposition is that these learning processes may be undermined by a feature of the modern food environment, which is the degradation of relationships between the taste properties of foods and their nutritive post-ingestive consequences [52]. Many modern food products are available in both high- and low-calorie versions, whether this is achieved through the use of non-nutritive sweetener or low-fat substitutes. This means that orosensory cues such as sweetness or creamy texture that would previously have been strongly associated with the delivery of sugar or fat calories may no longer have such predictive power because sweetness and creaminess might be predictive of calories or not depending upon the product being consumed. Thus, it is possible that the ability of these cues to elicit compensatory or anticipatory cephalic responses or contribute to portion size decisions is undermined, with possible consequences for food intake and weight gain. In support, there are studies showing that exposing laboratory rats to inconsistent relationships between a food cue and caloric consequences leads to increased food intake and weight gain [53•, 54]. This work suggests that consumption of low-calorie versions of food products may actually undermine attempts to reduce food intake. While similar studies have yet to be conducted in humans, one recent study has indicated that repeated exposure to a low energy-dense version of a food decreased liking for that product [55].

Episodic Food Memories

Memory for Recent Eating

The influence of memory for the predicted consequences of eating acquired over repeated experiences has been examined for some time but recently the idea that episodic memory for the most recent eating experience might also play a role in food intake has been proposed [56]. There is evidence that people encode in memory specific attributes of recently eaten foods and that this can occur without awareness [57]. It has been argued that information about recent eating encoded in memory is factored into decision making about future food intake and that the hippocampus is a potential neural substrate [58, 59]. In support, amnesic patients with damage to the hippocampus who are unable to recall a recent eating episode show impaired satiety and overconsume when offered multiple meals [60–62]. Furthermore, manipulating memory for recent eating in healthy volunteers by facilitating recall or disrupting encoding of food memories results in increased and decreased afternoon snack intake, respectively [63, 64].

Higgs and Woodward [64] found that participants who consumed their lunch while watching TV ate more at an afternoon cookie tasting session compared to participants who ate the same lunch but in the absence of any distraction. It was hypothesized that TV watching would result in participants paying less attention to their meal, with a concomitant impairment in encoding of the meal and poorer memory for the meal later. Consistent with this proposition, participants who had the TV lunch rated their meal memory as much less vivid that then control participants. A similar study by Oldham-Cooper et al. [65•] manipulated meal memory by asking participants to play a computer game while eating lunch. They also reported increased afternoon snack intake in the group who were distracted at lunch. Moreover, this group was less accurate when asked to recall the order in which they ate different food items at lunch, suggesting poorer lunch memory. Similarly, Mittal et al. [66] found over two experiments that a TV lunch led to greater food intake later than a no-TV lunch and that participants who had the TV lunch were also less accurate in recalling the amount of food consumed at lunch than the no-TV lunch. The results were not affected by varying the content of the TV program watched at lunch, suggesting that the effect cannot be easily explained by non-mnemonic factors such as effects of TV on mood or effects of TV content on dietary disinhibition. Taken together, the results of these studies suggest that environmental factors that impact encoding of information about a recent eating episode (eg, distractions such as TV or computer games) may contribute to overeating and possibly weight gain. While associations between sedentary activities such as TV watching and obesity have been well documented [67, 68], these new data point to a previously unappreciated contributor to the effect of these activities on body weight, which is the impact they have on food intake after the activity has finished.

If distraction during a meal causes increased later eating then improving memory for lunch should work in the opposite direction. Higgs and Donohoe [69•] recently tested this hypothesis showing that better lunch memory resulted in reduced afternoon snack intake. Three groups of participants were asked to consume a fixed lunch in the laboratory. One group ate their lunch in the absence of any concurrent activity (neutral control group). A second group ate the same lunch while reading an article about food (food cues control group). The third group ate their lunch while being instructed to focus on the sensory qualities of the lunch via a recording listened to on headphones (experimental group). All three groups returned to the laboratory a few hours later to take part in a cookie tasting session at which they were unaware their intake was being monitored. The group who had previously focused on their lunch while eating consumed fewer cookies in the taste test than the other two groups who did not differ. The food focus group also reported a more vivid memory of their lunch and the vividness ratings were negatively correlated with cookie intake, suggesting that enhanced food memory might have been responsible for the reduced cookie intake.

Altering lunch memories therefore appears to affect later snack intake. This is probably because in making decisions about snack intake people draw on information about the satiating effects of their lunch from memory. If the last meal is recalled to be satiating then this has an inhibitory effect on future intake. Using food memories in this way to inform decisions about future food intake allows us to use our wide experience of learning about the satiating effects of foods to predict the consequences of further eating. In support of this idea, research suggests that expectations about the satiating effects of a meal at the time the meal memory is encoded affects appetite after the meal. Participants who were led to believe that a fruit smoothie contained a large portion of fruit expected to feel more full than participants who were led to believe that a fruit smoothie contained a small amount of fruit. All participants then consumed an identical fruit smoothie but those with greater expected satiety felt fuller later than those with lower expected satiety [70]. One explanation for these results is that the participant's memory of how satiating the food was, influenced by their beliefs, affected later intake.

In summary, the data suggest that interventions based around improving meal memories and expectations about the satiating effects of food, such as encouraging an attentive approach to eating and avoiding distractions during eating, might prove effective for weight control. An interesting possibility is that slow eating, which is associated with reduced food intake, might increase attention paid to food as it is eaten and this enhances food memories. This idea is consistent with findings that reducing the rate of eating in obese children shows promise as a treatment for obesity [71]. How slow eating may bring about better appetite control is not known and an effect on food memory is only one possibility among others [72].

Poor food memory has been shown to affect consumption but there are also data suggesting the consumption of certain foods, in particular saturated fats and food high in sugar, may impair memory potentially creating a vicious cycle that promotes overeating [73]. Long-term consumption of a high saturated fat/sugar diet by laboratory rats impair hippocampal-dependent memory processes [74], which would be predicted to impair food memories and lead to overconsumption of food. These data are consistent with evidence that obesity is associated with cognitive impairments, including memory problems and early onset of Alzheimer's disease [75]. In the future it will be important to establish the relationship between individual differences in memory and eating patterns and how these may be modified by diet. In the meantime, evidence suggests that consuming a diet high in saturated fat and sugar may have implications not only for development of diseases such as diabetes and cardiovascular function, but also cognitive function and Alzheimer's disease.

Remembered Food Liking

Another factor affecting food choices is remembered enjoyment of consuming a particular food. Because food choices are usually made in the absence of direct sensory contact with foods, our memories of how much we enjoyed eating that food in the past are likely to be an important guide for behavior. In the case of food choice, remembered liking probably plays a more significant role than actual liking at the time of eating [76]. In support, recall of past enjoyment of foods and altering remembered enjoyment of a food affects future choice of the remembered foods [77•, 78]. It is also the case that specific aspects of a food or meal are likely to influence remembered enjoyment. Memories of food enjoyment are not an exact replica of the actual experience and tend to be biased toward remembering only certain parts of the experience. For example, memories of eating enjoyment have been shown to be influenced by the most liked and most recent part of an eating episode [79]. These data suggest that strategies based on improving remembered enjoyment for healthier food choices such as fruit and vegetables could be useful in limiting intake of high energy-dense foods to reduce energy intake. Laboratory-based studies suggest that strategies such as rehearsal of enjoyable aspects of eating immediately after consumption or recall of previous episodes where healthy choices were consumed and enjoyed should be tested in interventions.

Conclusions

Recent developments in understanding the psychological processes involved in the control of food intake are providing insights into the reasons why the modern food environment, in which a wide variety of palatable foods are easily available, might promote overeating leading to obesity. Individual differences in learning and memory processes provide a potential explanation as to why some people are more vulnerable to the effects of the food environment than others. Factors such as slow habituation to food stimuli, responsiveness to the rewarding effects of food stimuli perhaps combined with reduced downregulation of reward responses in satiety or reduced inhibitory control are likely to contribute to overeating in the face of food variety and tempting food cues. Reduced memory for recent eating may also play a role. Other aspects of modern lifestyles may also interact with these individual differences to promote overeating. Eating in the presence of distracting stimuli or on the go may decrease habituation to food stimuli, impair food memory, and reduce learning about the post-ingestive effects of eating, leading to increased intake over time. Targeting the food environment itself is likely to be most effective in combating obesity, but there is also potential in translating recent findings from psychological research into interventions to help people avoid overeating given the current environment.

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References

Papers of particular interest, published recently, have been highlighted as:
- • Of importance

1. World Cancer Research Fund and American Institute for Cancer Research. Food, nutrition, physical activity, and the prevention of cancer: a global perspective. Washington, DC: American Institute for Cancer Research; 2007.
2. Finucane MM, Stevens GA, Cowan MJ, et al. National, regional, and global trends in body-mass index since 1980: systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 9.1 million participants. Lancet. 2011;377:557–67.
3. Rokholm B, Baker J, Sorensen T. The levelling off of the obesity epidemic since the year 1999—a review of evidence and perspectives. Obes Rev. 2010;11:835–46.
4. Wang YC, McPherson K, Marsh T, Gortmaker SL, Brown M. Health and economic burden of the projected obesity trends in the USA and the UK. Lancet. 2011;378:815–25.
5. Swinburn B, Sacks G, Ravussin E. Increased food energy supply is more than sufficient to explain the US epidemic of obesity. Am J Clin Nutr. 2009;90:1453–56.
6. Swinburn BA, Sacks G, Hall KD, et al. The global obesity pandemic: shaped by global drivers and local environments. Lancet. 2011;378:804–14.
7. Cairns G, Angus K, Hastings G. The extent, nature and effects of food promotion to children: a review of the evidence to December 2008. Geneva: World Health Organization; 2009.
8. Epstein LH, Temple JL, Roemmich JN, Bouton ME. Habituation as a determinant of human food intake. Psychol Rev. 2009;116:384–407.
9. Temple JL, Giacomelli AM, Roemmich JN, Epstein LH. Dietary variety impairs habituation in youth. Health Psychol. 2008;27:S10–9.
10. Epstein LH, Robinson JL, Temple JL, Roemmich JN, Marusewski AL, Nadbrzuch RL. Variety influences habituation of motivated behavior for food and energy intake in children. Am J Clin Nutr. 2009;89:746–54.
11. Temple JL, Giacomelli AM, Roemmich JN, Epstein LH. Overweight children habituate slower than non-overweight children to food. Physiol Behav. 2007;91:250–4.
12. Epstein LH, Robinson JL, Roemmich JN, Marusewski A. Slow rates of habituation predict greater zBMI gains over 12 months in lean children. Eat Behav. 2011;12:214–8.
13. Epstein LH, Rodefer JS, Wisniewski L, Caggiula AR. Habituation and dishabituation of human salivary response. Physiol Behav. 1992;51:945–50.
14. • Epstein LH, Carr KA, Cavanaugh MD, Paluch RA, Bouton ME. Long-term habituation to food in obese and nonobese women. Am J Clin Nutr. 2011;94:371–6. *This study shows for the first time that repeated consumption of the same food over days leads to habitation of responding for that food and reduced food intake. These data suggest a mechanism whereby a reduction in meal variety might help decrease food intake in the long term.*
15. Morewedge CK, Huh Young E, Vosgerau J. Thought for food: imagined consumption reduces actual consumption. Science. 2010;330:1530–3.
16. Cornell CE, Rodin J, Weingarten H. Stimulus-induced eating when satiated. Physiol Behav. 1989;45:695–704.
17. Robinson TE, Berridge KC. The neural basis of drug craving: an incentive-sensitization theory of addiction. Brain Res Rev. 1993;18:247–91.
18. Berridge KC, Chao-Yi H, Richard JM, DiFeliceantonio AG. The tempted brain eats: pleasure and desire circuits in obesity and eating disorders. Brain Res. 2010;1350:43–64.
19. • Davis CA, Levitan RD, Reid C, et al. Dopamine for "wanting" and opioids for "liking": a comparison of obese adults with and without binge eating. Obesity. 2009;17(6):1220–5. *These authors report that obese binge eaters carry elevated frequency of the G allele of the OPRM1 gene A118G single nucleotide polymorphisms related to the opioid μ receptor, compared to obese controls who did not binge eat. Binge eating obese individuals were also more likely to express an A2 allele for the dopamine D2 receptor marker Taq1A. The results suggest that higher dopamine and opioid signaling in binge eaters may relate to their enhanced reactivity to the hedonic properties of food.*

20. Werthmann J, Roefs A, Nederkoorn C, Mogg K, Bradley BP, Jansen A. Can(not) take my eyes off it: attention bias for food in overweight participants. Heal Psychol. 2011;30:561–9.
21. Calitri R, Pothos EM, Tapper K, Brunstrom JM, Rogers PJ. Cognitive biases to healthy and unhealthy food words predict change in BMI. Obesity. 2010;18:2282–7.
22. • Yokum S, Ng J, Stice E. Attentional bias to food images associated with elevated weight and future weight gain: an fMRI study. Obesity. 2011;19:1775–83. *This study combined fMRI and behavioral measures of attention to food in lean and obese adolescent girls. Body mass index (BMI) correlated positively with speed of behavioral response to food stimuli but not to neutral stimuli. Greater lateral orbitofrontal cortex activation during initial orientation to appetizing food cues predicted future increases in BMI. These data suggest that young women who show elevated reward circuitry responsivity during food cue exposure are at increased risk for weight gain.*.
23. Stice E, Yokum S, Burger KS, Epstein LH, Small DM. Youth at risk for obesity show greater activation of striatal and somatosensory regions to food. J Neurosci. 2011;31:4360–6.
24. Finlayson G, Arlotti A, Dalton M, King N, Blundell JE. Implicit wanting and explicit liking are markers for trait binge eating. A susceptible phenotype for overeating. Appetite. 2011;57:722–8.
25. Havermans RC. “You say it’s liking, i say it’s wanting … ”. On the difficulty of disentangling food reward in man. Appetite. 2011;57:286–94.
26. Finlayson G, King N, Blundell JE. Liking vs. wanting food: importance for human appetite control and weight regulation. Neurosci Biobehav Rev. 2007;31:987–1002.
27. Berthoud H-R, Lenard NR, Shin AC. Food reward, hyperphagia, and obesity. Am J Physiol Regul Integr Comp Physiol. 2011;300: R1266–77.
28. Fulton S, Pissios P, Manchon R, et al. Leptin regulation of the mesoaccumbens dopamine pathway. Neuron. 2006;51:811–22.
29. Hallschmid M, Higgs S, Thienel M, Ott V, Lehnert, H: Postprandial administration of intranasal insulin intensifies satiety and reduces intake of palatable snacks in women. Diabetes, in press.
30. Castellanos EH, Charboneau E, Dietrich MS, et al. Obese adults have visual attention bias for food cue images: evidence for altered reward system function. Int J Obes. 2009;33:1063–73.
31. Nijs IM, Muris P, Euser AS, Franken IH. Differences in attention to food and food intake between overweight/obese and normal-weight females under conditions of hunger and satiety. Appetite. 2010;54:243–54.
32. Farooqi IS, Bullmore E, Keogh J, et al. Leptin regulates striatal regions and human eating behavior. Science. 2007;317:1355.
33. Malik S, McGlone F, Bedrossian D, Dagher A. Ghrelin modulates brain activity in areas that control appetitive behavior. Cell Metabolism. 2008;7:400–9.
34. Hare TA, Camerer CF, Rangel A. Self-control in decision-making involves modulation of the vmPFC valuation system. Science. 2009;324:646–8.
35. Chechlacz M, Rotshtein P, Klamer S, Porubská K, Higgs S, Booth D, Fritsche A, Preissl H, Abele H, Birbaumer N, Nouwen A. Diabetes dietary management alters responses to food pictures in brain regions associated with motivation and emotion: a functional magnetic resonance imaging study. Diabetologia. 2009;52:524–33.
36. Malik S, McGlone F, Dagher A. State of expectancy modulates the neural response to visual food stimuli in humans. Appetite. 2011;5:302–9.
37. Higgs, S. Rutters, F. Thomas, J. Naish, K. & Humphreys, G.W. Attention to food stimuli is enhanced by processing of food in working memory. Under review.
38. Fishbach A, Friedman RS, Kruglanski AW. Leading us not unto temptation: momentary allurements elicit overriding goal activation. J Pers Soc Psychol. 2003;84:296–309.
39. • Papies EK, Hamstra P. Goal priming and eating behavior: enhancing self-regulation by environmental cues. Heal Psychol. 2010;29:384–8. *This study took place in a naturalistic setting in which free meat snacks were offered from a tray in a store. Participants were subtly primed with a dieting goal (a poster for a slimming recipe on the outside of the store) or not primed. Unrestrained eaters were not affected by the dieting prime but restrained eaters ate less when primed with dieting than when not primed, suggesting that unobtrusively priming the goal of dieting can enhance self-regulation in tempting eating situations*.
40. Hare TA, Malmaud J, Rangel A. Focusing attention on the health aspects of foods changes value signals in vmPFC and improves dietary choice. J Neurosci. 2011;31:11077–87.
41. Herman CP, Mack D. Restrained and unrestrained eating. J Personal. 1975;43:647–60.
42. Demos KE, Kelley WM, Heatherton TF. Dietary restraint violations influence reward responses in nucleus Accumbens and Amygdala. J Cogn Neurosci. 2011;23:1952–63.
43. Nederkoorn C, Houben K, Hofmann W, Roefs A, Jansen A. Control yourself or just eat what you like? Weight gain over a year is predicted by an interactive effect of response inhibition and implicit preference for snack foods. Heal Psychol. 2010;29:389–93.
44. Appelhans BM, Woolf K, Pagoto SL, et al. Inhibiting food reward: delay discounting, food reward sensitivity, and palatable food intake in overweight and obese women. Obesity. 2011;19:2175–82.
45. Batterink L, Yokum S, Stice E. Body mass correlates inversely with inhibitory control in response to food among adolescent girls: an fMRI Study. NeuroImage. 2010;52:1696–703.
46. Houben K, Wiers RW, Jansen A. Getting a grip on drinking behavior: training working memory to reduce alcohol abuse. Psych Sci. 2011;22:968–75.
47. Bickel WK, Yi R, Landes RD, et al. Remember the future: working memory training decreases delay discounting among stimulant addicts. Biol Psychiatry. 2011;69:260–5.
48. Houben K. Overcoming the urge to splurge: Influencing eating behavior by manipulating inhibitory control. J Behav Ther Exp Psychiatr. 2011;42:384–8.
49. Woods SC, Ramsay DS. Pavlovian influences over food and drug intake. Behav Brain Res. 2000;110:175–82.
50. Booth DA, Lee M, McAleavey C. Acquired sensory control of satiation in man. B J Psychol. 1976;67:137–47.
51. Brunstrom JM, Rogers PJ. How many calories are on our plate? Expected fullness, not liking, determines meal-size selection. Obesity. 2009;17:1884–90.
52. Swithers SE, Doerflinger A, Davidson TL. Consistent relationships between sensory properties of savory snack foods and calories influence food intake in rats. Int J Obes. 2006;30:1685–92.
53. • Swithers SE, Ogden SB, Davidson TL. Fat substitutes promote weight gain in rats consuming high-fat diets. Behav Neurosci. 2011;125:512–8. *In this study, laboratory rats received potato chips as a supplement to their diet. One group was fed a mixture of high-fat chips and low-fat chips, containing the fat substitute olestra, while the other group received only high-fat chips. The group given the high-fat and low-fat chips gained more weight than the rats just fed the high-fat chips but only if they had concurrent access to a high-fat diet. Further, rats previously fed on both the high- and low-calorie chips had greater body weight gain when they were switched from a low-fat to a high-fat diet, suggesting that experience with the low-fat chips affected the ability to predict high calories based on the sensory properties of fat*.
54. Davidson TL, Martin AA, Clark K, Swithers SE. Intake of high-intensity sweeteners alters the ability of sweet taste to signal caloric consequences: Implications for the learned control

of energy and body weight regulation. Q J Exp Psychol. 2011;64:1430–41.
55. O'Sullivan HL, Alexander E, Ferriday D, Brunstrom JM. Effects of repeated exposure on liking for a reduced-energy-dense food. Am J Clin Nutr. 2011;91:1584–9.
56. Higgs S. Memory for recent eating and its influence on subsequent food intake. Appetite. 2002;39:159–66.
57. Moller P, Mojet J, Koster EP. Incidental and intentional flavor memory in young and older subjects. Chem Senses. 2007;32:557–67.
58. Higgs S. Memory and its role in appetite regulation. Physiol Behav. 2005;85(67):72.
59. Higgs S. Cognitive influences on food intake: the effects of manipulating memory for recent eating. Physiol Behav. 2008;94:734–9.
60. Higgs S, Williamson AC, Rotshtein P, Humphreys GW. Sensory specific satiety is intact in amnesics who eat multiple meals. Psychol Sci. 2008;19:623–8.
61. Hebben N, Corkin S, Eichenbaum H, Shedlack K. Diminished ability to interpret and report internal states after bilateral medial temporal resection: case H.M. Behav Neurosci. 1985;99:1031–9.
62. Rozin P, Dow S, Moscovitch M, Rajaram S. What causes humans to begin and end a meal? A role for memory for what has been eaten, as evidenced by a study of multiple meal eating in amnesic patients. Psychol Sci. 1998;9:392–6.
63. Higgs S, Williamson AC, Attwood AS. Recall of recent lunch and its effect on subsequent snack intake. Physiol Behav. 2008;94:454–62.
64. Higgs S, Woodward M. Television watching during lunch increases afternoon snack intake of young women. Appetite. 2009;52:39–43.
65. • Oldham-Cooper RE, Hardman CA, Nicoll CE, Rogers PJ, Brunstrom JM. Playing a computer game during lunch affects fullness, memory for lunch, and later snack intake. Am J Clin Nutr. 2011;93:308–13. *In this study, participants played a computer game while eating their lunch or ate the same lunch in the absence of distraction by the computer game. Eating while playing the computer game led to greater consumption of snacks a few hours later compared with eating in the absence of distraction. Furthermore, the authors used a quantifiable measure of lunch memory and found that serial-order memory for the presentation of the nine lunch items was less accurate in participants who had been distracted during lunch. These data add to previous reports that impairing lunch memory via distraction influences subsequent eating*.
66. Mittal D, Stevenson RJ, Oaten MJ, Miller LA. Snacking while watching TV impairs food recall and promotes food intake on a later TV free test meal. Appl Cogn Psychol. 2011;25:871–7.
67. Moray J, Fu A, Brill K, Mayoral MS. Viewing television while eating impairs the ability to accurately estimate total amount of food consumed. Bariatr Nurs Surg Patient Care. 2007;2:71–6.
68. Jackson DM, Djafarian K, Stewart J, Speakman JR. Increased television viewing is associated with elevated body fatness but not with lower total energy expenditure in children. Am J Clin Nutr. 2009;89:1031–6.
69. • Higgs S, Donohoe J. Focusing on food during lunch enhances lunch memory and decreases later snack intake. Appetite. 2011;57:202–6. *In this study, participants were instructed to pay attention to their lunch while eating or were instructed to eat the same food either in their usual manner or while reading a magazine article about food. Focusing on lunch as it was eaten led to a decrease in afternoon snack intake compared with usual eating or eating when reading. These results have implications for the design of interventions to prevent overeating that are focused on attentive eating strategies*.
70. Brunstrom JM, Brown S, Hinton EC, Rogers PJ, Fay SH. 'Expected satiety' changes hunger and fullness in the inter-meal interval. Appetite. 2011;56:310–5.
71. Ford AL, Bergh C, Sodersten P, et al. Treatment of childhood obesity by retraining eating behaviour: randomised controlled trial. Br Med J. 2010;340:b5388.
72. de Graaf C, Kok FJ. Slow food, fast food and the control of food intake. Nat Rev Endocrinol. 2010;6:290–3.
73. Kanoski SE, Davidson TL. Western diet consumption and cognitive impairment: links to hippocampal dysfunction and obesity. Physiol Behav. 2011;103:59–68.
74. Kanoski SE, Davidson TL. Different patterns of memory impairments accompany short- and longer-term maintenance on a high-energy diet. J Exp Psychol-Anim Behav Process. 2010;36:313–9.
75. Smith E, Hay P, Campbell L, Trollor JN. A review of the association between obesity and cognitive function across the lifespan: implications for novel approaches to prevention and treatment. Obes Rev. 2011;12:740–55.
76. Rode E, Rozin P, Durlach P. Experienced and remembered pleasure for meals: duration neglect but minimal peak, end (recency) or primacy effects. Appetite. 2007;49:18–29.
77. • Robinson EL, Blissett J, Higgs S. Recall of vegetable eating affects future predicted enjoyment and choice of vegetables. J Am Diet Assoc. 2011;111:1543–8. *This is the first study to show that recalling an event when vegetables were eaten and enjoyed can enhance anticipated enjoyment of vegetable eating and actual consumption of vegetables. This suggests that a simple, inexpensive event recall strategy could be used as an intervention procedure to increase enjoyment and consumption of vegetables*.
78. Robinson EL, Blissett J, Higgs S. Changing memory of food enjoyment to increase food liking, choice and intake. Brit J Nutr, in press
79. Robinson EL, Blissett J, Higgs S. Dietary restraint moderates the influence of peak and end effects on remembered enjoyment of eating. Appetite. 2011;57(207):212.