Available online at www.sciencedirect.com
ScienceDirect
Neuroscience and Biobehavioral Reviews 31 (2007) 987–1002
NEUROSCIENCE AND BIOBEHAVIORAL REVIEWS
www.elsevier.com/locate/neubiorev

Review

Liking vs. wanting food: Importance for human appetite control and weight regulation

Graham Finlayson*, Neil King, John E. Blundell

Biopsychology Group, Institute of Psychological Sciences, University of Leeds, Leeds LS2 9JT, UK

Received 27 November 2006; received in revised form 19 March 2007; accepted 20 March 2007

Abstract

Current train of thought in appetite research is favouring an interest in non-homeostatic or hedonic (reward) mechanisms in relation to overconsumption and energy balance. This tendency is supported by advances in neurobiology that precede the emergence of a new conceptual approach to reward where affect and motivation (liking and wanting) can be seen as the major force in guiding human eating behaviour. In this review, current progress in applying processes of liking and wanting to the study of human appetite are examined by discussing the following issues: How can these concepts be operationalised for use in human research to reflect the neural mechanisms by which they may be influenced? Do liking and wanting operate independently to produce functionally significant changes in behaviour? Can liking and wanting be truly experimentally separated or will an expression of one inevitably contain elements of the other? The review contains a re-examination of selected human appetite research before exploring more recent methodological approaches to the study of liking and wanting in appetite control. In addition, some theoretical developments are described in four diverse models that may enhance current understanding of the role of these processes in guiding ingestive behaviour. Finally, the implications of a dual process modulation of food reward for weight gain and obesity are discussed. The review concludes that processes of liking and wanting are likely to have independent roles in characterising susceptibility to weight gain. Further research into the dissociation of liking and wanting through implicit and explicit levels of processing would help to disclose the relative importance of these components of reward for appetite control and weight regulation.
© 2007 Elsevier Ltd. All rights reserved.

Keywords: Liking; Wanting; Food reward; Hedonics; Overconsumption; Appetite control; Weight regulation; Homeostasis

Contents

1. Introduction . . . 988
2. Neurobiological study of liking and wanting . . . 988
   2.1. How is liking measured? . . . 988
   2.2. How is wanting measured?. . . . 989
   2.3. Implicit and explicit components . . . 989
3. From core processes to constructs in human appetite . . . 989
4. Reward and appetite control: homeostatic and hedonic interplay . . . 990
   4.1. Homeostasis and hedonics: separate identities... . . . 990
   4.2. ...Or inseparable entities? . . . 991
5. Liking, wanting and ingestive behaviour: a re-examination of selected studies . . . 992
   5.1. Intake and meal size . . . 992
   5.2. Hunger and satiety . . . 992

*Corresponding author. Tel.: +44 113 343 5753; fax: +44 113 343 6674.
E-mail address: g.s.finlayson@leeds.ac.uk (G. Finlayson).

0149-7634/$ - see front matter © 2007 Elsevier Ltd. All rights reserved.
doi:10.1016/j.neubiorev.2007.03.004

5.3. Ratings of subjective liking . . . 993
6. Dual-component contributions to the study of reward in human appetite . . . 993
7. Further conceptual development . . . 995
7.1. Homeostatic-hedonic model of hunger . . . 995
7.2. Motivating operations in human appetite . . . 996
7.3. The tonic/phasic model of da system regulation . . . 997
7.4. Determinants of food choice model . . . 998
8. Implication for weight gain and obesity . . . 998
9. Conclusion . . . 1000
10. Postscript . . . 1000
Acknowledgement . . . 1000
References . . . 1000

1. Introduction

According to the French National Nutrition-Health Program (2001–2005) it is essential that an individual's food choice remains a 'free act' and that eating is recognised as a moment of pure pleasure. In order to fully appreciate these premeditated episodes of sensuality, it is not sufficient to focus only on the hedonic sensations arising from events in the mouth. The joy of eating can be as much to do with the preparation and effort that one invests in their chosen food and of course the expectancy and anticipation that intensify then peak in the final moments before ingestion. In summary, the pleasure of food can be seen as an interaction of liking and wanting, and experiencing one without the other—although pleasurable in isolation—stops short of full reward.

Advances in neurobiology are helping to characterise the substrate mediating hedonic processes of consumption, and they are precipitating the emergence of a new conceptual approach to reward where affect and motivation (a.k.a. liking and wanting) can be seen as the major force in guiding human eating behaviour. This concept is especially important for the study of ingestive behaviour in the modern world, where food is plentiful, cheap, energy-dense, and enticing, and physical activity is being reduced to a luxury afforded by environment and lifestyle. We have reached an age where weight control has been turned upside down from an instinctual, highly regulated system, to a process requiring considerable cognitive effort (Peters et al., 2002). Furthermore, where overweight and obesity have taken hold, losing weight and defending that loss (especially in an environment where our hedonic drives are encouraged and exploited) can change from a daily struggle, to a losing battle (e.g. Ikeda et al., 2005).

Parsing reward from a unitary process into distinguishable liking and wanting components in neurobiological studies (Berridge and Robinson, 2003) has struck a chord that is resonating across many disciplines and in different areas of research. In the field of ingestive behaviour alone it has implications for characterising eating disorders and obesity, identifying pharmacological targets, the psychology of appetite control, phenotypic profiling of resistance and susceptibility to weight gain, and industrial product development. Despite the possibilities of a dual process modulation of food reward, several issues remain to be addressed: How can these concepts be operationalised for use in human appetite research? Can they be translated into observable entities that reflect the neural mechanisms by which they may be influenced? Do liking and wanting operate independently to produce functionally significant changes in behaviour? Can liking and wanting be truly separated or will an expression of one inevitably contain elements of the other? In this review, current progress in applying processes of liking and wanting to the study of human appetite and ingestive behaviour are examined and the importance of these concepts for human appetite research are discussed.

2. Neurobiological study of liking and wanting

Our capacities in neuroscience can reveal—to some extent—the circuits responsible for the reward we derive from food. The picture emerging is that food reward, rather than being a unitary neurological entity, is represented functionally and structurally by distinct components. One such distinction, between processes associated with affective vs. motivational consequences of ingesting food, has received much recent attention (Berridge, 1996). With principle focus on opioid neurotransmission in the nucleus accumbens shell and the mesolimbic dopamine system, research has shown that core processes of 'liking' and 'wanting' can be separately manipulated in rodent models to produce patterns of behaviour that are either exclusively affective or motivational in conjunction with a food stimulus (see http://www.lsa.umich.edu/psych/research&labs/berridge/Publications.htm for a comprehensive list of publications).

2.1. How is liking measured?

In its simplest description, 'liking' is most commonly operationalised as the change in affect observed using a technique to analyse taste reactivity patterns in rats (Grill and Norgren, 1978). This involves a careful assessment of hedonic and aversive behavioural reactions thought to be universal affective expressions, some of which can also be

observed—allowing for speed and body size—in primates and human infants (Berridge, 2000). Taste reactivity patterns are thought to provide a relatively pure indication of affect because they can be isolated from the sensory properties of a taste (see Berridge, 2000), they can be dissociated from the desire to eat (e.g. Berridge and Valenstein, 1991; Berridge et al., 1989) and they often correspond to human subjective ratings of palatability.

2.2. How is wanting measured?

If liking is operationalised as the pattern of reactive behaviours associated with affective aspects of food reward, then wanting is commonly measured by changes in the propensity to eat that are independent of shifts in liking. Therefore, any measure that requires the subject to actively engage with its environment in pursuit of a known food stimulus can be said to be contain at least an element of wanting. It is worth emphasising at this juncture however that wanting (specifically termed by some as incentive salience attribution) is not adequately captured by appetitive drive or the non-specific desire for food in general. Wanting is hypothesised to be the consequence of an active process of assigning value to perceptual or representational events wherein sensory and cognitive inputs are transformed into desirable, attractive entities (Berridge, 1996). Hence wanting is likely to be modulated by sensory and/or cognitive influences which set it apart from other appetitive processes (e.g. needing). Wanting implies a direction, not just a force. Therefore, obtaining a measure of wanting that can be dissociated from a non-specific drive to eat (e.g. see Beagley and Holley, 1977; as cited in Berridge, 1996) can become awkward when a further separation from liking is required. In passing, the investigation of specific food cravings may provide a useful platform from which to further clarify these processes (e.g. Robinson and Berridge, 2003; Pelchat, 2002). Nevertheless, the more satisfactory measures of wanting concern the anticipatory or instrumental phase of reward seeking behaviour.

2.3. Implicit and explicit components

Through the separate examination of specific neural substrates in the brain, behavioural measures of liking and wanting are thought to reflect 'core' processes that can operate without conscious awareness. However, these implicit components clearly have their explicit counterparts which express themselves subjectively in the form of hedonic feelings from the ingestion of a specific food (conscious liking) and the intent or desire to consume a specific food (conscious wanting) (Berridge and Robinson, 2003). Therefore it is important that we know how core liking and wanting (operating at implicit and explicit levels of conscious awareness) might relate to influence behaviour.

Berridge (2004) used the term 'niceness gloss' (the pleasantness added by the brain to the sensory qualities of sweetness) in reference to the explicit component of liking, with implicit liking and its associated brain structures forming an unconscious core. In this review, implicit liking was presented as capable of producing objective reactions without subjective awareness of their underlying cause. For instance, the subliminal presentation of picture stimuli (positive, neutral or negative facial expressions) caused no change in self reported mood ratings, but the emotional valence of the stimuli was positively associated with pleasantness rating, consumption and rated value of a beverage (Winkielman et al., 2005).

As explained above, the implicit component of wanting is linked to the attribution of 'incentive salience' to external stimuli. Explicit wanting (truer to the colloquial understanding of the word), has been described as the conscious desire for a cognitively represented outcome (Berridge, 2004). Interestingly, implicit wanting has also been shown capable of producing objective reactions independent of any subjective awareness (Berridge, 2004). For instance, it has been demonstrated that animals can show 'irrational wanting' for food rewards. In one study, activation of the dopamine system caused a reward cue (conditioned stimulus) for sucrose to become a hyper incentive, temporarily outstripping the reward value of the sucrose presented alone or with an irrelevant cue (Wyvell and Berridge, 2000). Such findings suggest that implicit wanting could be a more compelling influence on behaviour than explicit (conscious) wanting (Berridge, 2004).

In summary, liking and wanting have been put forward as core processes with implicit and explicit features. Explicit liking (acutely perceived hedonic reaction) can be associated with explicit wanting (subjective desire for a perceived goal) and implicit wanting (incentive salience attribution). Implicit liking (unconscious affect) may also influence implicit wanting to influence ingestive behaviour without the subjective awareness of either process. Lastly, wanting can be irrational when implicit wanting for a reward is greater than explicit wanting, and not proportional to the experience or expectation of liking (e.g. Wyvell and Berridge, 2000).

3. From core processes to constructs in human appetite

If core processes of liking and wanting can independently modulate food reward both implicitly and explicitly, it is important to understand how they can be approached and rendered suitable for the study of ingestive behaviour in humans. Even to date, the role of food reward in human appetite behaviour is mostly treated as a single entity embodied by a 'palatability' or 'pleasantness' factor and its effects on appetite control (Yeomans, 1998). The logical view is that liking and wanting co-vary in a natural two-way sequence. Therefore, in behavioural terms we

assume that a change in liking—e.g. through manipulating palatability—will lead to proportional adjustments in wanting—e.g. amount of the commodity consumed (Bobroff and Kissileff, 1986; Yeomans et al., 1997), and likewise changes in wanting—e.g. indirectly, through manipulating hunger and satiety—will effect changes in liking—e.g. hedonic response (Cabanac, 1979; Carr and Wolinsky, 1993). A dissociation of liking and wanting in the brain suggests that this self-evident liking↔wanting sequence may not always hold true, and this could throw new light on the way human ingestive behaviour is understood.

Translating liking and wanting into constructs amenable to the study of human ingestive behaviour has a number of obstacles, and most concern the accuracy and accessibility of our subjective consciousness. Firstly, our feelings may not do justice to the complexity of their underlying processes. For example, is the hedonic strength of a response a consequence of one underlying dimension (e.g. pleasantness) or a convergence of two (e.g. pleasantness and unpleasantness) or three (e.g. pleasantness, unpleasantness, and salience) or numerous other dimensions? Affective taste reactivity in rats and infants can simultaneously assess hedonic (i.e. positive) and aversive (i.e. negative) aspects of affect (Berridge, 2000), but asking humans to introspectively provide the same information may be too contradictory and confusing. In human appetite research, it has been suggested that distinct sets of underlying processes can be interpreted as a single more general variable which is only then partitioned cognitively into the required domains (Booth, 1987a, b). The distorting potential of cognitions on affect is cautioned by Berridge (1996) where the accuracy of actively reconstructing emotional events is vulnerable to excessive cognitive processing. Hence—paradoxically—the more someone is encouraged to consider their feelings, the less reliable their responses may become. However, introspectionist constructions of underlying processes can refer to unconscious experiences as well as those in conscious awareness (e.g. hunger, craving, fullness, thirst), but it must be remembered that this method can only reveal a causal relationship between the construct and the behaviour for the elements of the experience that are explicit and subjective (Booth 1987a, b; Booth and Blair, 1988). An adequate resolution of this problem requires more than the examination of these constructs and their behavioural correlates. In the case of liking and wanting, the utility of introspective constructions can only be judged in the context of their interaction with objective mental/behavioural outputs and the ability to distinguish these from other environmental contingencies (e.g. Booth, 1990). Alternatively, measures that are able to better reflect the unconscious components of these constructs or are less reliant on the cognitions of the subject, or are specifically designed to test for the dissociability of distinct underlying processes, could greatly facilitate a dual-process approach to the study of reward in human appetite research.

4. Reward and appetite control: homeostatic and hedonic interplay

A key issue in the study of appetite control is the relationship between reward and homeostatic drives arising from biological needs (Yeomans et al., 2004a). Historically, hedonic processes have been viewed as a function of nutritional need-state. In a state of depletion, the hedonic response (experienced palatability) to energy providing foods is enhanced and when replete, the hedonic effect of these foods is reduced (Cabanac, 1989). This view is compatible with the link between energy density and palatability (Drewnowski, 1998) and also that the consumption of fats and sugars—rich sources of energy—may be under neuro-regulatory control (Levine et al., 2003). However, the idea of reward as a consequence of the fulfilment of nutritional need is not broad enough to explain non-homeostatic ingestive behaviour (non-compensated patterns of over or under consumption) and it is perhaps more useful to try and distinguish the neural substrates of homeostatic and hedonic systems and to assign them separate identities (Blundell and Finlayson, 2004).

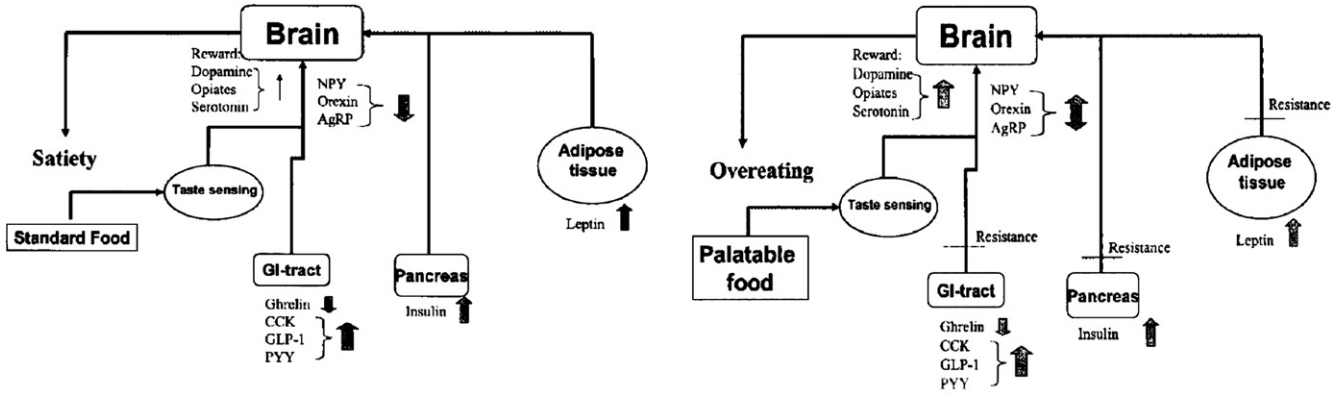
4.1. Homeostasis and hedonics: separate identities…

The homeostatic substrate comprises a network of neuropeptides and biogenic aminergic neurotransmitters which link peripheral and central components. This system has been well characterised (e.g. Schwartz et al., 2000) and involves insulin, leptin, NPY, AgRP, MSH, CART, GLP-1, orexins, ghrelins, PYY, and other peptides along with serotonin pathways and other aminergic systems. A biological substrate mediating the reward processes of consumption is also being characterised and ostensibly involves glutamate, benzodiazepines, endocannabinoids, opioids and dopamine pathways (e.g. Saper et al., 2002; Flier, 2004). The implication of distinct neural substrates for homeostatic and hedonic systems is that processes of reward can operate free from biological need, and the extent to which this occurs can be investigated. For example, pharmacological evidence suggests that these circuits are somewhat separate. In obese subjects, administration of the serotonin drug D-fenfluramine suppressed the sensation of hunger but had no effect on the appreciation of the pleasantness of food (Blundell and Hill, 1987). Conversely, an opioid antagonist reduced the rated pleasantness of palatable foods but had no effect on hunger (Yeomans and Gray, 2002). This double-dissociation concept indicates that hedonic aspects of reward are associated with a specific biological substrate that can be pharmacologically dissected from the substrate-mediating hunger (Rogers and Blundell, 1991). This is supported by evidence from animal studies. In one study (Giraudo et al., 1999), saline (control), NPY or an opioid agonist (DAMGO) was injected into the paraventricular nucleus of rats. The rats could freely consume from standard chow

and 10% sucrose solution. After injection of NPY, food intake was increased relative to saline, and the rats were found to consume approximately half their calories from the chow and half from the sucrose solution. Injection of DAMGO also stimulated intake, but in this condition 85% of calories came from the sucrose. Therefore, NPY and opiates may represent a demarcation between energy-driven versus reward-driven feeding. However, it is still possible for a functional interaction to occur when the manipulation is made through the natural commodity (food) rather than through more selective pharmaceutical manipulations.

4.2. …Or inseparable entities?

Advances in our understanding of the molecular and neural mechanisms behind appetite regulation are revealing how the reward system can interact with homeostatic mechanisms. As mentioned above, cannabinoid receptors and their endogenous ligands (e.g. anandamide) are implicated in the reward system. Peripheral and central administration of anandamide increased appetite in rodents, and this seemed to be related to alterations in incentive value (desire) for palatable foods (Kirkham and Williams, 2001). However, the cannabinoid system has been shown to interact with homeostatic processes in a number of ways (Stanley et al., 2005): Leptin signalling becomes defective when hypothalamic endocannabinoid levels are high (Di Marzo et al., 2001); activation of CB1 receptors prevent the melanocortin system from altering food intake (Verty et al., 2004); furthermore, CB1 receptors can be found on adipocytes where they may directly increase lipogenesis (Cota et al., 2003). Opioid neurotransmission also forms part of the biological substrate mediating reward processes of consumption. For example, endogenous opioids are associated with the reinforcing effect of food (especially when palatable) (Welch et al., 1996; Yamamoto et al., 2000). However, there is evidence to show that in a fasted state, the reinforcing effect of food can be reinstated in enkephalin and β-endorphin knock-out mice (Hayward et al., 2002). Therefore, homeostatic processes may interact with hedonic signalling to override selective reward deficit. Erlanson-Albertsson (2005) summarised how ingestion of palatable food can offset normal (homeostatic) appetite regulation. In the brain, research shows that energy deficit is registered in the hypothalamus leading to the release of hunger signals and the activation of their receptors. Consumption of 'standard' food generates information on its energy content and taste in the brain stem. This information is transmitted to the hypothalamus leading to the release or upregulation of various satiety peptides, causing consumption to cease. However, a different scenario is apparent when the reward system is activated by highly palatable food. With ingestion of palatable food, taste sensing is different than with standard food; information is transmitted to the reward circuit, leading to the release or upregulation of reward mediators like dopamine, endocannabinoids, and opiates. The reward circuit has connections with appetite-controlling neurones in the hypothalamus that can increase the expression of hunger peptides such as NPY and orexins, while blunting the signalling of satiety peptides like insulin, leptin and cholecystokinin. Therefore when food is highly palatable, the drive to eat is maintained, with continued eating now mediated by reward rather than biological need (see Fig. 1). Hence although homeostatic and hedonic systems can be given separate identities (Blundell and



Left. *Hunger and satiety signalling during intake of a standard meal. Hunger signals, such as ghrelin in the stomach and NPY, orexin, AgRP in the hypothalamus, are depressed after intake of standard food, while satiety signals like CCK, GLP-1, PYY, insulin and leptin are raised. Food intake is terminated as a result.*

Right. *Hunger and satiety signalling after a period on a diet of palatable food. Hunger signals are either depressed, like ghrelin in the stomach and NPY in the hypothalamus, in response to a meal consisting of palatable food or raised, as for orexin and AgRP in the hypothalamus. Satiety signals like insulin and leptin are increased. Palatable food induces resistance to several satiety signals, documented for CCK, insulin and leptin, resulting in overeating. Food intake is driven by an increased activity in the reward system (dopamine, serotonin and opiates), triggered by the attractiveness of the taste.*

Fig. 1. Hunger and satiety signalling during consumption of standard (A) and palatable (B) food (Erlanson-Albertsson, 2005).

Finlayson, 2004), they are also—to an extent—inseparable, with neural cross-talk permitting functional interactions which may influence the organisation of ingestive behaviour. From this standpoint, the interaction of homeostatic and non-homeostatic pathways in the neuro-regulatory control of feeding may be given more importance than the two systems studied in isolation. From behavioural and anatomical observations, Berthoud (2006) suggested that projections from the hypothalamus to the nucleus accumbens may modulate the motivation to feed via metabolic signals. Furthermore, direct and indirect projections from the accumbens to the hypothalamus may explain the ability for mesolimbic processes—activated by relevant environmental cues and incentives—to essentially hijack the homeostatic regulatory circuits and drive up energy intake. Further research is necessary to identify the pathways that mediate such interactions; however some progress has been made (see Berthoud, 2004).

5. Liking, wanting and ingestive behaviour: a re-examination of selected studies

Considering that most studies have repeatedly shown that palatability—a factor influencing the reward value of food—has an effect on intake, this would support the notion that reward plays a role in the process of satiation (De Graaf et al., 1999). However, it is uncertain how processes of liking and wanting might independently modulate the effect of reward on appetite to influence ingestive behaviour. With a dual process perspective on reward, it becomes possible to re-examine some of the previous research investigating its impact on human appetite.

5.1. Intake and meal size

Manipulating the palatability of test foods is a common way to investigate how reward processes can influence ingestive behaviour. Most research using such an approach has found increased palatability to have a stimulatory effect on intake and meal size, leading some authors to propose a proportional relationship between subjective shifts in palatability and corresponding adjustment of food intake in grams (e.g. Bobroff and Kissileff, 1986). However, differences in the palatability of foods do not provide a comprehensive indication of their reward value. In most cases a palatability manipulation (brought about by a change in the physical or sensory properties of a food) can only account for differences in liking for the food. Less can be assumed about the effects of the same manipulation on the desire or motivation to consume the food. It is interesting therefore, that a small number studies do not find an association between palatability and food intake. For example, supplements of monosodium L-glutamate enhanced the palatability of a soup preload, but had no effect on the consumption of a test meal delivered 2 or 30 min later (Rogers and Blundell, 1990). Similarly, manipulation of the palatability of fat or carbohydrate based meals did not significantly decrease intake despite reduced hunger and enhanced satiety (Warwick et al., 1993). In one study, manipulated palatability of the test foods correlated to overall intake in only half the subjects (Lucas and Bellisle, 1987). Some free-living studies also indicate that the role of liking in determining meal size may not be as crucial as many laboratory studies have suggested. For instance, in obese and lean subjects, no correlation was found between liking for preferred taste (sweet foods) and total intake of foods in that taste category, and there was very little variability between mean hedonic ratings for each taste category (sweet, savoury/salty, bitter, sour) in self-selected foods (Cox et al., 1999). Other research on free-living subjects in France and North America found a strong association between palatability and meal size, but demonstrated that over 70% of all recorded meals were rated at above neutral palatability (De Castro et al., 2000a, b). These studies suggest that the subjective pleasantness of meals is likely to play a role in food choice, but may be less important in accounting for the variability in amount consumed. Indeed, it is intuitive that in real life, where people have freedom of choice, palatability will be a relatively consistent factor in all meals. Therefore, in isolation liking for foods may not tell the whole story about reward driven food intake.

For example, in one quasi-experimental study, three different types of test meal (conventional four course, sandwich, and semi-liquid) at two levels of palatability (high or low) were compared after covert video recording (Guy-Grand et al., 1994). The palatability manipulation was only found to stimulate intake in the conventional meal, yet it produced comparable differences in subjective liking for all the meals. However, in the sandwich meal, bite rate was significantly greater in the lower palatability condition, and in the semi-liquid meal, pause duration was less. These behaviours seem at odds with the lower ratings of pleasantness for this level of palatability. Indeed, if bite rate and pause duration are taken as indicators of the subjects' overall motivation toward their food, then it appears that wanting was to some extent higher, despite the meals being less liked. Interestingly, studies that have employed some measure of eating rate in their methodologies have commonly found the palatability-dependent effects of rate on intake were confined to the initial stages of the eating bout (Bellisle et al., 1984; Bobroff and Kissileff, 1986; Spiegel et al., 1989; Yeomans, 1996)—presumably when motivation to eat is at a peak. Such measures may help to discern and separately track processes of liking and wanting within a meal.

5.2. Hunger and satiety

Previously in the literature, it has been disputed whether liking for food may have an influence on hunger and satiety (see De Graaf et al., 1999). One early study (Hill et al., 1984) found an enhancing effect of an equi-caloric but preferred meal on rated hunger. Even the sight of the

preferred food increased hunger suggesting that the expectancy or anticipation of food (wanting) was stimulating appetite before liking for the food could be confirmed. Other research has found a hunger-dependent effect of palatability on intake (Spiegel et al., 1989). When lean subjects were deprived of breakfast, there was no difference in intake when offered either low or high palatability foods at lunch. When breakfast was reinstated, intake of the high palatability food was greater. One proposal is that liking is an important factor in food intake within an acceptable threshold of hunger; once this threshold is crossed, the palatability of available foods is then secondary to perceived energy requirements.

Therefore, the mediating effect of liking on food intake may be overridden by potent homeostatic signals. Conversely, in a study where disguised high or low energy preloads were administered before tasty or bland test meals (Yeomans et al., 2001), subjects failed to compensate for the preloads when subsequent food was more palatable. It was argued that tasty foods prevented short-term responses to satiety cues. Thus, homeostatic signals may also be overridden by liking. However the system does appear to operate asymmetrically; although enhanced liking for foods can augment hunger (and therefore food intake), the presence of strong satiety developing over the course of a meal does not always downregulate a food's palatability (e.g. see Yeomans and Symes, 1999; Looy et al., 1992) or wanting for other available foods (Finlayson et al., under review).

5.3. Ratings of subjective liking

Nearly, all human studies interested in reward processes in appetite include some subjective measure of liking. Nevertheless, several studies have revealed that such introspective ratings may be of limited use. The most common tests involving subjective ratings of liking are brief exposure tests and fixed quantity tests. Although easy to conduct, these tests are considerably different to free-living eating situations where much larger quantities of food are eaten and in combination with other foods and drinks. Bellisle et al. (1984), Lucas and Bellisle (1987), Monneuse et al. (1991), Perez et al. (1994) and Zandstra et al. (1999) have all found brief exposure tests to give a biased estimation of the optimal palatability of a food. Zandstra et al. (1999) found consumption to correlate best with ad libitum tests and worst with taste-and-spit tests. This finding gains support from several other studies who find liking ratings alone insufficient to predict subsequent intake (Bellisle and Le Magnen, 1980; Hellemann and Tuorila, 1991). This point has been explored elsewhere (Mook and Votaw, 1992): by asking college students to rank or choose from a list of reasons for terminating a meal, the authors found hedonic factors to be of little perceived importance. They cautioned that studies employing subjective ratings may enhance the perceived importance of the hedonic properties of test foods that otherwise might not be considered. For instance, one study investigated repeated within meal interruptions (in order to make subjective appetite and hedonic ratings) and their effect on intake (Yeomans et al., 1997). Surprisingly, when meals were interrupted at every 50 g interval of intake, overall consumption was significantly greater compared to one continuous bout of feeding. It is possible that by repeatedly eliciting a conscious, subjective evaluation of liking for available foods, the incentive salience of the food may also have been inadvertently enhanced.

6. Dual-component contributions to the study of reward in human appetite

With a dual-process model of reward, retrospective evaluation of the literature may help to throw light on the relationship between food reward, appetite and ingestive behaviour. More recently, researchers are beginning to consider liking and wanting interpretations of their own study findings and some are specifically tuning their methodologies to allow for separations of motivation and affective responding to food. These studies provide examples of how liking and wanting may be operationally defined in human appetite research.

In one study (Zandstra et al., 2000), three levels of palatable food were consumed as open sandwiches each day for three weeks (5 days repeated exposure per level of palatability). As well as measuring ad libitum food intake and subjective appetite on each day, change in rated pleasantness of the food and change in rated desire-to-eat were assessed by subtracting post-meal ratings from ratings taken after the first bite. The study found that intake on the first two days reflected the manipulated palatability of the foods, with proportional differences between low, medium and high levels. On the third day of repeated exposure, intake between the medium and high levels was equivalent, and on the fifth day, intake did not vary between any of the conditions. Similarly, ratings of fullness suggested that satiety following the low palatability food was increasing with time. On examination of ratings of pleasantness and desire-to-eat, an interesting dissociation was observed. After a considerable reduction in desire-to-eat on the first day of exposure for the low palatability food relative to the other levels, this decline dissipated after a few days and ratings for all foods became equivalent. In contrast, the change in rated pleasantness for each level of palatability was stable over time. Therefore, the convergence of intake and satiety for three levels of palatability brought about over five days of repeated exposure was associated with changes in desire to eat (explicit wanting) and not rated pleasantness (explicit liking) for the foods. More recently, these explicit processes have also been dissociated by manipulating expectations by labelling or withholding information about fresh versus processed food (Zandstra et al., 2004). When consumed blind, foods were desired and liked similarly. However, when labelled as fresh or processed, desire for the fresh food was greater, with no

change in liking. The findings of both studies may justify the importance of differentiating questions of liking and desire (explicit wanting) for foods. The authors suggested that without the opportunity to rate liking and wanting aspects of the foods separately, subjects' ratings may have contained elements of both. In other words, they thought the explicit components of each underlying process may have been interpreted as single subjective feeling.

Another study compared lean and obese subjects on their liking for an array of food products (sandwiches or snacks) in hungry and satiated states (Snoek et al., 2004). Subjects tested and rated a selection of foods before consuming one food item to fullness as an ad libitum meal. Ratings of appetite that were specific (e.g. "appetite for a meal", "appetite for something sweet", "appetite for a snack" etc.) as well as non-specific (e.g. hunger, fullness) were recorded immediately before and after a sandwich or snack 'meal'. Results showed that obese and lean subjects did not rate the pleasantness of any of the food items differently after consuming the meal. However, differences were apparent in some of the appetite ratings; specifically "appetite for a snack" and "appetite for a meal" following consumption of either snacks or sandwiches, and also "appetite for something savoury" after snack consumption. The authors speculated that obese-lean differences referred to differences in wanting and not liking, on the basis of higher perceived energy needs of obese subjects. The authors were careful however, to point out that their measures "reflect conscious, rationalised liking and wanting" (p. 830). They suggested that to study the differences in obese and lean humans will require improved more objective techniques to measure liking and wanting, for example through neuroimaging techniques, or indirect methods that do not rely on subjective ratings.

One neuroimaging study (Small et al., 2001) used consecutive PET scans to track changes in brain activity as subjects consumed chocolate to beyond satiety. In this design, subjects ate one piece of chocolate at a time, followed by a rating of how pleasant or unpleasant they found it (explicit liking), and how much they would like or not like to have another piece of chocolate (explicit wanting). Each feeding period—with a scan followed by a rest period—was determined by the amount of chocolate it took to produce a two unit decrease in rated liking. Thus, as subjects ate chocolate from a state where it was highly motivating and pleasurable, to a state where motivation to stop eating was high and chocolate consumption unpleasant, it was possible to observe the differential recruitment of brain regions as the reward value of the chocolate decreased. The study found that subjects ratings of wanting decreased faster and to a greater extent than ratings of liking, suggesting that some separation of these constructs was occurring. In order to see if these processes could be identified physically in the brain, scan order was controlled for and regional cerebral blood flow was regressed against subjective ratings of liking and wanting. It was found that activity in a region of the retrosplenial cortex correlated to a greater extent to ratings of wanting than ratings of liking. The authors suggested that this region may form part of the substrate for a dissociation between liking and wanting. Indeed, studies that incorporate objective measures of brain activity in conjunction with sensory and motivational challenges may help to further characterise separable processes of reward. As these techniques become more sophisticated it is likely that more will be understood about the connection between implicit and explicit components of reward and their influence on ingestive behaviour in more complex scenarios.

One way to overcome the difficulties of extracting reliable subjective responses may be to find alternative indirect measures of motivation or affect. For example, Saelens and Epstein (1996) assessed the reinforcing value of food using a slot-machine-like progressive ratio computer task. In this paradigm, subjects' commitment to the task was rewarded with points that could be exchanged for amounts of tasty snack food or allotments of time that could be spent playing an enjoyable computer game. The reinforcing value of the tasty food was calibrated as the willingness to work for amounts of the food relative to the time playing the game. In a study comparing obese and lean subjects, the authors found that subjective ratings of liking for snack food items—including the most preferred item used in the progressive ratio task—did not differ; however, the obese subjects were found to work harder for food relative to playing the game, and this corresponded to the amount of calories consumed. These findings confirmed earlier research associating obesity with willingness to work for food in the presence of food cues (Johnson, 1974). These findings may also demonstrate important differences in reward-driven behaviour in these populations. Although liking for rewards may be involved in establishing their reinforcing value, it is possible for wanting to become a more significant factor in influencing ingestive behaviour (e.g. Robinson and Berridge, 2000). Furthermore, the lack of differences in subjective hunger ratings suggests that the differences in reward-driven behaviour were somewhat independent of homeostatic processes.

Using a similar paradigm, the effects of food deprivation on hedonics (liking) and reinforcing value (wanting) of foods was assessed (Epstein et al., 2003). Subjects were assigned to satiated (i.e. fed) or hungry (i.e. unfed) conditions and the subjective pleasantness of a range of drinks as well as the reinforcement value of a preferred snack item were measured before and after a satiating meal or equivalent time abstaining from food. There were no differences in temporal profiles of pleasantness between the two conditions. In contrast, the deprived subjects responded more for food on the progressive ratio task compared to fed subjects. The authors concluded that food deprivation influenced the motivation to eat, but not the hedonics of a range of drinks. These findings are consistent with a dual-component view of reward. Wanting was shown to increase under deprived conditions while liking did not change. In passing, liking was found to decrease for

the most pleasant drink in the fed state. Although non-significant, this finding may reveal certain conditions (e.g. satiated) where liking can decrease independently from wanting.

In our laboratory, some progress has been made in developing a methodology to separately assess and observe dissociation between liking and wanting (Finlayson et al., 2007). We designed a novel computer-based procedure to identify liking and wanting for the same target food stimuli through separate tasks consisting of a number of response trials. These separate procedures prevented cross-contamination in the evaluation of liking and wanting. The presentation of trials for both tasks was integrated and fully randomised into a single executable program. The stimuli were a number of photographic food items selected to vary in fat content and taste (two key dimensions associated with loss of appetite control and overconsumption). In this procedure, liking is measured explicitly using visual analogue scales combined with the prompt "how pleasant would it be to experience a mouthful of this food now?" this question was designed to carefully direct attention to the (imagined) hedonic experience in the mouth at the point of ingestion, rather than a more global rating of a foods pleasantness. In contrast, the wanting task was designed to provide an implicit measure derived from a forced choice task whereby stimuli are presented in pairs and responded to according to which food is most wanted at the present moment. The speed with which one stimulus is chosen rather than its alternative provides a quantifiable measure (reaction time) related to relative wanting for that food item. Reaction time of each decision can convey information about the degree (on a continuous, interval unit of measurement) to which a chosen stimuli is wanted relative to an alternative. Furthermore, mean reaction time for each food category can give an indication of whether motivation is increasing or decreasing independent of the other categories—in essence an implicit measure of wanting. The forced choice paradigm carries with it several advantages as an implicit measure of wanting. The visual nature of the food stimuli means that the dimensions of the categories can be simply adapted to compare motivation for innumerable combinations of food properties or specifically selected to complement a particular experimental intervention. Perhaps most crucially, the forced choice paradigm allows the concurrent measurement of both implicit and explicit elements of food reward by using dissimilar methodology to measure each process. Using the computer task, one recent study has revealed evidence to suggest that the sensory specific satiety phenomenon brought about by consumption of a standardised, uniform test meal may implicate different roles for liking and wanting in human appetite (Finlayson et al., under review). Our findings demonstrated that liking for foods in the task was reduced more for stimuli sharing properties similar to the test meal (e.g. taste), while wanting had increased for stimuli with contrasting properties. If a wanting component to sensory specific satiety exists, it may be activated or enhanced for foods with contrasting sensory properties. We suggest that the outputs of this computer-based procedure resemble and may correspond to separate processes of liking and wanting.

The studies reviewed above provide different examples of how cross-contamination between explicit measures of liking and wanting can be circumvented. Repeated exposure ratings within a bout of eating (Small et al., 2001) or over a number of days (Zandstra et al., 2000) can track separations in changes of liking or desire for a target food. Alternatively, distinguishing general appetite from appetite for a specific food category (Snoek et al., 2004) may isolate wanting from hunger. Finally, instrumental measures of motivation (e.g. Saelens and Epstein, 1996; Finlayson et al., 2007) for target food stimuli may provide operational definitions for the concept of implicit wanting (incentive salience).

7. Further conceptual development

Parsing reward into separable components provides a parsimonious theoretical framework within which to study ingestive behaviour. However, liking and wanting are not well understood constructs in human appetite. Berridge (1996) advocated a minimal definition of these processes, opting to focus instead on their role and function through expanding empirical study. However, as more investigations start to draw on dual component theories to tackle their research questions, and interpret their findings, there is a more pressing need to expand and develop theoretical conceptualisation of these constructs. Four recent theoretical models that—through the diversity of their approach—may help to enhance a description of liking and wanting are discussed below.

7.1. Homeostatic-hedonic model of hunger

In a discursive paper on the controversy over dietary restriction, Lowe and Levine (2005) distinguish physiological or homeostatic hunger (resulting from nutritional or energy need) from psychological or hedonic hunger (reward-driven eating when not hungry), which operates "beyond the need to counteract physiological signals of energy depletion" (p. 798). Although reward-driven hunger is thought to be an important factor in passive overconsumption and obesity (Blundell, 2002; Blundell and Gillett, 2001); the authors argue that both motivations are intuitively adaptive when viewed in terms of human evolution in a climate of food scarcity. Just as physiological hunger can strongly motivate food seeking behaviour in response to declining energy stores, hunger that promotes eating in the absence of an energy deficit is protective against future energy crises. Therefore, food seeking behaviour can be motivated by the presence or availability of food (especially when energy dense or palatable) as well as by genuine homeostatic need. A further implication is that restriction of wanted foods—even in the absence of an

energy deficit—can cause difficulties in suppressing further food seeking behaviours, similar to a denial of food that is genuinely required. For example, ''200 kcal of unflavoured hot cereal might result in short term satiation whereas 200 kcal of chocolate cake might not satisfy hedonic needs'' (p. 799). As the authors point out, the boundary between these two processes is unclear; the point at which hunger becomes reward-driven rather than necessity-based is subject to a host of inter and intra-individual factors. The model also provides an interesting perspective on the relationship between liking and wanting. The authors note that liking is largely a learned phenomenon, and food preferences that are learned generally retain their motivational properties independent of homeostatic needs. Hence, palatability seems to be maintained by oro-sensory factors of reward rather than the energetic delivery of the ingested nutrients and separate to their perceived energy value. It is argued that food intake behaviour is more ambiguous than other reward-driven behaviours because the need to consume food is innate. Therefore, need and reward based want are likely to interact. However, this assertion can be taken further: feeding behaviour is not alone in being innate (e.g. mating, parent-child attachment), but it is a habitual behaviour that is uniquely vital to continued survival. Therefore, the motivating processes behind feeding are likely to be different to non-essential reward driven behaviours. The authors emphasise the need to differentiate these behaviours (needing/wanting vs. liking/wanting) in the study of reward. Interestingly, this issue was potentially resolved in a recent symposium on non-homeostatic behaviour (Corwin and Hajnal, 2005). The speakers developed a collection of defining principles from which feeding could be included in an operational definition for all non-homeostatic behaviour. They proposed that over- or under-eating in response to a homeostatic drive for energy is normal and will vary within a certain regulatory range (termed 'compensated non-homeostatic eating'). When over- or under-eating becomes repetitive or excessive however, this is termed 'maladaptive non-homeostatic eating'. This distinction may represent a framework in which the reward mechanisms central to all non-homeostatic behaviours can be understood.

7.2. Motivating operations in human appetite

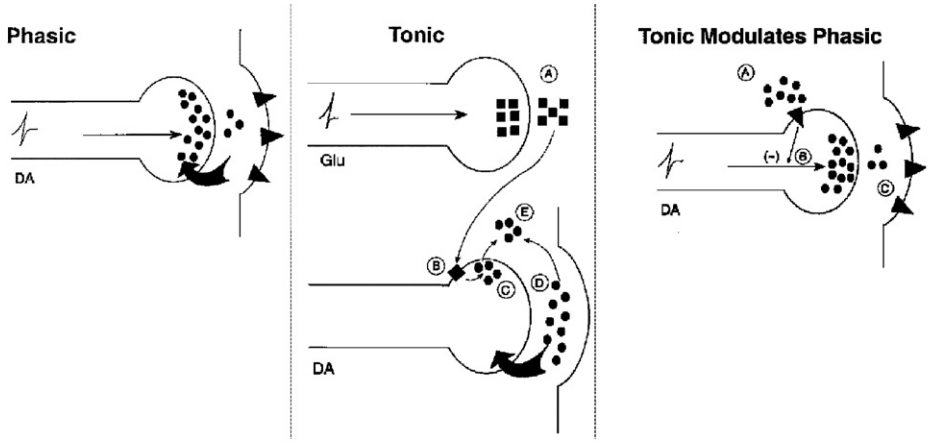
Advances in the field of behaviourism have led to an update in the theoretical principles that can be applied to the study of human appetite. In an interesting review of the literature the concept of 'motivating operations' (MO) was discussed (Tapper, 2005). The paper examines the extent to which MOs can account for different aspects of ingestive behaviour and discusses the conceptual overlap between this and liking/wanting components of reward. An MO is a motivating event, operation or stimulus that temporarily affects an organism on two levels. Firstly there is a value-altering effect: how reinforcing or punishing a relevant stimulus is. Secondly there is a behaviour-altering effect: whether a relevant behaviour is evoked or abated (Michael, 1993). In most circumstances, value-altering and behaviour-altering MOs occur simultaneously and independently. A straight-forward example of an MO is food deprivation. This physical state acts to increase both the reinforcing effectiveness of eating (i.e. enhances the experienced pleasure, Cabanac, 1989) and evokes food seeking behaviour (i.e. increases the motivation to consume). Similarly, a state of excessive-satiety would qualify as an MO; simultaneously reducing the reinforcing effectiveness of eating and the frequency of behaviours that lead to eating. Tapper points out that through behavioural analysis, it is possible to observe the independence of an MO's separate effects. In food deprivation for example, the frequency and intensity of food seeking behaviour increases prior to any physical contact with food. Hence the actual reinforcing value of the food is still unknown and cannot factor into the observed behaviour. Once oral contact is made with the food, these seeking behaviours are sometimes observed to intensify (e.g. the 'appetiser effect'; see Yeomans, 1996). This temporary increase in responding can be seen as the impact of the value-altering MO on ingestive behaviour. The author suggests that the strength of an MO's value-altering effects may not always be proportional to its behaviour-altering effects. Moreover, an MO could have value-altering effects in the absence of behaviour-altering effects. These possibilities are reminiscent of neural dissociations of liking and wanting. For example, dopamine depletion causes available food incentives to be ignored—behaviour-altering effect—but does not reduce the hedonic reaction to food—no value-altering effect (Berridge et al., 1989). Another feature of the MO model is that MOs can influence the reward value of neutral stimuli (termed discriminative stimuli, SD) that have since become associated with the availability of a reinforcer. To adapt the author's example, through repeated association with the availability of food, a photograph of a set meal on a restaurant menu (SD) may elicit some of the behaviours previously linked only to the food itself (e.g. salivation, stomach rumbling). MOs work to influence the strength of SDs, therefore, food deprivation could increase responsivity to the food photograph. From this simple scenario it is possible to envisage a situation where the presence of an MO could raise the power of an SD above that of the availability of the reinforcer alone. Consequently, in a state of energy need, the food photograph may elicit a more intense response than the actual availability of the food and disproportionate to the reinforcing value of consuming the food. In this way, the MO model could provide a methodological platform from which 'irrational wanting' (Wyvell and Berridge, 2000) might be simulated in humans. The MO model may also help to explain some non-homeostatic ingestive behaviour. According to the approach, it is possible for environmental factors to take on motive-like properties through repeated association with an MO. For

example, stimuli paired with hunger (an MO associated with homeostatic feeding) could eventually acquire similar value behaviour altering effects even in the absence of the MO.

7.3. The tonic/phasic model of da system regulation

In a paper written to inform research on craving, Grace (2000) presented a model in which aspects of drug craving can be explained by distinguishing tonic and phasic responses in the dopamine system (see Fig. 2). Phasic DA release refers to the action potential ('spike') dependent release of DA into the synaptic cleft. Phasic DA release is necessary for the behaviourally relevant actions of DA system activation including reward signals (e.g. Schultz, 1997, 1998). The phasic response causes a flood of DA to be released into the synapse which under normal conditions is quickly and efficiently dealt with by re-uptake mechanisms before it can diffuse into extra-cellular space. Tonic DA release on the other hand refers to the escape of very small concentrations of DA into extra-cellular space caused by sustained increases in DA neuron firing or presynaptic stimulation of DA terminals by glutamate. Although too low to stimulate post-synaptic targets, these levels of DA are sufficient to be detected by DA terminal auto receptors that regulate DA release from the terminal. These extra-cellular levels of DA are highly regulated by feedback systems. Through their effect on the DA auto receptors, increases in tonic DA can cause the inhibition of phasic DA release (Grace 1991, 1995).

Administration of potent sources of reward (e.g. psychostimulants and alcohol) differ in their mechanism of DA transmission, but what they have in common is that repeated administration causes the overflow of DA into the synapse which can result in the escape of extra-cellular DA. Since increased levels of tonic DA can diminish phasic release, stimuli that would normally be rewarding will instead produce a blunted reinforcing signal. This imbalance between phasic and tonic systems is detected by the organism which attempts to restore the equilibrium. The tonic/phasic model has interesting implications for the conceptualisation of liking and wanting. Firstly, it is possible to envisage at the molecular level how a 'natural' dissociation between these processes might occur. Chronic overstimulation of the reward commodity may cause a gradual increase of tonic DA. This disequilibrium of tonic and phasic systems could be responded to via psychological drivers such as craving (a potential form of wanting). Consequently, food cues become more salient and food seeking behaviours are initiated. However, due to inhibited phasic response, consumption of the desired food does not produce the expected rewarding effects. In this way, liking and wanting may become separated by subtle yet significant changes in tonic DA regulation.



The tonic/phasic model of DA system regulation. Top: phasic DA is defined as the release of DA into the synaptic cleft as a consequence of action potential discharge in the DA neuron. The action potential causes release of large amounts of DA (circles), resulting in intrasynaptic DA concentrations in the millimolar range where it can stimulate postsynaptic receptors (triangles). The DA is then rapidly removed from the synaptic cleft by the DA transporter (large arrow) before it can escape into the extrasynaptic space. Center: tonic DA is defined as the DA that is present in the extrasynaptic space. Tonic DA is proposed to be derived from two sources. Glutamate released from corticoaccumbens terminals (A; squares) diffuses to the DA terminal, where it can stimulate presynaptic glutamate receptors (B). This causes a release of DA from the synaptic terminal (C). In addition, a small portion of tonic DA is also likely to be derived by overflow from the synaptic cleft as a consequence of sustained activity in the DA terminal (D). The level of extrasynaptic DA (E) derived from these sources is maintained at low nanomolar concentrations by numerous regulatory processes. Bottom: the tonic DA levels in the extrasynaptic space (A) are too low in concentration to stimulate the DA receptors located in the synaptic cleft, but are sufficient to activate DA autoreceptors located on the DA terminal that regulate synthesis and release of transmitter (B). As a consequence, there is an inhibition of phasic, spike-dependent DA release into the synaptic cleft (C).Circles = DA; squares = glutamate/glutamate receptors, triangles = DA receptors.

Fig. 2. The tonic/phasic model of DA system regulation (Grace, 2000).

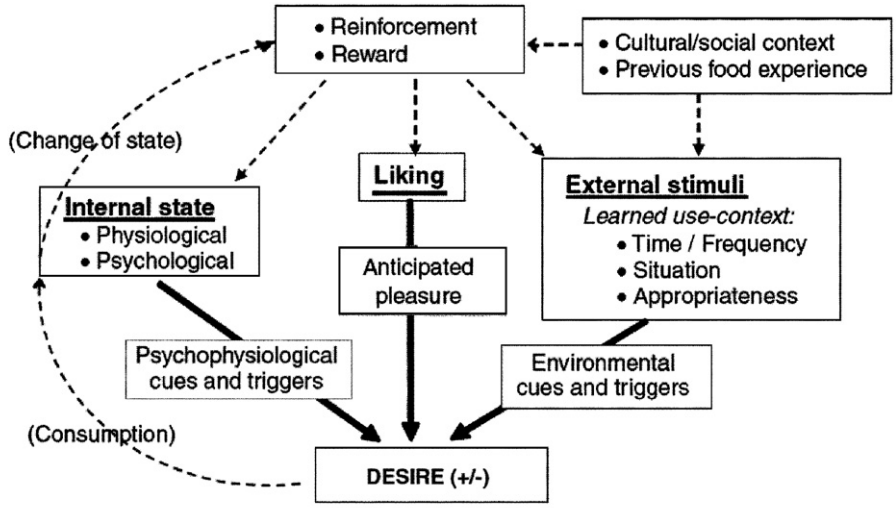
7.4. Determinants of food choice model

Mela (2001, 2006) developed a model to support the assertion that desire for food—a function of innate and learned liking, internal need state, and environmental cues—can be viewed as one of the major determinants of food choice in man. Liking is constructed as the experience or anticipation of pleasure either innately present or acquired through associative conditioning. In this way, liking is seen as an essential component contributing to desire. Desire is also influenced by environmental cues such as situation and appropriateness which may increase or decrease motivation to eat—possibly through a change in anticipated liking. In this way, it is suggested that external stimuli can become integrated into a system of cues that trigger motivation for specific foods under certain conditions. The third factor thought to influence desire is internal need state. This factor refers to psychological and/or physiological needs such as hunger, thirst and specific food cravings which may also reinforce the development of liking. The author uses this model to support the notion that food liking (as an isolated factor) is not a crucial process in weight gain and obesity, as liking is often a rather stable characteristic within an individual and less influenced by weight status (e.g. Cox et al., 1999; Snoek et al., 2004). The implication is that food intake is driven in part by the stimulation or suppression of desire that incorporates, yet remains distinguishable from liking. The presence of environmental cues, strength of internal need state and degree of liking, may all modulate the strength of desire which in turn determines what (and possibly how much) is consumed next (see Fig. 3).

The four models discussed here provide very different perspectives on processes of liking and wanting in reward. In the homeostatic–hedonic model, wanting is viewed as an adaptive response to the presence of rewarding food that is closely related to physical hunger stemming from nutritional need. The motivating operations account distinguishes value-altering effects from behaviour-altering effects to account for the variable impact of reward cues on food seeking behaviour and the possible dissociation of explicit and implicit wanting. The tonic/phasic model describes a possible mechanism by which wanting can be enhanced yet subsequent reward diminished through repeated stimulation of the reward commodity. Lastly, the determinants of food choice model conceptualises liking as a component of wanting, the strength of which can determine preference. All four models are compatible with reward driven (non-homeostatic) consumption, and permit a degree of independence between processes of liking and wanting. Such theoretical accounts may help to inform our understanding of how separate components of reward should be conceptualised.

8. Implication for weight gain and obesity

Thanks in part to a better understanding of the interaction of homeostatic and hedonic processes of appetite and the phenomenon of non-homeostatic consumption, reward is growing to be viewed as an significant risk factor in weight gain leading to obesity (Nasser, 2001; Yeomans et al., 2004a, b; Blundell and Finlayson, 2004; Erlanson-Albertsson, 2005). But what is the evidence that reward may play a role in the aetiology of obesity? Some studies have implicated individual variability in sensitivity to reward (STR)—a psychobiological trait linked to the mesolimbic dopamine pathway—in the development of obesity. Davis et al. (2004) used the Physical Anhedonia



Simplified operational schematic combining the influences of liking (pleasure), internal state (psychophysiology), and external stimuli (learned cues) in the acquisition and activation of desire for foods in everyday situations. Solid lines reflect proximate drivers, dashed lines are underlying processes. Modified from Mela (2001)

Fig. 3. The determinants of food choice model (Mela, 2006).

scale (Chapman et al., 1976) to measure the capacity to experience reward in normal, overweight and obese patients. The authors found an inverted U relationship, with overweight subjects scoring higher (more anhedonic) than obese and normal weight. Similarly, Franken and Muris (2005) demonstrated that STR (Torrubia et al., 2001) is associated with food cravings and BMI. More recently, the STR trait has been shown to correlate strongly with activation in relevant regions of the brain in response to appetising foods relative to bland, aversive and non-food stimuli (Beaver et al., 2006). Taken together, these findings suggest that a high STR may characterise those individuals who are at risk of weight gain due to exaggerated responding to rewarding food cues. Interestingly, because Davis et al. found no differences between normal weight and patients who had become obese, STR may represent a risk factor for weight gain (leading to obesity) without necessarily characterising an obese person. Therefore, STR could be a factor in enhancing susceptibility to weight gain, rather than a definitive trait of obese people. There is evidence to support this proposition. For example, PET scans of normal-weight healthy subjects eating a favourite meal revealed an increase in dopamine release that correlated with the degree of experienced pleasure. Another study demonstrated that the availability of the dopamine D2 receptor was decreased in very obese subjects in proportion to their BMI (Wang et al., 2001). These studies map onto the idea that there is an optimal level or inverted U relationship between the capacity to experience reward and dopamine activation (Volkow et al., 1999). Subjects administered the dopamine agonist methylphenidate found it either pleasant or unpleasant depending on their DRD2 receptor levels. Those subjects reporting pleasant effects had significantly lower levels of the dopamine receptor than those who found it unpleasant. It can therefore be construed that potent stimuli (including palatable foods) elicit a dampened positive hedonic response through the stimulation of dopamine activity in people of an anhedonic (low STR) pre-disposition. One way to overcome this might simply be to consume more of the rewarding commodity. However, there is no evidence to date that tests the notion that particularly hedonic individuals respond aversively to highly palatable foods. It has been suggested that dopamine activity relating to excessive food consumption might only involve activation of brain reward circuitry within normal limits, with other psychological factors exerting stronger effects (Robinson and Berridge, 1998).

Recent investigation of behavioural phenotypes characterised by habitual diet suggests that the hedonic response to palatable food can influence appetite control via effects on both food choice and energy intake. Groups of overweight and lean young males matched for age and the habitual high consumption of fat (high-fat phenotypes) were compared (Blundell et al., 2005; LeNoury et al., 2002). Although both groups were eating a diet which theoretically favours a positive energy balance, the overweight phenotypes consumed greater amounts of the high-fat foods in a test meal and reported greater feelings of pleasantness, satisfaction, and tastiness for the foods consumed. One interpretation of these data is that, for at least this group of overweight people (susceptible to weight gain), they habitually self-select (high-fat/palatable) foods with a high probability of generating a positive energy balance (on the basis of their energy density), consume these foods in greater amounts, and derive greater pleasure from eating these foods. This outcome also demonstrates that certain high-fat phenotypes—susceptible to weight gain—have a disposition to perceive foods as being more pleasant than their lean counterparts (who consume the same habitual high-fat diet). Interestingly, Salbe et al. (2004) found a heightened hedonic response to sweet and creamy solutions to be associated with subsequent weight gain in a sample of Pima Indians (a population highly prone to obesity). Furthermore, Drewnowski and Schwartz (1990) found higher liking for dietary fat in obese individuals compared to lean. Given this capacity to obtain a high level of pleasure from foods (and eating), it is not surprising that many obese people show a tendency to self-select high-palatability foods.

There is also evidence to suggest that obese people may differ in their motivation to eat. As mentioned previously, Saelens and Epstein (1996) found obese subjects invest more effort for liked foods on a progressive ratio task, with similar findings when a normal weight sample of subjects were food-deprived compared to fed (Epstein et al., 2003). Nasser et al. (2005) have also reported the propensity to binge eat to be associated with persistence of motivation to eat in a post-fed state. Furthermore, some neuroimaging studies have indicated greater neural activation at specific reward sites in the brain of weight gaining subjects after consumption of a meal compared to lean controls (Gautier et al., 2000, 2001). These findings were interpreted as weaker post-meal satiety signals in overweight subjects. Lastly, Tetley et al. (2006) have reported greater reactivity in overweight subjects (measured by self-estimated prospective consumption) when exposed to the sight and smell of a palatable food compared to lean controls. Cue reactivity was correlated to habitual portion size and TFEQ disinhibition. Taken together these studies provide some evidence to indicate that susceptible individuals may be characterised by a diminished ability to resist the foods they want, possibly due to greater responsiveness and motivation toward cues associated with tasty foods.

Indeed, individual differences in reward may lead to overconsumption relative to homeostasis via a number of routes. In terms of processes of liking, it is possible that some susceptible individuals may experience an exaggerated hedonic response to palatable foods, in that foods are enjoyed more and therefore eaten in greater amounts for longer periods of time. In addition to this, some people may have a diminished ability to experience pleasure from food and therefore greater consumption of palatable food

is promoted to satisfy an optimum level of stimulation. Processes of wanting may also leave some individuals vulnerable to weight gain through increased motivation (or reactivity) towards cues signalling the availability of food. Further to this, a reduced capacity to resist the motivation to eat when replete may also promote non-homeostatic eating. Finally, some individuals may simply habitually choose highly-palatable, energy-dense foods that promote overconsumption and lead to a positive energy balance. This latter scenario can be viewed as (unhealthy/weight gain promoting) preference and is a behavioural outcome likely to contain elements of both liking and wanting.

9. Conclusion

Prompted by findings on the neural structure of food reward in the brain, it is possible to take a fresh look at the role of reward in human appetite and weight regulation. Research shows that hedonic processes interact with the homeostatic system of energy regulation, and that this can influence the organisation of ingestive behaviour, but less is understood about how liking and wanting components of reward might work together or separately to modulate appetite. An important consideration is how these concepts can be operationalised for use in human appetite research. Most previous research has approached these processes on an explicit (subjective) level, although there have been some more recent attempts to explore implicit processes through behavioural measures and brain imaging techniques. There is some interesting overlap between a number of recent theoretical models and the dual process modulation of food reward, and these may help to flesh out a framework from which these processes can be better understood. Liking and wanting may have independent roles in characterising susceptibility to weight gain and obesity. Further research into the dissociability of these processes would help to assess the relative importance of these conceptual components of reward in appetite control and weight regulation.

10. Postscript

It should not be forgotten that—strictly speaking—liking and wanting should be seen to have the logical status of theoretical constructs. Our preferred view is that liking and wanting should be viewed as intervening variables that help us to understand the role of hedonics in appetite control. Their existence should not be taken to mean that these processes are structurally embodied in a neural substrate. Rather, that different neurochemical pathways can separately influence the events that can be measured objectively (in animals and humans) and which imply the existence of processes here referred to as liking and wanting. It is in this sense that we have used the terms in this review.

Acknowledgement

This research was supported by Medical Research Council (MRC) Case award G78/8223 in conjunction with NRC, Lausanne.

References

Beagley, W.K., Holley, T.L., 1977. Hypothalamic stimulation facilitates contralateral visual control of a learned response. Science 196, 321–323.

Beaver, J.D., Lawrence, A.D., van Ditzhuijzen, J., Davis, M.H., Woods, A., Calder, A.J., 2006. Individual differences in reward drive predict neural responses to images of food. Journal of Neuroscience 26, 5160–5166.

Bellisle, F., Le Magnen, J., 1980. The analysis of human feeding patterns: the edogram. Appetite 1, 141–150.

Bellisle, F., Lucas, F., Amrani, R., Le Magnen, J., 1984. Deprivation, palatability and the micro-structure of meals in human subjects. Appetite 5, 85–94.

Berridge, K.C., 1996. Food reward: brain substrates of wanting and liking. Neuroscience and Biobehavioural Reviews 20, 1–25.

Berridge, K.C., 2000. Measuring hedonic impact in animals and infants: microstructure of affective taste reactivity patterns. Neuroscience and Biobehavioural Reviews 24, 173–198.

Berridge, K.C., 2004. Pleasure, unconscious affect and irrational desire. In: Manstead, A.S.R., Frijda, N.H., Fischer, A.H. (Eds.), Feelings and Emotions: The Amsterdam Symposium. Cambridge University Press, Cambridge, pp. 43–62.

Berridge, K.C., Robinson, T.E., 2003. Parsing reward. Trends in Neuroscience 26, 507–513.

Berridge, K.C., Valenstein, E.S., 1991. What psychological process mediates feeding evoked by electrical stimulation of the lateral hypothalamus? Behavioural Neuroscience 105, 3–14.

Berridge, K.C., Venier, I.L., Robinson, T.E., 1989. Taste reactivity analysis of 6-hydroxydopamine-induced aphagia: implications for arousal and anhedonia hypothesis of dopamine function. Behavioural Neuroscience 103, 36–45.

Berthoud, H.R., 2004. Neural control of appetite: cross-talk between homeostatic and non-homeostatic systems. Appetite 43, 315–317.

Berthoud, H.R., 2006. Homeostatic and non-homeostatic pathways involved in the control of food intake and energy balance. Obesity 14, S197–S200.

Blundell, J.E., 2002. A psychobiological system approach to appetite and weight control. In: Brownell, K.D. (Ed.), Eating Disorders and Obesity. Guildford, New York, p. 49.

Blundell, J.E., Finlayson, G.S., 2004. Is susceptibility to weight gain characterised by homeostatic or hedonic risk factors for overconsumption? Physiology and Behaviour 82, 21–25.

Blundell, J.E., Gillett, A., 2001. Control of food intake in the obese. Obesity Research 9, S263–S270.

Blundell, J.E., Hill, A.J., 1987. Nutrition, serotonin and appetite: case study in the evolution of a scientific idea. Appettie 8, 183–194.

Blundell, J.E., Stubbs, R.J., Golding, C., Croden, F., Alam, R., Whybrow, S., LeNoury, J., Lawton, C.L., 2005. Resistance and susceptibility to weight gain: individual variability in response to a high-fat diet. Physiology and Behaviour 86, 614–622.

Bobroff, E.M., Kissileff, H.R., 1986. Effects of changes in palatability on food intake and the cumulative food intake curve in man. Appetite 7, 85–96.

Booth, D.A., 1987a. Cognitive experimental psychology of appetite. In: Boakes, R.A., Burton, M.J., Popplewell, D.A. (Eds.), Eating habits. Wiley, Chichester, pp. 175–209.

Booth, D.A., 1987b. Objective measurement of determinants of food acceptance: sensory, physiological and psychosocial. In: Solms, J.,

Booth, D.A., Pangbom, R.M., Raunhardt, O. (Eds.), Food acceptance and nutrition. Academic Press, London, pp. 1–27.
Booth, D.A., 1990. How not to think about immediate dietary and postingestional influences on appetites and satieties. Appetite 14, 171–179.
Booth, D.A., Blair, A.J., 1988. Objective factors in the appeal of a brand during use by the individual consumer. In: Thomson, D.M.H. (Ed.), Food acceptability. Elsevier Applied Science, London, pp. 329–346.
Cabanac, M., 1979. Sensory pleasure. Quarterly Review of Biology 54, 1–29.
Cabanac, M., 1989. Maximization of pleasure, the answer to a conflict of motivations. Comptes Rendus de l Academics des Sciences Serie III 309, 397–402.
Carr, K.D., Wolinsky, T.D., 1993. Chronic food restriction and weight loss produce opioid facilitation of perifornical hypothalamic self-stimulation. Brain Research 607, 141–148.
Chapman, L.J., Chapman, J.P., Raulin, M.L., 1976. Scales for physical and social anhedonia. Journal of Abnormal Psychology 85, 374–382.
Corwin, R.L., Hajnal, A., 2005. Too much of a good thing: neurobiology of non-homeostatic eating and drug abuse. Physiology and Behaviour 86, 5–8.
Cota, D., Marsicano, G., Tschop, M., Grubler, Y., Flachskamm, C., Schubert, M., Auer, D., Yassouridis, A., Thone-Reineke, C., Ortmann, S., Tomassoni, F., Cervino, C., Nisoli, E., Linthorst, A.C., Pasquali, R., Lutz, B., Stalla, G.K., Pagotto, U., 2003. The endogenous cannabinoid system affects energy balance via central orexigenic drive and peripheral lipogenesis. Journal of Clinical Investigation 112, 423–431.
Cox, D.N., Perry, L., Moore, P.B., Vallis, L., Mela, D.J., 1999. Sensory and hedonic associations with macronutrient and energy intakes of lean and obese consumers. International Journal of Obesity 23, 403–410.
Davis, C., Strachan, S., Berkson, M., 2004. Sensitivity to reward: implications for overeating and overweight. Appetite 42, 131–138.
De Castro, J.M., Bellisle, F., Dalix, A.-M., 2000a. Palatability and intake relationships in free-living humans: characterization and independence of influence in the French. Physiology and Behaviour 68, 271–277.
De Castro, J.M., Bellisle, F., Dalix, A.-M., Pearcey, S.M., 2000b. Palatability and intake relationships in free-living humans: characterization and independence of influence in North Americans. Physiology and Behaviour 70, 343–350.
De Graaf, C., De Jong, L.S., Lambers, A.C., 1999. Platability affects satiation but not satiety. Physiology and Behaviour 66, 681–688.
Di Marzo, V., Goparaju, S.K., Wang, L., Liu, J., Batkai, S., Jarai, Z., Fezza, F., Miura, G.I., Palmiter, R.D., Sugiura, T., Kunos, G., 2001. Leptin-regulated endocannabinoids are involved in maintaining food intake. Nature 410, 822–825.
Drewnowski, A., 1998. Energy density, palatability, and satiety: implications for weight control. Nutrition Reviews 56, 347–353.
Drewnowski, A., Schwartz, M., 1990. Invisible fats: sensory assessment of sugar/fat mixtures. Appetite 14, 203–217.
Epstein, L.H., Truesdale, R., Wojcik, A., Paluch, R.A., Raynor, H.A., 2003. Effects of deprivation on hedonics and reinforcing value of food. Physiology and Behaviour 78, 221–227.
Erlanson-Albertsson, C., 2005. How palatable food disrupts appetite regulation. Basic Clinical Pharmacology and Toxicology 97, 61–73.
Finlayson, G., King, N., Blundell, J., 2007. Is it possible to dissociate 'liking' and 'wanting' for foods in humans: a novel experimental procedure. Physiology and Behavior 90, 36–42.
Finlayson, G., King, N., Blundell, J., under review. The role of implicit wanting in relation to explicit liking and wanting for food: implications for appetite control. Appetite, under review.
Flier, J.S., 2004. Obesity wars: molecular progress confronts and expanding epidemic. Cell 116, 337–350.
Franken, I.H., Muris, P., 2005. Individual differences in reward sensitivity are related to food craving and relative body weight in healthy women. Appetite 45, 198–201.
Gautier, J.F., DelParigi, A., Chen, K., et al., 2000. Differential brain responses to satiation in obese and lean men. Obesity Research 9, 676–684.
Gautier, J.F., DelParigi, A., Salbe, A.D., et al., 2001. Effect of satiation on brain activity in obese and lean women. Diabetes 49, 838–846.
Giraudo, S.Q., Grace, M.K., Billington, C.J., Levine, A.S., 1999. Differential effects of neuropeptides Y and the mu-agonist DAMGO on 'palatability' vs. 'energy'. Brain Research 10, 160–163.
Grace, A.A., 1991. Phasic versus tonic dopamine release and the modulation of dopamine system responsivity: a hypothesis for the etiology of schizophrenia. Neuroscience 41, 1–24.
Grace, A.A., 1995. The tonic/phasic model of dopamine system regulation: its relevance for understanding how stimulant abuse can alter basal ganglia function. Drug Alcohol Dependence 37, 111–129.
Grace, A.A., 2000. The tonic/phasic model of dopamine system regulation and its implications for understanding alcohol and psychostimulant craving. Addiction 95, S119–S128.
Grill, H.J., Norgren, R., 1978. The taste reactivity test. I. Mimetic responses to gustatory stimuli in neurologically normal rats. Brain Research 143, 263–279.
Guy-Grand, B., Lehnert, V., Doassans, M., Bellisle, F., 1994. Type of test-meal affects palatability and eating style in humans. Appetite 22, 125–134.
Hayward, M.D., Pintar, J.E., Low, M.J., 2002. Selective reward deficit in mice lacking beta-endorphin and enkephalin. Journal of Neuroscience 22, 8251–8258.
Hellemann, U., Tuorila, H., 1991. Pleasantness ratings and consumption of open sandwiches with varying NaCl and acid contents. Appetite 17, 229–238.
Hill, A.J., Magson, L.D., Blundell, J.E., 1984. Hunger and palatability: tracking ratings of subjective experience before, during and after the consumption of preferred and less preferred food. Appetite 5, 361–371.
Ikeda, J., Amy, N.K., Ernsberger, P., Gaesser, G.A., Berg, F.M., Clark, C.A., Parham, E.S., Peters, P., 2005. The National Weight Control Registry: a critique. Journal of Nutrition Education and Behaviour 37, 169.
Johnson, W.G., 1974. Effect of cue prominence and subject weight on human food-directed performance. Journal of Personality and Social Psychology 29, 843–848.
Kirkham, T.C., Williams, G., 2001. Endogenous cannabinoids and appetite. Nutrition Research Review 14, 65–86.
LeNoury, J.C., Lawton, C., Blundell, J.E., 2002. Food choice and hedonic responses: differences between overweight and lean high fat phenotypes. International Journal of Obesity 26, S125.
Levine, A.S., Kotz, C.M., Gosnell, B.A., 2003. Sugars and fats: the neurobiology of preference. Journal of Nutrition 133, S831–S834.
Looy, H., Callaghan, S., Weingarten, H.P., 1992. Hedonic response of sucrose likers and dislikers to other gustatory stimuli. Physiolgy and Behaviour 52, 219–225.
Lowe, M.R., Levine, A.S., 2005. Eating motives and the controversy over dieting: eating less than needed versus less than wanted. Obesity Research 13, 797–806.
Lucas, F., Bellisle, F., 1987. The measurement of food preferences in humans: do taste-and-spit tests predict consumption? Physiology and Behaviour 39, 739–743.
Mela, D.J., 2001. Determinants of food choice: relationships with obesity and weight control. Obesity Research 9, S249–S255.
Mela, D.J., 2006. Eating for pleasure or just wanting to eat? Reconsidering sensory hedonic responses as a driver of obesity. Appetite 47, 10–17.
Michael, J., 1993. Establishing operations. Behaviour Analysis 16, 191–206.
Monneuse, M.O., Bellisle, F., Louis-Sylverstre, J., 1991. Responses to an intense sweetener in humans: immediate preference and delayed effects on intake. Physiology and Behavior 49, 325–330.
Mook, D.G., Votaw, M.C., 1992. How important is hedonism? Reasons given by college students for ending a meal. Appetite 18, 69–75.
Nasser, J., 2001. Taste, food intake and obesity. Obesity Review 2, 213–218.

Nasser, J.A., Geliebter, A., Pi-Sunyer, F.X., 2005. Persistence of food reinforcement after a caloric preload in women is correlated with binge eating score and hunger in the fasted state. Appetite 44, 329.

Pelchat, M.L., 2002. Of human bondage: food craving, obsession, compulsion, and addiction. Physiology and Behaviour 76, 347–352.

Perez, C., Dalix, A.M., Guy-Grand, B., Bellisle, F., 1994. Human responses to five concentrations of sucrose in a dairy product: Immediate and delayed palatability effects. Appetite 23, 165–178.

Peters, J.C., Wyatt, H.R., Donahoo, W.T., Hill, J.O., 2002. From instinct to intellect: the challenge of maintaining healthy weight in the modern world. Obesity Review 3, 69–74.

Robinson, T.E., Berridge, K.C., 2000. The psychology and neurobiology of addiction: an incentive-sensitization view. Addiction 95, S91–S117.

Robinson, T.E., Berridge, K.C., 2003. The neural basis of drug craving: an incentive-sensitization theory of addiction. Brain Research Review 18, 247–291.

Rogers, P.J., Blundell, J.E., 1990. Umami and appetite: effects of monosodium glutamate on hunger and food intake in human subjects. Physiology Behaviour 48, 801–804.

Rogers, P.J., Blundell, J.E., 1991. Mechanisms of diet selection: the translation of needs into behaviour. Proceedings of the Nutrition Society 50, 65–70.

Saelens, B.E., Epstein, L.H., 1996. Reinforcing value of food in obese and non-obese women. Appetite 27, 41–50.

Salbe, A.D., DelParigi, A., Pratley, R.E., Drewnowski, A., Tataranni, P.A., 2004. Taste preferences and body weight changes in an obesity-prone population. The American Journal of Clinical Nutrition 79, 372–378.

Saper, C.B., Chou, C.T., Elemquist, J.K., 2002. The need to feed: homeostaticand hedonic control of eating. Neuron 36, 199–211.

Schultz, W., 1997. Dopamine neurons and their role in reward mechanisms. Current Opinions in Neurobiology 7, 191–197.

Schultz, W., 1998. The phasic reward signal of dopamine neurons. Advances in Pharmacology 42, 686–690.

Schwartz, M.W., Woods, S.C., Porte, D., Seeley, R.J., Baskin, D.G., 2000. Central nervous system control of food intake. Nature 404, 661–671.

Small, D.M., Zatorre, R.J., Dagher, A., Evans, A.C., Jones-Gotman, M., 2001. Changes in brain activity related to eating chocolate: from pleasure to aversion. Brain 124, 1720–1733.

Snoek, H.M., Huntjens, L., Van Gemert, L.J., De Graaf, C., Weenen, H., 2004. Sensory-specific satiety in obese and normal-weight women. The American Journal of Clinical Nutrition 80, 823–831.

Spiegel, T.A., Shrager, E., Stellar, E., 1989. Responses of lean and obese subjects to preloads, deprivation, and palatability. Appetite 13, 45–69.

Stanley, S., Wynne, K., McGowan, B., Bloom, S., 2005. Hormonal regulation of food intake. Physiological Reviews 85, 1131–1158.

Tapper, K., 2005. Motivating operations in appetite research. Appetite 45, 95–107.

Tetley, A.C., Brunstrom, J.M., Griffiths, P., 2006. Individual differences in food-cue reactivity. Appetite 47, 278.

Torrubia, R., Avila, C., Molto, J., Caseras, X., 2001. The sensitivity to punishment and sensitivity to reward questionnaire (SPSRQ) as a measure of Gray's anxiety and impulsivity dimensions. Personality and Individual Differnces 31, 837–862.

Verty, A.N., Geller, F., Dempfle, A., Schauble, N., Friedel, S., Lichtner, P., Fontela-Horro, F., Wudy, S., Hagemann, S., Gortner, L., Huse, K., Remschmidt, H., Bettecken, T., Meitinger, T., Schafer, H., Hebebrand, J., Hinney, A., 2004. Ghrelin receptor gene: identification of several sequence variants in extremely obese children and adolescents, healthy normal-weight and underweight students, and children with short normal stature. The Journal of Clinical Endocrinology and Metabolism 89, 157–162.

Volkow, N.D., Wang, G.J., Fowler, J.S., Logan, J., Gatley, S.J., Gifford, A., et al., 1999. Prediction of reinforcing responses to psychostimulants in humans by brain dopamine D2 receptor levels. American Journal of Psychiatry 156, 1440–1443.

Wang, G.J., Volkow, N.D., Logan, J., Pappas, N.R., Wong, C.T., Zhu, W., et al., 2001. Brain dopamine and obesity. Lancet 357, 354–357.

Warwick, Z.S., Hall, W.G., Pappas, T.N., Schiffman, S.S., 1993. Taste and smell sensations enhance the satiating effect of both a high-carbohydrate and a high-fat meal in humans. Physiology and Behaviour 53, 553–563.

Welch, C.C., Kim, E.M., Grace, M.K., Billington, C.J., Levine, A.S., 1996. Palatability-induced hyperphagia increases hypothalamic dynorphin peptide and mRNA levels. Brain Research 721, 126–131.

Winkielman, P., Berridge, K.C., Wilbarger, J.L., 2005. Unconscious affective reactions to masked happy versus angry faces influence consumption behavior and judgements of value. Personality and Social Psychology Bulletin 31, 121–135.

Wyvell, C.L., Berridge, K.C., 2000. Intra-accumbens amphetamine increases the conditioned incentive salience of sucrose reward: Enhancement of reward 'wanting' without enhanced 'liking' or response reinforcement. Journal of Neuroscience 20, 8122–8130.

Yamamoto, T., Sako, N., Maeda, S., 2000. Effects of taste stimulation on beta-endorphin levels in rat cerebrospinal fluid and plasma. Physiology and Behaviour 69, 345–350.

Yeomans, M.R., 1996. Palatability and the micro-structure of feeding in humans: the appetizer effect. Appetite 27, 119–133.

Yeomans, M.R., 1998. Taste, palatability and the control of appetite. Proceedings of the Nutrition Society 57, 609–615.

Yeomans, M.R., Gray, R.W., 2002. Opioid peptides and the control of human ingestive behaviour. Neuroscience and Biobehavioural Reviews 26, 713–728.

Yeomans, M.R., Symes, T., 1999. Individual differences in the use of pleasantness and palatability ratings. Appetite 32, 383–394.

Yeomans, M.R., Gray, R.W., Mitchell, C.J., True, S., 1997. Independent effects of palatability and within-meal pauses on intake and appetite ratings in human volunteers. Appetite 29, 61–76.

Yeomans, M.R., Lee, M.D., Gray, R.W., French, S.J., 2001. Effects of test-meal palatability on compensatory eating following disguised fat and carbohydrate preloads. International Journal of Obesity and Related Metabolic Disorders 25, 1215–1224.

Yeomans, M.R., Blundell, J.E., Leshem, M., 2004a. Palatability: response to nutritional need or need-free stimulation of appetite? British Journal of Nutrition 92, S3–S14.

Yeomans, M.R., Tovey, H.M., Tinley, E.M., Haynes, C.J., 2004b. Effects of manipulated palatability on appetite depend on restraint and disinhibition scores from the Three-Factor Eating Questionnaire. International Journal of Obesity and Related Metabolic Disorders 28, 144–151.

Zandstra, E.H., De Graaf, C., van Trijp, H.C.M., van Staveren, W.A., 1999. Laboratory hedonic ratings as predictors of consumption. Food Quality and Preference 10, 411–418.

Zandstra, E.H., De Graaf, C., Mela, D.J., Van Staveren, W.A., 2000. Short- and long-term effects of changes in pleasantness on food intake. Appetite 34, 253–260.

Zandstra, E.H., Weegels, M.F., van Spronsen, A.A., Klerk, M., 2004. Scoring or Boring? Predicting boredom through repeated in-home consumption. Food Quality and Preference 15, 549–557.