Human hunger as a memory process

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Human hunger as a memory process

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Abstract

Hunger refers to: (1) the meaning of certain bodily sensations; (2) a mental state of anticipation that food will be good to eat; and (3) an organising principal, which prioritises feeding. Definition (1) and (2) is the focus here, as (3) can be considered their consequent. Definition (1) has been linked to energy-depletion models of hunger, but these are no longer thought viable. Definition (2) has been linked to learning and memory (L&M) models of hunger, but these apply just to palatable foods. Nonetheless, L&M probably forms the basis for hunger generally, as damage to declarative memory can eradicate the experience of hunger. Currently, there is no general L&M model of hunger, little understanding of how physiology intersects with a L&M approach, and no understanding of how definition (1) and (2) are related. We present a new L&M model of human hunger. People learn associations between internal (e.g., tummy-rumbles) and external cues (e.g., brand-names) and food. These associations can be to specific foods (episodic memories) or food-related categories (semantic memories). When a cue is encountered, it may lead to food-related memory retrieval. If retrieval occurs, the memory’s affective content allows one to know if food will be good to eat now - hunger - a cognitive operation learned in childhood. These memory processes are acutely inhibited during satiety, and chronically by multiple biological parameters, allowing physiology to modulate hunger. Implications are considered for the process of making hunger judgments, thirst, the cephalic phase response, and motivational and lay theories of hunger.

Keywords: Hunger, Declarative memory, Associative learning, Appetite, Ingestive behavior
Human hunger as a memory process

In humans, the term hunger has been defined as: (1) the meaning of certain bodily sensations such as a rumbling stomach; (2) a mental state of positive anticipation that food will be good to eat; and (3) an organising principal, which serves to prioritise feeding (e.g., Cofer & Appley, 1964; Reber, 1985; see Table 1). These usages are partially hierarchical, because (3) is typically regarded as a potential consequence of experiencing (1) and/or (2), but the relationship between (1) and (2) is less clear. It is (1) and (2) that are of central interest here.

Over the last two centuries there have been many theories of hunger and much empirical work. On this basis, scientific understanding of human hunger should be well advanced. The reality is more complex. Currently, there is some understanding of which historical theories are incorrect, notably homeostatic models of hunger, however these remain highly influential (Assanand, Pinel & Lehman, 1998). There is also some understanding of theories that are probably correct - learning and memory models - but their current application is limited in scope. It is also apparent that there are data that do not square well with existing accounts, as well as some significant omissions in understanding. These observations prompted the current endeavour, namely to develop a new general model of hunger that addresses these outstanding issues, one based upon learning and memory.

The human body requires an adequate supply of energy from food to maintain healthy functioning. It also requires certain specific fats (e.g., alpha linolenic acid), several essential amino acids, and a large number of different micronutrients (i.e., vitamins and minerals), all of which cannot be synthesised by the body. Because of these multiple requirements, it has been suggested that there may be several forms of hunger, where the person or animal deliberately seeks out food containing the specific depleted nutrient (Richter et al., 1938). However, the notion of multiple specific hungers, namely one for each bodily need, is
generally not well supported (Galef, 1991), with the exception of salt (e.g., Hurley & Johnson, 2015) and possibly the essential amino acids (Morrison, Reed & Henagan, 2012). Setting aside specific hun\m{gers, most interest – historical, current, and here - has focussed on the bodies need for energy. Indeed, it has been noted (Beaulieu & Blundell, 2021; Weingarten, 1985) that the term hunger has often been equated with a state of energy depletion. Before turning to the account of hunger presented in this manuscript, it is instructive to see what theories have come before. Not only do most contain the basis for the current proposal (i.e., learning and memory), but they also reveal the progressive shift in thinking away from the energy depletion models, which dominated much of the early work on hunger (Toates, 1986).

**Table 1:** Definitions of hunger

<table>
<thead>
<tr>
<th>Term (Source)</th>
<th>Definition(s)</th>
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| Hunger (Cofer & Appley, 1964; Reber, 1985) | (1) The meaning of certain bodily sensations (e.g., rumbling stomach)  
(2) A mental state of positive anticipation that food will be good to eat  
(3) An organising principal, which serves to prioritise feeding |
| *Hunger* (Cannon & Washburn, 1912) | A bodily state characterised by periodic contractions of the oesophagus, stomach and small intestine, reflecting an empty digestive system, which could be satisfied by any form of food |
| ‘Hunger’ (Cannon & Washburn, 1912) | Phenomenally the same as *Hunger*, but reflecting bodily energy needs (i.e., depletion of energy triggering ‘hunger’ – a needs-based homeostatic model) |
| ‘Appetite’ (Cannon & Washburn, 1912) | Attributed to Pavlov, whereby cues become associated with palatable food |
Theories of hunger

The scientific literature on hunger is extensive and at least 200 years old. In a seminal paper Cannon and Washburn (1912) identified three potential forms of hunger (see Table 1), which map on to the first (i.e., the meaning of certain bodily sensations) and second (i.e., anticipation that food will be good to eat) definition presented in the Introduction. They termed one hunger (in italics to reflect their meaning), which was differentiated from a second form ‘hunger’ (apostrophes to indicate their second meaning), with both of these differing from a third form ‘appetite’ (apostrophes to indicate their special meaning). Hunger was an interoceptive state characterised by periodic contractions of the oesophagus, stomach and small intestine, reflecting an empty digestive system, which could be satisfied by any form of food. Cannon and Washburn (1912) were at pains to distinguish hunger from ‘hunger’ which while phenomenally the same, critically reflected bodily energy needs (i.e., depletion of food substances in the blood triggering ‘hunger’ – a needs based homeostatic model). Cannon and Washburn (1912) poured scorn on this type of homeostatic ‘hunger’ concluding it was an absurd idea as hunger was both intermittent under normal conditions and could wane during prolonged fasts (op.cit. p442). In contrast to hunger and ‘hunger’, ‘appetite’ was attributed to Pavlov, it was learned, and occurred primarily for palatable foods. ‘Appetite’ was considered of little physiological significance and so did not feature beyond the second paragraph of their paper.

Mayer (1953), in another landmark paper, focussed on just two hungers - homeostatic ‘hunger’ and ‘appetite’ - with ‘appetite’ again getting just a passing mention. The historical importance of this paper is that it provides the first specific account of how the body might assess and regulate short-term energy needs. According to Mayer (1953), departures from energy homeostasis manifested as changes in blood glucose levels - glucostatic theory - these being detected by the brain and forming a physiological basis for experiencing ‘hunger’.
This theory has been adapted in various ways and has remained influential (Melanson et al., 1999; Campfield et al., 1996). A further homeostatic account was lipostatic theory (Kennedy, 1953), where variation in blood levels of fatty acids govern ‘hunger’. Other homeostatic models of ‘hunger’ have also emerged, based on body temperature (Brobeck, 1948/1997) and other markers of energy-use or fuel status (e.g., Friedman & Stricker, 1976; Mellinkoff et al., 1955). Common to all of these models is some physiological indicator of energy depletion, which when it departs from normality (set point) yields an internal bodily state that is by some means known as ‘hunger’. This in turn leads to an increased likelihood of food seeking behavior and ultimately ingestion (Kissileff & Van Itallie, 1982).

From the 1960s onwards there was a marked shift away from interest in ‘hunger’ and towards ‘appetite’. Schachter (1968) emphasised the importance of external food cues (e.g., seeing or smelling food – ‘appetite’) to people with obesity, with the premise that they could not properly perceive or utilise the interoceptive states that characterised homeostatic ‘hunger’ (e.g., reports of ‘hunger’ were reportedly not linked to stomach contractions in people with obesity). While Schachter’s (1968) focus on ‘appetite’ was used as the basis to explain excess weight gain, the definitive theoretical shift away from ‘hunger’ was made by Weingarten (1985). Here, the importance of internal indices of energy state as the basis for ‘hunger’ were downgraded, such that they only became operative in extremis. In normal day-to-day existence learnt external cues to food were considered the primary determinant of hunger (i.e., ‘appetite’; Weingarten, 1984).

This emphasis on ‘appetite’ was also adopted by Woods et al., (2000) but now with a special focus on feeding time as a combination of biological propensity (i.e., circadian rhythm dictating feeding in the day or night) and temporal cue (e.g., routine feeding time). According to this model, feeding times are learned, either being imposed externally by the experimenter in the case of rats or by society/caregivers in the case of humans (e.g., van den
Akker, Havermans & Jansen, 2017). Whether feeding times constitute an internal or external cue becomes blurred in this account, as people and rodents have access to multiple external (e.g., clocks, lights etc) and internal cues, which signal the passage of time (e.g., an empty stomach is one internal cue that time has elapsed since the last meal). A further feature of Wood’s account is the way it addresses another problem. Organisms need to prepare for the arrival of food, as the unsignalled influx of nutrients can cause major physiological disruption (Woods, 1991). This preparatory process was first studied by Pavlov, and involves using cues that signal the imminent arrival of food as a means to prepare the body to receive nutrients – the cephalic phase response (Power & Schulkin, 2008). Thus, cues to food - internal or external to the organism - can both initiate hunger and prepare the body to receive food.

Much of the work described in the previous paragraph centred on animal models. However, the ‘appetite’ concept has also emerged as important in the human literature, but generally without any explicit connection being made to the earlier historical work described above. Three influential contemporary human ‘appetite’ models warrant mention. Lowe and Butryn, (2007) developed the notion of hedonic hunger. This has an environmental cause, is cue dependent, just involves palatable foods, is not ‘needs’ based (i.e., non-homeostatic) and is learned. In this account, hedonic hunger is explicitly contrasted with a second form of hunger, namely homeostatic or ‘energy deficient’ ‘hunger’. In more recent expositions (Espel-Huynh, Muratore & Lowe, 2018), hedonic hunger has been linked to a highly influential motivational model, incentive salience theory (Berridge, 2009). Hedonic hunger can be fairly considered synonymous with Cannon and Washburn’s (1912) ‘appetite’.

A second human ‘appetite’ model is the elaborated intrusion theory of desire (Kavanagh, Andrade & May, 2005), which was originally developed to account for drug cravings. As applied to food (May, Andrade, Kavanagh & Hetherington, 2012), this model
identifies two processes a “sophisticated homeostatic physiological system to signal hunger…” (op.cit., opening line) – ‘hunger’ and a second process, which can be triggered by learned cues to palatable foods. Encountering such learned cues can create intrusive thoughts, which then become elaborated by working memory systems, generating affective mental imagery of consumption, which constitutes craving (May, Kavanagh & Andrade, 2015). This again seems closely allied to the ‘appetite’ of Cannon and Washburn (1912).

A third ‘appetite’ model has been advanced by Papies, Barsalou and Rusz (2020), which also envisages ‘appetite’ as being distinct from ‘hunger’ (see p194 op.cit.; and see Devos et al., 2022; Papies et al., 2021, 2022). In this account, ‘appetite’ emerges from the way that cues associated with a palatable food act to reinstate similar mental operations (i.e., memories), to those present when that food was previously consumed. These mental simulations encompass both unconscious and conscious elements, and like the elaborated intrusion model, allow for vivid sensory affective experience - imagining eating - which constitutes desire/craving. Yet again, the core notions expressed in this theory echo Cannon and Washburn’s (1912) ‘appetite’.

**Hunger, physiology, and energy depletion**

There are many reasons to question the basic assumptions of homeostatic energy depletion models of hunger. However, it would be ludicrous to suggest that bodily physiology has no influence on hunger. Indeed, a range of short-acting (e.g., gut fullness signals; Wood et al., 2018; Blundell & Burley, 1987) and longer-acting physiological parameters connected to metabolic rate (e.g., Hopkins et al., 2016), body composition (especially fat free mass; e.g., Hopkins et al., 2019), and energy expenditure (e.g., Mayer, Roy & Mitra, 1956), are all well-established correlates of how much people eat (e.g., Blundell et al., 2020). To the extent that hunger influences food intake, it seems likely that such physiological parameters will also influence hunger.
An important point to consider is the difference between physiological influences on hunger versus homeostatic models of hunger. Physiological parameters such as for example gut hormones, fat-free mass, fat mass or basal metabolic rate, are not presumed here to *regulate* hunger because they do not operate around a set-point or norm. Rather, they just exert excitatory or more often inhibitory influences, which (broadly) wax or wane in concert with the magnitude of that physiological parameter (Beaulieu & Blundell, 2021; Blundell et al., 2020).

One of the main reasons that the scientific literature has largely abandoned homeostatic energy depletion models of hunger is that their foundational premise is unlikely to be correct. Human bodies do not rapidly run out of fuel. This is because the human body is able to maintain a healthy functional fuel status for many hours, days or even weeks without food (e.g., Stricker, 1984; Strubbe & Woods, 2004; Woods et al., 2000). In particular, and over timespans of several hours, as typical for well-fed humans, there may be minimal change in fuel availability and thus no indicators of changing fuel status to form the basis for ‘hunger’.

Several other pieces of evidence are also problematic for an energy-need account. First, breakfast might be expected to be the largest meal of the day on the basis that it follows the longest interval without food, which should result in the greatest degree of energy depletion. Yet, it is usually the smallest (Kant, 2018). Second, people who receive total parenteral nutrition at home (i.e., liquid nutrition via an indwelling venous catheter) typically report no change in hunger across the infusion period, even though it meets all of their energy requirements (e.g., McCutcheon & Tennissen, 1989; Murray et al., 2006). Indeed, many people who receive total parenteral nutrition also experience frequent bouts of hunger. In some cases, these can be so intense as to be distressing, and result in eating even when this may cause pain and vomiting (e.g., Stratton & Elia, 1999). Third, animal data indicate that
when rats come to expect food at a certain time, and then that feeding time is missed with food presented at a later unexpected time, food intake decreases (e.g., Bousfield & Elliot, 1934). A depletion model would predict the reverse - something that Cannon and Washburn (1912) also remarked upon but in their case based on reports of human starvation. Fourth, actual depletion of blood glucose, either during diabetic hypoglycaemia (where neurological symptoms predominate; Deary et al., 1993) or when ‘hitting-the-wall’ during endurance exercise (extreme fatigue), are not reported to feel like ‘hunger’ (Rogers & Brunstrom, 2016). This is particularly troubling if one assumes that reports of hunger are driven by energy depletion of the sort identified in glucostatic theory. Fifth, if hunger did directly reflect energy need, it is hard to see why the experience of hunger can sometimes be brief and intermittent (see Murray & Vickers, 2009). This was also identified by Cannon and Washburn (1912) as a further argument against a homeostatic account of hunger.

Finally, perhaps the strongest reason to doubt energy depletion models is theoretical. It makes more functional sense to have an organism defaulted towards hunger (e.g., Assanand, Pinel & Lehman, 1998; Liu & Kanoski, 2018). After all, the natural environment is capricious and so feeding opportunities ideally need to be taken whenever they arise, assuming of course that the organism is capable of eating (i.e., available gut space – reminiscent of Cannon & Washburn’s hunger). From this perspective organisms should by default tend to eat whenever food is available - gut capacity not withstanding - and especially so if it is energy dense.

A longstanding assumption seems to have been that the hypothetical state of energy depletion in homeostatic ‘hunger’ models, should manifest as some form of bodily sensation (Cannon & Washburn, 1912; Monello & Mayer, 1967; Schacter, 1968), just as water depletion has as its correlate a dry mouth. For energy depletion, such a link to bodily sensation should take place because fuel changes are hypothesised to be occurring in the
body, and hence its phenomenological correlate will also be felt as arising in the body (i.e., as an interoceptive state). Setting aside the critical issue of how this (or any) particular bodily sensation becomes meaningful (Changizi, McGehee & Hall, 2002; Harshaw, 2008; Richter, 1927), the assumption that energy depletion is linked to bodily sensation yields two predictions. First, there should be commonality of the ‘hunger’ interoceptive state between people, if this has a common shared physiological basis. That is energy depletion should manifest and be detected in largely the same manner by everyone and so its phenomenological correlate/s in the body should also be broadly similar – like a dry mouth and thirst (e.g., Brunstrom, Tribbeck & MacRae, 2000). Second, because of this reliance on interoception for the experience of ‘hunger’, damage to the neural substrates of interoceptive processing should be particularly disruptive to the experience of hunger. It turns out that neither of these predictions are currently supported.

First, there is little agreement between individuals when asked to describe what hunger feels like. Monello and Mayer (1967) note that there are “…many and diverse sensations of hunger…” (op.cit., p261). More tellingly, Harris and Wardle’s (1987) study concluded that: “It proved impossible to identify a specific subset or constellation of hunger symptoms which were characteristically experienced by hungry people and no evidence was found for a dimensional structure for hunger” (op.cit., p154). The phenomenal experience of hunger seems then to be rather idiosyncratic.

Second, functional neuroimaging has consistently found insula activity to be associated with all aspects of interoception (e.g., Craig, 2009; Barrett et al., 2015). This has led to the insula being referred to as primary interoceptive cortex (Wilson-Mendenhall et al., 2018). However, insula lesions are linked to only subtle reductions in the ability to experience hunger (Grossi et al., 2014). The only type of brain lesions that seems to consistently impair the perception of hunger are those that affect the medial temporal lobes
(Berriman et al., 2016; Hebben et al., 1985; Higgs et al., 2008; Rozin et al., 1998; Zuniga et al., 2020). This raises the possibility that other cognitive processes, notably learning and memory that are supported by the medial temporal lobes, may be more significant substrates for hunger than the insula. Indeed, there seems to be little empirical or theoretical support for homeostatic depletion models of hunger.

**Memory loss, the medial-temporal lobes and hunger**

A powerful argument for considering a learning and memory-based account of hunger comes from the study of people with medial temporal lobe (MTL) damage. There is substantial evidence that the MTL supports a particular type of memory system, termed declarative memory, which is composed of episodic (self-referential events) and semantic (abstract knowledge) sub-systems (e.g., Bird & Burgess, 2008; Squire & Zola-Morgan, 1991; Tulving & Markowitsch, 1998). Additionally, particular structures within the MTL have been identified with more specific mnemonic functions, such as the hippocampus with spatial memory and the amygdala with aversive conditioning (Li et al., 2011).

A key study was reported by Hebben et al., (1985) on patient HM who had received a bilateral temporal lobectomy for intractable epilepsy, removing much of his medial temporal lobes. The study was motivated by the observation that HM rarely mentioned being hungry. Indeed, as Corkin said of HM “He never sought out food for himself, so his caregivers served him three meals a day” (Corkin, 2013, pp210). The implication of this statement is that if HM had not been in residential care, he would have died from inanition. Hebben et al., (1985) asked HM to rate his hunger prior to and after a series of meals. HM reported no change in hunger across his normal evening meal, with his pre-meal and post-meal ratings both suggesting he felt neither hungry nor full. On one evening, he was served a second complete dinner just 1 min after he had finished his first - he ate both. This second meal did
produce a small shift in ratings towards fullness. Hebben et al., (1985) attributed this impairment in hunger perception to his bilateral amygdala loss.

In a follow-up study focussed on multiple meal eating, Rozin et al., (1998) examined this phenomenon in two amnesic patients (BH & RH) with MTL damage. Not only did they eat multiple meals like HM, they also showed abnormal patterns of hunger ratings. Like HM, both patient BR and RH reported little or no change in hunger across their main meals. Looking at all of BR’s hunger ratings, revealed a variety of responses from ‘no hunger’ to ‘some’, which did not resemble the pattern in controls obtained over the same pre/post-meal time intervals. For RH, their ratings were consistently set at ‘hungry’ irrespective of food intake, a pattern which again did not resemble controls.

Higgs et al., (2008) examined two patients who had bilateral MTL damage, in addition to other brain areas including the insula, resulting from herpes simplex encephalitis. These patients also showed no change in hunger across a test meal, but they had preserved hedonic responsiveness to food while eating, in that they reported normal sensory specific satiety (i.e., liking for just the meal food declined across the meal). Pre-meal hunger ratings were not reported, so it is not possible to tell if these were abnormal. In a further study, Berriman et al., (2015) described two patients, PT with a selective right-sided MTL resection, and JW with selective bilateral MTL damage. Each patient and a close family member were briefly interviewed about their experience of hunger and other states. Both PT and JW reported no sensations of hunger, with JW’s family member noting that she had to regularly text JW to remind her to eat. JW had also lost weight over the preceding months. Finally, Zuniga et al., (2020) studied two patients with bilateral MTL damage. One patient, DW ate very little on an experimental meal, but the other JC who ate a similar amount to controls, reported no change in hunger across this meal, in contrast to controls. JC was also asked
about her desire to eat each of several palatable foods, with these ratings occurring before and after the test meal. These desire ratings did not change either, in contrast to controls.

In sum, these observations suggest a connection between MTL integrity and hunger, one that has also been suggested by neuroimaging findings. In a meta-analysis of the processing of visual food cues, van der Laan et al., (2011) reported that the largest and most significant area activated on exposure to food pictures, when contrasting fasted versus sated participants, were MTL structures, with the only other active area being the inferior frontal gyrus/orbitofrontal cortex. These observations of MTL involvement in hunger are at odds with the view that interoceptive processing is mediated primarily by the insula cortex (Craig, 2009; Barrett et al., 2015; Wilson-Mendenhall et al., 2018). Irrespective of this, the neuropsychological findings suggest that declarative memory processes are particularly important to the experience of hunger.

**Hunger – three problems**

A common theme that runs through the accounts of hunger reviewed earlier, is the presence of two processes. One is ‘hunger’, which is presumed to arise from energy depletion, and where the hypothetical interoceptive correlate (bodily sensation) is dependent upon some physiological index of fuel level (i.e., ‘hunger’ serving as a fuel gauge). Second, a learnt form of hunger, where cues predictive of palatable food generate ‘appetite’. As the case for an energy depletion model of hunger is unconvincing, this leaves ‘appetite’ as the one form of hunger upon which researchers agree. And this is pretty much where the psychological literature ends. To propel things forward three problems need addressing.

The first problem concerns the need for a general model of hunger. ‘Appetite’ offers only a limited model of hunger because it applies just to palatable food and especially highly palatable foods at that (Lowe & Butryn, 2007; May et al., 2012; Papies et al., 2020). Thus, there is a need to explain how hunger arises for the many foods that fall outside this category.
In addition, ‘appetite’ models do not consider how physiology affects hunger. They sidestep the need to do so by claiming - probably correctly - that high palatability overrides most physiological influences (Bilman, van Kleef & van Trijp, 2017). However, any general model would clearly need to encompass physiological influences. In terms then of the nature of a general model of hunger, it seems reasonable to presume a basis in learning and memory. This is partly because of the longevity and success of learning and memory based ‘appetite’ models in animals and humans, but more generally because of the serious effect on the experience of human hunger wrought by MTL damage (Parent et al., 2022; Seitz, Tomiyama & Blaisdell, 2021). Consequently, the first problem we address is what would a general - all food types - learning and memory-based model of hunger look like, and how would this integrate physiological information?

The second problem concerns the relationship between the first (i.e., the meaning of certain bodily sensations) and second (i.e., anticipation that food will be good to eat) definition of hunger presented in the opening sentence of the manuscript (see Table 1). The first definition has been considered as the phenomenological manifestation of an energy-depletion model of ‘hunger’ (Cannon & Washburn, 1912; Monello & Mayer, 1967; Schacter, 1968), while the second definition is strongly connected to the notion of ‘appetite’. However, as needs-based ‘hunger’ seems unlikely to exist (see above) it raises the question as to what these bodily sensations are that people report as hunger, and whether they can be explained within a common learning and memory framework?

The third problem concerns top-down processing (i.e., self/other initiated explicit hunger judgments; e.g., ‘are you hungry?’). There has been a great deal of emphasis here, and in the literature, on the role of internal and external cues to hunger. These constitute a bottom-up initiation of hunger (i.e., the cue initiates thoughts of food), and this is what is dealt with in the first two problems outlined above. It is not surprising that scientists have
had a bottom-up focus given that the modern environment exposes people to so many food cues in so many places and at so many times (e.g., Larson & Story, 2009). Equally, in the animal literature, internal and external cues are an integral part of feeding behavior (e.g., Weingarten, 1984), but top-down processing is not really a consideration. Indeed, people, but not non-human animals, commonly ask their family and friends - ‘are you hungry now?’ and many papers report asking research participants a similar question - ‘how hungry are you now?’ How then do people go about answering these top-down initiated hunger judgments, and how might this relate to a general learning and memory framework of hunger?

Problem 1: A general learning and memory model of hunger and how it might integrate physiological information

The basic aspects of the proposed model are summarised in Figure 1. Hunger is defined in this model as a state of knowing that food will be good (i.e., rewarding) to eat now. For hunger to occur, a person needs to encounter a cue that leads to the retrieval of a food-related memory, which is then briefly experienced. The consequent knowing that food will be rewarding to eat now, is based upon appraising the affective content of the retrieved food-related memory. This appraisal process is premised on understanding the predictive utility of the affect experienced during the food-related memory (i.e., tastes nice in memory is understood to signal will taste nice now in reality). This predictive utility is probably learned during development. If the experienced affect is positive, this will predict that the food will taste good to eat (i.e., rewarding) now - hunger. Hunger then is constituted of both the positive affect from the food-related memory and an awareness of its predictive value.

Whether this state of knowing results in food search and ingestion then depends upon strategic processes (i.e., central executive), which fall outside the scope of this model. These strategic processes involve evaluation of both the immediate and long-term costs and benefits of acting upon this instance of hunger (e.g., effort to obtain food vs. expected reward value;
weight-loss goals vs. expected reward value; expected reward value of one food vs. another etc). This two-stage process – knowing (i.e., hunger) food will be good to eat now, followed by evaluating/planning obtaining it – is probably why there is only a weak-to-moderate positive correlation between reports of hunger and acts of ingestion (e.g., Mattes, 1990; McKiernan, Houchins & Mattes, 2008).

**Figure 1:** A declarative memory model of hunger

A key issue is whether after encountering a cue, this leads to the retrieval of a memory where food is experienced as rewarding to eat (or not). The probability of retrieval, and the nature of what is retrieved, depends upon several factors that are outlined below.

The first is the reward value of the food associated with a particular cue. Reward value is dependent on two correlated properties. One is palatability, which is primarily caused by the stimulation of particular sensory systems when a food is placed in the mouth. These systems detect simple carbohydrates (gustation), protein (gustation), salt (gustation) and fat (mainly texture). Learning can augment this process in several ways, but especially by linking the olfactory component of food with these gustatory and textural sources of pleasure and/or with a food’s post-ingestive consequences. Indeed, highly palatable foods
are usually those with the highest energy density, which constitutes the other – hence correlated – aspect of reward value. Learning is more robust when a cue is linked to a highly rewarding event (e.g., Epstein et al., 2007; or as reflected by the $\beta$ parameter in the Rescorla-Wagner model, Rescorla & Wagner, 1972) and the neural representation of highly rewarding food is especially distinct (Londeree et al., 2021). Consequently, more rewarding foods – those high in sugar, sugar or salt and fat – are likely to be better recalled than less rewarding foods (e.g., many vegetables) due to the strong association between cue and food memory (see Seitz, Blaisdell & Tomiyama, 2021, for a recent demonstration of this).

Many cues for food are present in the environment and these fall into three hypothetical categories. Primary cues are those most directly linked to the identity of a particular food. These include such things as a food’s visual appearance, its name, and its packaging (e.g., seeing a chocolate éclair or reading or hearing its name). This type of cue is most likely to result in retrieval of a specific food memory. The smell of a food may be a primary cue in combination with visual or other identifiers, but smell alone may sometimes be unable to support individual item recognition (e.g., Jonsson & Stevenson, 2014), making it a secondary cue on some occasions (i.e., indicative of a category rather than an item).

Secondary cues do not link to a particular food, but rather to a food category. They can include, for example, such things as time of day (e.g., retrieving breakfast foods vs. lunch or dinner foods), shop signs (e.g., patisserie vs. butchers), particular places (e.g., food courts vs. fancy restaurants) and cuisine-types (e.g., Indian vs. Chinese). Secondary cues are more likely to lead to food memory retrieval than tertiary cues, which are those where the link to food is more distant (e.g., plates, fridges, a chef’s hat, etc) and/or tangential (e.g., seeing an éclair shaped cloud or meeting a woman called Claire).

A third factor, and one closely linked to the issue of cue type, is the nature of the retrieved memory. Primary cues are most likely to retrieve episodic memories, rich in
sensory affective detail and that have an autonoetic quality (Tulving, 1983; i.e., they provide a sense of what it would be like to eat that food even though it is not actually being eaten). Secondary and tertiary cues are more likely to retrieve semantic memories, covering categories of food with a more generic representation of their sensory and affective properties (e.g., Mexican or Indian cuisine; breakfast or lunch foods, etc). The nature of the affective experience may differ for semantic memories. Whilst they should provide knowledge about whether that class of food is generally liked or disliked, they lack both the autonoetic quality and the felt affect, which characterises episodic memories of particular foods. However, it is unusual to have any declarative memory experience that does not have some episodic and semantic qualities (e.g., Conway, 2009; Greenberg & Verfaellie, 2010; Irish & Piguet, 2013), and so it is their relative contribution which differs here (i.e., more semantic vs. more episodic).

A fourth factor, and the one most directly linked to retrieval, reflects the impact of both shorter acting (e.g., multiple physiological signals resulting from recent food intake; e.g., Dockray et al., 2009; Janssen et al., 2011; Woods et al., 2018) and longer acting physiological influences (e.g., physiological signals of bodily energy reserves, energy expenditure, fat-free mass, basal metabolic rate, etc [e.g., Blundell et al., 2020; Hopkins et al., 2016; Hopkins et al., 2019; Mayer, Roy & Mitra, 1956]). These are argued to summate to produce a greater or lesser degrees of inhibition (general inhibition). This summed inhibitory signal may serve to: (1) suppress food-memory retrieval; (2) it may favour retrieval of memories consistent with the current degree of general inhibition (i.e., with general inhibition serving as a retrieval context); and (3) it may suppress the affect linked to a food memory. While (1) and (2) inhibit retrieval, thus no food-related memory is available for a hunger appraisal, (3) would not affect retrieval, rather here inhibition would act upon
retrieved content. The upshot of all of this will be to dampen the capacity to experience hunger.

Below we focus on three particular aspects of this model - palatability, memory and inhibition.

**Palatability**

Cues for highly palatable foods will likely retrieve memories of that particular food associate (i.e., rewarding and food specific), in contrast to less palatable and less energy dense foods, which will be less likely to result in memory retrieval. To some extent this distinction maps on to that between episodic and semantic memory, because recollections of highly palatable foods are more likely to have episodic-like qualities because of their high memorability, just as would foods that have made a person sick (i.e., a conditioned taste aversion; e.g., Logue, Ophir & Strauss, 1981). Importantly, episodic memories of highly palatable foods would require equally high levels of inhibition if they were not to engender hunger. The conditions necessary to produce such high levels of inhibition may be quite rare, making these types of food particularly hunger-inducing irrespective of the body’s physiological state (Bilman, van Kleef & van Trijp, 2017).

Hunger for *specific foods* (i.e., episodic memory) can thus be conceptualized as the product of two interacting processes, the original reward value of the food encoded into memory and the level of physiological inhibition that is currently arising from the body. The predicted relationship between these two processes is illustrated in Figure 2, with the surface area of this graph representing hunger. The relationship is hypothesised to be non-linear. At minimal levels of inhibition, reward value of food may still be relevant to the degree of hunger generated. While some laboratory studies argue against this proposition based on brief periods of fasting (e.g., Hofling et al., 2009), none have come anywhere near the minimal levels of inhibition that occur during deep starvation. Documented reports of
starving people offer several instances of significant palatability concerns when there is potential for food choice (e.g., Mollinson, 1946). This behavior puzzled healthy observers who seem to have believed that any wholesome food would be ‘good food’ to a starving person (e.g., avoidance of black bread and bitter protein hydrolysates by concentration camp inmates during re-feeding; Lipscombe, 1945). Non-linearity may also occur at maximal inhibition. It was suggested earlier that there may often not be enough inhibition to fully counteract memories of high reward value foods.

Figure 2: Hunger as a product of food reward value and level of physiological inhibition for specific foods (episodic memory)

Memory

The key process that leads to hunger is an appraisal of the felt affect that accompanies a food related memory, which informs whether that food will be good (rewarding) to eat now. There is plenty of evidence to suggest that people remember eating experiences, and especially their hedonic component (e.g., Garbinsky, Morewedge & Shiu, 2014). Peak
enjoyment of an eating bout seems to be one important hedonic feature that is remembered (Munoz et al., 2018; Robinson et al., 2011, 2014), along with pleasure at the end of the bout (Garbinsky et al., 2014; Robinson, 2014). Duration of pleasure seems far less important (Rode et al., 2007). Remembered enjoyment correlates with hedonic reports made when eating (Munoz et al., 2018) and the former is an excellent predictor of future expected enjoyment (Robinson, 2014). There seems little doubt then that people remember how much they liked a particular food or meal. Such information can be contained both within an episodic (e.g., experience with a particular eating event; Conway, 2009) and a semantic memory (e.g., about a class of eating events; Gaillard & Urdapilleta, 2011), although the way affect is represented in each may differ.

For episodic memories of food, it seems likely that affect is directly experienced when the memory is retrieved and that this probably reflects peak/end enjoyment, suggesting a fairly brief mental experience - seconds - rather than several minutes that might reflect actual eating time. A central property of episodic memories is that they are presumed to be autonoetic, that is they are as if an event is being briefly relived (Tulving, 1983). ‘Appetite’ models make essentially the same claim. That is cues for high palatability foods bring to mind what it is like to consume that food (i.e., affect & sensation), describing this as either explicit ‘process simulation’ (Munos-Vilches et al., 2019, 2020a, 2020b) or as ‘mental imagery’ (Hoch & Loewenstein, 1991; May et al., 2012; May et al., 2015). There is considerable convergence between episodic memory, mental simulation and mental imagery, in that all involve internally generated sensory-affective experience (e.g., Hassabis & Maguire, 2007; PAPies et al., 2020, 2021). The MTL is likely to be central to this process, as it provides the memories from which mental images are constructed (e.g., Buckner, 2010; Maguire et al., 2010). Access to extant episodic memories (i.e., anterograde memory), formation of new episodic memories, and capacity for mental imagery are all affected by
damage to the MTL (e.g., Gilboa et al., 2006; McCormick et al., 2018) and it may be for these reasons that the capacity to experience hunger is also impaired.

That felt affect is probably a core component of episodic memory is implied by several findings on mental imagery and process simulation. Imagining a palatable food increases desire to consume that food (e.g., Devos et al., 2022; Munos-Vilches et al., 2019, 2020a, 2020b). Mental imagery of food can also: (1) increase salivation (Keesman et al., 2016); (2) increase the amount subsequently consumed (Papies, 2013); and (3) activate a similar neural network to that observed during actual consumption (Chen et al., 2016). One might also expect that people’s different food likes and dislikes would be accurately reflected in their mental simulations, and this also appears to be the case (Papies et al., 2021). In addition, a greater capacity for vivid mental images – along with its affective component – should result in greater appetite and hence a greater propensity for weight gain. This too has been observed (Patel et al., 2015). These findings suggest that affect probably occurs when someone retrieves an episodic memory of a previous encounter with a palatable food, with that affective component forming the basis for knowing whether or not that food will be good to eat now.

Secondary and tertiary cues are more likely to retrieve semantic memories, which will involve a more knowledge-based representation of food affect (e.g., Gaillard & Urdapilleta, 2011). As there is no absolute distinction between episodic and semantic memories - the two are usually intertwined (e.g., Conway, 2009; Greenberg & Verfaellie, 2010; Irish & Piguet, 2013) - this means in many instances there will still be some episodic component. Some episodic component allows for some felt affect, which could form the basis for knowing if food is good to eat now. It is also plausible that semantic-based affect-knowledge could support hunger judgments. However, a more likely alternative is cognitive elaboration, with the magnitude of ‘known liking’ for the retrieved semantic memory (e.g., ‘lunchtime’,
‘Indian cuisine’, ‘Patisserie’) increasing the likelihood of thinking about specific exemplars drawn from that category. For example, if you see an Indian restaurant and you really like Indian food [semantic memory], this will increase the likelihood of thinking about specific foods such as whether your favourite dish is available (conceptually like the idea of cognitive elaboration in the elaborated intrusion theory; May et al., 2012). Retrieving a specific instance - an episodic memory of a particular food - with its attendant affect, would then form the basis for knowing if food is be good to eat now - hunger.

*Inhibition*

Several authors have suggested the importance of inhibitory processes in understanding hunger-related states. Strubbe and Woods (2004) proposed two broad classes of physiological process. One they described as ‘tonic’ reflecting a general background propensity to react or not react to food cues. The other consisted of satiety factors associated with recent food intake, which is also identified as an inhibitory influence by Beaulieu and Blundell (2021). Blundell et al., (2021) identified several physiological sources of inhibition, as well as sources of excitation. As described earlier, these physiological influences are potent, and are presumed to operate on psychological processes (hunger) in an integrated manner. That is all of the physiological sources of inhibition (and excitation) summate, to produce general inhibition. And it will be *generally* inhibitory, as these forces are usually most dominant in healthy people.

At the broadest level, general inhibition will influence both the affect experienced when a food memory is retrieved and that experienced when eating food - noting the important distinction between *remembered* and *sensory*-based affect. One consequence of general inhibition influencing both memory and sensory affect is that for any given food, the affect experienced in memory is *generally* well correlated with that when eating commences (i.e., sensory affect). In studies of participants desire to eat palatable snack foods –
effectively a hunger rating made for just one food when looking at it – there is generally a correlation of between 0.8 and 0.9, to liking ratings made when tasting the first mouthful of it (e.g., Attuquayefio et al., 2016; Pender et al., 2019; Stevenson et al., 2020; Stevenson et al., 2017). As general inhibition increases over the course of a meal (i.e., satiation), this produces well documented effects on people’s hedonic reaction to food – pleasure declines. This has both food specific (sensory specific satiety; e.g., Sorensen et al., 2003) and food generic (alliesthesia; e.g., Cabanac, 1979) components, with the latter more likely reflecting the impact of general inhibition rather than habituation. Earlier, the word ‘generally’ was deliberately italicised, because when general inhibition tends to be high, as at the end of a meal, there is an important decoupling of the affect that occurs in a food memory, from the affect that occurs when that food is tasted in the mouth. This decoupling is illustrated in Figure 3.

**Figure 3:** Ratings of desire (memory-based affect) decline to a greater extent across a meal than ratings of sensory pleasure (data from Stevenson et al., 2017).

As is apparent in Figure 3, food engenders much less positive affect, as general inhibition increases from before (labelled hungry) to after a meal (labelled sated), and this is true for affective ratings made from both memory (i.e., desire ratings made when just looking
at a food) and from really tasting the actual food (i.e., sensory pleasure ratings). This general reduction in affect presumably reflects the influence of increasing general inhibition on all food-related affect - namely alliesthesia – as different foods were used to generate satiety from those used viewed and tasted, before and after this meal (i.e., sensory specific satiety is not likely to be important here; Stevenson et al., 2017).

The other effect illustrated in Figure 3 – and the one we wish to emphasise - suggests that a decoupling can occur between memory-based affect (desire in Figure 3) and that generated by food in the mouth. This affective discrepancy effect is large and robust, having been replicated several times, with memory-based affect declining far more rapidly across a meal than sensory based affect (Attuquayefio et al., 2016; Chan et al., 2020; Pender et al., 2019; Stevenson et al., 2020; Stevenson et al., 2017).

A key assertion here is that the affective discrepancy effect is dependent on the function of the medial temporal lobes, and by the same token that alliesthesia and sensory specific satiety are independent of the function of the medial temporal lobes. The latter assertion seems well supported, because as noted earlier sensory specific satiety still occurs in people with MTL lesions (Higgs et al., 2008; Zuniga et al., 2020). A number of findings also support the former assertion that the affective discrepancy effect is dependent on the MTL. First, the magnitude of the affective discrepancy effect is correlated with performance on declarative memory tasks (Attuquayefio et al., 2016). Second, disruption of hippocampal-dependent memory function by exposure to a Western-style diet, results in a reduction in the affective discrepancy effect – a reduction that correlates with the reduction in performance on a test of declarative memory (the Hopkins Verbal Learning Test; Stevenson et al., 2020). Third, the affective discrepancy effect has been found to be absent in a patient with bilateral damage to her medial temporal lobes (Zuniga et al., 2020). The upshot of these observations is that the affective discrepancy effect is one consequence of increasing general inhibition,
but crucially it is a consequence that manifests through the declarative memory system. This mode of inhibition is then hypothesised to cause a substantial reduction in hunger, when sated participants view food.

Much of the thinking about the role of inhibition in memory has been suggested by a now extensive series of animal learning studies performed by two labs, namely that of - Davidson (e.g., Davidson et al., 2009; Davidson et al., 2010) and Parent (e.g., Henderson et al., 2013; Parent, 2016), with a focus on the medial temporal lobes, and especially the hippocampus. A key premise, which is now well supported, is that the hippocampus has all of the necessary hormonal (Lathe, 2001; Zhao et al., 2004; Kanoski et al., 2013) and neural inputs (e.g., lateral and ventromedial hypothalamus; striatal reward areas; orbitofrontal and prefrontal cortices; e.g., Cavada et al., 2000; Risold et al., 1997) to monitor metabolic related processes. With this in mind, Parent has suggested that the hippocampus encodes a memory of recent food intake, and this then serves to inhibit feeding behavior in the immediate post-meal period (Hannapel et al., 2019; Parent, 2016; Parent, Darling & Henderson, 2014).

According to Davidson, physiological satiety cues from recent food intake serve to inhibit retrieval of excitatory associations to food – food memories in other words (e.g., Davidson et al., 2019; Hargrave, Jones & Davidson, 2016).

The form in which this inhibition occurs in humans is not understood and at least three possible types of inhibitory processes are suggested here. First, inhibition could suppress retrieval of just food-related memories. Second, degree of general inhibition present at retrieval, could favour recovery of food-related memories encoded with the same degree of general inhibition. Third, inhibition may act to suppress the affect linked to just food-related memories. It is not known if these hypothetical processes would differentially impact episodic and semantic memory subsystems. We have tended to assume that episodic
remembered affect is likely to be more impacted than known semantic affect (e.g., I may not want any more Indian food after eating it for lunch, but I still know I like it).

The first possibility would suggest that in a well-nourished person after a filling meal, coming across primary, secondary or tertiary food cues would be far less likely than when hungry, to lead to the retrieval of food-related memories. This version is closest to the type of animal model envisaged by Davidson (e.g., Davidson et al., 2019; Hargrave, Jones & Davidson, 2016) as it operates on the retrieval process and is presumed to have a hippocampal substrate. However, if a food-related memory was retrieved, its affective and sensory content would not presumably differ from the same memory generated when fasted.

The second possibility relates to a class of memory process where the context of retrieval (i.e., presence of a certain degree of inhibitory state) governs what is retrieved (i.e., what it is like to eat this food with this level of inhibition). This would seem to be a form of state dependent memory (Eich, 1995). This possibility is premised on the idea that memories of eating, which include the pleasantness of the food, are encoded near satiation (i.e., greater inhibition), as are memories of eating the same food when physiological satiety cues are largely absent (i.e., less inhibition). Whether one type of memory or the other is retrieved will then depend on the retrieval context (i.e., bodily physiology), and hence on the inhibition of similar competing memories that are not consistent with the current contextual state. This type of inhibitory process is also consistent with hippocampal-related function in people (e.g., Kennedy & Shapiro, 2009) and animals (e.g., Holland & Bouton, 1999). However, it is not established if people encode changing orosensory affect across a meal (i.e., having memories of the same food that are positive at one point in a meal and less so at another).

The third possibility is that inhibitory processes operate solely on the affective component of the retrieved food-related memory. In this case, the same memories are as likely to be retrieved when there is significant inhibition as when there is far less, but what
differs is that the inhibition just impacts the positive affective component of the memory. Thus, the memory of a pleasant tasting food might be experienced as less pleasant during the immediate post-meal phase, because the positive affective component of this memory is selectively inhibited. Another way of envisaging this is that the satiety state itself is affective, and it thereby precludes the experience of any similar affective state, such as that from a food memory.

Conclusion for problem 1

The proposed account suggests that the affect experienced when a declarative memory is retrieved, forms the basis for hunger - namely will food be good to eat now? Many aspects of the proposed model are hypothetical, with mechanisms that remain to be tested, but it is grounded both by its reliance on learning and memory (i.e., a model in the ‘appetite’ lineage), and by its use of inhibition as a means of manifesting the impact on psychological processes (hunger) of critical physiological parameters (e.g., energy stores, recent feeding etc). This type of inhibitory relationship between ingestive physiology and psychology, has been suggested by several authors (e.g., Beaulieu & Blundell, 2021; Strubbe & Woods, 2004).

An important consideration is whether the account proposed here advances understanding beyond that of the three current human ‘appetite’ models (Lowe & Butryn, 2007; May et al., 2012; Papes et al., 2020). One major reason why it does is that these ‘appetite’ models are restricted in scope, as they cede much to an illusory process - homeostatic hunger. Consequently, they have little need to address how physiology affects hunger, no need to account for hunger for less palatable foods, and no requirement to explain how bodily sensations that people report as hunger, arise. A core feature of these current ‘appetite’ models, the involvement of learning and memory, is also central to the model presented here, but we extend this in several novel ways to enable a general model of hunger.
We do so by starting to specify how physiology impacts hunger at a psychological level via memory inhibition, and how this in turn alters desire for foods that vary across the spectrum of palatability (i.e., Figure 2). We also offer a classification of cue types that can trigger retrieval and hunger, and link this classification to the two major sub-systems of declarative memory. Finally, we provide a unifying perspective that connects the meaning of certain bodily sensations such as a rumbling stomach with a mental state of positive anticipation that food will be good to eat - the focus of the next section.

**Problem 2: Bodily sensations and hunger**

Interceptive states that people describe as connoting hunger, such as an empty stomach, gut contractions, irritability, gastric rumbling or light-headedness etc (Harris & Wardle, 1987; Monello & Mayer, 1967), may all rely on learning processes that occur during development. In addition, other bodily (i.e., interoceptive) states, which can lead to eating, but that are not typically thought of as hunger, such as pain, low mood, or stress-related arousal, may also draw upon a similar learning process. The basic contention here is that many interoceptive states - including all of the examples just listed - can potentially come to serve as secondary cues. That is, they can lead to the retrieval of food-related semantic memories (i.e., for classes of food or food situations; Ross & Murphy, 1999). This provides one important link between the two definitions of hunger presented in the opening line of the manuscript (i.e., the meaning of certain bodily cues and positive anticipation that food will be good to eat).

There is a second and further link between the two meanings of hunger presented at the start of the manuscript. This concerns a key aspect of the acquisition process hypothesised to occur during development, and which is predicted to be common to both interoceptive cues for hunger, and to external cues for food. This concerns learning that the affect in a food memory, retrieved following a primary or secondary cue, is **predictive** that
food will be good to eat now. This then involves both learning the process – taking note of
the affect in a food memory – and the outcome, namely that the affect in food memories is
predictive of how pleasant a food will taste now.

One occasion where bodily sensations become very pronounced is during starvation.
This is discussed at the end of this section as it raises some of the same issues about the
meaning of interoceptive states that is a key theme here. It also illustrates how there is no
need to invoke homeostatic models as a cause of hunger even in extremis.

*Linking bodily sensations to hunger during development*

How internal bodily sensations become linked to food/hunger is largely unstudied.
Nonetheless, the idea that children may learn about hunger states from their parents has been
suggested before in the literature, alongside the broader notion that people and animals have
to learn what particular interoceptive states mean – perhaps with the exception of pleasure
and pain (e.g., Dupuy et al., 2021; Harshaw, 2008). In animals, food-deprived rat pups only
demonstrate food-orientated behavior when they have learned that the feeling of ‘not having
eaten for several hours’ means food will be good to eat now (Changizi, McGehee & Hall,
2002). In humans, evidence that this type of learning occurs is currently indirect. One piece
of evidence comes from patients with semantic dementia, who have degeneration of their
anterolateral temporal lobes. As this form of dementia develops, patients progressively lose
awareness of what their bodily sensations mean, including those relating to hunger (Gan, Lin,
Samimi & Mendez, 2016). This is consistent with the idea that the meaning of these bodily
sensations was originally learned.

For interoceptive states termed hunger, we suggest acquisition occurs during early
childhood, post-weaning (i.e., 18M to 3 years). A child may be explicitly informed by their
caregivers that a rumbling tummy (for example) signals the need to eat, enabling the child to
learn an association between this internal sensation and food. A similar situation could also
be envisaged with a child complaining of feeling tired or showing irritability, and again being informed by their caregiver that this indicates hunger and the need for food. In all of these hypothetical situations the result would presumably be that the internal state becomes linked to the idea of needing food, and hence the internal state acquires the capacity to retrieve food-related memories (see left-hand pathway in Fig. 4). As this learning process does not involve specific foods, rather just *food in general*, it is for this reason that such interoceptive states are presumed to serve as secondary cues in the proposed model.

While the process described above is explicit and based on caregivers drawing a child’s attention to their internal state, it is plausible that a similar form of learning could occur more implicitly and earlier in development. Infants could learn to associate what an empty stomach feels like with milk-food being rewarding. If this were to occur, it is hard to know how functionally useful it would be. This is because the infant would have to communicate this state to their mother and they in turn would need to understand its meaning. It is currently unclear if infants do communicate distinct hunger signals to their mother (Hetherington, 2017). This is one reason why we suspect that interoceptive hunger cue learning may occur when the infant is older.

We stated above that not only were interoceptive cues like a rumbling stomach linked to a general learning and memory framework by their capacity to retrieve food-related semantic memories, but that they were also linked by a common aspect of the acquisition process. This requires considering how children learn that the affect in a food memory is
predictive of the affect experienced during eating that food. When a child is offered or shown food and asked, ‘are you hungry?’ or ‘would you like some?’ this may provide the foundational cognitive process for learning that the affect experienced in a food memory can be predictive that a food will be good to eat now (see right-hand pathway in Fig. 4). We suggest that in young children, this judgment process may at first be deliberative, occurring perhaps when: (i) a child is just offered food; (ii) asked if they would like to eat a particular food’ (iii) asked if they are hungry; or (iv) asked if they would like to eat now. This may invite reflection on how that eating might feel. This cognitive process may then be crucially reinforced by subsequently finding out that food is good to eat now or as the case may be – is not. This sequence of events may serve over time to turn the process of asking the question ‘how might eating feel now?’ into a habit, which occurs on every occasion that a food-related memory comes to mind – irrespective of whether this memory arises from an internal (tummy rumble) or external (seeing food) cue.

The rudiments of this type of judgmental process may start early in development. One possible illustration of this is provided in Hetherington (2017; see her Figure 1, upper three photos), with an infant being presented with a spoonful of a novel vegetable, looking at it, and then turning her head away in a clear act of refusal. While novelty (rather than experience - memory) may be the cause of rejection in this instance, this is still presumably based on the infant’s inference (i.e., mental modelling or simulation) that this food will not be good to eat. However, it is important to note that this behavior may involve brain systems outside the MTL, as infants may not yet have a fully functional hippocampus.

Other types of interoceptive cue may induce a ‘hunger-like’ state, drawing upon a related but different learning process that probably occurs later in development. Here, negative mood states such as boredom, stress and mild pain (e.g., menstrual dysphoria), may be temporarily alleviated by eating a palatable food (e.g., see Blass & Shah, 1995, for the
basic premise). Through this process the person comes to learn that the consequences of eating while dysphoric are rewarding because it alleviates that state. Thus, when boredom, stress or mild pain occur again, they come to serve as a cue to retrieve memories for whatever class of food had been eaten before to alleviate that state. Those food memories will then be experienced as rewarding. While this will probably not be labelled as hunger, it may function in largely the same way to promote eating.

To the extent that these various forms of learning outlined above actually occur, it would suggest that people should possess a rather idiosyncratic set of internal states that can serve as cues to retrieve food-related memories. This is because during development caregivers could in theory draw a link between any of several different bodily states that the child may experience and the need for food - perhaps based on the bodily states linked to food that they were taught by their caregivers. This in turn would imply a low degree of agreement between strangers as to what internal state is meant by the term hunger due to the idiosyncratic nature of the learning process, but a higher degree of agreement between child-caregiver and child-sibling pairs. These predictions seem broadly consistent with the pattern of observations described earlier in the manuscript, but there does not appear to be data as yet on child-caregiver and child-sibling pair similarity.

*Interoceptive states during starvation*

A number of authors have suggested that *in extremis* low fuel status can create a ‘hunger’ interoceptive state so potent it almost always leads to eating if food is available (e.g., Weingarten, 1985). It is suggested here that this need to fall back on a ‘hunger’ model *in extremis* is unnecessary because it relates directly to the issue of what an internal state *means*, and the types of process described in the previous section. One of the characteristic features of starvation is that people’s desire to eat is compelling and impossible to ignore, because it constitutes an internal state as affectively aversive as pain (e.g., Collins, 1995;
Leyton, 1946; Keys et al., 1950). We suggest there are two components to this starvation experience. One is the presence of several internal states associated with changes in body composition, gut function, metabolism, and fuel status (Heymsfield et al., 2011; Prentice, 2005). All of these result in aversive pain-like states, such as feeling intensely cold, severe gastrointestinal pain, fatigue, and low mood (Kalk et al., 1993; Keys et al., 1950). The other component reflects an intense desire to eat. This results from the lifting of all inhibitory physiological influences, such that exposure to primary, secondary or even tertiary cues all result in the generation of food-related memories, producing an incessant stream of ‘food is rewarding’ evaluations.

Both these components together may be labelled as hunger - but only because the meaning (i.e., cause) of the aversive physical state is so readily apparent - starvation. Importantly, this is not the same as saying there is a ‘drive to eat’ caused by the metabolic changes that accompany starvation. This is because the aversive internal signals have no intrinsic meaning relating to food and eating without their interpretive context (i.e., the knowledge one is starving, and the incessant stream of food-related memories each providing a ‘food is rewarding’ evaluation, resulting from minimal inhibition). In the absence of starvation, feeling intensely cold, severe gastrointestinal pain, fatigue, and low mood would be labelled as an infection or some other serious health problem. It would not, however, be called hunger.

Conclusion for problem 2

A learning and memory approach can potentially account for the presence of the various interoceptive states that people report as hunger, and bring together the two definitions presented at the start of this manuscript under one model. However, there has as yet been very little work to examine if the acquisition processes we envisage actually occur.
Problem 3: Top-down initiation – judging hunger

Psychologist often ask their research participants to report their current level of hunger. From the perspective of energy depletion models this makes sense, in the same way that a petrol tank gauge in a car indicates the amount of fuel left in the tank. However, in the context of the model presented here, an overall judgment of hunger probably has several potential determinants, because the context in which this question is asked can often be quite different to the cue-driven situations where hunger normally arises. It is for this reason that we suspect that laboratory-based hunger judgments may have several determinants (and see Friedman, Ulrich & Mattes, 1999), some of which include processes related to the proposed model, and others that do not.

Several processes could contribute to laboratory-based hunger judgments. Participants may try to imagine themselves eating, as retrieval of both episodic and semantic memory can be intentional (e.g., Addis & Schacter, 2012). In trying to imagine eating, one route may be to recall a recent eating bout - an episodic memory - and use this as the basis for making a judgment if such food would be good to eat now. Another route would be if the participant knew what they were going to eat in the future, focussing on this food instead. It is also possible that in trying to make a general hunger rating, this invokes a search of the milieu intérieur, with attention focussed inwards to identify any internal cue believed to indicate hunger. The presence of such an internal cue may then form the basis for a judgment, and/or it could trigger a food-related memory. A further possibility is that a person may use both the time of day (i.e., lunchtime) and how long it has been since they last ate, to estimate how hungry they should feel. Finally, a large number of other parameters may also influence general hunger ratings, such as current weight-related food restriction (i.e., dieting), the amount of food that a participant believes they have consumed at a previous meal (e.g., Brunstrom et al., 2011), the sensory properties of food eaten at a prior meal, and the degree of
attention paid to eating that food (e.g., Yeomans et al., 2017; Morris et al., 2020). All or any of these factors (and probably others too) may then contribute to or influence general hunger ratings. In sum, this would suggest hunger ratings may not always be measuring the same thing in different people within the same experiment, or between people in different experiments (i.e., where the study parameters may favour one causal factor over another).

One possible example of this issue can be seen in a recent series of studies. When participants are asked to recall a meal from today, this reliably suppresses their food intake on a subsequent test eating bout, relative to participants asked to recall a meal from the day before (e.g., Collins & Stafford, 2015; Higgs, 2002; Higgs, Williamson & Attwood, 2008; Robinson et al., 2013; Szypula, Ahern & Cheke, 2020). Although food intake is reduced on the test eating bout in participants recalling their meal today, their hunger ratings, obtained before and after the test eating bout, are not affected by this manipulation (Higgs, 2002; Higgs, Williamson & Attwood, 2008). However, when ratings are made of participants desire to eat specific foods - essentially hunger for individual items - these ratings are significantly suppressed by the meal today manipulation, while general hunger ratings remain unchanged (Arthur, Stevenson & Francis, 2021). This difference may arise because general hunger ratings may have a broader set of causes to the hunger ratings made for individual foods.

The causal basis of general hunger ratings is also important for the proposed model. This is because abnormalities on general hunger ratings in people with MTL lesions, formed part of the rationale for focussing on declarative memory as a core basis for hunger. If general hunger ratings have multiple causes, and if some of these arise outside of the proposed model, then it could be argued that it is these extraneous causes that result in the abnormal hunger ratings in MTL patients. One reason to think otherwise comes from the Zuniga et al., (2020) study. Here patient JC exhibited abnormalities on both hunger ratings
for specific foods (i.e., desire) - one aspect of the proposed model - as well as on general hunger ratings, suggesting some commonality of impairment. One patient in one study does not of course settle this matter, and while there are several other good reasons to think that learning and memory are critical for understanding hunger, it will be important to identify exactly why evaluations of general hunger are so disrupted in people with MTL damage.

**Broader implications of a learning and memory approach to hunger**

**Relationship to animal hunger, wanting and liking**

The excitatory associations between say a tone and food that support ‘appetite’ in animals (Weingarten, 1985), do not seem to depend on the hippocampus in the rat, while the inhibitory processes that suppress excitatory responding during satiety do (e.g., Benoit et al., 1999; Chan et al., 2001). Rather simple excitatory associations in the rat - tone-food - may instead depend upon a striatal and/or an amygdala based circuit (e.g., Liljeholm & O’Doherty, 2012; Petrovich & Gallagher, 2003). In the model proposed here, while the inhibitory processing may be similarly dependent on the hippocampus (e.g., Stevenson et al., 2020), the food-related memories that form the basis for hunger rely upon the declarative memory system, whose substrate is the medial temporal lobes, and especially the hippocampus (e.g., Squire, 2004; Squire & Zola-Morgan, 1991). The import of this is that ‘appetite’ in rats - to the extent that this is modelled by say tone-food pairings - probably utilises a rather different set of brain substrates to those that form hunger (i.e., ‘appetite’) in humans (but noting there may be occasions where rodents do have episodic-like memories; see Crystal & Suddendorf, 2019).

In rats, a key neural distinction has been proposed, which underlies two often closely linked psychological states. One mediates orosensory affective responding to food (liking) and the other mediates desire for food (wanting). This neural distinction, and its implications, comprise the core components of incentive salience theory (e.g., Berridge,
The same wanting/liking distinction has been suggested to occur for humans, and this idea has seen quite broad application in the field of ingestive behavior (e.g., Finlayson, King & Blundell, 2007; Stice & Yokum, 2016). A potential parallel then between the model presented in this manuscript, and incentive salience theory, is that hunger - remembered felt affect - parallels wanting, and orosensory affect on consumption, parallels liking. The latter parallel is uncontroversial, but there are some problems with the former. If hunger in animals reflects wanting, which seems to be a clear presumption of incentive salience theory (expressed as ‘appetite’; Berridge, 1996; Berridge, 2009), and the neural basis of hunger in animals does not parallel the neural basis of hunger in humans, then presumably neither does wanting.

Even if the substrates of human hunger were to parallel those of animal hunger, the wanting/liking distinction of incentive salience theory may have little relevance to the model proposed here and vice versa. This is because wanting and liking in incentive salience theory are usually very closely coupled, except when neural sensitization occurs – such as with drugs of addiction (Berridge, 2009). In this case, wanting becomes a pathological motivational force driving consumption even in the absence of liking. It is very unclear at the moment if foods can reliably cause neural sensitization in humans in the manner that certain drugs clearly do, and relatedly, it is also unclear if any food has a truly ‘drug-like’ effect on the human brain (Havermans, 2012; Rogers, 2017; Ziauddeen et al., 2012). Indeed, even highly palatable foods are not pharmaceuticals, and they exert their effects on the brain indirectly via the perceptual system.

If neural sensitization is unlikely to occur for foods, incentive salience theory would expect wanting and liking for foods to be very tightly coupled. To the extent that wanting maps on to hunger (as described here), this would imply that hunger and liking should also be closely coupled. They certainly are when several hours has elapsed since a prior meal, but
following a meal, hunger very markedly dissociates from orosensory affect (see Figure 3 and associated discussion). Clearly, this is not a dissociation anticipated by incentive salience theory. This too suggests that the wanting of incentive salience theory is unlikely to be akin to hunger as presented in the proposed model.

*Cephalic phase response*

Several papers by Wood’s (e.g., Ramsay & Woods, 2016) have argued that learning is very important for understanding eating behavior in rats. One of the reasons for this is that consuming a meal represents a significant physiological challenge (Woods, 1991). Eating produces potentially disruptive increases in blood sugars, fats and other digestive products as the organism attempts to break down, move, utilise and store these nutrients. In humans, there is some limited evidence that disrupting the normal ingestive time course (i.e., anticipation, followed by oro-sensory cues, stomach filling etc) is problematic, with data mainly coming from the study of patients receiving total parenteral nutrition (TPN). While many TPN patients are severely unwell and hospitalized, a proportion are ambulatory and use TPN at home. Metabolic complications from TPN are common, including hyperglycemia, transient hypoglycemia on starting an infusion, hypoglycemia during an infusion, reactive hypoglycemia post-infusion, and longer-term, a build-up of fatty tissue in the liver from abnormal carbohydrate metabolism (Btaiche & Khalidi, 2004a & b). While not all of these metabolic complications can be laid at the door of an absent cephalic phase response, it is probable that a proportion do result from unsignalled nutrient influx.

There is then a potentially useful consequence of having a hunger system predicated on learned cues, because they can also serve as a signal for the body to prepare for nutrient intake. While a proportion of the human cephalic phase response is initiated by the presence of food in the mouth, more distal food cues (e.g., sight, smell) also produce a response both for insulin (Wiedemann et al., 2020) and other digestive hormones (Skvortsova et al., 2021).
The relationship between the experience of hunger on exposure to food cues, and the occurrence of the cephalic phase response has not been studied (i.e., is the latter dependent on the former?), nor has the impact of multiple exposures to food cues in the absence of food receipt (i.e., does cue exposure end up blunting the cephalic phase response and relatedly what does it do to hunger?).

**Automaticity**

It has been argued that a combination of automatic (e.g., attentional bias) and controlled processes (e.g., restraint) are involved in regulating food intake (e.g., Aulbach, Knittle & Haukkala, 2019; Cohen & Babey, 2012). Consequently, we might expect that this distinction would also be relevant in the context of the model presented here. In essence the proposed model suggests a cue retrieves a declarative food memory, the affect in this memory is experienced, and this forms the basis for knowing that food will be good to eat now (i.e., hunger). We can fractionate this into a *retrieval part* (i.e., retrieving memory), a *content part* (i.e., affect from the retrieved memory) and a *process part* (i.e., does the affect indicate food will be good to eat now). We suggested in Problem 2: Bodily sensations and hunger, that children may initially be deliberative about the *process part* but that with repetition, this becomes automated so that when food-related affect is experienced its meaning (hunger [or not]) is known without the need for deliberation (i.e., food will be good to eat now). In other words, the supposition here is that the *process part* becomes automatic.

For declarative memories, retrieval can occur automatically, as well as via controlled processing (e.g., Ciaramelli, Grady & Moscovitch, 2008). This would suggest that the same would hold for the *retrieval part* here. The impact of automatic retrieval for memory inhibition is also important to consider because of its centrality to the proposed model. Memory inhibition may be more effective with automatic retrieval than with controlled retrieval, as inhibition might be overridden if a person actively wishes to retrieve a food-
related memory. In addition, to the extent that the human declarative memory system maps on to rodent memory systems, we might expect that retrieval in animal models would be more of the automatic sort, and clearly inhibition can occur in this case (e.g., Davidson et al., 2009; Davidson et al., 2010; Henderson et al., 2013; Parent, 2016).

The *content part* is most interesting from the perspective of controlled and automatic processing. This is because we have assumed that hunger is explicit (as with declarative memory), being something that is explicitly known and reportable about oneself. *If* the *content part* can occur with minimal awareness, as with retrieval and processing, then one could come to know one was hungry but not have this accompanied by any feeling (i.e., *content part*) or awareness of what had taken place (i.e., *retrieval part* and *process part*). This could also include, potentially at least, not knowing what cue had initiated this chain of events. In sum, we suggest that automaticity is theoretically possible for all components, and that it is probably routine for the *process part*, frequent for the *retrieval part*, and probably less likely for the *content part* and triggering cue - while stressing much remains unknown.

*Thirst*

In addition to HM’s inability to report hunger, he also had problems with experiencing thirst and fatigue (Corkin, 2013; Hebben et al., 1985). This suggests a more general impairment of interoceptive processing arising from his medial temporal lobe (MTL) damage. There have been few investigations of interoception in patients with MTL damage, beyond the hunger-related studies described earlier. However, poorer performance on tests of declarative memory - a proxy for MTL integrity - have been linked to diminished experience of thirst, poorer heart-rate perception, and more generally to a poorer capacity to predict future internal states across several interoceptive domains (e.g., Brannigan, Stevenson & Francis, 2015; Dudley & Stevenson, 2016; Edwards-Duric, Stevenson & Francis, 2020; Stevenson et al., 2018). Such findings are puzzling from the perspective of contemporary
accounts of interoception, which consider the insula as the primary interoceptive processor (e.g., Craig, 2009; Barrett et al., 2015; Wilson-Mendenhall et al., 2018). They are less puzzling if a significant role for learning and memory is considered as a routine part of interoceptive processing, as suggested here for hunger.

Beyond hunger, thirst is a good starting point for considering the role of learning and memory processes. Like hunger originally, thirst has also been partitioned into homeostatic and non-homeostatic components (Rolls & Rolls, 1982). While much fluid ingestion occurs in anticipation of future need, there is no empirical or scientific problem with the idea of a needs-based homeostatic thirst. However, for the latter to occur an organism still has to learn the association between the internal state(s) indicative of water need, retrieved affect-rich memories of drinking, and its satiation by particular actions (i.e., drinking). Learning and memory are also likely to be important for non-homeostatic drinking, especially that involving anticipation/prediction of future needs. Finally, a lot of fluid consumption involves sugar-sweetened beverages or drinks containing caffeine. Cues to these drinks (e.g., brand names etc) may lead to the retrieval of episodic memories. The affect associated with that memory could then serve to indicate the likely reward value of consumption. This process would come closest to that described for hunger.

Lay theories of hunger

Lay theories about psychological processes can have significant real-world impacts, such as metaphors about memory being like a tape-recorder providing a rationale for recovered memories. As scientific study of hunger has been heavily dominated by homeostatic regulatory thinking, it would not be surprising to find that this view of hunger has also come to dominate lay people’s beliefs about the origins of hunger, and the impacts of failing to heed its occurrence. We were able to find only one study directly exploring lay people’s beliefs about hunger (Assanand, Pinel & Lehman, 1998). It suggests that many
people do indeed believe that hunger is an internal state that reflects the body ‘running low on fuel’. There has been no research to determine the impacts of holding this belief.

**Trusting hunger**

Since Cannon (1932) coined the term ‘wisdom of the body’ there has been a popular belief in the idea that the body knows best in terms of what, when and how much to eat. A modern rendition of this idea can be seen in the intuitive eating movement, which arose from a popular book (500,000 copies sold according to the publisher) of the same name (Tribole & Resch, 1995). A key claim of the intuitive eating approach is to get people to trust, attend to and act on their internal hunger and satiety signals (e.g., Schaeffer & Magnuson, 2014; Tylka & van Diest, 2013; Van dyke & Drinkwater, 2014).

Eating when hungry makes sense from a homeostatic perspective, but less sense from a learning perspective, where hunger emerges as a consequence of cue exposure rather than bodily need. On this basis one would expect that teaching people to act on hunger signals would not be a very effective approach to managing body weight. Indeed, a recent systematic review found no benefit of this approach for weight control (Grider et al., 2021). The model proposed here suggests a more cautionary approach to using hunger as a signal to eat, irrespective of whether the cue is internal or external. This is particularly so in individuals who may be most drawn to intuitive eating, namely people with obesity, who will probably have dysfunctional memory inhibition due to diet-induced MTL impairment (e.g., Davidson et al., 2022). In such individuals, hunger will occur irrespective of whether they have eaten (i.e., impaired memory inhibition) and so eating in response to either internal or external cues will favour positive energy balance.

While the intuitive eating movement has been aimed primarily at adults, the same ideas have also emerged in the infant feeding literature (e.g., Birch & Davison, 2001; Hodges et al., 2013). The notion here is that parental feeding practices that emphasise ‘finishing
one’s plate’ detract from the child learning to use internal cues to regulate food intake. The long-term impact of this is argued to be excess weight gain and obesity. However, if internal cues for hunger are learned, and do not represent ‘honest signals’ of energy need, then reliance on such cues would not be an ideal manner to regulate body weight. Not surprisingly then - and consistent with the adult literature - a recent systematic review concluded that controlling parental feeding practices (i.e., ‘finish your plate!’) were linked to lower weight outcomes over time (Beckers et al., 2021). While much remains to be learned about which parental feeding practices are best at promoting healthy eating, those favoring major reliance on internal cues may not be among them.

**Generative impact**

Table 2 outlines key issues that arise from the model. These cover the nature of hunger, its development, inhibition, neuropsychology and psychopathology. Some of these domains have received minimal attention, with little understanding of why MTL lesions impair hunger, how (indeed if) children learn hunger, how inhibition functions, and the impact of this model for understanding eating psychopathology. Indeed, this last point is particularly pertinent, as work in both animals and humans has demonstrated how a Western-style diet impairs MTL function thereby dysregulating both appetitive inhibition and also the perception of hunger – something common to MTL damage as first observed in HM (Hebben et al., 1985). What is beginning to emerge from this research endeavour is the broader link between diet, hunger, interoceptive impairment, MTL-based memory impairment and over decades, cognitive decline with its links to dementia (e.g., Francis & Stevenson, 2013; Hargrave, Jones & Davidson, 2016; Hsu & Kanoski, 2014; Wieckowska-Gacek et al., 2021). This may be the most important reason for understanding how MTL-related learning and memory are involved in hunger, as abnormalities in hunger may be a very early sign for more significant neurodegenerative disease of later life.
### Table 2: Implications from the memory model of hunger

<table>
<thead>
<tr>
<th>Domain</th>
<th>Issue</th>
<th>Expectation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nature of hunger</td>
<td>Repertoire of 1° cues that elicit hunger</td>
<td>Should be diet-dependent</td>
</tr>
<tr>
<td></td>
<td>Cue type and memory</td>
<td>Different cue types activate different subtypes of declarative memory</td>
</tr>
<tr>
<td></td>
<td>Interoceptive hunger cues</td>
<td>Multiple, idiosyncratic but reliable</td>
</tr>
<tr>
<td></td>
<td>Self-evaluations of hunger</td>
<td>Should have multiple causes, and so should be readily manipulable</td>
</tr>
<tr>
<td></td>
<td>Impact of deprivation on cue types</td>
<td>Increasing deprivation should lawfully affect 1°, then 2°, then 3° cues</td>
</tr>
<tr>
<td>Development</td>
<td>Children learn interoceptive hunger cues</td>
<td>Parent-child similarity in this regard</td>
</tr>
<tr>
<td></td>
<td>Children learn causes of hunger</td>
<td>Parent-child similarity in this regard</td>
</tr>
<tr>
<td></td>
<td>Children learn hunger</td>
<td>Learning should be observable via parent-child transmission</td>
</tr>
<tr>
<td>Inhibition</td>
<td>Nature of memory inhibition</td>
<td>Is it retrieval inhibition, state dependent memory and/or affect suppression</td>
</tr>
<tr>
<td></td>
<td>Impact on memory types</td>
<td>Greater on episodic, less on semantic</td>
</tr>
<tr>
<td></td>
<td>Improving operationalisation</td>
<td>Extension beyond affective discrepancy effect</td>
</tr>
<tr>
<td>Neuropsychology</td>
<td>MTL lesions and hunger</td>
<td>Understanding nature of impairment and testing for loss of memory inhibition</td>
</tr>
<tr>
<td></td>
<td>Insula lesions and hunger</td>
<td>Minimal, and hence inconsistent with current interoception models, but very few tests</td>
</tr>
<tr>
<td></td>
<td>Aging and hunger</td>
<td>Yoking of declarative memory loss to impairments in memory inhibition</td>
</tr>
<tr>
<td>Psychopathology</td>
<td>Impairment of inhibition</td>
<td>Should be evident in people with obesity and anorexia due to MTL dysfunction</td>
</tr>
<tr>
<td></td>
<td>Parental eating psychopathology</td>
<td>Should impact child interoceptive hunger development and hunger processing</td>
</tr>
<tr>
<td></td>
<td>Alzheimer’s disease</td>
<td>Loss of inhibition (increased appetite) should precede loss of hunger cues (loss of appetite)</td>
</tr>
</tbody>
</table>
Conclusion

The central premise of this manuscript is that hunger results from experiencing a food related declarative memory and determining if the affect experienced in that memory indicates food will be good to eat now. This model is consistent both with historical theoretical trends, which have placed increasing emphasis on ‘appetite’ over ‘hunger’ and with neuropsychological findings, which implicate the declarative memory system in hunger. The model also engages with contemporary animal models of food-memory inhibition, and recent human theorising about the influence of satiety on appetite, to propose that the mnemonic basis of hunger is inhibited by a range of physiological parameters linked to body weight, body composition and recent ingestive behavior. The model reconciles the two definitions of hunger presented in the opening paragraph, by considering internal states as learned, and linking them to the same declarative memory-based processing system as utilised by external cues, with both having common developmental origins. Finally, a consideration of the processes involved in making general hunger ratings suggest they may have multiple causes, only some of which directly connect to the model proposed here.
References


