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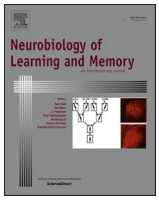

Review

Cue-potentiated feeding in rodents: Implications for weight regulation in obesogenic environments

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ABSTRACT

Cue-potentiated feeding (CPF) describes instances where food intake is increased by exposure to conditioned cues associated with food, often in the absence of hunger. CPF effects have been reported in a range of experimental protocols developed by researchers working across diverse fields spanning behavioural neuroscience, social psychology and ecology. Here we review the evolution of research on cue-potentiated feeding in animal models to identify important behavioural parameters and key neural circuits and pharmacological systems underlying the effect. Overall, evidence indicates that social, discrete and contextual stimuli can be used to elicit CPF effects across multiple species, though effects are often subtle and sensitive to procedural variables. While regular exposure to food cues is thought to be a key risk factor for overeating in so-called 'obesogenic' environments, further work is needed to identify whether CPF promotes positive energy balance and weight gain over the longer term. We suggest several methodological and conceptual areas for inquiry to elucidate the contribution of CPF to the regulation of food choice and energy intake.

1. Introduction

It has long been acknowledged that choices about what, when, and how much to eat can be influenced by the surrounding environment. The idea that initially neutral stimuli associated with food could come to elicit eating was articulated in a 1937 textbook of psychology that explained how:

> *"The baby who has acquired the habit of eating when offered fruit or candy, even in absence of hunger, will rapidly develop interest in the fruit or candy store, in doing this or that to earn the tidbits, in pennies that will procure them, in uncles who furnish the pennies."* (Dashiell, 1937, p. 121.).

This quote captures the broad, anecdotal manner in which external cues were often described. Indeed, as examples were relatable and easy to generate, what constituted an 'external' cue was not defined rigorously but instead encompassed anything other than internal hunger signals (Herman & Polivy, 2008). Several experiments early in experimental psychology's history showed that various external stimuli could trigger eating, alternatively referring to food-paired cues and contexts as feeding-related stimuli (Valle, 1968), signals for feeding (Zamble, 1973) and external events or situational factors (Grant & Milgram, 1973), Yet there were only sporadic attempts to directly study the effects of *conditioned* cues on eating until the 1980s, when Weingarten (1983) published what is generally seen as the seminal demonstration of cue-potentiated feeding. Interest in the effects of external cues grew across the late 20th century as obesity emerged as a global public health challenge, reflected by the incorporation of food cues in theories of energy balance and body weight regulation (e.g. Herman & Polivy, 2005).

Here we review literature on the modulation of eating by conditioned food cues in animal models, beginning with studies of social facilitation and drive theory, in which food intake *per se* was not necessarily the primary interest (enhanced eating was considered a hindrance in some designs), but simply a practical variable to manipulate and measure. Although this work used 'food cues' that were inherently less controlled (e.g. social stimuli), it is informative given that associative processes were often invoked to explain the effects of cues on eating. We then review the evolution of research on cue potentiated feeding from the largely behavioural approaches of early studies to current models employing pharmacological and neural techniques. Most recent studies use conditioned food cues that are intentionally separated

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from physical properties of the food itself (e.g., smell) or the act of eating, such as discrete stimuli (e.g. lights, tones, buzzers) or contextual stimuli (e.g. environments with distinct tactile, olfactory and/or visual features). Finally, we discuss methodological and conceptual issues that future studies might address to better model human eating and overeating. We discuss select work in humans to provide historical context and comparisons but, for comprehensive reviews of effects in humans, please see Bilman et al. (2017) and Larson & Story (2009).

2. The evolution of research on cue-potentiated feeding

2.1. Social facilitation of feeding

Social facilitation describes instances where the presence of a conspecific performing a behaviour initiates or increases that behaviour in another animal (Clayton, 1978; Guerin, 1993, 2010). While social facilitation can occur with a range of behaviours, a large literature has studied eating. Animals will eat novel foods more readily in the presence of a conspecific eating nearby (Dally, Clayton, & Emery, 2008), even with animals from different species (Rubenstein, Barnett, Ridgely, & Klopfer, 1977). Group housing typically increases feeding rate in pigs and cows (Keeling & Hurnik, 1995; Phillips, 2004) and in laboratory studies in rats (Harlow, 1932) and juvenile chicks (Tolman, 1964). While some studies found that social facilitation of feeding required direct social contact between animals (Tolman & Wilson, 1965), suggesting the effect relied on active competition for food, subsequent studies demonstrated enhanced eating with designs that removed competition (e.g., through the use of barriers; Hoyenga & Aeschleman, 1969; Harlow & Yudin, 1933; Dally et al., 2008; Strobel, 1972). Socially facilitated feeding was also shown in studies where sated animals resumed eating upon the introduction of a hungry companion, observed in hens (Fischel, 1927; Bayer, 1929), puppies (Ross and Ross, 1949) and adult dogs (James, 1954). A study in monkeys found that introducing fresh food to a pair of animals stimulated consumption of stale feed by observers in surrounding cages (Galloway, Addessi, Fragazy, & Visalberghi, 2005).

Social hierarchies within groups can moderate social facilitation effects, with stronger effects on eating reported in dominant pigs (Hsia & Wood-Gush, 1984) and submissive cattle (Harb et al., 1985), mixed findings in puppies (James, 1953; James & Cannon, 1955), and no differences between dominant and submissive hens (Bayer, 1929). Group feeding sessions might establish social hierarchies rather than measure pre-existing ones; indeed, this was the explicit aim of group feeding in some studies (Harlow & Yudin, 1933).

Learning and conditioning processes were often invoked to explain social facilitation, with some accounts describing the introduction of another animal as a conditioned, excitatory stimulus that facilitated eating (Harlow & Yudin, 1933). The emergence of social facilitation effects across repeated sessions was cited as evidence that learning and conditioning underlay the effect. For example, James and Gilbert observed that puppies raised in isolation only exhibited social facilitation after repeated group feeding sessions, hypothesising that companion animals became secondary reinforcers, or discriminative stimuli associated with food, that elicited eating (James, 1954; James & Gilbert, 1955). However, in other models, social facilitation was evident in the first group feeding session and stable thereafter (Tolman, 1964).

There were several inherent challenges when interpreting socially facilitated feeding. Apparent social facilitation in group housing may be an artefact of lower food intake by individually housed animals (Clayton, 1978) – though single-housed rats tend to exhibit greater food intake (Schipper, Harvey, van der Beek & van Dijk, 2018) – or reduced stress due to the presence of conspecifics, especially in novel environments (Harlow & Yudin, 1933). Few studies were able to quantify intake by individual animals or identify which specific aspects of eating behaviour were augmented. Social settings may energise other appetitive behaviours besides eating (Keeling & Hurnik, 1996) or behaviours that preclude eating, such as play (e.g. Harlow, 1932, Experiment 3), underscoring the importance of understanding the behavioural repertoire of the species under study (Guerin, 1993). Nonetheless, social facilitation studies demonstrate that the presence of conspecifics can enhance and prolong eating across species. In humans, meal size, duration and even food choices can be affected profoundly by social facilitation and other social processes such as modelling (De Castro, 1997; Stroebele & De Castro, 2004).

2.2. Drive theory

In his 1943 work *Principles of Behavior*, Clarke Hull defined drive as a theoretical construct motivating an animal's behaviour in response to its biological needs. For example, depriving an animal of food was thought to increase its hunger drive, which motivated food-seeking and consumption, meaning that food consumption could be used to infer the strength of the hunger drive (or water intake to the thirst drive, etc.). The concept of drive was defined in varied and largely descriptive terms until Hull refined it within a model that generated testable predictions (Bolles, 1967).

An important question within this framework was the extent to which drive states such as hunger could be *conditioned*, *acquired*, or *externalised* to cues. This was tested by pairing environmental stimuli with a drive state and measuring behaviour in that environment. For example, Calvin, Bicknell & Sperling (1953a) exposed rats to a striped box for 30-min/day after either 1-h or 22-h food deprivation, intended to pair the box with low- and high-hunger drive, respectively. In feeding tests held after 12-h deprivation, the 'high-drive' group ate significantly more, with authors concluding that the motivating properties of the hunger drive became associated with the box, increasing consumption at test (Calvin et al., 1953a). However, this 'conditioned hunger' effect was not replicated in two later studies (Siegel & MacDonnell, 1954; Scarborough & Goodson, 1957) and the broader literature found only weak evidence for the existence of conditioned drives (Bindra, 1978; Bolles, 1967; Morgan, 1979). Weingarten (1985) argued that there was no more reason to expect an environment paired with the absence of food to elicit eating than copulation, or any other behaviour that had been unavailable.

Notably, in the same year, Calvin and colleagues published a similar study in which the same striped box contained food (Calvin, Bicknell & Sperling, 1953b). Pairing the box with food was intended to establish it as a secondary reinforcer through its association with the primary reinforcer (food), rather than with the hunger drive. One group of rats was fed daily in the box; a second was fed in the home cage and placed in the empty box 1-h later; the third always remained in home cages. When testing chow consumption in the box, authors hypothesised that if the secondary reinforcer (the box) functioned like a primary reinforcer, it should reduce the associated primary drive (hunger) and reduce intake. Instead, food intake was greater for rats previously fed in the box, suggesting that what was conditioned to the box was not a reduction in hunger drive, but the act of eating.

2.3. Resistance to satiation

A fundamental prediction of drive theories was that manipulating the strength of a drive should produce corresponding changes in behaviours under its control. This was often tested by training hungry animals to make an instrumental response for food, then assessing instrumental responding in the absence of hunger (Capaldi & Myers, 1978). 'Resistance to satiation' described instances where instrumental responding persisted in sated animals (Morgan, 1974). To minimise the possibility that residual hunger might drive continued responding, tests were often preceded by weeks of *ad-libitum* food in the home cage as well as acute pre-feeding of the food reward (Morgan, 1979). A recurring problem, however, was that animals frequently continued to eat in the food-paired environment despite extensive satiation procedures.

Morgan (1979, p. 190) observed that "*in practice it is often very difficult to eliminate consummatory responding in a situation where the animal has learned to eat; and then matters become much more complicated*".

In most studies, food intake was simply a means to satiation and consumption data were rarely reported or analysed (Morgan, 1974). However, consumption was measured in a series of studies by Elizabeth Capaldi and colleagues, who trained hungry rats to traverse an alley to locate food pellets in a goal-box (e.g. Capaldi & Myers, 1978). Despite unrestricted home-cage chow and pre-feeding of the pellets prior to test, sated rats continued to traverse the alley and eat the goal-box pellets on most trials. A later study compared groups given reinforced alley training to those placed directly into the goal-box with pellets inside, alongside two groups not pre-exposed to the alley or pellets (Capaldi, Davidson, & Myers, 1981). Sated rats only ate pellets if they were previously fed pellets in the apparatus, with the effect persisting for at least 10 test days with four daily trials. The presentation of pellets during pre-feeding and placement in the running alley were viewed as cues that elicited the consummatory response in satiated animals by 'force of habit' (Capaldi & Myers, 1978). However, no studies appear to have tested whether consumption would continue in *any* environment, such as alleys never paired with pellets (Capaldi et al., 1981).

2.4. Conditioned food cues using discrete stimuli and contexts

While food intake was a means to an end in studies of drive theory and resistance to satiation, other studies across the 20th century were designed to test explicitly whether food-paired cues could enhance consumption. Drew (1937) tested the potential of an extensive range of stimuli to stimulate eating in satiated rats, including turning a tap on and off and training the rat to tug on a piece of food held by the experimenter or to retrieve food from adjacent tables. The only manipulation that reliably induced eating was placement in an environment where eating had previously occurred, interpreted as an ongoing 'habit' fostered by prior training. Valle (1968) fed hungry rats chow in a distinct white box for 1-h/day for 15 days. At test, rats either remained in the home cage or were re-exposed to the context immediately prior to a 10-min test of pellet consumption in the home cage. Pellet intake was significantly greater in rats re-exposed to the context than those kept in the home-cage. In a similar study, rats were exposed to a distinct context containing food pellets for 30-min/day for 6 days, with one group given unrestricted access to pellets in the home-cage and another fed only in the context and for 30-min afterwards (Grant & Milgram, 1973). Tests of pellet consumption in the context after 3 days of unrestricted food showed greater intake in rats previously exposed hungry than satiated; however, this group weighed significantly less than the satiated group at test, suggesting energy depletion may have driven greater intake (Grant & Milgram, 1973).

While Valle (1968) and Grant and Milgram (1973) used contexts paired with food, Zamble (1973) applied a discrete cue procedure to rats in their home-cage. At an unpredictable time each day, animals received a 20 g food ration for 30 min. For one group, the housing room light turned off 15 min prior to feeding ('signalled' feeding), whereas offset of the light and feeding were unpaired for another group. Over 25 days, rats given 'signalled' feeding sessions ate more of their food ration and lost less weight than those given unpaired feeding. Experiment 2 compared forward and backward pairings of an auditory cue with unpredictable feeding sessions spaced 4–44 h apart. Once again, the cue enhanced food intake only when it preceded food availability, leading to body weight differences despite equivalent access to food. In fact, excessive weight loss in the backward-paired group led to premature termination of the experiment.

In what appears to be the first within-subjects demonstration of cue potentiated feeding, Lovibond (1980) trained rats to associate two contexts with food and two with no food. Rats were exposed to one context in each pair while hungry, and to the other after minimal food deprivation. At test, the two food-paired contexts increased food intake, rates of lever pressing and general activity. Lovibond (1980) suggested that the food-paired contexts aroused a single central appetitive system directed toward procuring food. A second experiment in this study was based on the Zamble (1973) study described above, and found that a discrete food-paired cue (tone or light) elevated food intake by 11 % relative to an unpaired cue and by 13 % relative to when no cue was presented.

Research around this time began to explore the involvement of satiety peptides in feeding routines. Schallert, Pendergrass and Farrar (1982) tested the effects of the digestive peptide cholecystokinin (CCK) on food intake in rats maintained for 3 weeks on a feeding schedule involving two daily 'cued' meals wherein the housing room light was switched off and a light adjacent to each cage turned on, before the experimenter offered pellets to the animal. This series of 'meal cues' continued to elicit food approach and consumption in minimally deprived rats, and CCK injections increased the latency to eat, reduced intake and eating time, and inhibited food intake.

2.5. Weingarten's model

If not the first, then likely the most influential demonstration of cue-potentiated feeding was published in *Science* by Harvey Weingarten in 1983, marking the first of four studies on the effect over a 6-year period. The first study (Weingarten, 1983) reported two experiments in which individually housed rats received all training and testing in the home cage. Rats were fed six daily liquid meals that were each preceded by a 270-s conditional stimulus (CS) consisting of a light and buzzer compound cue; CS+, with a CS- (tone) presented between meals. The liquid meals were delivered into a food-cup during the last 30-s of the cue and were rats' sole food source, providing a total of 70 % of *ad-libitum* intake (thus, training occurred in a state of hunger). Latency to approach the food-cup upon CS+fell rapidly to 5-s over the 11-day training phase (Weingarten, 1983, Fig. 1A). During the subsequent 21-day test phase rats had free access to the liquid diet from a bottle within the cage, with CS+and CS- cues presented once daily to measure approach to the food-cup and consumption. Despite free access to the liquid diet via the bottle, rats continued to reliably approach the food-cup within 5-s of CS+onset, with time exploring the food-cup and consumption both comparable to training. Presentations of the CS- had no such effects (Weingarten, 1983, Fig. 1B). Demonstrating that the CS+initiated consumption at the food cup despite unrestricted access to the same food from another source (the bottle) was a departure from previous designs that had tested effects of food cues after a discrete satiation phase. Training and testing in the home-cage removed the need for context shifts and the necessary handling that had been shown to contribute to conditioning effects (Valle, 1968).

Experiment 2 examined the contribution of cue-induced feeding to overall energy intake, an important advance from comparisons within a single test or against a given control. After an identical training phase, rats were again allowed unrestricted access to the liquid diet from a bottle inserted into the cage. On some days, a single presentation of the CS+was followed by delivery of a 15-ml meal to the food-cup, stimulating around 20 % of rats' total daily energy intake. However, rats compensated for cued meals by consuming less at other times, such that total energy intake was no different on days with versus without a CS+presentation (Weingarten, 1983, Fig. 2). Weingarten suggested that although his animals compensated for cued meals, in other situations "*…persistent responding to conditioned cues would result in positive energy balance and obesity.*" (Weingarten, 1983, p. 432).

The effect was further characterised in a series of experiments using the same training protocol published the following year (Weingarten, 1984a). Experiment 1 (actually the same experiment as Weingarten [1983]), showed that the magnitude of cue-induced feeding persisted across 21 test days, despite free access to food and the fact that CS- tests were also reinforced (rats often ate this meal, but well after the CS-played). Subsequent experiments showed that the CS+elicited meals of

comparable size to spontaneous meals from the *ad-lib* bottle (Experiment 2), even if the food cup contained stale milk from the previous day (Experiment 3). Experiment 4 showed that rats compensated for cue-elicited meals regardless of whether the CS+was played once or five times per day. In the latter case meals triggered by the CS+comprised 50 % of daily energy intake, though not every cue presentation prompted intake (Weingarten, 1984a; Fig. 6).

The third study examined whether cue-induced feeding was robust to pharmacological interventions. Injections of the cholinergic antagonist, atropine methyl nitrate, did not alter latency to eat or amount eaten in response to the CS+ (Weingarten, 1984b, Figure 2), whereas CCK injections reduced meal size elicited by the cue and the time spent eating, but not anticipatory food-cup behaviour or the latency to eat (Weingarten, 1984b; Figure 3), thus, broadly consistent with the effects of CCK reported by Schallert et al. (1982) and later studies of CCK in instrumental responding (Balleine, Davies, & Dickinson, 1995).

In his fourth study, Weingarten and Martin (1989) tested whether various interventions could dissociate the anticipatory and consummatory aspects of CPF. Conditioning an aversion to the liquid milk diet (via pairings with lithium chloride injection to induce nausea) reduced meal size but did not alter conditioned approach to the food-cup (Weingarten & Martin, 1989, Figure 2 and Table 1). The dopamine antagonist α-flupentixol blunted anticipatory food-cup activity, but had no effect on meal size; conversely, the opioid antagonist naloxone reduced meal size, but not food-cup activity (Weingarten & Martin, 1989, Table 2 and Table 3), consistent with subsequent reports implicating dopamine in incentive motivation processes and opiates in the hedonic processing of rewards (Berridge, Robinson, & Aldridge, 2009). Both anticipatory and consummatory behaviour were blunted by oral gavage of the liquid diet, suggesting that satiety more completely suppressed behaviour than either of the pharmacological treatments (Weingarten & Martin, 1989, Table 4). Weingarten's research directly inspired at least one study of CPF in humans; Birch, McPhee, Sullivan, and Johnson (1989) found that contexts paired with food enhanced consumption in preschool children. In rats, however, there was little further published research on cue-potentiated feeding until the late 1990 s.

2.6. Food cues in energy balance theory

The studies of cue-potentiated feeding described thus far formed part of a larger evidence base that challenged prevailing theories of food intake and weight regulation in the 20th century. These 'set-point' or 'depletion' models proposed that short-term food intake and long-term body weight regulation were homeostatically regulated and tied to circulating glucose levels and body fat content, respectively (Mayer, 1955; le Magnen, 1981). Kennedy (1953) hypothesised that body weight was regulated by a negative feedback loop controlled by the hypothalamus, which mediated changes in energy intake or expenditure in response to signals from adipose tissue. Around this time, researchers had observed hyperphagia and obesity following lesions to the ventromedial sub-region (Kennedy, 1950; Corbit & Stellar, 1964), or electrochemical stimulation of the lateral hypothalamus (e.g. Epstein, 1960; Grossman, 1960), and complete inanition following lesions to the lateral hypothalamus (Anand and Brobeck, 1951). The discovery of leptin in 1994 (Zhang et al., 1994) realised this prediction of set-point models, with much subsequent work in this space (for review, see Speakman et al., 2011).

Nonetheless, purely homeostatic models could not account for the many, varied circumstances in which rats or humans could be prompted to eat (Kanarek, 1981; Toates, 1981) with reports that manipulating blood glucose did not induce meals under certain conditions (Friedman, 1981; Davis, 1981; Woods & Ramsay, 2000) and that predictions borne out in food-deprived animals did not hold in those fed *ad-libitum* (Weingarten, 1985). Together, this suggested that homeostatic mechanisms were one of several interacting determinants of eating behaviour and directed attention towards external factors.

An influential theory of internal and external cues was developed by Stanley Schachter in the 1960s. Schachter proposed that obesity was characterised by a reliance on external cues to initiate eating, resulting from an inability to sense internal signals of hunger and satiety. Schachter and colleagues devised experiments in which normal-weight participants reduced their food intake (often of crackers and under the guise of taste tests) after various manipulations, such as pre-feeding with sandwiches, threat of an inescapable shock, or manipulating a clock to run fast to deceive participants that dinnertime was near (Schachter, 1968). In each case, consumption by participants with obesity was either unaffected by the manipulation, or, in the latter clock study, *increased*. Schachter reported similar findings across a range of observational, situational and experimental studies (for review, see Schachter, 1971).

Critics of Schachter's theory argued that internal and external control over eating was not a dichotomy and that sensitivity to one form of cue over the other was not determined only by weight (Rodin, 1981). For example, Meyers and Stunkard (1980) reported that body weight did not alter the influence of external cues on dessert choices in an observational setting at a hospital cafeteria. Some of Schachter's students modified his hypothesis to distinguish between types of external cues (see Herman & Polivy, 2008, for review) and several theories proposed that eating was best explained by interactions between internal and external cues (e.g., Toates, 1981). With limited work on cue potentiated feeding at this time, discussion of learning and conditioning processes often centred on flavour-nutrient learning, conditioned satiety and sensory-specific satiety (Bellisle, 1979; Booth, 1981; Rolls et al., 1981). Weingarten proposed a 'two-factor' theory involving a slow-onset, nonspecific *internal* hunger induced by energy depletion and an *external* hunger triggered by conditioned cues which rapidly provoked a desire for a specific food (Weingarten, 1985). This distinction is analogous to conceptualisations of homeostatic versus hedonic eating systems (e.g. Saper, Chou, & Elmquist, 2002) and distinct 'metabolic' versus 'cognitive' brain systems thought to underlie them (Berthoud, 2007, 2012).

Interest in external cues and food intake has grown as many observe that the recent dramatic rise in the global prevalence of obesity points to environmental rather than genetic causes. The term 'obesogenic environment' was coined in 1999 to describe the myriad ways in which technological, social and environmental features of modern societies promote energy intake and reduce energy expenditure (Swinburn et al., 1999). A key feature of obesogenic environments is ready access to palatable, energy-dense foods signalled by salient cues (Hetherington, 2007). This has been conceptualised as an evolutionary mismatch wherein easy access to food and food cues overwhelms brain circuits evolved to attend to signals for food in times of scarcity (Berthoud, 2007). Indeed, meta-analyses have identified that food cue reactivity prospectively predicts eating behaviour (Boswell & Kober, 2016).

Recent theories ascribe slightly different roles for external cues. In their 'boundary model', Herman and Polivy (1984, 2005) propose that external cues are the primary determinants of food intake, aside from instances of complete satiation or significant hunger (i.e., the boundaries). A similar theory suggested that environmental cues only influence body weight within a 'settling zone' determined by biology (Levitsky, 2005). Other accounts propose that responding to food cues established through Pavlovian conditioning is an adaptive process that helps organisms prepare for meals (Pavlov, 1927; Woods & Ramsay, 2000). De Castro (1997, 2010) has argued that environmental cues are the primary determinant of food intake, and that physiological mechanisms exert subtle effects that are evident only over the longer term. Davidson and colleagues suggest that food cues act within a vicious cycle of obesity and cognitive decline mediated by the hippocampus. In their model, food cues prompt consumption of palatable but ultimately unhealthy foods that promote obesity and impair hippocampal function; in turn, individuals are less able to inhibit responding to food cues and overeat further (Davidson, Kanoski, Walls, & Jarrard, 2005; Davidson, Sample, & Swithers, 2014; see also Parent, 2016). Consistent with this

idea, work in rats indicates a bidirectional relationship between diet-induced obesity and the motivation to work for palatable food (e.g. la Fleur et al., 2007; Robinson et al., 2015) and food cues can similarly promote consumption and operant responding for food in humans with these effects being heightened in individuals with overweight or obesity (Kanoski & Boutelle, 2022). In sum, it is clear that external food cues feature prominently in many contemporary theories of food intake, especially in relation to the risk of obesity.

2.7. *Neural substrates of CPF*

A productive body of research in the early 2000 s, led by Peter Holland, Michela Gallagher and Gorica Petrovich, identified neural circuits underpinning cue potentiated feeding (for review, see Petrovich, 2013). The experimental procedure typically used by these researchers resembled Weingarten's method in that rats were trained hungry and tested satiated. In this model, food-deprived rats undergo daily training sessions held in conditioning chambers (not in the home-cage as in Weingarten's experiments). Pavlovian training usually begins with two sessions in which 10-s presentations of an auditory cue (conditional stimulus; CS, most often a tone or white noise) terminate with delivery of two 45-mg food pellets to establish this cue as a CS+. Subsequently, rats undergo ~ 10 days of discrimination training involving intermixed presentations of the CS+and non-reinforced presentations of an auditory CS- cue. Most studies use between-subjects designs wherein a single cue is rewarded for a *Paired* group, with an *Unpaired* group given equivalent, non-contingent presentations of the cue and pellets.

After training rats receive unrestricted access to chow in the home cage for 7–14 days to return body weight to baseline levels, with additional acute pre-exposure to the reward pellets, prior to the CPF tests. Pre-feeding often involves placement in the test chamber for 5–10 min with an ample supply of pellets in the food-cup. In some studies, this pre-feeding procedure is repeated; in others, rats are pre-fed in home cages. Rats are ultimately returned to the chamber where a large number of pellets are now available for a 5–10 min test typically involving ten 10-s presentations of the cue. Cue-potentiated feeding is demonstrated by higher consumption in the CS+test relative to the CS- test (in within-subjects designs) or relative to rats given unpaired training (in between-subjects designs). In contrast to Weingarten's model, which showed that the CS+triggered spontaneous meals in free-feeding rats, this method provides stricter control over food intake immediately prior to the test to show that cues prolong or extend consumption despite ample pre-feeding. This is reflected in terminology: whereas Weingarten referred to meals elicited or *induced* by cues, the effect is often described as cue-*potentiated* feeding by Petrovich and colleagues.

2.8. *Cue potentiated feeding: Neurocircuitry*

Holland, Petrovich and Gallagher (2002) used this method to show that CPF was abolished by lesions to the basolateral (BLA) but not central nucleus (CeA) of the amygdala, with no differences in training performance or pre-feeding between BLA-, CeA- and sham-lesion groups. Sham- and CeA-lesioned groups showed CPF both when pellets were presented in the trained location (food-cup) and in a bowl on the other side of the chamber, indicating that CPF was not driven by conditioned approach behaviour (Holland et al., 2002, Fig. 2). Several other studies found CPF to be impaired by BLA lesions (Holland, Hatfield, & Gallagher, 2001; Galarce, McDannald, & Holland, 2010) and unaffected by CeA lesions (Holland & Hsu, 2014). Notably, one of these revealed a double dissociation between BLA versus CeA lesions on CPF and Pavlovian-to-Instrumental Transfer (PIT) tests, showing that while BLA but not CeA lesions abolished CPF, CeA but not BLA lesions abolished the elevation in instrumental responding produced by the CS+ (i.e., single stimulus PIT; Holland & Gallagher, 2003, Fig. 3).

Petrovich, Setlow, Holland, and Gallagher (2002) demonstrated that the role of the BLA in CPF depended on its connections with the lateral hypothalamus (LHA). Rats received lesions to the BLA and LHA in contralateral or ipsilateral hemispheres. Because LHA-BLA connections are mostly ipsilateral, contralateral lesions abolished functional connectivity between the two structures whereas ipsilateral lesions retained it in one hemisphere, while equating total tissue damage. Rats with ipsilateral or sham lesions showed significantly greater intake in the presence of the CS+, whereas those with contralateral lesions ate similarly low amounts in CS+and CS- tests (Petrovich et al., 2002, Fig. 3). However, groups did not differ in a second-order conditioning task where a new (visual) cue was followed by presentations of the original (auditory) CS+, suggesting that BLA-LHA disconnection blocked the ability of the CS+to promote eating but not to permit new learning (Petrovich et al., 2002).

Subsequent studies characterised the role of cortical regions in CPF. Petrovich, Holland, and Gallagher (2005) injected the retrograde tracer FluoroGold into the lateral hypothalamus on the day after standard Pavlovian training. Food intake by sated rats during the CS+and CS- was measured in two 5-min tests held 25-min apart on the same day (order counterbalanced) to align with maximum mRNA induction of two immediate-early genes, H1a and Arc, respectively. Greater intake in the CS+than CS- test was coupled with co-expression of FluoroGold and increased expression of H1a and Arc related to the CS+in the basolateral/basomedial amygdala and orbitomedial frontal cortex, indicating that projections from these frontal regions to the LHA were activated during the expression of CPF (Petrovich, Holland & Gallagher, 2005, Fig. 2 and Fig. 5).

Two other studies examined the role of the frontal cortex in CPF. The first by McDannald, Saddoris, Gallagher and Holland (2005) showed that CPF was intact in rats given bilateral lesions of the lateral orbitofrontal cortex (OFC). However, OFC-lesioned rats were poorer in a differential outcome-expectancy task that tested lever pressing for distinct outcomes during various discriminative stimuli. Poorer performance by OFC-lesioned rats on this task suggested that CPF did not require the retrieval of specific outcome representations (McDannald, Saddoris, et al., 2005), a result with implications for the specificity of cue-potentiated feeding. Whereas McDannald, Saddoris and colleagues targeted the lateral OFC, another study found that lesions to the ventromedial prefrontal cortex that incorporated the *medial* OFC abolished CPF when a context (rather than discrete cue) was paired with food (Petrovich, Ross, Holland, & Gallagher, 2007a).

2.9. *Cue potentiated feeding: Neuropharmacology*

Many peptides involved in the regulation of eating have been shown to influence the control of eating by learned cues. The peptide orexin/hypocretin is produced within the lateral hypothalamus (LH) and appears important for the expression of CPF, as indicated by a study in which systemic injections of an orexin antagonist blocked the effect in rats (Cole, Mayer, & Petrovich, 2015). Another study found that presentations of a conditioned food cue induced neuronal activation in orexin neurons within the LH (Petrovich, Hobin, & Reppucci, 2012). The same study found no effects on LH melanin-concentrating hormone (MCH) neurons, a result which contrasts two other studies reporting that deletion of the MCH-1 receptor blocked CPF in mice (Johnson, 2011; Sherwood, Holland, Adamantidis, & Johnson, 2015). The effects of orexin depend, at least in part, on signalling within the medial prefrontal cortex (mPFC) as disconnection of the LH and mPFC, or orexin 1 receptor antagonism within the mPFC each abolished CPF (Cole, Keefer, Anderson & Petrovich, 2020).

Several studies have assessed how CPF is moderated by ghrelin, an orexigenic peptide secreted from the stomach. One study adapted Weingarten's (1983) original protocol to show that ghrelin microinjections to the ventral hippocampus enhanced the number of meals induced by a food-paired CS+relative to a CS- paired with no food (Kanoski, Fortin, Ricks, & Grill, 2013). Notably, CPF was not observed following vehicle injections, suggesting that the cue exerted only weak effects on

eating under baseline conditions (Kanoski et al., 2013, Fig. 4). Walker, Ibia and Zigman (2012) showed that pharmacological antagonism of ghrelin blocked CPF in C57BL/6J mice, and that ghrelin receptor knockout mice (growth hormone secretagogue receptor [GHSR] −null) increased food intake during presentations of a CS- in addition to the CS+, an effect interpreted as an inability to discriminate between cues. Peripheral administration of a ghrelin antagonist had no effect on CPF as measured by consumption, but delayed the onset of eating in response to the CS+relative to vehicle-treated rats (Dailey, Moran, Holland, & Johnson, 2016). Finally, peripheral ghrelin administration treatment immediately prior to context-food pairings in sated mice was insufficient to recapitulate a CPF effect in C57Bl/6J mice (Reed et al., 2023).

CPF has also been demonstrated in preparations where contextual cues, rather than discrete stimuli, are paired with food. These studies typically present food in a chamber adorned with distinct olfactory, tactile and/or visual cues. In some instances, this training is intermixed with exposure to a second distinct context containing no food. Recent work has used contextual cues to reveal new circuitries involved in the learning of cue-food associations and potentiated feeding effects. A study in mice showed that a single exposure to a context containing food in a distinct location activated dopamine D2 receptor-expressing neurons in the hippocampus, and that projections from this population of neurons to the septal area mediated the encoding of the food-place association (Azevedo et al., 2019). Using a brief training protocol involving two 30-min exposures to a food-paired context, another study found that sated mice increased food intake in this context relative to another context not paired with food, coupled with increased neuronal activation in the lateral hypothalamus, central amygdala, insular cortex and lateral septum (Stern et al., 2020). The CPF effect produced with this rapid training protocol was blocked by inactivating the insular cortex with the GABA agonists muscimol and baclofen, and was not observed in mice trained without food-deprivation or given massed training (Stern et al., 2020). Stern and colleagues subsequently showed that the CPF effect produced using this protocol relied on a subpopulation of nitric oxide synthase-1-expressing neurons projecting from the insular cortex to the central amygdala (Stern et al., 2021).

A recent study adapted Stern and colleagues' protocol to reveal a key role of hypothalamic agouti-related peptide (AgRP) neurons in CPF (Reed et al., 2023). After demonstrating that the activity of AgRP neurons was sensitive to placement in the food-paired versus unpaired context, subsequent experiments showed that optogenetic inhibition of AgRP neurons in hungry mice during training was sufficient to promote a CPF effect in sated mice. Notably, optogenetic inhibition of AgRP activation in the context was sufficient to stimulate increased eating at test even when no food was provided during training sessions. Altogether, these data indicated that the acute suppression of AgRP neuronal activity in the context was necessary and sufficient to elicit increased eating in this environment. This evidence demonstrates that the expression of cue-potentiated feeding is sensitive to diverse neural circuits, including the amygdala, hypothalamus, hippocampus and insular cortex. In addition to altering total intake, many of these studies demonstrate more subtle effects of circuitry or pharmacological manipulations on the distribution of meal patterns. While both discrete stimuli and contextual cues are able to induce CPF, few if any studies have compared methods directly and as such, it remains to be determined whether these involve distinct or common mechanisms.

3. Future directions to improve translation

An important question for understanding the extent to which food cues contribute to overeating and translate to human obesity is whether their effects are specific or general. That is, do cues prompt consumption only of the food they have previously signalled or of a wider range of foods? Studies in people generally show specific effects following exposure to olfactory or taste cues. For example, participants given a morsel of pizza or ice cream selectively increased consumption only of that food when subsequently offered a choice between the two (Cornell, Rodin & Weingarten, 1989) and the smell of pizza or cookies selectively increased consumption of that food in a study of restrained eaters (Federoff, Polivy, & Herman, 2003). However, Ferriday & Brunstrom (2008, 2011) have found that an olfactory pizza cue increased the desire to eat and the anticipated portion sizes of other savoury foods as well as pizza, but not sweet foods, suggesting that effects may transfer to similar-tasting foods.

Most work in rodents has found that the effect of conditioned audiovisual or contextual cues on intake is *selective* or *specific* to the paired food. Few early experiments tested this since the paired food in training was often rats' maintenance diet (e.g. Zamble, 1973; Weingarten, 1983). Petrovich and colleagues (2007a, 2007b) showed that a context paired with pellets increased intake of those pellets but not a novel pellet formula or home-cage chow, relative to an unpaired control group. Whereas these studies examined the different foods across several tests, reinforcer-specific effects of cues have also been demonstrated in experiments where the paired pellets and home-cage chow are available simultaneously at test (Petrovich et al., 2012). A similar study found that a food-paired cue enhanced pellet but not chow consumption over a longer 4-h test, but only when rats were tested satiated and not food-deprived (Reppucci & Petrovich, 2012). This latter effect may have been due to a ceiling effect when consumption was high in both groups. Experiment 2 in this paper showed that the CPF effect persisted for the first two of four tests over a 2-week period but that increased intake by the Unpaired group occluded any effect on latter tests.

While these studies suggest that conditioned food cues do not increase intake of less palatable alternatives like chow, one study found that a context previously paired with Oreos increased intake of chow over a 24-h period relative to a context paired with chow, and that providing a morsel of Oreo (2 g) enhanced this effect (Boggiano, Dorsey, Thomas, and Murdaugh, 2009). However, as chow was provided in the Oreo-paired cage during training, the context had the opportunity to also become associated with chow, making it difficult to conclude whether the overeating of chow constitutes a non-specific CPF effect.

Evidence for the specificity of CPF also comes from studies pairing separate auditory cues with sucrose and maltodextrin solutions, where rewards are delivered intermittently during longer CS presentations (typically 2-min). Studies using this method have shown that consumption of each reward is enhanced only by its predictive CS and not by the CS that predicts the alternative reward (Galarce, Crombag, & Holland, 2007; Delamater & Holland, 2008; Galarce & Holland, 2009; Holland, 2014; Holland & Hsu, 2014). The specificity of CPF in these experiments mirrors effects on instrumental responding found in studies of Pavlovian-instrumental transfer (PIT). In PIT the primary outcome measure is performance of an instrumental response (e.g., lever press) rather than consumption, however, these findings are related as in many situations an animal will have to work to procure food before they can consume it. In such experiments animals are trained to perform two instrumental responses to earn two rewards, and, in a separate phase, undergo training where two CS are paired with each of those rewards. At test, the responses are available, and for the first time, the stimuli are periodically presented and the impact on instrumental responding assessed. What is typically observed is that presentation of a Pavlovian cue increases responding only of the action earning the same reward; i.e., specific PIT (e.g. Corbit & Balleine, 2005). The specificity of PIT seems to rely on animals being trained with two actions earning distinct rewards; when animals are trained with two stimuli predicting different rewards, but instrumental training with only one reward, both stimuli invigorate responding (Holland, 2004). It is not known whether similar effects might be found with CPF. For example, cues may first direct choice toward the associated food when it is available (and presumably increase its consumption). Additionally, presenting a cue paired with an outcome not used as a reward in instrumental training can produce a general elevation in responding (Corbit & Balleine, 2005). A parallel effect in CPF might arise if only a different food is available, cues might

just enhance consumption (analogous to general PIT; Corbit & Balleine, 2005; Holland, 2004). While these ideas remain to be tested, it is clear that the associative history of a food can influence the circumstances in which animals will work for and consume it in the presence of other cues.

Contextual food cues in day-to-day life may become associated with multiple tastes and flavours. To test the effects of variety on CPF, we trained rats to associate a distinct 'Plus' context with either one palatable food (Single group) or multiple palatable foods across days (Variety group) alongside exposures to a second 'Minus' context containing no food (or chow; Kendig, Boakes & Corbit, 2018). Notably, we found that when testing novel palatable foods not used during training, the Plus context increased intake in the Variety, but not in the Single group. Thus, a cue trained with a variety of foods led to increased consumption of other foods. Together, this evidence indicates that cues consistently paired with a single food selectively influence consumption only of that food, whereas effects may generalise to other foods for cues paired with a variety of tastes and textures. While more work is needed to study food cues paired with variety, one implication is that environments containing an abundant variety of food options may be more likely to provoke overeating.

3.1. Does CPF increase the risk of obesity over the long-term?

As global obesity rates soar, the idea that environmental factors contribute to this epidemic is appealing because food cues are ubiquitous and examples of the temptation they produce are easy to imagine. Thus, while it is known that food-paired stimuli can increase acute food intake in rodents and humans (Petrovich, 2013; Boswell & Kober, 2016), an important question is to what extent individuals compensate for cue-triggered snacks or meals, and the consequences for body weight. CPF and overeating may be dissociable to the extent that some individuals may be able to fully compensate for meals elicited by cues so as to maintain body weight. However, even very small increases in daily intake may increase long-term weight gain (Hill, Wyatt, Reed, & Peters, 2003; Hall et al. 2011) and compensation is likely to be challenging given that advertisements for food overwhelmingly signal palatable, energy-dense foods (see Levitsky, 2005). Indeed, Sun and colleagues (2015) found that neural activity in the amygdala in response to the taste of a palatable milkshake when sated, but not hungry, predicted prospective weight gain a year later in healthy adults, but only for those not carrying an A1 copy of the Taq1A polymorphism.

People learn about and encounter food cues across a range of hunger states in everyday life. Accordingly, the form of food intake stimulated by cues might vary widely (e.g., snacks versus meals). The research reviewed here shows that most studies have modelled CPF using designs where animals are trained hungry and tested sated, with some evidence that food-deprivation is necessary to evoke effects (Stern et al., 2020; Reed et al., 2023). There is scope to explore how hunger and satiety mediate the effects of food cue exposure on choice and intake.

Further, as many CPF effects are modest in magnitude, it is possible that a ceiling effect may prevent detection of CPF in situations where consumption is already high. For example, a recent study showed that diet-induced obese mice (12 weeks' access to a purified high-fat diet) failed to exhibit CPF (Lewis-Sanders et al., 2024). It may, however, still be the case that learned cues still direct what is eaten and when eating takes place, which could be at odds with dieting attempts.

3.2. How does CPF relate to the length of training?

It is not known at what point cues acquire the ability to potentiate eating and how this changes over extended training. This is likely to depend on deprivation state and palatability of the food reward. Recent studies indicate that CPF effects can be produced after as few as two conditioning trials (Stern et al., 2020; 2021). There is evidence for one-trial learning in aversive paradigms (e.g., Fanselow, 1990) and some evidence from appetitive procedures (e.g. Parkes et al., 2014). Nonetheless, most rodent CPF experiments have used closer to 20 training sessions, with some exceptions, for example, 50 exposures to food-paired contexts (Lovibond, 1980). A study in people found that pairing chocolate consumption with a specific time of day enhanced desires to eat at that time after 15 but not 5 conditioning sessions (van den Akker, Havermans, & Jansen, 2017).

Much is known about how instrumental and Pavlovian conditioning are affected by extended training. Goal-directed control of an instrumental response is reduced with extended training (Adams, 1982; Lingawi & Balleine, 2012), by access to high-fat, high-sugar diets (Furlong, Jayaweera, Balleine, & Corbit, 2014; Kendig, Boakes, Rooney, & Corbit, 2013) and is acutely impaired in environments previously paired with palatable food (Kendig, Cheung, Raymond, & Corbit, 2016). This suggests the interesting possibility that the specificity of CPF might wane over extended training. Related studies of Pavlovian-instrumental transfer (PIT) have found that the magnitude of the PIT effect grows with extended training (Holland, 2004; Corbit & Janak, 2016) although, Pavlovian cues retain the ability to selectively increase performance of instrumental responses which earn the same outcome even after extended training (Holland, 2004). A notable exception is where rats are trained that two cues predict two separate foods, but are only trained to perform a single instrumental action. Under these conditions, presentations of a cue paired with either the same, or a different food as earned by the lever increased responding (Holland, 2004), which may be attributed to a general form of PIT (Corbit & Balleine, 2005). As with the other training conditions, the invigorating effects of both cues grew in magnitude with extended training (Holland, 2004). Therefore, a parametric analysis manipulating the amount of training would be highly interesting in terms of the size and specificity of CPF. Consideration of the impact of training cues with a variety of foods might also yield important insights, as will testing effects of varying CS-US contingencies on CPF. For example, Derman and Ferrario (2018) showed that a partially-reinforced CS (50% CS-US contingency) produced a CPF effect, whereas a fully predictive CS (100% CS-US contingency) did not. Given PIT, CPF and sensitivity to devaluation can feasibly be tested within the same experiment (e.g., Delamater & Holland, 2008), comparisons between these tasks would also be informative. For example, do animals that exhibit the strongest CPF effect also show enhanced PIT (for review see Gladding et al., 2023; Kanoski & Boutelle, 2022), or impaired sensitivity to devaluation? This might inform how distinct incentive versus general motivational processes interact to produce CPF. More broadly, studying CPF across extended training will be informative for understanding its relevance to overeating and obesity, since food-cue associations are formed and strengthened across months and years in humans.

3.3. Effect size

CPF is often tested in animals with free access to food in the home cage given additional pre-feeding of the test food. Effects are thus working against general and sensory-specific satiety, with the latter effect shown to persist up to five hours after pre-feeding (Parkes, Marchand, Ferreira, & Coutureau, 2016). The palatability of test foods means there is little disincentive for animals *not* to eat when in control conditions or in the presence of a control cue. While these factors mean that effect sizes are often small, incremental increases in energy intake produced by food cues may foster long-term weight gain if organisms do not compensate adequately at other times. Additionally, training conditions that do not produce large effects may be opportune for detecting effects of interventions that enhance eating in response to external cues (e.g. Kanoski et al., 2013). Nonetheless, acute effects in a single or small number of tests are limited in their ability to explain things like overeating and obesity, and as noted above, longer-term studies examining more chronic effects of cue exposure on eating are likely to have more translational potential.

3.4. Consistency of effects across subpopulations

Finally, it will be important to explore how CPF effects vary across development, specific populations and disease states, since most studies have looked at effects within healthy adult animals. It will also be important to test for potential sex differences, given that males have historically been used over five times as often as females in most biological science research (Beery and Zucker, 2011) and increasing appreciation of the importance of including sex in future studies as a biological variable in both preclinical and clinical research (Miller et al., 2017). Indeed, of the rodent CPF studies reviewed here, less than 10 % included females (4/45 studies: Boggiano et al., 2009; Kendig et al., 2018; Cole et al., 2020; Lewis-Sanders et al., 2024). Finally, future work should explore how CPF effects are sensitive to acute and chronic stress, which have demonstrated effects on food intake (see Razzoli et al., 2017).

4. Conclusions

A substantial literature demonstrates that environmental stimuli can increase eating, even in the absence of hunger. Evidence has been found from multiple species and using a range of different stimuli (social, contextual, discrete cues) indicating that CPF is a widespread phenomenon. Nonetheless, there is still much to learn about CPF. Most notably, a better understanding of when, and to what degree CPF promotes overeating and potential weight gain in humans will be critical for assessing its translational utility. While preclinical CPF effects appear subtle and sensitive to experimental parameters, similar effects have been observed in humans, and even small energy surplus, if repeated over time, could lead to weight gain and so the small acute effects should not be taken as grounds for dismissing the model. Identifying how acute intake triggered by food cue exposure contributes to energy intake over the longer-term will be important in this regard. Against a global backdrop of high rates of metabolic disease, understanding the factors that initiate eating and direct choices about what is consumed is essential to refine strategies to limit overconsumption and promote healthy eating.

CRediT authorship contribution statement

Michael D. Kendig: Writing – review & editing, Writing – original draft, Methodology, Formal analysis, Conceptualization. **Laura H Corbit:** Writing – review & editing, Writing – original draft, Methodology, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

No data was used for the research described in the article.

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