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Implications of learning theory for developing programs to decrease overeating

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Abstract

Childhood obesity is associated with medical and psychological comorbidities, and interventions targeting overeating could be pragmatic and have a significant impact on weight. Calorically dense foods are easily available, variable, and tasty which allows for effective opportunities to learn to associate behaviors and cues in the environment with food through fundamental conditioning processes, resulting in measurable psychological and physiological food cue reactivity in vulnerable children. Basic research suggests that initial learning is difficult to erase, and that it is vulnerable to a number of phenomena that will allow the original learning to re-emerge after it is suppressed or replaced. These processes may help explain why it may be difficult to change food cue reactivity and overeating over the long term. Extinction theory may be used to develop effective cue-exposure treatments to decrease food cue reactivity through inhibitory learning, although these processes are complex and require an integral understanding of the theory and individual differences. Additionally, learning theory can be used to develop other interventions that may prove to be useful. Through an integration of learning theory, basic and translational research, it may be possible to develop interventions that can decrease the urges to overeat, and improve the weight status of children.

Keywords

child; obesity; learning; food cue; extinction

Obesity is a serious and refractory problem that is associated with multiple medical and psychological comorbidities and risks. Recent data suggest that 4–5 million children are overweight or obese, representing 33% of children ages 6–19 years in the United States (Ogden, Carroll, Kit, & Flegal, 2014). Overweight and obese children are at an increased

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risk for many negative health complications in childhood and adulthood, including orthopedic and endocrine conditions, cardiovascular disease, cancer and all-cause mortality (Dietz, 1998; Dixon, 2010; Flegal & Kalantar-Zadeh, 2013; Key et al., 2004; Micic, 2001; Must et al., 1999). Obesity tracks well from childhood to adulthood, as 80% of overweight youth will be overweight as adults (Guo, Roche, Chumlea, Gardner, & Siervogel, 1994; Guo, Wu, Chumlea, & Roche, 2002). As a consequence of rising obesity rates, obesity-related medical costs in the United States are expected to increase by \$48–66 billion per year by the year 2030 (Wang, McPherson, Marsh, Gortmaker, & Brown, 2011). Considering the high prevalence rates, escalating medical costs, and significant health consequences, it is of utmost importance that more potent models are developed to intervene on behaviors that contribute to obesity.

Family-based behavioral treatment (FBT) is considered the standard weight loss treatment program for overweight and obese children. FBT for childhood obesity is provided to parents and the target child, and includes nutrition and physical activity education, in addition to behavior therapy strategies and parent management skills (Epstein, 1996). The behavior therapy component incorporates techniques to assist the children and their parents in changing the energy balance equation, including goal-setting, self-monitoring food intake and activity, stimulus control, problem solving, cognitive restructuring and relapse prevention. Reviews suggest that interventions such as FBT can result in weight reduction for overweight and obese children (Altman & Wilfley, 2014; Ho et al., 2012; Whitlock, O'Connor, Williams, Beil, & Lutz, 2010). Additionally, sustained weight loss is observed in a proportion of children completing FBT, with 1/3 of children no longer being overweight in adulthood (Epstein, Valoski, Wing, & McCurley, 1994). However two thirds of children do not respond over the long-term. This lack of response could be due to underlying mechanisms that remain unaddressed by FBT, such as the retention of previously-learned behavior patterns. We believe that a more integrated understanding of learning theory and how it relates to appetite and overeating could allow for the development of effective interventions that target the etiological and maintenance mechanisms that drive overeating.

Societal advances have created the “obesogenic” environment (Brownell, 2004; Kessler, 2009; Lowe, 2003) that encourages excess energy intake (Berthoud, 2004). Calorically dense foods are easily available, highly variable, tasty, relatively inexpensive and portable (Drewnowski, 2004, 2010; Duffey & Popkin, 2013; Nielsen & Popkin, 2003; Popkin, Duffey, & Gordon-Larsen, 2005). Excessive caloric intake is one of the most proximal causes of rising obesity rates during the past three decades (Jeffery & Harnack, 2007; Swinburn et al., 2009). Overweight and obese children, compared to healthy weight children, have higher levels of eating in the absence of hunger in the laboratory (Fisher & Birch, 2002) and at home (Hill et al., 2008; Moens & Braet, 2007). Thus, from a pragmatic intervention perspective, in terms of weight loss, it could be easier to focus on reduction of caloric intake, especially when sated, than it is to exercise enough to burn off the extra calories.

Unfortunately, neurophysiological and higher order cognitive systems do not effectively decrease food intake under conditions of food abundance (Ahima et al., 1996). The motivation to eat is not only influenced by the basic internal, physiological signals from the

body, such as perceptions of hunger or hormone systems, but also by other factors that are not directly related to nutrient homeostasis, including sensory experiences, cravings, reward expectations, emotional and social factors (Berthoud, 2004, 2007; Grill & Kaplan, 2002; Lutter & Nestler, 2009; Nogueiras et al., 2012; Saper, Chou, & Elmquist, 2002; Schachter, 1968; Schwartz, Woods, Porte, Seeley, & Baskin, 2000; Small, 2009). The stronger the non-homeostatic influences, the harder it is to maintain body weight. Eating beyond energy requirements, i.e. overeating, is believed to drive the obesity epidemic to a much greater extent than metabolic changes (Berthoud, 2011; Small, 2009; Volkow & Wise, 2005). Recent estimates suggest that a moderate reduction of only 220 calories per day could sufficiently result in successful weight loss over time (Hall et al., 2011).

Learning opportunities in the obesogenic environment

Energy intake in humans is a reflection of homeostatic, hedonic and cognitive mechanisms (Hall, Hammond, Rahmandad, 2014). Hedonic eating, which depends on basic learning processes, is a significant contributor to eating past physiological needs (Petrovich & Gallaher, 2007; Berridge, Ho, Richard, DiFeliceantonio, 2010). Today's food environment is full of effective opportunities to overeat through associations of cues in the environment with food. Pavlovian conditioning occurs when a neutral stimulus (e.g., a couch) is paired with a biologically significant stimulus (i.e. food), causing the neutral stimulus to acquire eliciting functions (e.g. salivation and/or cravings when sitting on the couch). Such "food cues" are thought to become directly associated with the food outcome (a cue-food association) and elicit the kinds of responses discussed above. However, today's food environment is also full of opportunities to learn to associate eating or food-seeking actions with the reinforcing effects of eating (operant action-outcome learning). Such operant learning can also play a role in increasing consumption in an obesogenic environment (e.g., Bouton, 2011). It is important to recognize that these two learning processes act in concert: Pavlovian and operant conditioning are constantly occurring at the same time (e.g., see Bouton, 2009 for one overview). In addition to eliciting the many kinds of responses described above, the presentation of Pavlovian food cues can increase the rate or vigor of operant eating or food-seeking through a process known as "Pavlovian-Instrumental Transfer" (Holmes, Marchand, & Coutureau, 2010; Rescorla & Solomon, 1967). Presentation of food cues can make humans work faster and harder for food (e.g. Lovibond & Colagiuri, 2013) by either increasing general arousal or by creating the expectancy of a specific food and consequently biasing choice toward actions that earn the same food (e.g., Watson, Wiers, Hommel, & de Wit, 2014).

Food cues can also acquire secondary reinforcing properties through their direct association with food (e.g., Williams, 1994). According to basic behavior principles, individuals may therefore be motivated to work in order to earn them. Stimuli that are present when operant actions are reinforced can further function as so-called "discriminative stimuli"; in this case, they increase operant responding by "setting the occasion" for the action-outcome relation (cue: action-outcome) rather than eliciting or motivating behavior through their simple direct association with food (see Hogarth et al., 2014 for a recent demonstration). Finally, with extended and repetitive practice, stimuli associated with operant responding can eventually elicit the operant behavior directly—as they do when they control mindless stimulus-

response habits that can develop with extensive repetition and practice (Balleine & O'Doherty, 2010; Thraillkill & Bouton, 2015a; Dickinson, 1985; Tricomi, Balleine, & O'Doherty, 2009). Thus, there are many ways in which stimuli associated with snacks and meals can augment future eating: They can elicit approach behaviors, increase motivation to eat, guide choice, provide conditioned reinforcement, set the occasion for eating, and elicit mindless eating out of habit.

Food cues can stimulate feeding in animals and humans in a manner that is independent of physiological hunger. In animals, specific cues, such as a sound or light paired with food, and contextual cues, such as a feeding environment, have been shown to potentiate feeding in sated states (Holland, Petrovich, & Gallagher, 2002; Petrovich, Ross, Holland, & Gallagher, 2007; Petrovich, Setlow, Holland, & Gallagher, 2002; Weingarten, 1983). In these settings food cues drive consumption of the signaled food (Delamater & Holland, 2008; Galarce, Crombag, & Holland, 2007; Petrovich, Ross, Gallagher, & Holland, 2007; Petrovich, Ross, Holland, et al., 2007). In humans exposure to food cues can prime an individual to engage in eating behavior and it can increase the amount of food that is selected and subsequently consumed (Ferriday & Brunstrom, 2008). Research shows that exposure to the sight and smell of food increases reported cravings (Ferriday & Brunstrom, 2008; Nederkoorn, Smulders, & Jansen, 2000; Oakes & Slotterback, 2000) and initiates cephalic phase responses, including release of insulin and changes in salivation, heart rate, gastric activity and blood pressure (Nederkoorn, Smulders, Havermans, & Jansen, 2004; Nederkoorn et al., 2000; Nirenberg & Miller, 1982; Overduin, Jansen, & Eilkes, 1997). Enhanced cephalic phase responses are associated with perceived food cravings and eating in adults (Nederkoorn et al., 2000) and with overeating in 8–12 year old obese children (Jansen et al., 2003). Reactivity to food cues has been shown to be heightened in obese subjects compared to lean subjects (Ferriday & Brunstrom, 2010). Food responsiveness, along with satiety responsiveness, differentiated three latent profiles of overweight and obese children, and those with the highest levels of food responsiveness had a higher BMI (Boutelle, Peterson, et al., 2014).

Food cue reactivity can be evaluated by assessing attentional bias and neural changes to food cues, in addition to self-report measures. Attentional bias is a measurement of how quickly and how much attentional resources are devoted to a cue. In general, these studies suggest that overweight and obese people have differential processing of food cues compared to healthy weight people; they initially orient to food cues more and then sustain their attention to them longer (Castellanos et al., 2009; Graham, Hoover, Ceballos, & Komogortsev, 2011; Loeber et al., 2012; Nijs, Muris, Euser, & Franken, 2010; Werthmann et al., 2011). Attention bias is correlated with increased craving and food consumption (Werthmann et al., 2011) and predicts increases in BMI in college students over one year (Calitri, Pothos, Tapper, Brunstrom, & Rogers, 2010). Attention bias to food pictures is associated with activation in brain regions related to attention and food reward and changes in BMI in adolescent girls (Yokum, Ng, & Stice, 2011). A one-session attention bias training program, compared to a control training program, differentially decreased how much overweight and obese children ate in the laboratory (Boutelle, Kuckertz, Carlson, & Amir, 2014). Additionally, a recent case series with 9 overweight and obese adults who endorsed binge eating showed that a 12-week attention bias training program resulted in

weight loss (Boutelle, Monreal, Strong, & Amir, under review). However, the previous study needs to be replicated with a control group before conclusions can be drawn.

Neural food cue reactivity, as measured by electroencephalography (EEG) and functional MRI (fMRI), has also been shown to differentiate overweight and obese individuals from lean individuals. A recent study using EEG showed that overweight and obese women, compared to healthy weight women, showed greater right parietal ERP P200 amplitude (early attentional processes) and shorter right parietal ERP P300 (enhanced maintenance of attentional processes; Hume, Howells, Rauch, Kroff, & Lambert, 2015). Imaging research, using fMRI, suggests that obese individuals, compared to lean individuals, show increased responsiveness in the gustatory (insula, frontal operculum), reward valuation regions (amygdala, ventralmedial prefrontal cortex, striatum) and cognitive control regions (orbitofrontal cortex) in response to images of palatable food (Bruce et al., 2010; Martin et al., 2010; Nummenmaa et al., 2012; Rothmund et al., 2007; Stice, Yokum, Blum, & Bohon, 2010; Stoeckel et al., 2009), cues that predict delivery of food (Stice, Spoor, Bohon, Veldhuizen, & Small, 2008), and to palatable food receipt (Boutelle et al., 2014; Ng, Stice, Yokum, & Bohon, 2011; Stice, Spoor, Bohon, Veldhuizen, et al., 2008). Neural activation to food cues has been associated with BMI in several studies (Rothmund et al., 2007; Stice, Spoor, Bohon, & Small, 2008) and with food intake (Burger & Stice, 2013).

As can be imagined, psychological and physiological food cue reactivity can be challenging for adults to manage, let alone children, and can stimulate overeating even in the sated state. Successful dieting is characterized by resisting tempting foods and food cues over and over again throughout the day. This resistance requires either the reduction of food cue reactivity (possibly through Pavlovian extinction) or inhibition of operant behaviors that typically occur when exposed to these foods (resisting the urge to eat, even in the presence of continued urges). In general, FBT recommends supporting overweight and obese children in losing weight by having healthy foods at home and reducing exposure to highly palatable, calorically dense foods. However, this is close to impossible in obesogenic environments. Children are exposed to highly palatable foods or food cues at school, at events, at birthday parties and holidays, at friend's houses, etc. Considering that self-control and executive functioning are developing throughout childhood (see for review Best & Miller, 2010), and that parents can not always supervise their child in every situation, it is important to develop interventions to reduce food cue reactivity, to give children tools for managing their cravings and motivation to eat.

One question that is pertinent to this discussion is can neural food cue reactivity be changed, or is it fixed? Cross-sectional neuroimaging studies of successful dieters, compared to healthy weight individuals, have shown mixed results to date regarding responses of the reward system to food cues (Cornier et al., 2009; DelParigi et al., 2007). However, one study showed that successful dieters, compared to currently obese and healthy weight controls, had greater activation to high-calorie food pictures in prefrontal regions implicated in inhibitory control (McCaffery et al., 2009). Studies that evaluated weight loss programs in overweight and obese adults have shown changes in neural responsivity for high-calorie vs. non-food pictures in regions involved in food reward and motivation, including the insula, thalamus, putamen, interior frontal gyrus, posterior cingulate, and cingulate gyrus

(Murdaugh, Cox, Cook, & Weller, 2012; Nock, Dimitropoulos, Tkach, Frasure, & von Gruenigen, 2012). In a small randomized controlled trial comparing participants in a 6-month weight loss intervention to a waitlist control, results showed changes in reward system activity (right ventral putamen and left dorsal putamen) in participants in the behavioral weight-loss program compared to the wait-listed control. Thus, food cue reactivity may change with weight loss, but further information is necessary. Additionally, it seems that inhibition may play a central role. Thus, interventions targeting a reduction in food cue reactivity have the opportunity to improve a child's ability to manage their cravings for palatable food.

Using learning theory to reduce food cue reactivity

Given that weight gain and obesity are associated with differential food cue reactivity, and that food cue reactivity is developed through learning and conditioning processes, it seems natural to turn to learning phenomena to evaluate intervention options which can be used to reduce food cue reactivity. In Pavlovian learning, *extinction* occurs when the conditioned stimulus, which was previously associated with a biologically significant stimulus, is presented repeatedly without the significant stimulus. In operant learning, extinction similarly occurs when the action is allowed to occur without producing the rewarding significant stimulus. For example, after eating chips in front of the TV every night, the TV becomes a conditioned stimulus and elicits cravings or feelings of wanting to eat chips; it might also set the occasion for eating as an operant response. In extinction, one might repeatedly sit in front of the TV without eating chips. Although the TV will elicit cravings and an urge to eat at first, over trials these responses will decrease, potentially reaching zero. It is possible that directly altering food cue responsiveness this way may have important implications for managing the omnipresent food cues for children who are trying to manage their weight.

Extinction theory can be applied in interventions to decrease food cue reactivity, to ultimately decrease overeating and weight. These interventions are typically referred to as Exposure and Response Prevention or Cue-Exposure Treatment (CET). Laboratory explorations showed that repeated cue exposure successfully reduced chocolate self-reported cravings and salivation in female chocolate cravers (Van Gucht, Vansteenwegen, Beckers, Hermans, et al., 2008). In another study with healthy weight college students, cravings for chocolate were acquired through cue-chocolate pairings, but these cravings did not extinguish over the duration of the study. However, expectations for chocolate were also acquired, and did in fact extinguish, suggesting potential differential effects of cue-exposure treatment on cravings and expectations (Van Gucht, Vansteenwegen, Beckers, & Van den Bergh, 2008).

We are in the process of developing a “cue-exposure treatment” for food (CET-Food) for overweight and obese children (Boutelle et al., 2011; Boutelle, N. Zucker, et al., 2014; Boutelle, Liang, et al., 2014). These methods were initially described decades ago in a pilot trial which showed that cue-exposure treatment led to abstinence as compared to self-control training with adults who binge eat and/or have bulimia nervosa (Jansen, Broekmate, & Heymans, 1992). CET-Food considers cravings for food a conditioned state or response that

is amenable to extinction. The goal of CET-Food is to extinguish or decrease the strength of the relationship between the food cues and cravings, expectations and physiological urges to eat when sated. It is important to note that we focus on extinguishing these urges when the child is full, as it is important to teach growing children to eat when they are physically hungry. The goal of CET-Food is to extinguish the relationship between having an accessible highly craved food and consumption. In brief, CET-Food is based on experiential “exposure” exercises, during which children (and their parents) are exposed to their highly craved foods, while increasing both psychological and physiological cue reactivity by holding, smelling and taking a small taste of the food during the exposure session, but not consuming the food. Following the exposure session, the food is thrown away in a trashcan. Initial pilot studies suggest that CET-Food shows some efficacy in decreasing overeating in overweight and obese children (Boutelle et al., 2011) and we are currently in the process of conducting a series of studies based on extinction theory to enhance CET-Food with children (Boutelle, Liang, et al., 2014). Additionally, recent data suggest that this method may also have promise with adults. Results from a pilot study with 28 overweight and obese binge eaters who participated in a 4-month program that included CET-Food showed significant weight change from baseline to post-treatment and continued weight loss 3 months post-treatment, which is remarkable considering the lack of diet or physical activity involved in the program (Boutelle, Peterson, Carlson, Bergmann, Knatz, under review). Although we believe that this treatment program has promise, either as a stand-alone treatment or in conjunction with behavioral weight loss, we recognize that more work grounded in the founding mechanisms is needed to make CET-Food more effective.

Extinction is a well-known phenomenon, and its use in cognitive-behavior therapies has a history. However, to further improve extinction-based treatments, it will be important to drill deeper into the implications of what is known about extinction based on contemporary basic laboratory research. One of the most critical themes of modern research on extinction is that extinction does not erase the original learning (Bouton, 2004, 2011, 2014; Laborda, McConnell, & Miller, 2011; Quirk & Mueller, 2008; Todd, Vurbic, & Bouton, 2014a; Vurbic & Bouton, 2014), which will present inherent challenges in managing food cue reactivity. That is, even after the successful elimination of a craving or behavior in extinction, the original learning continues to exist, and the response can reappear under many circumstances (see below). In either Pavlovian or operant learning, the reduction in behavior that occurs during extinction is the result of a form of context-specific retroactive inhibition process rather than unlearning. To extend our previous illustration, when seeing the TV, the child may indeed feel cravings and an urge to eat potato chips. However, after extinction/inhibition learning, cravings and urges would be reduced, but not erased; something akin to *resisting eating* has been learned in the presence of the TV.

The modern view that extinction depends specifically on a context-specific form of inhibition stems from several important response-recovery or “relapse” phenomena that are now considered central to the understanding of extinction (see (Bouton, 2004, 2014; Vurbic & Bouton, 2014) for reviews). In *renewal*, extinguished responding returns when the CS or the response is tested in a different context. Several forms of renewal have been demonstrated (ABA, ABC, and AAB, where the three letters refer to the contexts of original

learning, extinction, and testing, respectively; e.g. Bouton, Todd, Vurbic, & Winterbauer, 2011). This means that even though a child has learned to resist eating in front of the TV, when she sees one in someone else's home, craving and urges to eat in front of the TV might return. In *spontaneous recovery*, extinguished responding returns after a period of time has elapsed after extinction (e.g., Brooks & Bouton, 1993; Rescorla, 2004). Thus, extinguished cravings and urges to eat can return if the TV is visited sometime later; the passage of time is considered a change in temporal context. In *resurgence*, an extinguished behavior that has been replaced by a new behavior returns when the new behavior is itself extinguished (e.g., Bouton & Schepers, 2014; (Shahan & Sweeney, 2011; Winterbauer & Bouton, 2010). Thus, when eating is extinguished and replaced with a new activity such as exercise, not exercising one day can cause cravings and urges to eat to return. In *reinstatement*, simple exposure to the biologically-significant stimulus after extinction can cause responding to a food cue to recover. This effect depends at least in part on receiving the significant stimulus in the context where testing then occurs; one way to think of it is that when the context (e.g., the family room) has been recently associated with food again, it arouses an expectancy of food that allows the food cue to trigger eating again. Finally, in *rapid reacquisition*, extinguished responding can quickly return when the food cue or action is paired with the reinforcer again. Thus, extinguished cravings and urges can quickly return with full force if one eats in front of the TV again after extinction. Rapid reacquisition may be especially important in understanding the persistence of overeating, because although phenomena like renewal and spontaneous recovery can cause an initial lapse in behavior, individuals who lapse necessarily expose themselves to new cue-food and action-food pairings, and begin the "spiral" into true relapse. Rapid reacquisition can also be considered a context effect because recent cue-food (or action-food) pairings were part of the background "context" during the original learning. Thus, resuming cue-food and/or action-food pairings will return the individual to the original context of eating (Bouton, 2011). Context can thus be conceptualized as the immediate environment, or any "background stimuli", including other behaviors, emotions, time, physiological states, and recent events (Bouton, 2011; Vurbic & Bouton, 2014).

Because behavior is suppressed in extinction in a context-specific way, rather than being unlearned, there is an inherent bias or asymmetry in how strongly craving and the urge to eat will be evoked across contexts. Conditioned responding (especially operant responding) may itself be context-specific to a degree (see Bouton, Todd, & Leon, 2014; Bouton et al., 2011; Thrailkill & Bouton, 2015a), but extinction learning is demonstrably even more so (Bouton & Todd, 2014). The same asymmetry seems to be true of many learning paradigms in which new learning is used to replace old. For example, in counterconditioning, responding based on cue-food learning can be suppressed by pairing the cue with a different significant stimulus instead of a lack of reinforcement. Animal experiments suggest that renewal, spontaneous recovery, and reinstatement can occur after counterconditioning, in which a tone was first paired with food and then with shock or vice versa (Bouton & Peck, 1992; Brooks, Hale, Nelson, & Bouton, 1995; Peck & Bouton, 1990). Thus, like simple extinction, counterconditioning can leave the individual vulnerable to lapse and relapse. However, it is worth noting that in some human studies, counterconditioning has shown some preliminary promise in impacting cue-induced cravings (Van Gucht, Baeyens, Vansteenwegen,

Hermans, & Beckers, 2010). In the Van Gucht study, extinction of the relationship between a cue and chocolate consumption did not impact cravings for chocolate, however, cravings were reduced after counterconditioning (pairing the cue that was previously paired with chocolate with an aversive liquid instead). Notably, this effect persisted (and did not spontaneously recover) after one week. However, counterconditioning with children is an ethical dilemma, and more research is needed before studies using counterconditioning will be conducted with children. And as just noted, the basic animal research suggests that counterconditioning can be context-specific and prone to relapse over sufficient time, like extinction.

In operant conditioning, recent experiments have similarly shown that responding reinforced by food can be renewed after it has been suppressed by response-contingent punishment (Bouton & Schepers, 2015). Punished responding recovered when the context was changed (both ABA and ABC renewal were demonstrated), and the results suggested that, as in operant extinction, the organism learns to inhibit a specific response in a specific context. Thus, suppression of behavior by punishment, like by extinction, is specific to its context. Finding renewal after punishment (see also Marchant, Khuc, Pickens, Bonci, & Shaham, 2013) is important as it relates to eating behavior, because people may often stop overeating because they begin to appreciate its aversive consequences rather than extinction. The phenomenon also suggests that nagging a child about overeating (a form of punishment that is used by many parents) might not be effective at suppressing the child's overeating in other contexts.

Overall, the results of basic research thus suggest that several strategies that can be used to suppress or inhibit learned performance (extinction, counterconditioning, punishment) do not produce unlearning, but instead cause a context-specific suppression of behavior. We suggest that this might be one important but overlooked reason why assisting children in losing weight and maintaining healthy eating is so challenging. The behavioral power of a food cue (as a stimulus controlling physiological arousal, craving and approach, motivation, conditioned reinforcement, or setting the occasion for an operant) is always ready to return.

Although research suggests that behavior change can be impermanent and context-dependent, recent research suggests a little good news. One finding is that extinction (inhibition of responding) in the presence of one cue can sometimes inhibit responding in the presence of other very different cues (Vurbic & Bouton, 2011). The case in operant conditioning seems especially instructive. First, in laboratory experiments, when an operant behavior is occasioned by two separate and distinct stimuli (e.g., a tone and a light), extinction of the behavior in the presence of one of the stimuli transfers and suppresses behavior in the presence of the other stimulus, too (Bouton, Carranza-Jasso, & Trask, in preparation). Although this transfer is not necessarily complete (there is still a little responding to the alternate stimulus), far less transfer of inhibition occurs when the alternate stimulus occasions a different operant behavior. Thus, operant extinction learning involves the suppression of the specific response. Translated to the overeating child, a boy who learns to resist eating in the presence of a bag of chips might also inhibit responding when given the opportunity to eat them out of a bowl. Thus, it might not be necessary to extinguish all stimulus-response combinations to suppress overeating somewhat generally. A second

recent finding is that when different behaviors are linked together in a “behavior chain,” extinction of one response can also weaken the other. For example, a person may buy junk food at a convenience store (a procurement response) and then eat it later (a consumption response); the two behaviors are different but linked. Recent laboratory research suggests that extinction of procurement alone can weaken a linked consumption response, and that extinction of consumption alone can weaken a linked procurement response, when the consumption and procurement responses are tested in isolation (Olmstead, Lafond, Everitt, & Dickinson, 2001; Thraillkill & Bouton, 2015b). However, we should note that responses can still return (be renewed) if the corresponding cues are presented in a different context (Todd, Vurbic, & Bouton, 2014b).

How to improve extinction learning

These facts about extinction (and related behavior change paradigms) suggest that we need to be realistic about what extinction treatments can accomplish. However, there are ways to improve extinction learning that can be applied in behavioral weight loss treatments (Bouton, 2007). In general, in order to improve extinction outcomes, we can theoretically either (1.) strengthen inhibition learning that occurs in extinction, or (2.) try to increase the generalization of learning from the extinction context to other potential relapse (Bouton, Woods, Moody, Sunsay, & Garcia-Gutierrez, 2006). Each of these strategies is discussed below.

Strengthening inhibition learning

One way to optimize extinction learning is to strengthen the inhibition learning that is thought to occur in extinction. Extinction is now widely understood to result, at least in part, from the correction of prediction error. According to most models of conditioning (Pearce & Hall, 1980; Rescorla & Wagner, 1972; Wagner, 1981; 2003), learning occurs on a conditioning trial when there is a discrepancy between what is predicted to occur on that trial and what actually occurs; this discrepancy is often called a prediction error. Learning is viewed as a means of reducing such prediction error; on every trial, the associative strength of cues is adjusted to bring what is predicted and what actually occurs into alignment. In extinction, we learn inhibition because the cues present on a trial predict that a significant stimulus will occur, and yet one does not. Over trials, the prediction is gradually corrected until what is predicted (“nothing”) is aligned with what actually occurs. The importance of prediction error has been supported by a great deal of research on both acquisition and extinction. In a number of extinction experiments, cues that were separately conditioned have then been combined and presented together on extinction trials (Janak & Corbit, 2011; Kearns, Tunstall, & Weiss, 2012; Rescorla, 2006; Thomas & Ayres, 2004). Because the prediction made by separate cues are thought to summate, the compound created an even larger prediction and hence, a larger prediction error. For that reason, compounding separately-conditioned cues can increase the amount of inhibition that is learned to them in extinction. In the extinction of overeating, we might present separate food cues simultaneously while conducting extinction. It is worth mentioning that a role for prediction error has received especially strong support from experiments in which two cues have been separately conditioned and then combined and paired with the same significant stimulus on

compound conditioning trials (Kremer, 1978; Lattal & Nakajima, 1998; Rescorla, 2007). During the latter compound trials, even though the stimuli are still paired with the significant stimulus, the compound predicts a larger event than actually occurs, and partial extinction therefore occurs. Such “overexpectation” has further implications for the extinction of overeating. Of note, anecdotally, adult participants in CET-Food remark how the food doesn’t taste as good as they thought it would. If the expectation of food created by cues is more positive than what is actually experienced when eating is slowed, prediction error and overexpectation lead us to hypothesize that focusing on taste or slowing eating may be another way to allow the development of inhibition.

Extinction learning might also be further optimized by manipulating the spacing of extinction trials. It is widely known that trials that are spaced over a period of time result in stronger learning than temporally massed trials; this is true for appetitive (food) conditioning (e.g., Sunsay & Bouton, 2008; Sunsay et al., 2004) as well as other kinds of conditioning (e.g., Barela, 1999; Domjan, 1980). At a theoretical level, spaced trials may enhance learning by increasing the novelty or surprisingness of the cue and the significant outcome (Sunsay & Bouton, 2008); they might also improve memory strength if it is partially forgotten between learning episodes (Bjork & Bjork, 1992) or the learning in extinction of inhibition if error correction (like responding itself) spontaneously recovers over time (e.g., Leung & Westbrook, 2008). Although there is ample theory to suggest that spaced extinction trials would improve extinction learning, trial spacing during extinction training has produced inconsistent results in animal experiments (e.g., compare results of Cain, Blouin, & Barad, 2003; Moody, Sunsay, & Bouton, 2006b; Urcelay, Wheeler, & Miller, 2009).

Variability during exposure could also potentially enhance inhibition of responding to food cues through enhanced generalization. Our understanding of the impact of variability on exposure are mainly drawn from anxiety disorders at this time, although the basic behavioral processes involved in fear and appetitive extinction do not appear to be different; for example, the basic extinction and recovery phenomena emphasized in this article have been repeatedly demonstrated in both (e.g., see Bouton, 2004; Vurbic & Bouton, 2014, for reviews). Variability may increase the strength of the inhibitory learning through several mechanisms (see for review Craske, 2008; Bjork, 1988). For example, it would increase the number of cues associated with the lack of eating, with more cues making the likelihood that these cues will be present (retrieval cues, see below) when this learning is needed. Variation might also promote the development and application of rules that capture the commonalities between tasks (Schmidt & Bjork, 1992). Studies in phobic samples have shown that exposure to varied phobic stimuli led to better maintenance of treatment gains (in terms of spontaneous recovery) at follow-up than did exposure to a constant stimulus (Lang & Craske, 2000; Rowe & Craske, 1998). Additionally, greater variability in fear levels throughout exposure enhanced outcomes in anxiety about contaminants and public speaking (Culver, Stoyanova, & Craske, 2012; Kircanski et al., 2012).

Extinction in multiple contexts may also improve the effectiveness of extinction learning (Bouton, Woods, Moody, Sunsay, & Garcia-Gutierrez, 2006). Since extinction learning is context-dependent, changing contexts repeatedly during extinction would repeatedly renew

responding and hence prediction error, facilitating further inhibitory learning (Holmes & Westbrook, 2013; as we note below, extinction in multiple contexts might also increase the generalization to new contexts.). It is possible that exposure in multiple contexts can also be done by video or online (Vansteenwegen et al., 2007). Unfortunately, humans have numerous pairings between food cues and eating, which results in cravings in many different contexts, with many different cues in each context; original learning experiences in many contexts may enhance renewal effects when in new contexts (Gunther, Denniston, & Miller, 1998; Todd, Winterbauer, & Bouton, 2012b).

Another method that might enhance learning in an “extinction” phase might be to pair the food cue or operant behavior with an aversive stimulus (counterconditioning or punishment, respectively). Such treatments can be highly effective at suppressing performance in the short run. But as we noted earlier, administering aversive events raises certain ethical questions that might be difficult to justify in the sense that both counterconditioning and punishment, like simple extinction, can still be vulnerable to lapse and relapse after temporal and physical context change (e.g., Bouton & Peck, 1992; Bouton & Schepers, 2015; Peck & Bouton, 1990).

New technologies might also eventually provide new ways to enhance extinction and eliminate undesirable learned behavior. For example, drugs that are known to facilitate learning could plausibly enhance extinction learning. D-cycloserine (DCS), a partial agonist of the NMDA receptor that is involved in long-term potentiation, a cellular model of learning, has been shown to increase the rate at which Pavlovian fear extinction is learned (e.g., Walker, Ressler, Lu, & Davis, 2002), although it might not qualitatively change the nature of extinction learning or eliminate its inherent context-dependency (Bouton, Vurbic, & Woods, 2008; Woods & Bouton, 2006). Thus, relapse effects might still occur. DCS can also facilitate the extinction of operant food-seeking (e.g., Leslie, Norwood, Kennedy, Begley, & Shaw, 2012), although the evidence here to date may be less consistent (e.g., see Vurbic, Gold, & Bouton, 2011; Thanos et al., 2011). A second type of new technology is suggested by a phenomenon known as *reconsolidation* (e.g., Nader, Schafe, & LeDoux, 2000). Here, the claim is that when a memory is retrieved, it becomes temporarily vulnerable to disruption by certain drugs if they are administered before the memory returns to a stable state (e.g., Nader et al., 2000; Kindt, Soeter, & Vervliet, 2009). A review of the reconsolidation literature is beyond the scope of this article, but it is worth noting that in fear conditioning (cue-shock learning), where the phenomenon has been studied most extensively, drugs seem most effective at weakening memories that are old or have been conditioned with only a small number of cue-shock trials (e.g., Wang, de Oliveira Alvares, & Nader, 2009; in contrast, food-cue learning can presumably involve a very large number of repeated conditioning trials.) Conducting extinction soon after a memory is retrieved might also disrupt memory reconsolidation (Monfils, Cowansage, Klann, & LeDoux, 2009; Xue et al., 2012), but this effect is poorly understood, and there have been published failures to replicate it (e.g., see Chan, Leung, Westbrook, & McNally, 2010; Kindt & Soeter, 2013; Soeter & Kindt, 2011).

Bridges to other contexts

The second general way to optimize learning is to provide a bridge from the extinction context to other contexts. The simplest way to accomplish this is to conduct exposure treatments in the context(s) where overeating is most probable and therefore problematic. For example, extinction could focus on the major contexts in which food cues are most likely to be encountered-- the kitchen, dining room, favorite restaurant or time (evening), or mood (bored). But in addition to this, as noted above, another method to create bridging might be to conduct extinction in multiple contexts. Contexts are complex stimuli composed of many features; by conducting extinction in multiple contexts, one increases the range of features that are connected with extinction—and might be encountered again in a new context.

Still another way to bridge across contexts is by providing objects that serve as retrieval cues (Bouton et al., 2006). In animals, retrieval cues that were present during extinction trials attenuate context-based renewal effects if they are presented again at the time of testing (Brooks & Bouton, 1993, 1994), and they are more effective than relatively novel cues or cues that were present during conditioning rather than extinction. Collins and Brandon (2002) showed that the return of alcohol cue reactivity after extinction due to a context-change could be reduced by the use of retrieval cues that were present during extinction. A related possibility is to mentally reinstate the extinction context. Some research suggests that individuals can “mentally reinstate” extinction contexts (i.e. the physical surroundings where exposure took place, word that therapist said to them; Craske, 1999). One study suggested that remembering the environmental context in which the exposure treatment was conducted resulted in less renewal of spider fear in a different context (Mystkowski, Craske, Echiverri, & Labus, 2006).

A less intuitive, but interesting way to provide better bridging between extinction and possible relapse contexts is to occasionally reinforce the food cue or the operant response during extinction (Bouton, Woods, & Pineno, 2004; Woods & Bouton, 2007). This method might provide especially effective protection against relapse in the form of rapid reacquisition. As noted earlier, resuming cue-food or action-food pairings after extinction could cause behavior to return quite rapidly because recent cue-food or action-food pairings were part of the context of original conditioning (Ricker & Bouton, 1996). By including occasional cue-food or action-food pairings among a very large number of extinction trials, one can suppress behavior while at the same time associating these contextual cues with extinction or behavioral suppression rather than just conditioning. Consistent with this idea, occasional reinforced trials have slowed relapse in the form of rapid reacquisition in both Pavlovian cue-food (Bouton, 2004) and operant action-food (Woods & Bouton, 2007) learning in animals. In a perhaps related way, cigarette smoking in humans has been reduced by giving smokers the opportunity to smoke only occasionally at predetermined intervals (Cinciripini et al., 1994; Cinciripini et al., 1995). The idea is to associate a reinforced trial (a minor lapse) with further suppression or even abstinence from the behavior. In the long run, this may promote the persistence of extinction by (in the case of eating) protecting it against the pernicious effects of new cue-food or action-food pairings.

The possible clinical effectiveness of using partial reinforcement in extinction is not to be confused with another well-known phenomenon in learning theory, the so-called Partial Reinforcement Extinction Effect (PREE). In this phenomenon, subjects who are reinforced only occasionally (“partially”) during conditioning are slower to later extinguish their responding than subjects who are consistently reinforced (see van den Akker, Havermans, Bouton, & Jansen, 2014) for a recent demonstration in food conditioning with humans). The focus of the PREE is on the persistence of conditioned performance in extinction rather than the persistence of extinction performance in reacquisition, which we were discussing immediately above. But the PREE may have its own important implications for overeating. As recently noted by Van den Akker et al. (2014), when an individual (for example) goes on and off a diet repeatedly, he or she may be exposed to a partial reinforcement schedule in which repeated extinction trials (not eating in the presence of food cues while dieting) are intermixed with reinforced trials eating in the presence of food cues while not dieting. By creating a PREE, such a schedule would only make the suppression of eating more difficult during a longer run of extinction trials. Because people can never be fully “abstinent” from eating, eating can always occur in the presence of a given food cue at least some of the time. The PREE may thus be very difficult to avoid, which might be one of the reasons why inhibitory learning with food is so challenging.

Role of individual differences

Although this discussion describes opportunities to apply learning theory to interventions designed to reduce overeating in children, it would not be complete without the recognition that there may be individual differences that play a role in learning relationships with food cues. At this time, we are just beginning to understand the role of individual differences in food cue reactivity. For example, recent studies suggest that some animals have especially strong tendencies to approach and attribute “incentive salience” to cues that are associated with food (e.g., Flagel et al., 2010; Saunders & Robinson, 2013). The same animals appear to be prone to drug addiction-related processes in that they are also strongly affected by cues associated with cocaine (Saunders & Robinson, 2010, 2011; Yager & Robinson, 2013; see Saunders & Robinson, 2013 for review). Interestingly, preliminary results suggest that a similar “cue-reactive phenotype” might also exist in humans. For example, the extent to which human smokers report craving in the presence of smoking cues correlates with the extent to which they also report cravings in the presence of food cues (Mahler & de Wit, 2010; Styn, Bovbjerg, Lipsky, & Erblich, 2013). Human traits, such as those that are driven toward reward, have been shown to be associated with neural activation to food cues in a fronto–striatal–amygdala–midbrain network (Beaver et al., 2006). In another study examining a weight loss in a 12-week weight loss program, obese individuals who had higher levels of activation in brain regions implicated in reward, such as the nucleus accumbens, anterior cingulate, and insula, were less successful in losing weight (Murdaugh, Cox, Cook, & Weller, 2012). These brain responses seem to interact with genetics to predict overeating and weight gain (Stice, Spoor, Bohon, & Small, 2008). Furthermore, neural activity in response to food cues in the ventralmedial prefrontal cortex and the dorsolateral prefrontal cortex at baseline, and a stronger functional connectivity in these regions is associated with dietary success, impulse control and weight loss (Weygandt et al., 2013).

Finally, other recent studies suggest that metabolic factors may mediate the relationship between ventral striatal activity in response to food cues and body weight (Malik, McGlone, Bedrossian, & Dagher, 2008; Simon et al., 2014).

Although there may be individual differences that could moderate or mediate learning and weight gain, there also could be individual differences in learning itself. A recent study showed that obese women, compared to healthy weight women, had difficulties learning to inhibit their responses to food cues, even in the presence of signals for nonreward (Zhang, Manson, Schiller, & Levy, 2014). Another study showed that overweight college students, compared to healthy-weight college students, swallowed more to an innocuous cue paired with chocolate milk, compared to cues paired with water or no liquid after only 9 pairings (Meyer, Risbrough, Liang, & Boutelle, 2014). Notably, the healthy weight students did not acquire a response to the innocuous cues paired with chocolate or water. Neuroimaging data also support the role of individual differences, as one study showed that adolescents who have the greatest escalation in the ventral pallidum to innocuous cues paired with a taste of food and the greatest decrease in the caudate to the taste of food had significantly larger increases in BMI over two years (Burger & Stice, 2014). From this small body of literature, it seems that individual traits, cognitive skills, differential learning rates or abilities, metabolic factors, genetics and neural responsivity may all impact food cue reactivity on an individual level. Understanding these individual differences, and the impact on food cue reactivity, will require further research, and will be key to developing interventions.

Other learning mechanisms relevant to overeating

We have emphasized extinction processes as one method from learning theory for addressing overeating. It is worth mentioning that a learning-theory perspective also recommends other kinds of treatments. The following review of other lines of research using learning theory may not be exhaustive, but will highlight some areas of research that could offer potential avenues for intervention development. As one example, Epstein and colleagues have emphasized the effects of habituation to foods in the control of eating (e.g., see Epstein, Temple, Roemmich, & Bouton, 2009 for a review). In habituation, repeated exposures to a stimulus (in this case, a food), decreases the amount of responding the stimulus elicits or controls. Thus, when given the opportunity to earn bites of food by playing a simple game on a computer, children's rate of responding for the food decreases as more and more food is eaten. Habituation is a well-studied phenomenon, and it has many well-known characteristics (Epstein, Temple, Roemmich, & Bouton, 2009; Rankin et al., 2009). For example, the effect is stimulus-specific; responding recovers when the habituated stimulus is changed. Thus, children whose responding had habituated for bites of cheeseburger worked hard again when they were offered bites of apple pie (Epstein et al., 2003)—the familiar dessert effect. Another related phenomenon in the habituation literature is the variety effect. When a participant is exposed to stimuli that vary from trial to trial, habituation is slower than when he/she is exposed to the same stimulus repeatedly. Thus, responding for food in children given a mixture of foods is slower to decrease than that in children repeatedly given the same food (Temple, Giacomelli, Roemmich, & Epstein, 2008). Epstein et al. (2009) framed the findings within the terms of a comprehensive memory model of conditioning and habituation (Wagner & Brandon, 1989) which emphasizes that

responsivity to food declines to the extent that the food is still remembered in short-term memory when it is presented again on the next trial.

The idea that eating is affected by habituation processes has led to a number of important insights about eating and overeating. As one example, in overweight children, eating in both the laboratory and in the home can be suppressed by repeated exposure to the same food instead of a variety of foods (Epstein et al., 2013). Other research suggests that children are slower to habituate to food (and consequently eat more of it) while watching TV or listening to audiobooks (Epstein, Saad, Giacomelli, & Roemmich, 2005; Temple, Giacomelli, Kent, Roemmich, & Epstein, 2007). Theoretically, this effect occurs because watching or book listening takes up more space in short-term memory, which has a limited capacity, making it more difficult for the memory of food to persist from trial to trial and cause habituation. Research has shown that children who are slower to habituate to food in a laboratory task, compared to those who habituate faster, gain more weight over one year (Epstein, Robinson, Roemmich, & Marusewski, 2011). And recent evidence suggests that the rate of a child's habituation to food is correlated with the child's short-term memory capacity; children with better short-term memory habituate to food faster (Epstein, Carr, Scheid, Gebre, O'Brien, Temple, & Bouton, under review). One might imagine the engineering of a healthy diet in which consumption of unhealthy high-energy-dense foods is reduced by decreasing their variety and the consumption of healthier low-energy-dense foods is conversely enhanced by increasing their variety.

The role of memory in habituation fits nicely with other work focusing on the role of memory processes and encoding of food memories. Episodic memories of the ingestive consequences of eating (memory for earlier eating episodes) affect decisions regarding future food consumption (Higgs, 2005; Robinson, Aveyard, et al., 2013). A number of studies have provided evidence that the inhibition of food intake is influenced by the memory of a recent meal. In these studies, healthy weight participants are asked to recall what they had for lunch, and the impact on eating later in the day is measured. Those who recalled the lunch they ate decreased snack consumption a few hours later compared to subjects in the control condition (Higgs, 2002, 2008). Additionally, studies evaluating the impact of food memories have shown that those who are distracted during a lunch meal ate more at a later afternoon snack compared to those that were not distracted (Higgs & Woodward, 2009). In healthy weight college students, deliberately holding food items in working memory, as opposed to just attending to food items, showed facilitated detection of food items (Higgs, Rutters, Thomas, Naish, & Humphreys, 2012). Considering the importance of encoding food memories, college students who were instructed to focus on the sensory properties of the food they were eating after consuming a lunch showed greater suppression of intake when given a snack a few hours later compared to subjects that read an article about food or were not given any specific task after eating the same lunch (Higgs & Donohoe, 2011). Interestingly, memory of a recent lunch exerts a stronger influence on subsequent intake suppression than does the actual energetic content of the meal itself (Brunstrom et al., 2012). Additionally, a recent pilot study suggested that a smart-phone app that trains both eating attentively and reviewing previous eating events before meals reduced weight in the 12 overweight participants in the pilot study (Robinson, Higgs, et al., 2013).

Considering that memory of a recent meal has been shown to reduce subsequent intake, it is possible that memory problems might be linked to overeating. Recent findings suggest that hippocampal-dependent learning and memory mechanisms contribute to the control of food intake (Davidson, Kanoski, Schier, Clegg, & Benoit, 2007). The hippocampus is important in spatial memory, declarative memory (formation and recall of memories about events and facts) and to resolve predictable ambiguities that exist when a stimulus signals different outcomes dependent on the presence/absence of other cues (such as the relationships between food cues and satiety signals). For example, the hippocampus functions in learning that satiety signals predict when food cues will not be followed by an appetitive postingestive outcome (Davidson, Kanoski, Walls, & Jarrard, 2005).

Davidson and colleagues have demonstrated in animal experiments that eating a “Western diet”, specifically diets high in saturated fats and refined carbohydrates, interferes with hippocampal functioning. Thus, they have pointed to a “vicious cycle of obesity and cognitive decline” (Davidson et al., 2005; Kanoski & Davidson, 2011) in which eating a Western diet disrupts hippocampal functioning, and this disruption impairs the individual’s ability to inhibit memories of the relationships between postingestive consequences of energy intake and environmental food cues. This impairment would increase the likelihood that those cues would evoke additional cravings and overeating of the Western diet, which will further decrease functioning of the hippocampus. Studies also suggest that diet-induced hippocampal deficits precede weight gain (Davidson, Sample, Swithers, 2014). Additionally, Kanoski and colleagues (Kanoski et al., 2011) showed that consumption of added sugars, particularly high fructose corn syrup, negatively impacted hippocampal functioning and metabolic outcomes in animals.

Other variations in what foods are consumed can lead to increased overeating through learning, including non-caloric sweeteners and non-caloric fat substitutes. In a normal environment, the perception of a sweet taste or fat taste on the tongue would be correlated with (and thus potentially predict) the intake of calories. But this natural connection between sweet tastes and calories has been weakened by the addition of noncaloric sweeteners in animals in the modern Western diet (e.g., Davidson, Tracy, Schier, & Swithers, 2014). That is, children who consume foods with noncaloric sweeteners could experience extinction or partial reinforcement trials in which sweet tastes are not associated with an increase in calories, which can create a number of negative consequences. Evidence suggests that animals given yogurt snacks flavored with saccharin (rather than glucose), so that the sweet taste had a weaker predictive relationship with the intake of calories, wind up eating more and gaining more weight over time (Swithers & Davidson, 2008). Their ability to suppress intake of their maintenance chow after they were given a different, but sweet, snack was also compromised. Similar results have been reported with animals exposed to non-caloric fat substitutes (Swithers, Doerflinger, & Davidson, 2006). Such results continue to reinforce the idea that fundamental learning processes can play a role in increasing food consumption in the contemporary food environment. However, to date, similar human studies have not been conducted.

Concluding comment

While addressing these problematic features of the Western diet and promoting primary prevention of obesity will require tremendous changes to today's food and physical activity environment, it will be decades before such changes are realized. Thus, it is necessary to develop more potent intervention strategies to assist children in managing the current toxic food environment. In this article, we have attempted to document some of the many learning mechanisms and processes that can contribute to overeating—and perhaps its suppression and inhibition. We expect that extinction might be an especially useful therapeutic tool to help decrease the impact of food cue reactivity and ultimately decrease overeating and weight gain. However, extinction theory is not simply implemented, and a number of factors play a role in individual's learning to food cues, and their ability to manage their food cue reactivity in today's environment. Moreover, our understanding of basic learning and extinction processes will continue to advance and evolve. Further research to translate learning mechanisms to interventions, utilizing an in-depth and up-to-date understanding of learning theory, will provide additional knowledge on how to help children decrease overeating and weight.

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Highlights

Learning theory can be used to develop interventions to decrease overeating

We discuss a modern view of extinction and mechanisms to enhance extinction

We also review other learning mechanisms relevant to overeating

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