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## **Regulating Food Craving: From Mechanisms to Interventions**

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### **Abstract**

Craving, defined here as a strong desire to eat, is a common experience that drives behavior. Here we discuss the concept of craving from historical, physiological, and clinical perspectives, and review work investigating the effects of cue reactivity and cue-induced craving on eating and weight outcomes, as well as underlying neural mechanisms. We also highlight the significance of cue reactivity and craving in the context of our “toxic food environment” and the obesity epidemic. We then summarize our work developing the Regulation of Craving (ROC) task, used to test the causal effects of cognitive strategies on craving for food and drugs as well as the underlying neural mechanisms of such regulation. Next, we review our recent development of a novel ROC-based intervention that trains individuals to use cognitive strategies to regulate craving, with promising effects on subsequent food choice and caloric consumption. We end by discussing future directions for this important line of work.

### **Keywords**

Craving; cue reactivity; regulation; choice; value; training

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At the 2019 annual meeting of the *Society for the Study of Ingestive Behaviors* in Utrecht, the last author presented our lab’s work on the regulation of craving task, as well as the development and validation of a cognitive training procedure based on this task. In this review, we provide a synthesis of the background and motivation for those studies, summarize important relevant work, and discuss future directions for refining and disseminating this training.

We will begin with a demonstration. Please look at this freshly baked brownie, and imagine that it is on a plate, right in front of you (Figure 1). You can see there are chocolate chips and nuts on top, and warm fudge running down the sides. Now, please imagine picking up

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this warm brownie, bringing it close to your nose, and inhaling the sweet scent of chocolate. Imagine bringing it to your mouth and taking a bite. Can you feel the rich texture of the dough, and the indulgent taste? As you imagine this, you may feel your mouth watering, perhaps your heart rate quickens ever-so-slightly. And with this, you may notice that *you really want to eat a brownie*. If so, then what you are experiencing is known as *craving*.

Craving can be defined in a myriad of ways. In our conceptualization – as in most common conceptualizations – craving is defined as a strong desire, urge, or wanting [e.g., 1, 2–4]. For example, in the current 5<sup>th</sup> edition of the Diagnostic and Statistical Manual of Mental Disorders [DSM-5; 3], craving is listed as a diagnostic criterion for substance use disorders and is defined as “a strong desire or urge to use drugs.”

As a human experience, craving has been troubling philosophers for centuries. In the Buddhist Four Noble Truths, craving is said to be the cause of all suffering [5]; the philosopher Baruch Spinoza said that it is “the very essence of man” [6]; and Bertrand Russell said that it prompts all human activity [7]. These thinkers join in the belief that craving is a very common experience (as confirmed by modern epidemiological studies showing that craving is experienced by ~99% of adults [8]) that also drives behavior [9].

## Craving as a Learned Response

Psychologically, we consider craving to be a complex, multidimensional construct that can be understood using a learning-based model of behavior. To conceptualize this, one could think back to the classical experiments of Russian physiologist Ivan Pavlov. In the late 19<sup>th</sup> century, Pavlov began researching salivation in dogs in response to feeding. He started with the fundamental observation that his dogs naturally salivated in response to eating food, and often as soon as it was placed in front of them. Then, Pavlov started to ring a bell each time before placing food in front of the dogs. After a number of repeated bell-food pairings, Pavlov rang the bell without placing food in front of the dogs, and observed that they salivated in response to the sound of the bell, even when food did not follow. Pavlov formalized his observations into a paradigm now known as classical or Pavlovian conditioning [10]. In this framework, ingested food is considered to be an “unconditioned stimulus” and salivation is considered to be an “unconditioned response” (that is, these “unconditioned” factors do not depend on or require any learning; salivation is a natural response to food). When Pavlov introduced the bell before food, he was adding a “neutral stimulus” that previously did not elicit a salivation response from the dogs. After repeated pairing with the food (the unconditioned stimulus), the bell became a “conditioned stimulus” that predicts food presentation, and came to elicit a “conditioned response,” salivation. As such, bell-cued salivation is a learned stimulus-response connection.

In the years since, research has repeatedly shown similar effects in animals as well as in humans. Indeed, after repeated exposure, food-related cues (e.g. labels/package for brownies, the sight of brownies) that are present at the time of eating acquire the ability to predict the ingested food, becoming conditioned stimuli that evoke conditioned responses. Specifically, experimental work in animals and humans has shown that cues paired with food elicit strong conditioned responses, including increased salivation [10, 11], heart rate [12],

gastric activity [13], hormone secretions [14, 15], dopamine neuron firing and release [16, 17], and subsequent neural activity in regions typically associated with reward [18].

Food cues that become conditioned (e.g., food pictures, advertisements, labeling) are often external. However, for humans in particular, conditioned food cues can also include internal factors such as the experience of stress [19], negative emotion [20], hormonal changes [21], and food-related thoughts [22]. Such conditioned cues reliably evoke conditioned bodily responses including increased salivation [11], heart rate [12, 23], gastric activity [12, 23] and neural activity in the ventral striatum [VS; a region typically associated with reward; 18, 24]. Further, these physiological conditioned responses are often accompanied by the conscious experience of craving, which in this context is termed “cue-induced craving” [25, 26]. Thus, we can think of craving as a type of cue reactivity, or as an emergent psychological property of physiological cue reactivity, or more broadly as a conditioned response to food cues. Accordingly, experimental work in humans has demonstrated that exposure to food cues strongly and consistently produces the conscious experience of craving, and that these associations are learned quickly [27]. To illustrate, even previously-neutral stimuli (e.g., serving trays) can come to elicit cue-induced craving for chocolate when presented alone, after just a single Pavlovian conditioning session in which they were paired with chocolate [e.g., 27].

Notably, craving can be experienced for specific foods (e.g., chocolate), for groups of foods (e.g., desserts), and for food in general. Across such contexts, craving is traditionally assessed by asking individuals to self-report their current desire to eat, or the current intensity of their craving. To this end, researchers have used both multi-item questionnaires and single-item scales. For example, some studies have used the Food Cravings Questionnaire-State to assess current levels of craving for specific foods, which includes questions such as “I have an intense desire to eat [one or more specific foods]” [e.g., 28, 29–32]. Other studies have used the Food Craving Inventory to assess frequency of cravings for four specific categories of foods (sweets, fats, carbohydrates, and fast food fats); scores can also be calculated for food in general [e.g., 31, 33, 34, 35]. Other scales have also been used to assess frequency and intensity for food cravings, such as those developed by Weingarten and Elston [36, 37], and Hill [37, 38]. Further, many studies have assessed craving (including cue-induced craving) for specific foods by collecting responses to single-item, Likert-type or Visual Analog Scale, with questions such as “how much do you want to eat this food” or “rate the intensity of your craving” [e.g., 2, 39, 40–45].

## **Food cues, Craving, Eating, and the “Toxic Food Environment”**

In the literature, it has been consistently documented that food and drug cues increase craving. For example, an early meta-analysis by Carter & Tiffany (1999) showed that across drugs of abuse, participants demonstrated significant increases in self-reported craving when exposed to drug-related vs. neutral stimuli [46]. Similar data have been reported for food, such that exposure to food cues significantly increases craving for food [e.g., 25, 32, 40, 47–50]. Importantly, a large body of work has shown that food cue exposure not only increases physiological responses and craving, but also increases eating. Further, this body of work has also shown that the magnitude of cue reactivity and of cue-induced craving consistently

and reliably predicts subsequent eating as well as weight gain in both adults and children [9, 51]. We recently assessed this systematically in a meta-analysis that quantified the effects of food cue exposure, cue reactivity, and the experience of craving on eating and weight-related outcomes in both the short- and long-term [9]. Overall, we found medium effect sizes of food cue exposure, cue reactivity, and craving on eating and weight outcomes, suggesting that cue exposure and craving lead to eating and weight gain. Taken together, these data are consistent with the idea that food cues increase craving, and that craving then leads to eating. Another meta-analysis focused on a subset of studies that used food advertisements as cues; this meta-analysis reported a small-to-moderate effect size of advertisement exposure on eating, such that participants ate more after exposure to food advertising [51]. Together, these findings show that cue exposure increases eating and that the magnitude of cue reactivity and craving reliably predicts eating and weight-related outcomes. This suggests that they are important targets for the prevention and treatment of obesity.

Indeed, the implications of such findings extend to the natural ecology, where food cues for energy-dense “unhealthy” foods – such as advertisements for fast food – are abundant. We now live in an environment with pervasive advertisements (e.g., TV commercials, billboards) for low-nutrient, high-calorie foods [52, 53]. According to the Rudd Center for Food Policy & Obesity, food companies spent \$1.28 billion advertising snack foods in 2014, an increase of 4% from the \$1.23 billion spent in 2010 [54]. During this time, exposure to such advertising increased by 10% and 29% for children and teens, respectively [54]. In addition, the number of advertisements viewed by adults has steadily increased since 2007, growing from ~5,500 in 2007 to >7,000 in 2014 [55]. On a population level, this increase in food cues leads to increases in eating: not surprisingly, both adults and children who live in a food environment with advertised, high-calorie foods (e.g. fast food) eat those foods more frequently and have higher Body Mass Index [BMI; 56, 57–59]. Some have deemed this a “toxic food environment,” in which widespread food cues lead to increased food consumption and weight gain [60–62]. These outcomes have important public health implications. Today, over two-thirds of the United States population is overweight or obese, and obesity is the second leading cause of preventable morbidity and mortality in the United States [63, 64].

## Neural Mechanisms Underlying Cue Reactivity and Craving

Given the link between food cues, craving, and eating – and the possible role this has in the obesity epidemic – much neuroimaging work has been devoted to understanding the neural mechanisms underlying these processes. This work has revealed that cue reactivity and cue-induced craving express in brain regions that could serve as targets for modulation.

Prior brain imaging work in drug users has shown that cue exposure consistently increases craving along with neural activity in regions including the ventral tegmental area [VTA; e.g., 43, 65], ventral striatum [VS; e.g., 43, 66], amygdala [65, 67], insula [67, 68], orbitofrontal cortex [OFC; e.g., 43, 65, 66, 67], and anterior cingulate cortex [ACC; e.g., 43, 65, 66], as well as its subgenual region [sgACC; e.g., 43, 66]. These regions have previously been associated with emotion [particularly, the amygdala, VS, OFC, ACC; 69, 70, 71] and the experience of drug effects, which have been shown to activate the dopamine reward pathway

[e.g., 72, 73]. Importantly, these regions have also been implicated in food cue exposure and craving: similar studies have been conducted with food stimuli, and the regions involved are largely similar to those for drugs, in both healthy individuals and those with eating pathology [18, 24, 74].

Over the past decade, several meta-analyses have been conducted to elucidate the robustness and reliability of brain networks and regions involved in food cue reactivity. One meta-analysis identified the insula, a region commonly associated with gustatory/taste processing [75], to consistently respond to olfactory as well visual food cues [76]. Another meta-analysis focusing on response to visual food cues identified the OFC, insula, and fusiform gyrus, with hunger modulating the response in the amygdala and OFC, and energy content of the food modulating the response in the VS [77]. Other meta-analyses have focused on these processes in individuals with obesity, and found that these individuals exhibit greater activation of VS and ACC, along with lower activation in dorsolateral prefrontal cortex (dlPFC) in response to visual food cues, compared with healthy individuals [78, 79]. Studies have also suggested that self-reported craving correlates with neural activity in regions including VS and OFC [e.g., 43, 80]; results from one study further suggest that neural response to food cues in the VS predicts future weight gain [81]. Notably, this network of regions that are linked with cue reactivity and craving lies along the reward-related dopamine pathway [82], overlaps with the default mode network [83], is commonly activated in both positive and negative emotion [e.g., 70, 84], and can serve as a target for modulation. Indeed, these are the regions in which we would expect to see reduced activity if craving is effectively regulated.

## Strategies and Interventions to Reduce Craving and Eating

To address obesity on a population level, some public-health interventions have attempted to change the structure of the food environment by altering food prices (e.g., soda tax) or reducing availability of foods failing to meet specific nutrition standards [e.g., junk food bans; 85]. Other approaches have attempted to alter the choice architecture surrounding eating behaviors by “nudging” people towards healthy behaviors [i.e., by presenting food in smaller portions or having healthy optimal defaults in menus; 85, 86–88]. Unfortunately, these interventions have had relatively little success in real-world settings because they are limited in implementation and generalizability [89–91]. Further, even if such changes were successfully implemented, ultimately, most people would still be exposed to tempting food cues in situations in which these nudge strategies have yet to be implemented (e.g., in our own kitchens). Therefore, it is critical to consider the mechanisms underlying this epidemic and identify targets for interventions on an individual level.

For example, how can we reduce craving in an environment rife with food cues? Under tempting circumstances, we could exert self-control over our thoughts, feelings, and behaviors, which can be facilitated through the application of cognitive strategies [92]. Consistently, cognitive strategies for the regulation of craving are an important component of cognitive-behavioral treatments (CBTs) for obesity and eating disorders [93], which are known to be effective [94, 95]. We developed the Regulation of Craving (ROC) task to

experimentally measure the specific causal effect of regulation strategies on craving, as well as the neural mechanisms underlying craving and its regulation [e.g., 42, 43].

In the original study of the ROC task, cigarette smokers and healthy adults were presented with photographs of cigarettes and foods that had been previously shown to induce craving [42]. During each trial of the ROC task (see Figure 2 for schematic representation), participants were exposed to one of these cues, and were instructed to follow one of two instructions: NOW – focus on the immediate feelings associated with consuming the item (e.g., it will taste good), or LATER – focus on the long-term negative consequences associated with repeated consumption (e.g., this increases my risk for heart disease). Then, participants were asked to rate their craving for the specific food or cigarette they just saw, using a 1 (not at all) to 5 (very much) Likert-type scale (Figure 2). Importantly, the LATER instruction is a strategy drawn from CBTs for addiction, eating disorders, and obesity [e.g., 96, 97, 98]. In addition, its prospective thinking component is related more broadly to Episodic Future Thinking (EFT), which involves engaging episodic memory to vividly simulate future events [99, 100], and has been shown to reduce monetary delay discounting and caloric consumption [101–103]. Behaviorally, participants reported significantly reduced cravings for both food and cigarettes after applying the LATER strategy vs. the NOW instruction. In a follow up study, we used functional magnetic resonance imaging (fMRI) to probe the neural systems underlying these effects [43]. In this study, we replicated the original results, finding significant reductions in craving in the LATER condition. Further, we found that this cognitive down-regulation of craving was associated with decreased activation in regions implicated in craving (e.g., VS & sgACC, vmPFC), along with recruitment of regions associated with cognitive control (e.g., dorsolateral and ventrolateral prefrontal cortex; dlPFC, vlPFC). In addition, these two neural effects were found to be related to each other: decreases in behavioral reports of craving correlated with decreases in VS activity and were inversely correlated with increases in dlPFC activity. Importantly, VS activity fully mediated the relationship between dlPFC activity and reported craving, suggesting that the dlPFC could exert an inhibitory effect on VS, which in turn affects the experience and reports of craving.

The downregulatory effects of cognitive strategies on craving have been replicated many times, in both healthy populations and drug-using populations, for both food and drugs [44, 104–113]. In addition, the role of the dlPFC has been well established in the context of emotion regulation, and regulation of craving for drugs and food [e.g., 43, 71, 109, 114–117], as well as in EFT [100]. In subsequent studies, we also tested slightly different instructions words (e.g., NEGATIVE instead of LATER) and replicated the original findings as well (see below).

These promising effects led us to develop training protocols targeting cue reactivity and cue-induced craving, with the hopes that such preemptive training in cognitive strategies, prior to temptation, would improve subsequent eating behavior [P50 DA09241; 44, 118]. This approach has several advantages. First, cognitive strategies can be generated by the individual and are adaptable based on the situation, unlike externally-applied, situation-specific nudges. Second, rather than modifying the environment, training in cognitive strategies aims to alter the internal architecture of food choices, including how individuals

crave and value foods, allowing individuals to *nudge themselves* toward change. Such training may also alter the subjective valuation of healthy vs. unhealthy options during food choice. Indeed, it has been proposed that value computation of choice options is a key factor in successful self-control [119–122]. Consistent with this idea, computational work has suggested that food choices are driven (at least to some degree) by estimations of the benefit or value of possible outcomes. In turn, these estimations are based on (a) the value assigned to each item on various dimensions (e.g., the healthiness and tastiness of foods), and (b) the weight assigned to each dimension (e.g., importance of healthiness vs. tastiness) by any particular individual, at any particular timepoint [107, 122–126]. In turn, this further suggests that training in cognitive strategies – as described below – could alter either the value of options or the weight placed on dimensions of health and taste, thus leading to sustainable change.

To test this, we conducted several studies to develop an intervention to improve food choices and eating behavior [44]. First, we developed and tested two cognitive strategy instructions that frame foods as “bad for you” or “good for you.” The *NEGATIVE* strategy involves thinking about negative consequences of eating food (e.g. health consequences or disliking the taste), whereas the *POSITIVE* strategy involves thinking about the positive aspects of eating food (e.g. health benefits or liking the taste). Across two studies, we exposed participants to both healthy and unhealthy foods, and compared participants’ self-reported craving for these foods (assessed on a 1 to 5 Likert-type scale following each specific food) after following these instructions to a control *LOOK* instruction (i.e., just look at the food and respond naturally).

Consistent with prior work, we found that the *NEGATIVE* strategy decreased craving for both unhealthy as well as healthy foods. In contrast, the *POSITIVE* strategy increased craving for healthy as well as unhealthy foods. Importantly, we found that both strategies also influenced the subjective valuation of both healthy and unhealthy foods, operationalized as willingness-to-pay (WTP), such that the *POSITIVE* strategy increased WTP by 21–69 cents, and the *NEGATIVE* strategy decreased WTP by 56–80 cents, compared to the *LOOK* instruction. To illustrate this, participants in one of our studies were willing to pay \$1.28 (US dollars) more for healthy foods like broccoli, after using the *POSITIVE* vs. the *NEGATIVE* strategy. They were also willing to pay \$1.75 less for unhealthy foods like brownies after using the *NEGATIVE* strategy. Notably, this magnitude of value change is far greater than reported effects of other population-level interventions; it is at least 2.5–10x more than that of the proposed penny-perounce soda tax [127]. Finally, these cognitive strategies were effective across individual differences in BMI.

Based on these strategies (and ongoing work with cigarette smokers), we developed Regulation of Craving Training (ROC-T) as a novel and brief intervention [44]. ROC-T contains both a framing (informational essay) and a training component, and it trains participants to use cognitive strategies to either (a) increase craving for healthy foods (*POSITIVE* ROC-T condition), or (b) decrease craving for unhealthy foods (*NEGATIVE* ROC-T condition; conditions are italicized; strategies are not italicized). In the next set of studies, we tested whether ROC-T, compared to a *CONTROL* no-training “look-only” condition, could change choices of healthy vs. unhealthy foods as well as food consumption.

In these studies, participants completed a binary food choice task (e.g., healthy food vs. unhealthy food) before and after ROC-T (See Figure 3 for schematic representation of one choice; see [44] for schematics of the full studies). In one of the studies, participants also completed a “food taste test” following the second food choice task, in which we measured caloric intake. Across studies, we found that after completing ROC-T, participants in both the *POSITIVE* and *NEGATIVE* ROC-T conditions chose significantly more healthy vs. unhealthy foods, and that these participants consumed 93-121 fewer calories compared to participants *CONTROL*. Finally, in another study, we compared ROC-T to a framing-only control condition, and found that the training component of ROC-T is an important “active ingredient”; that is, the training component was necessary for maximal change in food choice to take place.

## Future Directions

Taken together, this work shows that cognitive strategies can be used effectively to regulate craving. Based on that initial insight, we developed a targeted, mechanism-focused, cognitive strategy-based intervention to prevent and reduce unhealthy eating that also alters the valuation of healthy and unhealthy foods. We are currently pursuing several avenues of work to extend these findings. For example, we are testing the boundary conditions of ROC-T, such as optimal “dose” and durability of effects under time pressure, stress, and cognitive load. In addition, we are investigating the neural mechanisms underlying the promising effects of ROC-T. We are also beginning to test the longevity of these effects, including the cumulative effects of repeated sessions and their effect on long-term outcomes such as weight, and to consider the effects of ROC-T on other constructs such as food reinforcement.

Further, dismantling and comparing the efficacy of specific components of the *POSITIVE* and *NEGATIVE* strategies is important to explore. Overall, there is limited work examining the efficacy of different strategies and their components in the context of craving, and existing evidence does not clearly indicate that certain strategies are differentially effective. For example, prior research in cigarette smokers has tested the efficacy of gain-framed (i.e., benefits of quitting smoking) vs. loss-framed messages (i.e., costs of continuing to smoke) on smoking cessation. This work suggests that gain-framed messages may be more effective in the short-term [128–130], but not the long term [128], and that this difference is influenced by gender and risk perceptions associated with quitting smoking [130]. In our own work testing regulation of craving training (ROC-T), we found that the *NEGATIVE* ROC-T and *POSITIVE* ROC-T condition were equally effective in improving food choice across three of four studies [44]. Further, in another study testing the regulation of food craving participants were allowed to select cognitive strategies from a suggested list [113]. The authors reported that the most commonly applied strategies were (1) focusing on the negative consequences of eating the food (as in our ROC task), and (2) imagining that something was wrong with the food [113]. However, reduction in craving did not vary by strategy [113]. Thus, it is important to explore additional strategies that may be differentially effective (e.g., mindfulness), and to further ask whether there are individual differences in strategy choice and efficacy.



In addition, because the food environment often presents more complex situations than making binary decisions between “healthy” and “unhealthy” foods, one useful next step would be to investigate foods that are often thought of as healthy but are also energy-dense (e.g., nuts, avocados) and thus might be detrimental to goals related to weight loss. Thus, we hope to make ROC-T adaptable to different eating and weight goals. Ultimately, we hope that ROC-T can serve as a standalone computerized intervention as well as “homework” as part of longer interventions for healthy eating, weight loss, obesity, and potentially for eating disorders.

## Conclusion

Craving – in the context of food – is defined as a strong desire to eat, and has troubled philosophers for centuries [5–7]. It is a common experience [8] that drives eating behavior and predicts weight gain over time [9]. Craving can be understood using a learning-based model of behavior, where it has been formalized within the framework of classical or Pavlovian conditioning [10]. Within this framework, food cues (such as sight or smell of food, food pictures, and advertisements) that are present at the time of eating become conditioned stimuli that acquire the ability to predict eating, and evoke conditioned responses such as increased salivation [11], heart rate [12, 23], gastric activity [12, 23], and neural activity in regions typically associated with reward [e.g., VS; 18, 24]. In humans, these responses are often in tandem with the conscious experience of craving, or “cue-induced craving” [25, 26]. As such, craving can be considered to be a type of cue reactivity, or an emergent property from it.

This is important clinically because we now live in an environment with pervasive food cues, such as TV advertisements [52, 53]. Over the past decade, industry spending on advertisements for low-nutrient, high-calorie foods and population exposure to such advertisements have steadily increased [54, 55]. Experts have deemed this a “toxic food environment,” in which abundant food cues lead to increased eating and weight gain [60–62], exacerbating the obesity epidemic [63, 64]. Indeed, we and others have used systematic and quantitative meta-analytic techniques to show that cue exposure increases eating, and that cue-induced craving reliably predicts eating and weight outcomes [9, 51]. Thus, they are promising targets for the prevention and treatment of obesity.

Thus, we developed and tested an individual-level, internally-applied intervention based on prior work showing that cognitive strategies for the regulation of craving successfully reduce self-reported craving and reward-related neural reactivity to unhealthy food cues [e.g., 43]. We first demonstrated that cognitive strategies decrease craving for unhealthy foods by emphasizing their negative consequences, increase craving for healthy foods by emphasizing their positive benefits, and importantly, change food valuation (willingness to pay) for both healthy and unhealthy foods [44]. In addition, we showed that Regulation of Craving Training (ROC-T) – a novel intervention based on these cognitive strategies – significantly increased healthy food choice and reduced caloric consumption [44]. Finally, we found that the training component of ROC-T was necessary for maximal change in food choice [44]. To extend these promising findings, we are now investigating the boundary conditions, neural mechanisms, and longevity of ROC-T. Taken together, these findings and future directions

have important theoretical, public health, and clinical implications for the prevention and treatment of obesity and eating disorders.

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### Highlights

- Craving – defined as a strong desire to eat – is a common experience
- Food craving predicts eating and weight gain
- Cognitive strategies can be used to regulate craving
- Training in cognitive strategies reduces eating and improves food choice



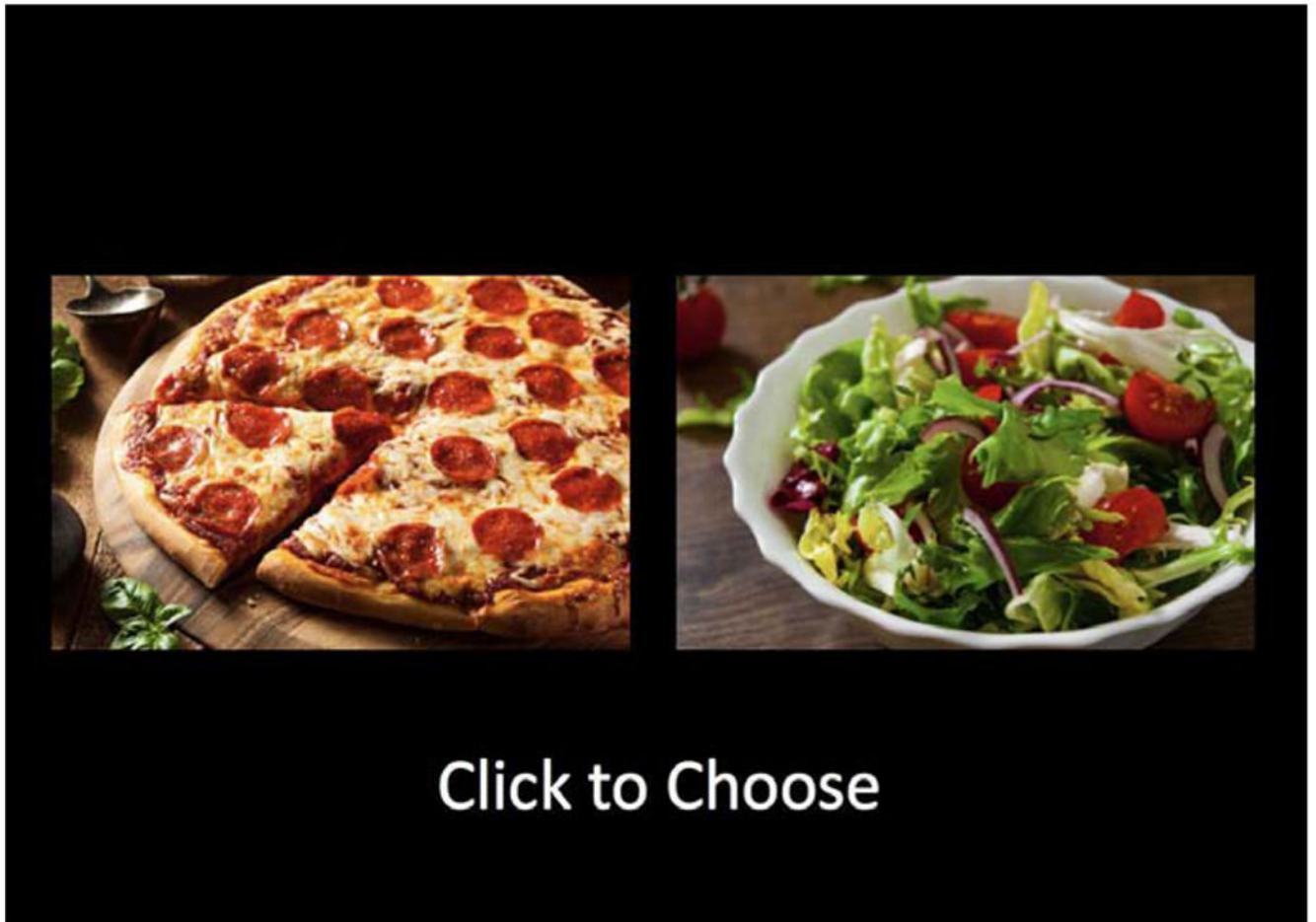
**Figure 1.**  
Brownie for craving demonstration. Image courtesy of [istockphoto.com/portfolio/anthonysp](https://www.istockphoto.com/portfolio/anthonysp).



**Figure 2.**

Regulation of Food Craving (ROC) Task

Schematic representation of one trial from the original Regulation of Craving (ROC) task. Participants first saw a fixation cross, followed by an instruction. Specifically, NOW instructed participants to focus on the immediate feelings associated with consuming the item (e.g., it will taste good). LATER – a strategy drawn from CBT treatment protocols – indicated that they should focus on the long-term negative consequences associated with repeated consumption (e.g., this increases my risk for heart disease). Instructions were followed by an image, including unhealthy foods, after which participants rated their craving. Pizza image courtesy of [istockphoto.com/portfolio/bhofack2](https://www.istockphoto.com/portfolio/bhofack2).



**Figure 3.**

Food Choice Task

Schematic representation of one trial from the Food Choice Task. Before and after ROC-T, participants completed binary food choices, most of which were between healthy and unhealthy foods (other choices were between healthy vs. healthy or between unhealthy vs. unhealthy foods). Pizza image courtesy of [istockphoto.com/portfolio/bhofack2](https://www.istockphoto.com/portfolio/bhofack2). Salad image courtesy of [istockphoto.com/portfolio/semenovp](https://www.istockphoto.com/portfolio/semenovp).