Research Report

Cue-elicited craving for food: a fresh approach to the study of binge eating

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Abstract

Recent research has indicated that craving for food can be elicited by exposure to food cues, suggesting that exposure to food cues may represent a useful experimental paradigm to investigate mechanisms related to binge eating. The first objective of the present research was to replicate previous reports that exposure to food cues elicits craving for food. In addition, this investigation was designed to extend the extant literature by testing the effects of 'priming' portions of food, by examining the association between reactivity to food cues and indicators of binge eating, and to examine the role of a putative genetic factor previously found to be associated with cue-elicited craving for alcohol and tobacco. In Study 1, 48 individuals completed measures of craving and mood after exposure to control cues, after exposure to food cues, and after consuming each of three small portions of food. In Study 2, 31 individuals with subclinical symptoms of binge eating completed the same procedures. The results suggested that food cues reliably elicited craving, increased attention to the cues, and decreased positive affect in both samples, although reactivity was greater among the sample with greater eating pathology. Correlational analyses suggested that reactivity to food cues was correlated with binge eating and body mass index among women but not men. Results also suggest that the DRD4 VNTR polymorphism influences cue-elicited craving for food, although the influence of the DRD4 may depend on the population under study.

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Obesity is recognized as a major health concern in the United States. It is associated with numerous serious health problems such as heart disease, diabetes, certain cancers (colon, breast), and stroke (Pi-Sunyer, 1999) and the raised mortality (Allison, Heo, Kaplan, & Martin, 1999). Despite the overwhelming evidence that obesity is a major contributor to mortality and morbidity and the associated socioeconomic costs, the prevalence of obesity continues to rise (Mokdad et al., 2000). According to the National Health and Nutrition Examination Survey 1999–2000, the estimated prevalence of obesity (BMI greater than or equal to 30.0) has doubled between NHANES II (1976–1980) and NHANES (1999–2000), from approximately 15% to an estimated 31% (Flegal, Carroll, Ogden, & Johnson, 2002).

Obesity is clearly a complex problem that must be broken down into its constituent parts in order to better understand the etiology of the larger problem. Binge eating is one behavior that is central to the etiology and severity of obesity (Bulik, Sullivan, & Kendler, 2002). Not surprisingly, several studies have found an association between nonpurging binge eating behavior obesity (Crow, Kendall, Praus, & Thuras, 2001). Furthermore, research has demonstrated a dangerous cycle: as weight increases, the frequency of binge episodes increases in adults (Telch, Agras, & Rossiter, 1988) as well as in children (Morgan et al., 2002). Up to 46% of people who are obese report binge eating (for review see Bulik et al., 2002). The concurrent presence of binge eating and obesity tends to

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predict greater medical and psychological pathology (Bulik et al., 2002), suggesting both a greater need for treatment and possibly greater obstacles to treatment success.

Although obesity and binge eating are major targets of prevention and treatment efforts, current efforts are only modestly effective. To develop more effective prevention and treatment efforts, it is important to develop a better understanding of the specific mechanisms that underlie binge eating and obesity. It is equally important to develop a better understanding of individual differences in these mechanisms that might guide future efforts to match particular individuals with the most effective treatments or guide efforts to target at-risk populations to alter problematic eating behaviors before weight has significantly increased. The precursors and mechanisms of binge eating are still largely unknown, and genetic factors or other individual differences that may influence binge eating remain unidentified. Perhaps, most importantly, a laboratory paradigm to study these mechanisms has been lacking.

One paradigm that has some promise has been recently adopted from the addiction literature (e.g. Carter, Bulik, McIntosh, & Joyce, 2002; Legenbauer, Vögele, & Rüddel, 2004). This paradigm involves exposure to cues associated with alcohol and drug use (or binge eating) to elicit craving and related responses. In the addiction literature, numerous studies have demonstrated that exposure to conditioned stimuli associated with drug use leads to conditioned appetitive responses. For example, there is an extensive body of research demonstrating that exposure to drug-related stimuli increases craving for a variety of drugs (for reviews see Carter & Tiffany, 1999; Niaura et al., 1988). Furthermore, increased reactivity to cues has been shown to predict increased likelihood of use of the cued substance (for reviews, see Jensen, 1998). Although several studies have examined craving for food and responses to external food cues as possible correlates of binge eating (Carter et al., 2002; Legenbauer et al., 2004), it is unclear whether cue-elicited craving precipitates loss of control over eating and/or a binge episode. However, several studies have shown that people with restrained eating habits are more likely to overeat after exposure to an external cue such as the thought or smell of a food on which they typically binge (Rogers & Hill, 1989; Fedoroff, Polivy, & Herman, 1997). Other studies have demonstrated that physiological responses to food ingestion can be elicited by exposure to cues such as odor, smell, and taste (Wardle, 1990; Woods, 1991). A recent study demonstrated reactivity to food cues, including increases in craving for food, and a positive correlation between this reactivity and eating disorder symptomatology (Carter, Bulik, McIntosh, & Joyce, 2002). Finally, while many studies have examined the influence of genetic factors on obesity (e.g. Barsh, Farooqi, & O’Rahilly, 2000), very few have investigated the influence of genetic factors on cue-elicited craving and loss of control over food consumption in humans.

**Biological mechanisms associated with obesity and binge eating**

Much research has been dedicated to finding possible biological antecedents to the highly prevalent problems of binge eating and obesity. Researchers have implicated such biological correlates to obesity and binge-eating as leptin and the leptin receptor (Adami, Campostano, Cella, & Ferrandes, 2001; Comuzzie & Allison, 1998) and the beta3 adrenergic receptor (Clement et al., 1995; Widen et al., 1995), both of which are biological differences involving metabolism and the role of uncoupling proteins in energy regulation (Gura, 1998). More recently, the role of genetic factors in the etiology of eating pathology, including obesity, has been studied with limited success (Barsh et al., 2000). The heritability of BMI is estimated between 0.4 and 0.7, suggesting genetic factors are important in the development and possibly maintenance of high body weight (MacDonald & Stunkard, 1990; Stunkard, Harris, Pedersen, & McClearn, 1990). Obesity and binge eating are likely multi-genetic, with many systems involved in the expression of eating pathology. The genetic contribution to binge eating and obesity may overlap, and both have been shown to be heritable (Bulik, Sullivan, & Kendler, 2003), however, the specific pathways of the genetic and behavioral contributions to binge eating and obesity remain unclear.

In order to better identify genes that are associated with behavioral disorders such as obesity and substance abuse, behavioral scientists have begun to emphasize the identification of a behavioral phenotype, or endophenotype, that is related to the larger disorder, rather than searching for a gene that expresses a behavioral disorder itself (Burmeister, 1999; Hill & Neiswanger, 1997; Hutchison, LaChance, Niaura, Bryan, & Smolen, 2002; Hutchison, McGearry, Smolen, Bryan, & Swift, 2002; Leboyer et al., 1998; Malhotra & Goldman, 1999, 2003). To increase power to detect significant associations with particular candidate genes and facilitate the interpretation of the findings, an ideal endophenotype should be narrowly defined, easily identifiable, empirically related to the clinical manifestation and the biological mechanism of the disorder, and theoretically related to a candidate gene. With respect to binge eating and obesity, cue-elicited craving for food and the acute loss of control over eating may represent a useful endophenotype that meets these criteria.

It is likely that there is overlap between the biological mechanisms that mediate appetitive motivation for alcohol and drugs and the mechanisms that mediate appetitive motivation for food (Robinson & Berridge, 2000). With respect to mechanisms involved in the appetitive motivation for alcohol and tobacco, recent studies have suggested that activation of mesolimbic dopamine substrates is critical to the development of the motivational and appetitive properties of tobacco, alcohol, and other drugs. This biological conceptualization of craving, which has recently been articulated as an incentive sensitization model of addiction,
stipulates that mesolimbic dopamine activation influences the motivational and appetitive properties of alcohol and drugs by controlling the attribution of incentive salience to neural representations of drug related stimuli (Berridge & Robinson, 1998; Wise, 1988). Other studies have implicated these same mechanisms in terms of the appetitive and motivational properties of food. For example, single cell recording studies in primates have documented the role of mesolimbic dopamine neurons in response to food cues (Schultz, 2001, 2002). Research with rodents has also indicated a role of mesolimbic dopamine in response to food cues (Bassareo & Di Chiara, 1997). Thus, the acquisition and sensitization of incentive salience for food may follow a similar process.

Consistent with animal models, recent research with humans has suggested that consumption of small doses of alcohol increases craving relative to consumption of non-alcoholic beverages and that this craving is attenuated by an atypical dopamine antagonist that acts on D2 and D4 receptors (Hutchison, McGeary, Smolen, & Wooden, 2001; Hutchison et al., 2003). Likewise, genetic factors that may influence the function of dopamine receptors (e.g. the DRD4 VNTR) also appear to influence cue-elicited craving for alcohol and tobacco (Hutchison & LaChance, 2002; Hutchison, McGeary, Smolen, Bryan, & Swift, 2002). Given the putative role of the DRD4 VNTR and the more general role of dopamine, it seems likely that some of these same pharmacological and genetic factors are also involved in craving for food and loss of control over eating.

Objectives of the current study

The first objective of the present research was to replicate previous reports that exposure to food cues elicits craving for food. In addition, this investigation was designed to extend the extant literature by testing the effects of ‘priming’ portions of food, by examining the association between reactivity to food cues and indicators of binge eating, and by investigating the role of a putative genetic factor previously found to be associated with cue-elicited craving for alcohol and tobacco. Finally, to further validate the usefulness of this experimental approach, the experiments were conducted in two different samples; one that was screened for the presence of eating pathology (e.g. dietary restraint and binge eating) and one that was not.

Methods

Participants

In Study 1, a total of 48 (20 male, 29 female) participants were recruited from introductory psychology courses. Participants were not aware of the study’s objectives until arrival to the experiment. Participants reported no current or past treatment for eating disorders as defined by DSM-IV-TR (American Psychiatric Association, 2000), no current substance abuse or dependence, and were English speaking and able to read and write. The sample was primarily Caucasian, and the ethnicity of the remaining subjects was Asian, African-American, and other minority groups, which reflected the ethnic breakdown of the University of Colorado at Boulder.

In Study 2, a total of 31 female participants between the ages of 18 and 35 were recruited for by advertisements for an ‘Eating Study’ posted on campus and the surrounding areas. The sample was primarily Caucasian, and the ethnicity of the remaining subjects was Asian, African-American, and other minority groups. Inclusion criteria were as follows: self-reported binge eating at least once per month, Body Mass Index between 18 and 26, no current or past treatment for eating disorders as defined by DSM-IV-TR (American Psychiatric Association, 2000), no current substance abuse or dependence, and not currently prescribed psychotropic medications, and must be English speaking, and able to read and write. Both studies were reviewed and approved by the University of Colorado Human Research Committee.

Baseline measures

Beck Depression Inventory-II. The BDI-II consists of 21 scaled statements designed to assess symptoms of depression, with a coefficient alpha of 0.92 (White & Labouvie, 1989).

Binge Eating Scale (Hawkins & Clement, 1980). The BES is a 16-item measure assessing binge eating behavior and feelings surrounding a binge. It successfully discriminates between participants with no, moderate, and severe binge eating problems as judged by a trained interviewer. It also has moderately high internal consistency (0.85) (Gormally et al., 1982).

Body Shape Questionnaire (Cooper, Taylor, Cooper, & Fairburn, 1987). The BSQ consists of 34 statements designed to assess individual behaviors and thoughts about eating and body shape.

A demographics instrument was used to determine gender, age, detailed ethnicity, SES, occupation, and income.

Three Factor Eating Questionnaire (Stunkard & Messick, 1985). The TFEQ is a 51-item self-report measure with three subscales: cognitive restraint, perceived hunger, and disinhibition of eating.

Experimental measures

Attention Form. The attention form consists of three items designed to assess attention to the cue. Craving and Mood Questionnaire. The CMQ consists of eleven items that were rated on a scale of 0–100 and that were combined to form a scale measuring craving for food, adopted from the same measure designed to measure craving for cigarettes (Hutchison, Niaura, & Swift, 2000). Cronbach’s alpha for the CMQ in the present study was greater than 0.90, suggesting good internal consistency.
**Profile of Mood States** (McNair, Lorr, & Dropleman, 1971). The POMS uses bipolar adjective scales to assess mood on a number of dimensions (e.g. vigor, tension, depression, happiness).

**DNA Collection and Analysis.** Genomic DNA was isolated from buccal cells collected with cheek swabs (Freeman et al., 1997; Lench, Stanier, & Williamson, 1988). The 48 bp VNTR in the third exon of the DRD4 was assayed using previously reported methods (Sander et al., 1997). Details of this collection procedure and the assay for the DRD4 VNTR are described elsewhere (Hutchison & LaChance, 2002; Hutchison & McGearry, 2002). Consistent with previous studies, individuals with at least one copy of the 7 repeat allele (or longer) were classified as DRD4 L and individuals with both copies of less than 7 repeats were classified as DRD4 S.

**Procedures**

If participants met the previously mentioned inclusionary criteria, they were scheduled and asked what foods they had experienced craving for and enjoyed eating in the past. They were also instructed to finish eating what they considered to be a normal meal 3 h prior to coming in for the experimental session. Participants came to the lab for one 2-hour session. Upon arrival, participants filled out a battery of assessment forms on mood, substance use, and risk-taking behavior, and had a DNA sample taken (further explained below).

Participants consumed one serving of food (as defined by FDA labeling of Nutrition Facts) split into three small portions. Prior to the experiment, the participant was asked to report examples of food s/he enjoyed most and had experienced cravings for in the past. Experimenters were nearly always able to provide the participant with one of the specified foods.

The experimental portion of the session consisted of five trials: pencil cue (neutral stimulus cue, designed to provide a baseline measure of craving), food cue (in which the subject was presented with her preferred food, and instructed to focus on it, think about what it would be like to eat it, but not taste it), and three eating trials. Each cue and eating trial lasted 3 min. Then the participant filled out forms assessing craving, mood, and rating of the food when applicable, with approximately 8 min between the trials. Participants consumed each serving of food at an even pace over 3 min and then relaxed for 3 min before beginning a battery of self-report measures. The second and third eating trials followed the same procedures.

**Results**

*Study 1—non-clinical population*

In Study 1, we tested the protocol using a non-clinical population from an introductory psychology subject pool.
the food cue (see Fig. 1b). There was no significant effect for the DRD4 or an interaction ($p > 0.05$).

**Changes in Mood.** We restricted our analyses to the elation, depression, and tension subscales of the POMS. We found a significant main effect of DRD4 on elation (POMS), $F(1,44) = 4.8, p < 0.05$, such that those with the risk allele reported, on average, significantly lower ratings of elation than participants without the risk allele. We also found a significant main effect of Cue on elation (POMS), $F(1,44) = 16.1, p < 0.0001$, such that happiness decreased after presentation of the food cue (see Fig. 1c). The interaction was not significant ($p > 0.10$). We also found a significant main effect of Cue on tension (POMS), $F(1,44) = 11.45, p < 0.002$, such that tension increased after presentation of the food cue (see Fig. 1d), but no significant main effect for DRD4 or a significant interaction.

**Effects of ‘Priming’ portions**

**Craving.** There was a significant main effect of the DRD4 on craving (CMQ), $F(1,43) = 9.48, p < 0.004$, such that participants with the risk allele reported significantly more craving, on average across the three portions, than participants without the risk allele. There was also a significant main effect for Portion, such that craving generally decreased across the three trials (see Fig. 2a), $F(2,88) = 15.39, p < 0.01$. The interaction was not significant ($p > 0.10$).

**Mood.** We found a significant main effect for the DRD4 (see Fig. 2b), $F(1,41) = 4.86, p < 0.05$, such that participants with the risk allele felt, on average, more depressed (POMS) versus participants without the risk allele. We found no significant effects with the happiness or tension subscales ($p > 0.10$).

**Study 2**

In Study 2, we tested the protocol on a population with sub-clinical eating concerns (e.g. binge eating) but who did not meet full criteria for an eating disorder as defined by the DSM-IV-TR.

**Cue reactivity**

**Craving.** Analysis revealed a significant main effect for Cue on craving (see Fig. 3a), $F(1,27) = 18.86, p = 0.0002$, such that craving increased significantly after presentation of the food cue. The interaction and main effect of the DRD4 were not significant ($p > 0.05$).

**Attention.** We found a significant main effect of Cue on attention (see Fig. 3b), $F(1,27) = 9.87, p = 0.004$, such that attention increased significantly after presentation of the food cue. There was no significant effect for the DRD4 or an interaction ($p > 0.05$).

**Mood.** We found a significant main effect of trial on happiness (see Fig. 3c) (POMS), $F(1,27) = 7.47, p = 0.011$, such that happiness decreased after presentation of the food cue. There was no significant effect of the DRD4 or an interaction ($p > 0.05$).

**Effects of ‘Priming Portions’**

**Craving.** We found a significant Portion by DRD4 interaction (see Fig. 4a), $F(1,27) = 4.13, p < 0.02$, such that participants with the risk allele reported significantly decreased levels of craving on subsequent eating trials than participants without the risk allele. There was no significant main effect for the DRD4 ($p > 0.05$).

**Mood.** We found no significant effects of depression, happiness, or tension (POMS).
Analyses of sample differences, gender differences, and correlations between cue-reactivity and binge eating and BMI

Subsequent analyses compared the individuals in the two samples (those screened for the presence of binge eating and those not explicitly screened for binge eating) on a number of eating-related variables (see Table 1). With respect to eating pathology, the sub-clinical group reported significantly more eating pathology on the Eating Attitudes Test ($p < 0.05$), significantly higher levels of body shape concerns on the Body Shape Questionnaire ($p < 0.05$), significantly higher levels of disinhibition on the disinhibition subtest of the Three-Factor Eating Questionnaire ($p < 0.05$), and significantly higher levels of binge eating pathology on the Binge Eating Scale ($p < 0.05$). In addition, analyses revealed that the two different populations were significantly different in terms of their craving and tension levels.

Table 1

<table>
<thead>
<tr>
<th>Measure</th>
<th>Study 1 (non-clinical) mean (STD)</th>
<th>Study 2 (sub-clinical) mean (STD)</th>
<th>T-test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Binge eating scale</td>
<td>2.02 6.45</td>
<td>6.45 10.07</td>
<td>$t(77) = 6.93, p &lt; 0.0001$</td>
</tr>
<tr>
<td>Disinhibition (TFEQ)</td>
<td>8.57 10.07</td>
<td>10.07 13.32</td>
<td>$t(78) = 3.34, p &lt; 0.05$</td>
</tr>
<tr>
<td>Body Shape questionnaire</td>
<td>80.60 103.32</td>
<td>103.32 113.34</td>
<td>$t(77) = 2.8, p &lt; 0.05$</td>
</tr>
<tr>
<td>IAQ</td>
<td>46.94 56.77</td>
<td>56.77 65.77</td>
<td>$t(77) = 4.28, p &lt; 0.0001$</td>
</tr>
<tr>
<td>EAT</td>
<td>110.19 133.47</td>
<td>133.47 141.47</td>
<td>$t(78) = -4.54, p &lt; 0.05$</td>
</tr>
<tr>
<td>Hunger (TFEQ)</td>
<td>9.12 7.16</td>
<td>7.16 7.16</td>
<td>$t(78) = 3.17, p &lt; 0.05$</td>
</tr>
</tbody>
</table>
after exposure to the food cues ($p<0.05$) as well as after consuming the priming portions ($p<0.05$).

When combining the two samples, female participants reported significantly higher rates of binge eating behavior ($p<0.05$), greater BMI ($p<0.05$), disinhibition ($p>0.05$), overall eating pathology on the EAT ($p<0.05$), and greater body shape concerns ($p<0.05$). Male and female participants did not differ significantly in terms of reactivity to the food cues ($p>0.05$).

Correlations were calculated to determine whether reactivity to food cues was associated with the presence of binge eating behavior and BMI. Correlations were calculated separately for men and women given the gender differences in BMI and binge eating behavior. Among females, cue-elicited craving (i.e. the increase in craving after presentation of the food cues) was significantly associated with binge eating behavior ($p<0.05$), such that greater increases in craving after exposure to food cues was positive correlated with greater frequency of binging (see Table 2). In addition, the correlation between change in craving across the three priming portions and BMI was marginally significant ($p=0.07$) and the correlation between change in craving and binge eating behavior was significant ($p<0.05$), such that greater decreases in craving across the three portions were related to lower BMI and lower levels of binge eating behavior. In terms of mood reactivity, greater levels of tension were significantly correlated with lower BMI, perhaps suggesting that women who experience greater tension during the presence of food are also the women who are more likely to restrict their diets, thereby maintaining a lower BMI. Among men, none of the correlations were significant. However, this may have been related to low statistical power due to a small number of men who completed the experimental protocol.

### Discussion

In the present study, we built on previous investigations of reactivity to food cues by assessing craving for food after the presentation of a food cue as well as after consuming ‘priming portions’ of a preferred food and by testing the influence of the DRD4 gene. Consistent with previous studies (e.g. Carter et al., 2002), the results demonstrated that craving for food could be manipulated using laboratory procedure similar to those used in studies of reactivity to alcohol and tobacco cues. The findings of the present studies indicate that reactivity to food cues can be readily elicited in the laboratory, that reactivity to food cues is associated with binge eating and BMI, and suggests that the DRD4 VNTR polymorphism influences cue-elicited craving for food, although the influence of the DRD4 may depend on the population under study. Overall, the results suggest that this experimental paradigm has considerable promise as a means of identifying and testing basic behavioral and biological mechanisms related to binge eating.

To investigate and elucidate mechanisms that underlie binge eating and obesity, it is important to first establish the validity of a theory-based experimental paradigm that can be used to systematically manipulate factors that putatively underlie loss of control over eating. The present study demonstrated that reactivity to cues differs in a sample of individuals explicitly screened for the presence of problematic eating behavior and one that was not. Furthermore, the results indicated that a greater degree of binge eating behavior was correlated with greater cue-elicited craving for food and greater craving across the presentation of priming doses of food. Thus, the present study suggests that this experimental paradigm may be sensitive to the processes and mechanisms that distinguish individuals with and without loss of control and binge eating. The implication of these findings is that this experimental paradigm may be used in the future to examine how individuals with different types of symptoms differ in terms of reactivity to food cues, to screen psychosocial or pharmacological interventions that may alter reactivity to food cues, or to screen genetic factors that may influence reactivity to food cues, loss of control, and binge eating.

The finding that the DRD4 VNTR influenced craving for food reinforces the notion that this paradigm may be useful
for identifying genetic factors that may influence binge eating. Interestingly, analyses revealed that the genetic effect on craving differed between the two populations on which we tested this protocol. One possible explanation of why participants in the subclinical sample responded differently than participants in the non-clinical sample is that people with more eating pathology may be more affected by cognitive factors such as conscious attempts to deny hunger or by the loss or inability to recognize hunger cues. Participants in the subclinical group reported higher levels of binge eating, disinhibition, body shape concerns, and eating pathology in general. Other reports have suggested that women with these concerns may actually report less desire for food yet binge eat at greater levels (Waters, Hill, & Waller, 2000). Thus, cognitive factors may influence the self-reported desire for food among these women and create an inconsistency between their self-reported desire for food and actual behavior. In turn, this may result in a different finding regarding the association of the DRD4 VNTR with self-reported craving. The implications of this finding are two-fold: this experimental paradigm may be sensitive to cognitive factors that influence self-reported craving for food and cognitive factors that influence self-reported craving must be considered when investigating genetic factors that may be associated with self-reported craving. Thus, the inclusion of psychophysiological measures that are less susceptible to artifact and behavioral measures that may reflect the effect of cue exposure on subsequent binge eating behavior (e.g., changes in respiration or salivation, measurement of startle response and heart rate, measures of post cue exposure food consumption) may be particularly important for future iterations of this experimental paradigm.

Although more research is necessary to compare and contrast the mechanisms influencing craving for food as opposed to craving for alcohol and other drugs, these results are consistent with the results of a recent studies suggesting that the DRD4 VNTR influences craving for alcohol (Hutchison, LaChance et al., 2002; Hutchison, McGearly et al., 2002; Hutchison et al., 2003) and tobacco (Hutchison, LaChance et al., 2002) and consistent with previous studies demonstrating that appetitive motivation for food is mediated by some of the same mechanisms as appetitive motivation for alcohol and drugs (Kelley et al., 2002; Pelchat, 2002). Irrespective of speculation about the implications of these findings, it is clear that this experimental paradigm holds promise in terms of identifying and testing basic behavioral, biological and cognitive mechanisms that underlie eating pathology. Further research should be undertaken with this experimental paradigm. Limitations of the present investigation that should be addressed in future studies include a narrow range of BMI across participants and insufficient numbers of male participants, which limits the generalizability of these findings.

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