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Negative affect influences the computations underlying food choice in bulimia nervosa

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This study makes a **valuable** contribution to understanding how negative affect shapes food-choice decision making in bulimia nervosa by using a mechanistic drift diffusion model to quantify the weighting and temporal integration of tastiness and healthiness attributes. The approach is **solid** and has clear potential to advance understanding of the decision processes underlying pathological food choices. The evidence is strengthened by the randomised crossover design and appropriate statistical analyses. The results are consistent across different analytic approaches, increasing confidence in the robustness of the findings.

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Abstract

Individuals often consume tasty, calorically dense foods in response to negative emotions, a phenomenon exemplified by notions of "stress eating" and "comfort food." While this link between food and mood can become pathological in binge eating, the decision-making processes underlying this link are poorly understood. Here, we investigated the impact of acute increases in negative affect on when and how strongly the perceived tastiness and healthiness of foods influence food choices in healthy adults and individuals with bulimia nervosa (BN), an eating disorder characterized by cycles of over- and underconsumption of food. In a randomized crossover design, 25 women with BN and 21 healthy controls completed two sessions where they received either a neutral or negative affect induction and then completed a food choice task. Using a time-varying diffusion decision model, we assessed how negative affect influences food choice dynamics for high- and low-fat foods. In the neutral affect condition, individuals with BN considered tastiness relative to healthiness of high-fat foods sooner than healthy controls but maintained a restrictive food choice policy by reducing the weight on tastiness. After a negative affect induction, both groups showed a stronger bias towards considering tastiness before healthiness, but this bias was exaggerated in individuals with BN. This affect-induced bias for high-fat foods predicted more frequent subjective binge episodes over three months. These results provide insights into how negative emotion influences food choices and may explain why binge eating in BN is more likely during high negative affect, while dietary restriction is more likely during low negative affect.

Introduction

Every day, multiple times a day, people must make decisions about what to eat. A growing body of research suggests that these decisions involve considerations of different attributes of the available food options (e.g., healthiness and tastiness), and that these attributes often present the decision-maker with challenging conflicts (e.g. "Should I choose the healthier, less delicious food or the better tasting, less healthy food?"). Difficulties resolving these conflicts can take on pathological forms in individuals with eating disorders, and some eating disorders are characterized by food choices that are biased toward one extreme (e.g., toward subjectively less tasty, but healthier, low-fat foods in anorexia nervosa; (Foerde et al., 2015)). However, food choices seem to episodically oscillate *between* extremes in the case of bulimia nervosa (BN), which is commonly marked by prolonged periods of rigid dietary restriction punctuated by out-of-control binge eating of highly palatable foods. Although subclinical forms of this oscillation are ubiquitous in examples of "yo-yo dieting" and "dietary lapses," surprisingly little cognitive neuroscience research to date has attempted to explain it.

Self-report data suggest that rising negative affect is one reliable predictor of the switch from more to less restrictive food choices (Konttinen et al., 2010). Although some individuals reduce intake in response to stress (Hill et al., 2022), research using both animal models and healthy adult samples show that stress can precipitate the overconsumption of highly palatable foods (Dionysopoulou et al., 2021; Jacques et al., 2019; Tomiyama, 2019). This connection between negative emotion and food choice is even more pronounced in binge eating. Inducing negative affect in the laboratory environment disproportionately increases food intake among individuals with binge eating (Telch & Agras, 1996). Ecological momentary assessment data show that binge eating in the natural environment is also much more likely to occur in the context of increasing and unstable negative emotions (Alpers & Tuschen-Caffier, 2001; Berg et al., 2013; Haedt-Matt & Keel, 2011; Hilbert & Tuschen-Caffier, 2007; Smyth et al., 2007). In addition, reactivity to negative emotions, or negative urgency, has been shown to predict or correlate with more frequent binge eating in individuals with BN (Fischer et al., 2013; Schnepper et al., 2021).

Although momentary increases in negative emotions are closely tied to the overconsumption of highly palatable foods, the cognitive processes underlying this tight link, particularly in the case of binge eating, are not well understood. Studies to date have failed to find support for the notion that increased negative affect, specifically stress, impairs inhibitory control processes in healthy adults (Allen et al., 2022) or in BN (Dreyfuss et al., 2017; Westwater et al., 2021). While another study found that healthy adults and individuals with BN differed in how stress impacted visual processing-related neural activity, they failed to find support for the hypothesis that increased negative affect abnormally increased limbic neural responses to palatable food stimuli in BN (Collins et al., 2017).

We recently tested the possibility that negative affect abnormally impacts decision-making in BN by promoting food choices that include the high-fat, appetitive foods that are typically consumed during binge-eating episodes (Gianini et al., 2019). Individuals with BN and healthy controls completed a food choice task after a negative and neutral affect induction (Figure 1). Counter to expectations, negative affect did not influence ultimate food choices in either group, and women with BN showed restrictive food choices whether neutral or negative affect was induced: they chose low-fat foods more often (Figure 2A), and these choices were strongly predicted by healthiness ratings (Figure 2B). However, these analyses focused primarily on individuals' ultimate choices and may have overlooked the possibility that even small changes in negative affect could influence more subtle aspects of the food decision-making process leading up to those choices.

These more subtle features can be quantified with computational cognitive modeling. Sequential sampling models leverage both choice and response time data to estimate parameters that capture individual differences in decision-making dynamics (Forstmann et al., 2016; Ratcliff & Smith, 2004). These models assume that decision-making involves the noisy accumulation of evidence over time until a predetermined threshold is reached, leading to a response. Therefore, both

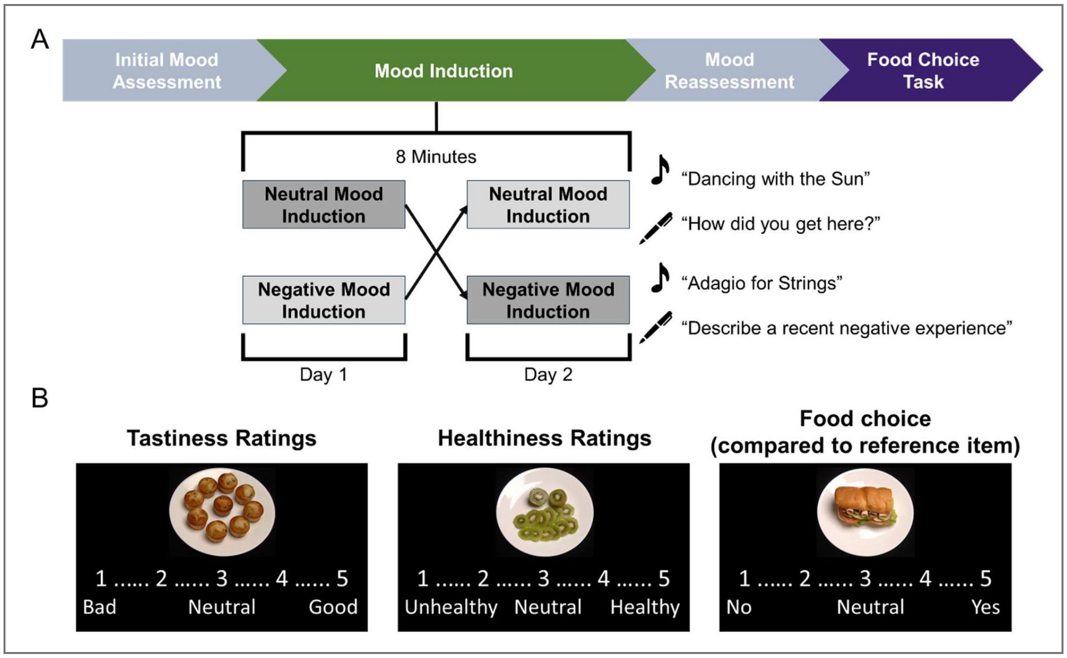


Figure 1. Affect induction and task design.

(A) Study timeline. BN and HC participants completed the study tasks on two separate days. On session 1, participants were randomly assigned to the neutral mood or negative mood induction before completing the Food Choice Task. On session 2, participants experienced the alternative mood induction before completing another run of the Food Choice Task. The mood inductions involved combinations of music and autobiographical writing. **(B) Food Choice Task**. During the Food Choice Task, both BN and HC participants rated 43 food items across three phases. In the Tastiness Ratings and Healthiness phases, participants rated each item on a 5-point Likert scale from Bad to Good and Unhealthy to Healthy, respectively. In the Choice phase, participants indicated their strength of preference for a presented food item, compared to a personally tailored neutral reference item.

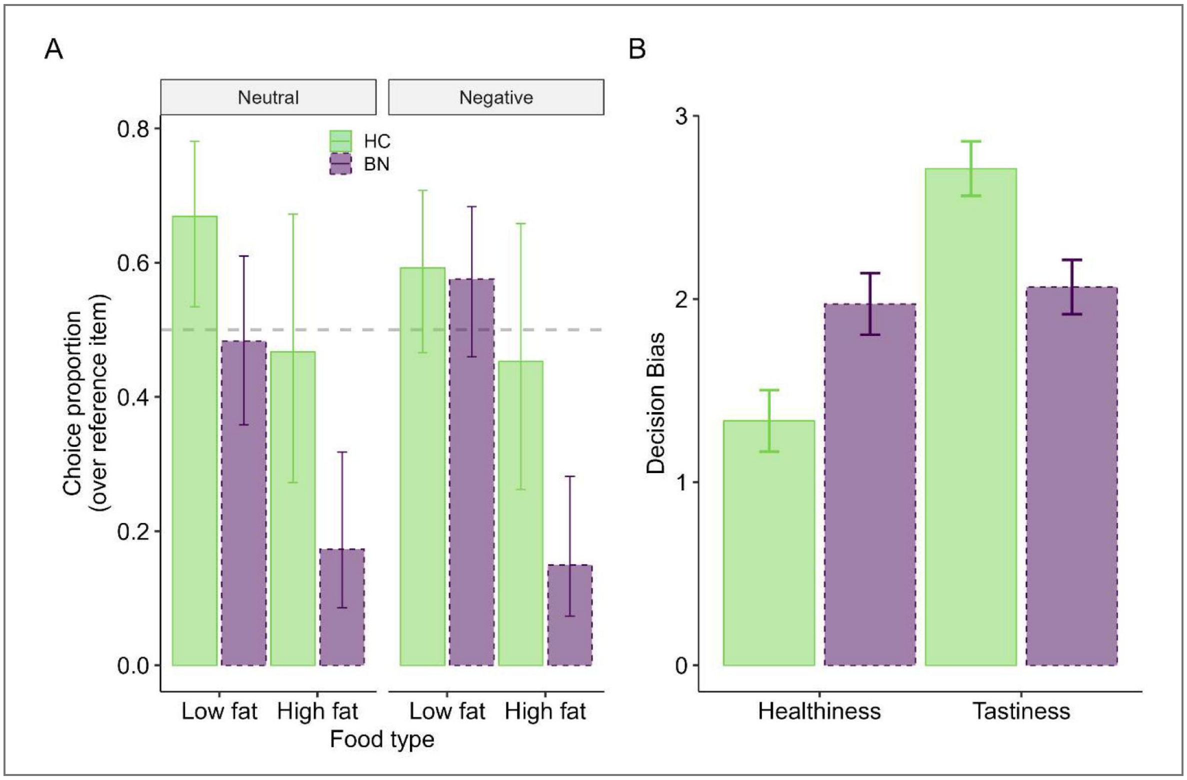


Figure 2.

(A) Food choice task behavior estimated from regression models. Re-analysis of the raw data excluding outlier response times (2.5% of trials *n* = 85) replicated original findings (Gianini et al., 2019). While both groups were less likely to choose high-fat foods (over the neutral reference item) than low-fat foods (over the neutral reference item), the BN group was even less likely than the HC group to choose high-fat food items. However, we did not identify any significant effects of the Affect Condition on choices. Error bars indicate 95% confidence intervals of the estimated effects. **(B) Influence of health and taste ratings on food choice**. Health ratings influenced food choice more in the BN group than in the HC group. Within the HC group, food choice was influenced more strongly by taste ratings than health ratings. Error bars indicate standard errors of the estimated coefficients. *Note.* Corresponding statistics are presented in Table S9. HC = healthy controls; BN = bulimia nervosa.

ultimate responses and response times are meaningful signals about the cognitive processes underlying decision-making. Though originally developed to describe perceptual, psychomotor, and memory tasks, these models can be applied to valuebased decision-making tasks by incorporating participants' ratings of different attributes for the stimuli presented in a trial. This allows researchers to examine how and when these attributes influence the decision-making process (Busemeyer et al., 2019; Clithero, 2018).

Research using sequential sampling models indicates that food choices are influenced not only by the weights assigned to attributes like healthiness and tastiness, but also by the time at which these attributes enter the decision-making process (Chen et al., 2022; HajiHosseini & Hutcherson, 2021; Hutcherson & Tusche, 2022; Maier et al., 2020; Sullivan et al., 2015; Sullivan & Huettel, 2021). Specifically, using time-varying sequential sampling models, studies have demonstrated that healthy individuals assign different subjective weights to healthiness and taste attribute ratings of foods (i.e., they have differing degrees to which they influence the evidence accumulation rate); that healthiness and taste attribute information can enter the decision process at different times; and that these relative weighting strengths and timing of attribute consideration have distinct influences on ultimate food choices (Maier et al., 2020). This notion is consistent with theories of both emotion- and food-craving regulation, which posit that there are separate attention deployment and stimulus valuation steps involved in these regulation processes (e.g., (Giuliani & Berkman, 2015; Gross, 2015; Han et al., 2018).

Notably, even if one of these decision parameters (e.g., weights on healthiness or tastiness, attribute onsets) biased individuals toward selecting a low-fat (or high-fat) food, the other parameter could compensate for it to ultimately generate the opposite response (Figure 3). For example, if taste information was considered before healthiness information, but healthiness information was weighted strongly enough, a decision-maker could choose a healthier, instead of a tastier, food (Figure 3B). These latent aspects of the decision-making process, either orthogonally or in parallel, could be sensitive to state changes like increases in negative affect and could be altered in BN. Specifically, these latent decision parameters could help explain why dysregulated consumption of tasty, high-fat foods in BN is more likely during periods of high negative affect, but restricted intake of lower-fat foods is more likely during periods of low negative affect. In one case, increasing negative affect could alter an individual's attribute weights, either reducing their weight on healthiness (dashed blue line) or increasing their weight on tastiness (dashed red line), which would increase their likelihood of choosing binge-type foods that are considered to be highly palatable yet also unhealthy (Figure 3C). Alternatively, increasing negative affect could cause individuals with BN to consider healthiness information later in the decision-making process, giving taste information more time to bias choices towards binge-type foods (Figure 3D). Clarifying these mechanisms is critical for translational efforts because data suggest that the weighting of attributes and the time at which they are considered have different neural substrates (Maier et al., 2020), and the treatment offered to patients should depend on which scenario more accurately reflects their pathology.

Here, we applied a diffusion decision model (DDM) with a time-varying drift rate to food choice and response time data from our controlled, randomized crossover study in women with BN and healthy controls (Figure 1A). This model allowed us to investigate the potential exaggerated influence of negative affect on the dynamics of food-specific decision-making in BN. Specifically, we tested how food type (low-fat/high-fat) and affective state (neutral/negative) influenced decision-makers' attribute weights and onsets.

Our hypotheses were derived from the clinical phenomenology underlying BN. As outlined above, individuals with BN seem to vacillate between unstable decision dynamics: many individuals with the disorder maintain a relatively restrictive diet until negative emotional states induce binge-eating episodes and subsequent compensatory behaviors (i.e., purging). Given this instability, we predicted that our BN participants' food choices would be influenced by components of the decision-making process whose opposing dynamics could be disrupted by increasing negative affect.

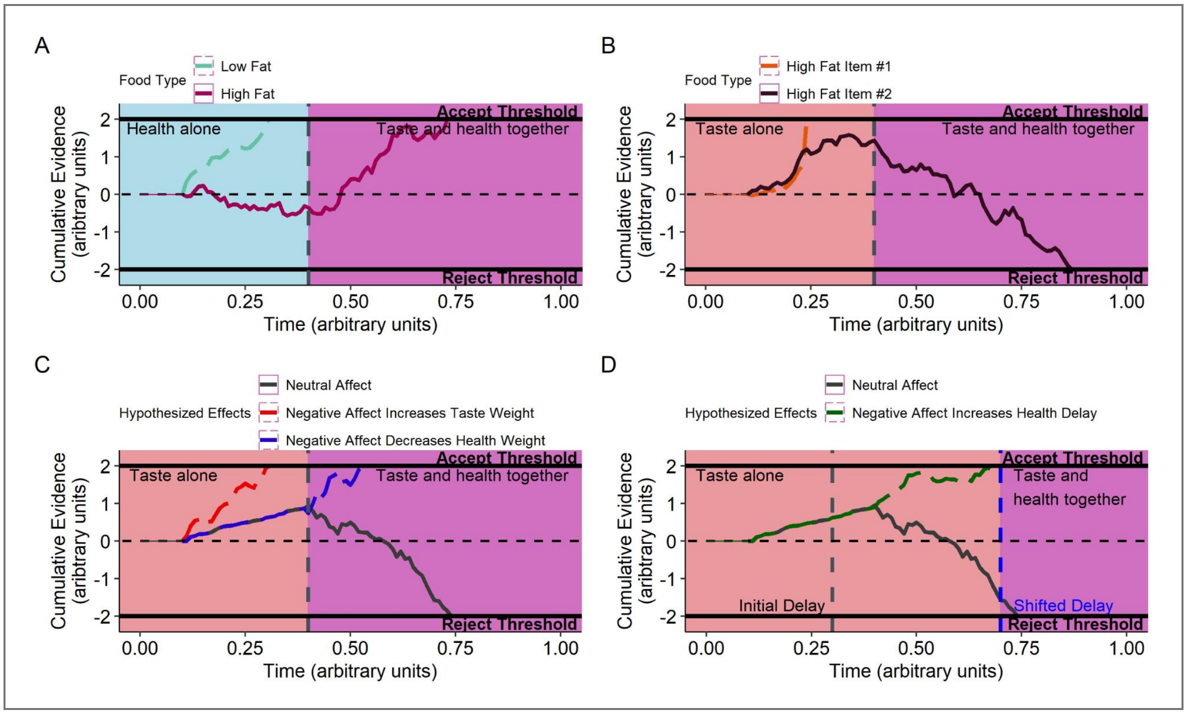


Figure 3. Example evidence accumulation trajectories predicted by the starting time diffusion decision model (stDDM).

Each trajectory is a simulated agent that considers one attribute alone before beginning to consider both tastiness and healthiness attributes together. **(A) Tastiness onset delay**. The dashed, green trajectory represents a case where positive healthiness information about a low-fat food item quickly biases the agent towards the accept threshold before taste information has time to influence the evidence accumulation process. The solid purple trajectory illustrates a case where evidence related to a high-fat food is initially biased towards the reject threshold while the aversive healthiness information dominates the evidence accumulation process. Once information about the item's appetitive tastiness comes online, the evidence accumulation changes its trajectory towards the accept threshold. **(B) Healthiness onset delay**. The dashed, orange trajectory illustrates a case where evidence related to the highly appetitive tastiness attribute dominates the evidence accumulation process, influencing the trajectory to terminate at the accept threshold of a high-fat food before the healthiness attribute is considered. For the solid brown trajectory, a less appetitive high-fat food item is ultimately rejected once aversive healthiness information enters the evidence accumulation process. **(C-D) Hypothesized model of affect-induced binge-eating**. Each trajectory is a simulated agent with BN making a decision involving a high-fat food. During neutral affect (gray solid line), the high-fat food is trending towards the accept threshold until the onset of aversive healthiness information biases the trajectory towards the reject threshold. **(C) Attribute-weight hypothesis**. During negative affect, either the attribute weight for taste information increases (red dashed line) or the attribute weight for healthiness information decreases (blue dashed line). In both cases, the trajectory is ultimately biased towards the accept threshold. **(D) Attributeonset hypothesis**. During negative affect, the initial delay shifts and taste information is accumulated longer before healthiness information comes online. With a longer delay in the onset of healthiness information, the evidence accumulation for the high-fat food has enough time to reach the accept threshold.

We first hypothesized that in states of neutral affect and for high-fat foods, one of these opposing dynamics would compensate for the other to promote ultimate choices that are restrictive. This could be achieved by either a) weighing taste information more strongly but considering it later than healthy controls, or b) weighing tastiness less strongly but considering it earlier than healthy controls. Second, we hypothesized that, in BN, increasing negative affect would abnormally impact at least one of these latent aspects of decision-making to increase the bias towards high-fat foods. Even though prior analyses indicate that high-fat foods were not ultimately chosen more often, we predicted that after a negative affect induction, individuals would either a) be slower to consider healthiness information, but still weight taste information just as strongly, or b) they would put less weight on healthiness information, but still consider taste information sooner.

Because these combinations require one feature of the decision process to compensate for another, they would result in a much less stable dynamic compared to one in which information weights and onset times are concordant. For example, when healthiness information is considered sooner *and* is more strongly weighted than taste information, foods low in healthiness would almost always be rejected. In contrast, when healthiness information is considered later but is more strongly weighed than taste information, food choice behavior can be more easily perturbed with state-based changes in weights or the delay in attribute onsets. Here, we show evidence for these unstable decision dynamics that can account for a bias in BN toward low-fat, healthier foods in states of relatively low negative affect, but toward high-fat, tastier foods in states of high negative affect. Although negative affect delayed the onset of the consideration of healthiness information among both HC and BN participants, this effect was more pronounced in the BN group. Ultimately, we found that although individuals with BN consistently weighed healthiness information more strongly, negative affect delayed its entry into the evidence accumulation process. With this longer delay, taste information has more time to bias decision-makers towards high-fat food choices before healthiness information can come online.

Results

Negative affect aberrantly delays consideration of healthiness information among women with BN

We analyzed data from individuals with BN (n = 25) and healthy controls (HC, n = 21) who completed a computerized Food Choice Task following negative and neutral affect inductions, across two visits (Figure 1). Mood was assessed before and after the affect inductions using the Profile of Mood States (POMS; (McNair et al., 1989)). Negative affect scores were calculated by summing the five negative subscale scores (e.g., anger, confusion, depression, fatigue, and tension) and subtracting the positive subscale score (e.g., vigor). Negative affect scores increased significantly for both groups following the negative affect induction (Gianini et al., 2019, reproduced in Table S1). Critically, post-induction negative affect within the BN group was significantly higher following the negative affect induction than after the neutral affect induction (mean difference = 17.40, SE = 4.21, t = 4.13, p < 0.001, Cohen's d = 0.83; see Supplementary Materials for full details), confirming that BN participants completed the food decision task under meaningfully distinct affective states across the two sessions.

To quantify the specific computations performed during Food Choice Task decision-making, we used a time-varying diffusion decision model (DDM; (Ratcliff & McKoon, 2008)). This time-varying model introduces a starting time parameter that indicates the delay with which different attributes enter the evidence accumulation process (Chen et al., 2022; Lombardi & Hare, 2021; Maier et al., 2020). Using this starting time DDM (stDDM), we can determine how individuals weigh healthiness versus taste information and when those attributes influence food-related decision-making in BN and HC groups in each affective state. The stDDM was fitted to data that included individual-level response times, choices, z-scored healthiness and tastiness ratings, and affect induction.

We specified a stDDM where individual parameters were drawn from four separate hyperparameters that varied both by group (BN or HC) and by condition (neutral or negative affect induction). To test our hypotheses about the interactions between affect and food type (low-fat or high-fat) on food choice, we allowed each attribute weight parameter (ω_{Taste} and $\omega Health$) and the relative-starting time parameter τ_s) to vary as a function of food type. The drift rate determining the evidence update can be written as follows:

$$v(t) = \begin{cases} \omega_{Taste} \cdot VD_{Taste} & \text{If } \tau_s < 0 \wedge 0 < t < |\tau_s| \\ \omega_{Health} \cdot VD_{Health} & \text{If } \tau_s > 0 \wedge 0 < t < \tau_s \\ \omega_{Taste} \cdot VD_{Taste} + \omega_{Health} \cdot VD_{Health} & \text{If } t < |\tau_s| \end{cases} \tag{1}$$

where ω_{Taste} is the subjective weight given to tastiness, ω_{Health} is the subjective weight given to healthiness, VD_{Taste} and VD_{Health} are the value differences in taste and healthiness attribute ratings, respectively, and τ_s is the time at which the taste and healthiness attributes come into the evidence accumulation process. If $\tau_s > 0$, healthiness information is accumulated first, and evidence from taste comes into consideration at time τ_s, whereas $\tau_s < 0$ means that taste information is accumulated first and evidence from healthiness information starts to come into the decision process at time $|\tau_s|$. To test our hypotheses, medians of the posterior distributions for subject-level parameters were used as the dependent variables in our statistical analyses. We ran three separate, fully factorial (Group-by-Affect Condition-by Food Type), mixed-effects linear regressions, one for each parameter of interest. Each of these binary categorical variables were contrast coded using the values -1 and 1, allowing us to separately assess group differences across condition.

Results exploring additional parameters (boundary separation *(α),* non-decision time (τ_{ND}), and starting point bias (z)) are presented in the Supplementary Materials along with model comparisons, model identifiability, posterior predictive checks, and parameter recovery exercises.

First, we assessed how relative start time parameters varied across groups as a function of food type in each condition. We found a significant three-way interaction between group, condition, and food type (Table S2; $\beta = 0.32$, $t = 2.23$, $p = 0.028$), indicating that the two groups differed in how negative affect influenced their attribute bias patterns across food types. To confirm the presence of a three-way interaction, we calculated a difference-in-difference score for each participant's τ_s parameter: (negative condition, high-fat – negative condition, low-fat) – (neutral condition, high-fat – neutral condition, low-fat). A Wilcoxon rank-sum test comparing these scores between groups confirmed that BN participants showed significantly larger food-type-specific changes in attribute onset timing following negative affect induction relative to HC (W = 156, p = 0.018). Visual inspection of the data suggested this interaction was driven by group differences across food types in the neutral condition that disappeared in the negative affect condition, and by a more pronounced influence of negative affect on attribute biases in BN (Figure 4A). Specifically, in the neutral condition, the HC group showed greater taste bias for low-fat versus high-fat foods (M = -0.456 vs. -0.314; Table S3), while individuals with BN showed greater taste bias for high-fat versus low-fat foods (M = -0.314 vs. -0.462; pairwise difference $t_{132} = -2.33$, p = 0.022). The negative affect induction eliminated this crossover pattern, increasing taste biases across both food types and groups, but with greater increases observed in the BN group (Tables S2, S3).

Reduced tastiness weights are abnormally unaffected by negative affect in BN

Our second and third regression models assessed how each attribute weight varied across groups as a function of food type in each condition (Figure 4B-C). For taste weights, the HC group placed consistently higher weight on taste compared to the BN group (Table S4–S5; $\beta = -0.39$, t = -6.86,$p < 0.001$; all pairwise contrasts $p \leq 0.002$), and both groups placed less weight on taste for high-fat versus low-fat foods $\beta = -0.23$, $t = -4.05$, $p < 0.001$; all pairwise contrasts $p < 0.001$). A significant interaction between group and condition ($\beta = 0.18$, $t = 2.46$, $p = 0.015$) reflects divergent

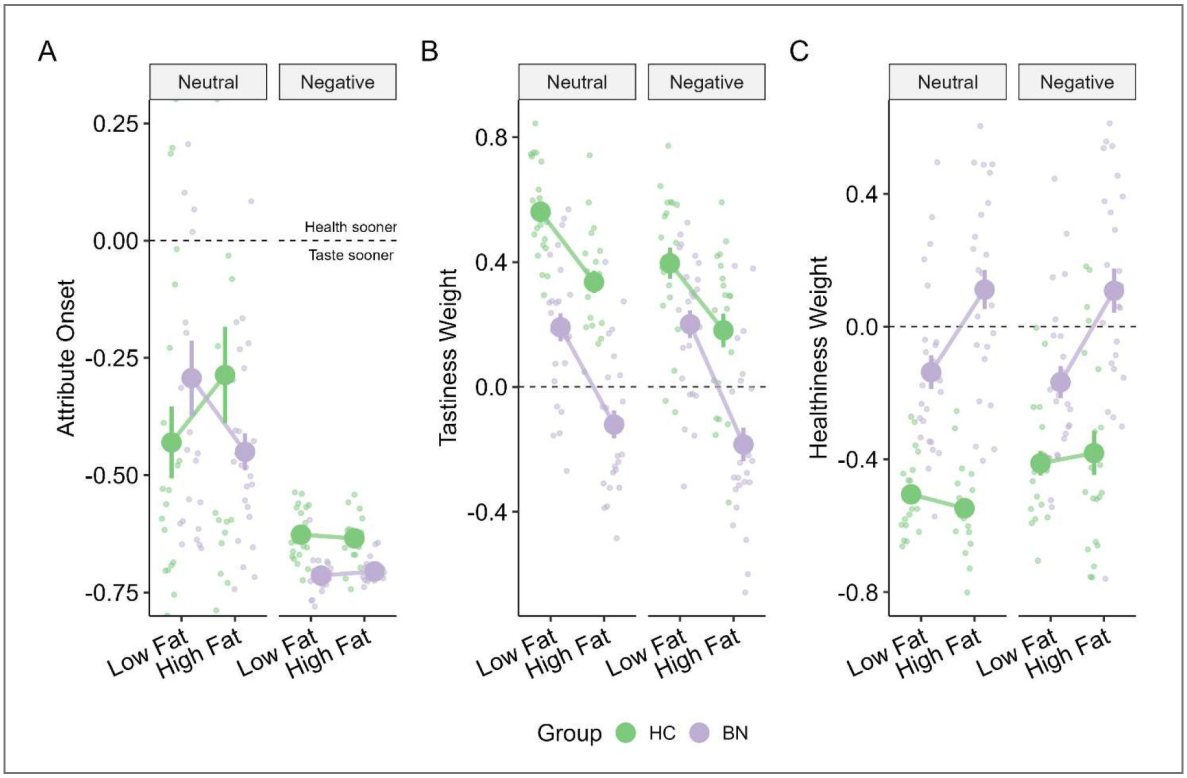


Figure 4. **Parameter estimates.**

Colors indicate diagnosis: purple = bulimia nervosa (BN); gree*n* = healthy controls (HC). Error bars represent the standard error of the mean. **(A) Attribute onset**. In the neutral condition, we observed a Group by Food Type cross-over effect: while the BN group showed a greater initial bias towards accumulating tastiness information of high-fat foods than low-fat foods, the HC group showed a greater tastiness information bias for low-fat foods than high-fat foods. After the negative affect induction, any Food Type-based distinctions disappeared, and both groups' biases towards tastiness information increased, but this effect was more pronounced in the BN group. **(B) Weight on tastiness information**. The HC group put more weight on tastiness information than the BN group. The negative affect induction reduced tastiness weights for the HC group, but not the BN group. **(C) Weight on healthiness information**. The BN group put more weight on healthiness information than the HC group, especially for high-fat foods. The negative affect induction did not have significant effects on healthiness weights for either group.

responses to negative affect: HC showed a significant reduction in taste weighting following the negative affect induction (t_{132} = 3.20, p = 0.002), but the BN group did not (t_{132} = 0.61, p = 0.542). Consequently, the group difference in taste weighting, while significant in both conditions (all pairwise contrasts $p < 0.001$), was numerically smaller in the negative affect condition (M = 0.21) than in the neutral condition (M = 0.39; interaction: t_{132}= 1.95, p = 0.053). In contrast, healthiness weights were insensitive to negative affect induction across both groups. However, a significant interaction between group and food type (Table S6–S7; β = 0.28, t = 3.09, p = 0.002) indicated that the BN group placed disproportionately higher weights on health information for high-fat relative to low-fat foods (t_{132} = -4.40, $p < 0.001$), while the HC group showed no difference in health weighting across food types (t_{132} = 0.01, p = 0.897).

More severe symptoms of BN are linked to affect-induced delays in processing healthiness information

To assess the clinical significance of our findings, we sought to connect stDDM parameters to retrospective self-reported symptom severity. For these exploratory analyses, we used median values of the individual-level posterior distributions of the attribute onset parameter x_s, the parameter which showed an exaggerated response to the affect induction in the BN group. These parameter values were then separately added to mixed-effects negative binomial regression models with Group and Food Type to assess their association with two symptom severity measures in the BN group: the frequency of objectively large binge eating episodes (OBEs) and subjectively large binge eating episodes (SBEs) in the past three months. We examined both types of out-of-control eating episodes because they both contribute to a diagnosis of BN according to ICD-11 (World Health Organization, 2022), and some data suggest that SBEs are more strongly related to negative affect (Brownstone et al., 2013; Brownstone & Bardone-Cone, 2021; Fitzsimmons-Craft et al., 2014). Indeed, we found that that greater negative affect-based decreases in τ_s for high-fat foods relative to low-fat foods were associated with more frequent SBE in the past three months (Table S8; τ = -46.39, t = -2.80, p = 0.005). In other words, those who focused for longer on the tastiness of high-fat foods after the negative affect induction reported more SBE episodes in the past three months (Figure 5). Conversely, affect-based changes in x_s were unrelated to OBE frequency (Table S8).

Discussion

Negative affect is a known precursor to over-eating and dietary rule-breaking (Frayn & Knäuper, 2018). Healthy adults, and even rodents, can exhibit dysregulated food consumption and altered food choices after periods of stress or other negative emotions (Dionysopoulou et al., 2021; Jacques et al., 2019; Konttinen et al., 2010; Tomiyama, 2019). Negative affect's influence on eating behavior is particularly pronounced among individuals with clinical diagnoses like BN, who tend to have large and out-of-control eating episodes in states of strong negative emotion (Cardi et al., 2015; Wonderlich et al., 2022). Prior studies have demonstrated that outside of binge-eating episodes, and in states of lower negative affect, individuals with BN typically engage in restrictive eating behaviors, consuming some low-fat foods and largely avoiding high-fat foods (Alpers & Tuschen-Caffier, 2004; Bjorlie et al., 2022; Elran-Barak et al., 2015; Kales, 1990). We previously found that women with BN demonstrated these restrictive food choices on a food-related decision task, regardless of their affective state (Gianini et al., 2019). However, by leveraging a computational model that captures the decision-making process underlying ultimate choices, we found that negative affect more strongly biased individuals with BN than healthy adults, increasing the amount of time that taste information alone influenced their decisions involving food. This affect-induced delay in the consideration of healthiness versus tastiness may help account for the fact that negative affect is a common precursor to taste-driven overeating in healthy adults, and the fact that it is a particularly strong predictor of taste-driven, out-of-control eating in individuals with BN. Consistent with this notion, we found that negative affect-induced delays in accumulating health information relative to taste information for higher fat foods were associated with more frequent out-of-control eating episodes in BN.

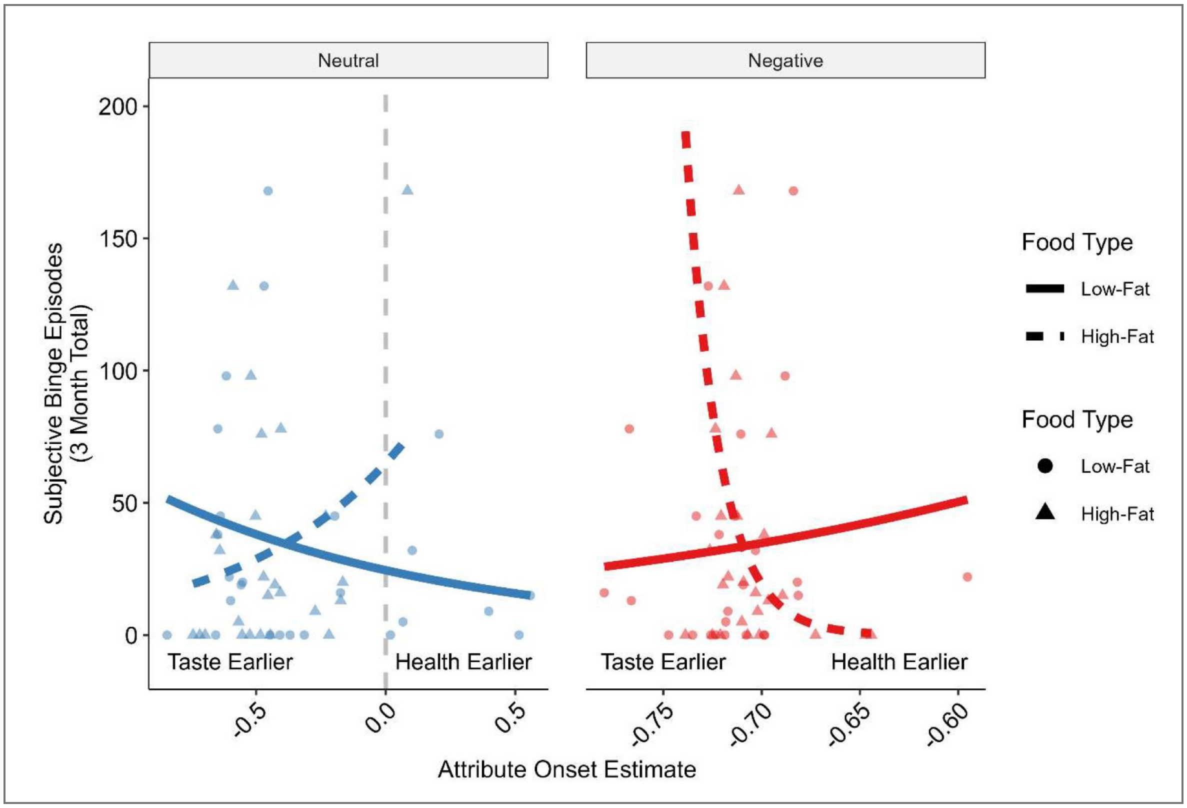


Figure 5. Affect-induced changes in information onset were associated with more frequent subjective binge episodes.

In the negative affect condition (right facet), longer delays in accumulating healthiness information (i.e., reduced τ_S) for high-fat foods compared to low-fat foods was associated with more frequent subjective binge episodes. Line type and shape refer to Food Type: Solid lines and circles = Low fat foods; dashed lines and triangles = High fat foods.

Previous studies have shown that in states of neutral affect, individuals with BN are more likely to consume foods that are low-fat and considered to be healthy (e.g., salads, fruits), as well as use healthiness information to guide their food choices (Alpers & Tuschen-Caffier, 2004; Bjorlie et al., 2022; Elran-Barak et al., 2015; Kales, 1990; Neveu et al., 2018; Schnepper et al., 2021). Our results demonstrate that in a neutral mood state, despite an initial tendency to focus on taste information before healthiness information, the BN group had stronger weights on healthiness information and lower, and in fact negatively signed, weights on taste, especially for high-fat foods. This negative weight on taste information (i.e., an aversive assignment to the value of taste) may have facilitated the BN group's restrictive food choice policy compared to healthy controls. Individuals with BN may compensate for their tendency to focus on the tastiness of high-fat foods by negatively reappraising or deemphasizing the relative importance of taste and highly weighting their food choices based on health.

During binge-eating episodes, which often follow increases in negative affect, individuals with BN tend to consume large amounts of high-fat foods that were previously avoided (Alpers & Tuschen-Caffier, 2001; Berg et al., 2013; Collins et al., 2017; Haedt-Matt & Keel, 2011; Hilbert & Tuschen-Caffier, 2007; Smyth et al., 2007). Although prior studies examined food consumption associated with negative emotions in BN (Cardi et al., 2015), discrete food choice behaviors had not been investigated, and the influence of negative affect on specific aspects of the decision-making process were unclear. We showed that this affect-related change in eating behavior may occur because negative affect disrupts the delicate balance between how much weight tastiness and healthiness carry in influencing food choices and the relative speed with which they begin to exert their influence (Barakchian et al., 2021; HajiHosseini & Hutcherson, 2021; Hutcherson & Tusche, 2022; Lim et al., 2018; Maier et al., 2020; Schubert et al., 2021; Sullivan et al., 2015; Sullivan & Huettel, 2021). When in states of high negative affect, both healthy adults and individuals with BN may consider food's appealing taste for longer, biasing them towards choosing tastier, high-fat foods. If this temporal advantage for tastiness is long enough, and a positive weight on tastiness is strong enough, individuals with BN could become more vulnerable to out-of control eating because they could decide to eat tastier or high-fat foods before ever considering their perceived healthiness. This notion is consistent with our finding that greater affect-induced delays in processing healthiness information were associated with more frequent subjective binge episodes— episodes of dysregulated eating that may not include a large amount of food, but like objectively large binge-eating episodes, are dominated by carbohydrates and high-fat foods that are highly palatable (Presseller et al., 2023), and are often spurred by negative affect (Alpers & Tuschen-Caffier, 2001; Berg et al., 2013; Haedt-Matt & Keel, 2011; Hilbert & Tuschen-Caffier, 2007; Smyth et al., 2007).

Our findings reveal new insights into the dynamics of food-related decision-making in both healthy adults and BN and may help to inform new interventions. When attribute weights are inconsistent with a larger goal, decision-makers can use shifts in the timing of attribute consideration to achieve a desired outcome (Amasino et al., 2019; Maier et al., 2020). For example, earlier shifts in the relative onset time of healthiness information could help an individual with stronger weights on tastiness adhere to a diet. Perhaps targeting affect-induced shifts in the timing of attribute consideration could help regulate the over- and under-controlled eating seen in binge-fast cycles. For example, past research in individuals without eating disorders indicates that cueing attention towards the healthiness of foods during a choice task both increased the weight on healthiness information and increased its onset time during decisionmaking (Barakchian et al., 2021; Maier et al., 2020) but see (Sullivan & Huettel, 2021). Several interventions already focus on modifying attentional biases in individuals with obesity and binge eating (Boutelle et al., 2016; Brockmeyer et al., 2019; Stojek et al., 2018). Our results suggest that such interventions could be targeted even more precisely. One way for individuals to avoid emotion-driven binge eating may be to focus attention away from the taste attributes of tastier, high-fat foods during states of high negative affect. Conversely, during states of low negative affect, increased attention on the positive taste attributes of all foods may be instrumental in motivating individuals with BN to engage in less restrictive eating.

Although we found an aberrant influence of negative affect on the computations that contribute to food choice in BN, in our paradigm, the influence of negative affect was not strong enough to change final choices in either group. Even after the negative affect induction which decreased taste weights for HC and not BN, choice outcomes in BN were ultimately determined by the advantage in weighted evidence for healthiness as opposed to the advantage in relative timing for tastiness. We speculate that this may have been for two reasons. First, the affect induction method may not have been potent enough. In the current study, post-induction increases in negative affect (d_{HC} = 1.34; d_{BN} = 1.19) were smaller than those reported after other induction methods, such as viewing affectively salient images with congruent music (d = 3.33; (Zhang et al., 2014)), and they may not have been powerful enough to effect changes in ultimate choices. Although the Food Choice Task was completed immediately after the affect induction, we do not know how long its effect lasted because mood was not repeatedly assessed throughout the task. This and previous studies using task-based designs have only assessed mood ratings immediately following affect inductions (Rouhani et al., 2025; Werthmann et al., 2014), but future studies could include continuous assessments throughout the experiment to better quantify potential group differences in how induced affect changes persist and/or fluctuate. In addition, our negative-affect inducing music and writing exercise may have created an emotional experience that inadequately represents the negative affect that typically precedes emotional eating or binge eating. Qualitative studies of patients with BN indicate that interpersonal stressors and related negative emotions (e.g., resentment) often precipitate binge episodes (Bohon et al., 2021; Wasson, 2003). Perhaps larger increases in negative affect intensity or exposure to the specific types of negative affect which typically precede binge eating could produce a large enough asynchrony in onset timing of attributes and changes in attribute weights to change individuals' choices (Figure 6). Second, participants knew they would be offered a snack-sized amount of food from a randomly selected trial to consume at the end of the task. This quantity of food may not have provided participants with access to the type of binge-eating experience that negative affect would otherwise precipitate. Further work is needed to test whether the same processes identified in the current study also drive binge eating outside the lab (Wonderlich et al., 2022). Future adjustments to incentive compatibility, with designs that allow for increased consumption beyond snack-sized portions and/or guaranteed access to a wider-array of high-fat foods could clarify whether opportunities to overconsume highly palatable food would generate different patterns of food choice behavior on the task. Additionally, future research should include continuous assessments of other relevant factors such as cravings (Konova et al., 2018; Wonderlich et al., 2017) to enrich the connection between mood, discrete food choice, and binge eating.

Of note, the modest sample size (n = 46) composed exclusively of female participants may limit the generalizability of our findings. Replication in larger samples that include male participants is needed. Nonetheless, the study's repeated-measures design benefits from the added statistical power and sensitivity of within-subjects statistical tests, which reduce error variance by controlling for individual differences (Charness et al., 2012; Judd et al., 2017; Westfall et al., 2014). Moreover, we collected a large quantity of data per subject, which enhances the validity of the findings by providing a more precise estimate of the effects of the experimental manipulation, despite the smaller sample size (Baker et al., 2021; Smith & Little, 2018).

Overall, our results advance our understanding of how changes in affective states may shift individuals with BN away from typically restrictive dietary choices and towards binge eating. Specifically, negative affect can bias individuals with BN to focus on taste information for longer, and the strength of this effect for high-fat foods was associated with symptom severity. Our results add to recent data suggesting that computational modeling can detect subtle alterations in decision-making processes in BN that are sensitive to state changes (Berner et al., 2023), and future research should continue examining the potential connections between negative affect and eating-related decision-making using these analytic approaches.

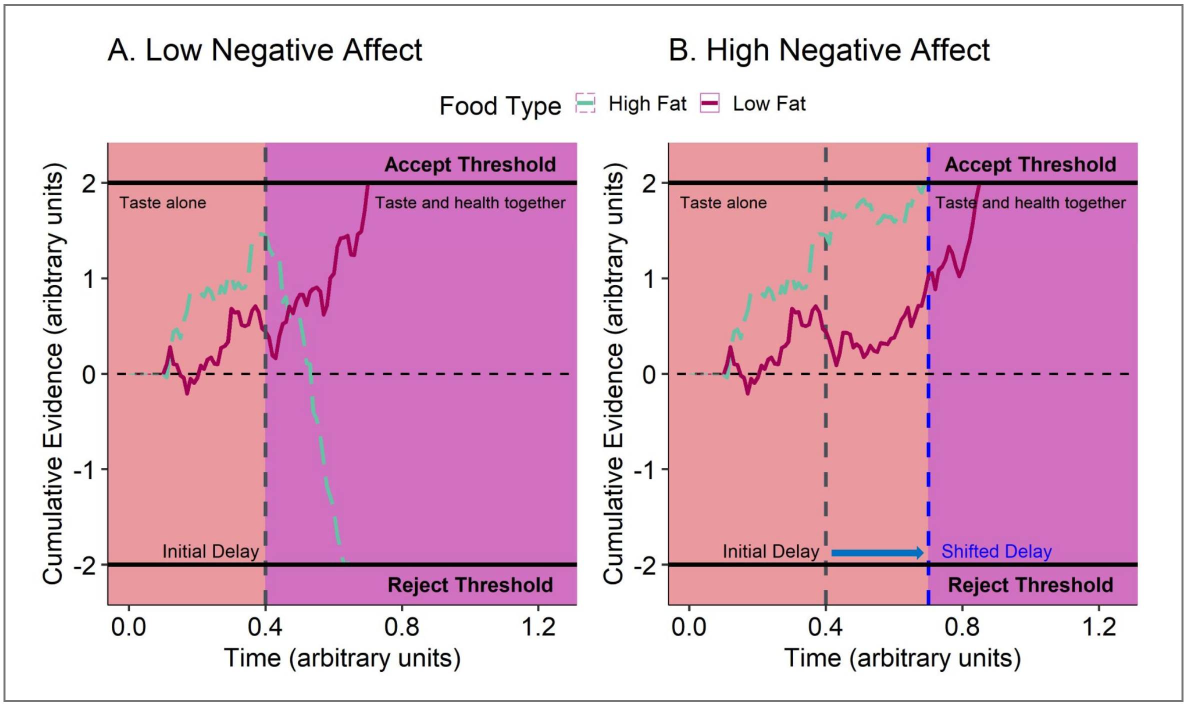


Figure 6. **Revised model of affect-induced binge-eating.**

Each trajectory is a simulated individual with BN who considers the taste attribute alone (pink shaded sides of the panels) before considering both tastiness and healthiness attributes together (blue shaded sides of the panels). In both panels the solid purple trajectory illustrates a decision involving a low-fat food, where tastiness information has a weak, positive weight and healthiness information has a strong, positive weight. The dashed blue trajectory illustrates a decision involving a high-fat food, where tastiness information has a strong, positive weight and healthiness information has a strong, negative weight. **(A) Low Negative affect**. During neutral affect, the high-fat food is trending towards the accept threshold until the onset of aversive healthiness information biases the trajectory towards the reject threshold. **(B) High Negative affect**. During negative affect, the initial delay is shifted and tastiness information is accumulated longer before healthiness information comes online. With a longer delay in the onset of healthiness information, the evidence accumulation for the high-fat food has enough time to reach the accept threshold.

Materials and Methods

Participants

Data were collected from 25 individuals who met *DSM-5* diagnostic criteria for BN and 21 healthy controls, group-matched for age and BMI. All participants were female and aged 18-40 years. The sample consisted of 27 participants who identified as Caucasian (58.7%), 7 as Hispanic (15.2%), 6 as Asian (13.0%), 3 as Black (6.5%), and 3 as mixed race (6.5%). For details on recruitment and exclusions, see (Gianini et al., 2019).

Materials and procedures

Participants completed the tasks over two separate study sessions, each lasting approximately four hours. On each study day, participants were instructed to abstain from consuming food or drinks except for a standardized meal consumed two hours preceding the study session.

Participants completed a Food Choice Task (Steinglass et al., 2015) on two separate days, in two counterbalanced states: after a neutral affect induction, and after a negative affect induction. This task is composed of three phases: Healthiness Rating, Tastiness Rating, and Choice. In each phase, participants viewed the same 43 food items, each categorized as either low-fat (< 30% kcals from fat) or high-fat (> 30% kcals from fat). During the Health Rating phase, participants used a 5-point scale to rate the healthiness of each food item from 1 (Unhealthy) to 3 (Neutral) to 5 (Healthy). During the Taste Rating phase, participants used a 5-point scale to rate the tastiness of each food item from 1 (Bad) to 3 (Neutral) to 5 (Good). The order of the Health Rating and Taste Rating phases was counterbalanced. After both phases were completed, one food item that was rated Neutral for both health and taste was selected as a Reference Item for use in the Choice phase. If no item was rated Neutral for healthiness and tastiness, then a food item rated 3 on health and 4 or above on taste was selected following previously established procedures (Steinglass et al., 2015). During the Choice phase, participants made choices between the neutral Reference Item and other food items presented on the computer screen. Participants indicated their choice on a 5-point scale from 1 (No - select the Reference Item) to 3 (Indifference) to 5 (Yes - select the shown food item). Participants were informed that one randomly selected trial would be selected for payout at the end of the experiment and would receive the food item they selected on that trial.

The experiment included an affect induction that included a combination of music and autobiographical writing (Werthmann et al., 2014). During the negative affect induction, participants were asked to write about a recent negative experience while listening to “Adagio for Strings” by Samuel Barber for 8 minutes. During the neutral affect induction, participants were asked to write about the route they took to get to the study site while listening to “Dancing with the Sun” by Celia Felix for 8 minutes. The 65-item Profile of Mood States scale was administered before and after the affect inductions to assess changes in affect (McNair et al., 1989).

Participants also completed the following measures: Eating Disorder Examination Questionnaire (EDE-Q; (Fairburn & Beglin, 1994)); Difficulties in Emotion Regulation Scale (DERS; (Gratz & Roemer, 2004)); Urgency, Premeditation, Perseveration, Sensation Seeking, and Positive Urgency Behavior ScaleNegative Urgency subscale (UPPS-P Negative Urgency; (Lynam et al., 2006)); Beck Depression Inventory (BDI; (Beck et al., 1961)); State Anxiety Inventory (STAI; (Spielberger et al., 1983)).

For additional information, please see (Gianini et al., 2019).

Data analysis

Data transformations and exclusions: Following the procedure from (Gianini et al., 2019), participants’ responses from the Choice phase of the FCT were binarized from their 5-point scale. Responses marked 1 or 2 (i.e., “no”) were determined to be choices of the reference item, responses marked 4 or 5 (i.e., “yes”) were determined to be choices of the presented food item, and responses marked 3 (i.e., “indifferent”) were omitted (total n =599; BN n = 292, HC n = 307).

In contrast to the original study, we excluded trials containing outlier responses times (RTs). Outlier response times likely reflect phenomena outside the decision-making processes of interest, including attention lapses or accidental responses (Cousineau & Chartier, 2010; Ratcliff, 1993). We excluded outlier responses using cutoffs of ±3 standard deviations (SD) from the mean (Berger & Kiefer, 2021). For each participant, the mean and standard deviation of their RTs were calculated and trials containing responses larger/smaller than the mean ±3 SDs were excluded. Additionally, we removed trials where the RTs were greater than 10,000 ms or less than 250 ms after the mean ±3 SDs treatment. Using this method, we removed 2.5% of trials (total n =85; BN n = 51, HC n = 34).

Computational modeling: We used a time-varying diffusion decision model (DDM; (Ratcliff & McKoon, 2008) to study the dynamics of the decision-making during the Food Choice Task. This time-varying model introduces a starting time parameter that indicates the delay with which different attributes enter the evidence accumulation process. This model was previously fitted to food choice data (Lombardi & Hare, 2021; Maier et al., 2020) and lottery choice data (Chen et al., 2022) from healthy adults.

The starting time DDM (stDDM) was estimated using a hierarchical, Bayesian framework implemented with the R package *Rjags*, which uses the JAGS MCMC sampling algorithm (Plummer, 2003). Our fitting scripts were adapted from those developed by Hsiang-Yu Chen, Gaia Lombardo, and Todd Hare (Chen et al., 2022). This fitting method simultaneously estimates both group- and individual-level parameters, which improves the reliability of parameters estimated from data with low trial counts per participant (Ratcliff & Childers, 2015; Wiecki et al., 2013). Parameter estimates were fitted to data that included individual-level response times, choices, z-scored health and taste ratings, and Affect Condition. We specified a model where individual parameters were drawn from four separate group-level distributions that varied both by Group (BN or HC) and by Affect Induction (neutral or negative). Within this model, attribute weight parameters (ω_{Taste} and ω_{Healt}) and the relative-starting time parameter (τ_s) were estimated separately based on food-types (low-fat or high-fat). Using model comparisons and posterior predictive checks, we confirmed that this model best fit our data compared to simpler models (see Supplementary Materials).

Following previously described protocols (Chen et al., 2022), we used group-level priors for attribute weight parameters *(*ω_{Taste} and ω_{Heal}), the relative-starting time parameter (τ_s). and the affect induction parameters that were drawn from Gaussian distributions with mea$n$ = 0 and standard deviatio$n$ = 1. These parameter values were then divided by their standard deviations before fitting the model. The priors for boundary separation *(a)* and non-decision time τ_{ND} were drawn from uniform distributions with ranges 1.0×10^{-4} to 5 and 0 to 10, respectively. The priors for starting point bias (z) were drawn from a beta distribution where both the shape and scale parameters were set to 2. All individual-level priors were drawn from gamma distributions with shape parameters of 1 and scale parameters of 0.1. Posterior estimates were drawn from three chains, each with 100,000 samples (85,000 discarded as burn-in) and thinning every 10 samples. Convergence among chains was assessed using the Gelman-Rubin statistic (Gelman & Rubin, 1992) using a threshold of 1.1. Model fit was quantified with the Widely Applicable Information Criterion (WAIC) (Watanabe, 2010), which penalizes for model complexity based on the number of parameters to prevent overfitting. For parametric tests involving parameter estimates, we used the medians of the individual-level posterior distributions. See the Supplementary Materials for model information on model comparisons, model identifiability, posterior predictive checks, and parameter recovery.

Multilevel linear regressions were run to evaluate our hypotheses regarding the influence of Food Type, affect induction, BN diagnosis, and their interactions on parameter estimates. Each regression model included Food Type (coded -1/1 for low-fat/high-fat foods), Affect Condition (coded -1/1 for Neutral/Negative), and Group (coded -1/1 for HC/BN), and their interactions as independent variables. For these models, the intercepts were treated as random effects and statistical significance was evaluated using α = 0.050.

Because the parameters of interest (i.e., ω_s, ω_{Taste}, ω_{Health}) were estimated separately per condition and food type, each participant contributed exactly four observations to these models (2 affect conditions × 2 food types), creating a fully balanced but structured repeated-measures design. Standard random-intercept-only models implemented in *lme4* (Bates et al., 2015) and *lmerTest* (Kuznetsova et al., 2013) do not explicitly model the residual covariance among these four within-person observations. To address this, models were re-implemented using the *nlme* package (José C. Pinheiro et al., 2022), which permits explicit specification of the within-subject residual covariance structure.

We evaluated three candidate covariance structures. We first considered a nested random effect of affect condition within subject, which would directly encode the pairing of Low-Fat and High-Fat observations within each session, but this model failed to converge due to the limit. With only two observations per session per subject, the session-level and residual variance components cannot be separately estimated. Next, we implemented a compound symmetry structure using the *corCompSymm* function, which assumes equal correlations among all four within-person observations. Finally, we implemented an unstructured covariance matrix using the *corSymm* function, which places no constraints on the pattern of correlations among the four within-person observations and represents the most general feasible specification for this design. Model comparisons via likelihood ratio test and information criteria (LRT: $\Delta df = 5$, $p = 0.057$; AIC (-119.08 vs. -118.34) marginally favored unstructured; BIC (-57.99 vs. -73.33) favored compound symmetry) did not strongly favor either tractable structure, and we selected the unstructured model as the more conservative option, as all constrained covariance structures are nested within the unstructured model and represent testable restrictions on the covariance parameters (Molenberghs & Verbeke, 2000; Pinheiro & Bates, 2000). All models additionally included heterogeneous residual variances across the four condition-by-food-type cells using the varIdent function, allowing each cell to have its own residual standard deviation.

Difference-in-differences scores were additionally computed for each participant and parameter to provide a complementary analysis that encodes the within-session pairing directly by construction, bypassing the covariance specification problem entirely. These analyses and their results are described in the Supplementary Materials.

To unpack observed interaction effects, we ran exploratory simple effects analyses to separately evaluate the influence of independent variables at specific values of other independent variables (Aiken et al., 1991; Brambor et al., 2006; Jaccard & Turrisi, 2003; Winer et al., 1991). We used the R package emmeans to compute estimated marginal means for specified factor combinations (Lenth, 2025), evaluating statistical significance using $\alpha = 0.050$.

Additional fixed-effects regressions were run to assess the relationship between parameter estimates and symptom severity. For these exploratory analyses, we used negative binomial models to test the association between parameter estimates and the three-month frequency of retrospectively reported Objective Binge Episodes (OBE) and Subjective Binge Episodes (SBE). We implemented these models using the R package *glmmTMB* (Brooks et al., 2017), with a zero-inflated term included for the model predicting SBE. For each of these two models, individual-level parameter estimates from each condition and food-type were included as independent variables. We applied a Bonferroni correction to control for the FWE of these exploratory analyses, using $\alpha = 0.025$.

Supplementary Information

Supplementary Methods

Behavioral analyses

We used a similar analytic approach to assessing choice and response time data to the methods described in (Gianini et al., 2019). A multilevel logistic regression model was fit to trial-level choice data (coded 1/0 for yes/no) using Food Type (coded -1/1 for low-fat/high-fat foods), Affect Condition (coded -1/1 for Neutral/Negative), Group (coded -1/1 for HC/BN), and their interactions as independent variables. We fit a second logistic regression model to trial-level choice data, also using Affect Condition, Group, and their interaction as independent variables. In this second model, we also included z-scored health and taste ratings, and their independent interactions with group, as independent variables.

We assessed self-control by selecting trials where participants made decisions involving highly appetitive unhealthy foods (i.e., taste rating = 5, health rating = 1) or unappetizing healthy foods (i.e., taste rating = 1, health rating = 5). The use of self-control (coded 1 for rejecting appetitive unhealthy foods or selecting unappetizing healthy foods and 0 otherwise) was assessed using a multilevel logistic regression with Affect Condition, Group, and their interaction entered as independent variables.

A similar multilevel linear regression was fit to trial-level response time data (log-transformed) using the same independent variables. This model tested the main effects and interactions of Food Type, Affect Condition, and Group with a dummy choice variable (coded -1/1 for choice of reference item or food item).

For all models, any within-individual variables (i.e., the intercept, main effects of z-scored health and taste ratings, Affect Condition, and Food Type, and their interactions) were treated as random effects. Regression analyses were conducted using the R packages *lme4* (Bates et al., 2015) and *nmle* (Pinheiro et al., 2022) and marginal means were estimated using *emmeans* (Lenth et al., 2024) and *sjPlot* (Lüdecke et al., 2024).

Model comparison

We assessed the model fit of variants of the time-varying DDM to determine which model parameters best explained the influence of Food Type on behavior. The null model (M0) assumed decision parameters were invariant towards food type. The attribute weight model (M1) assumed both ω_{taste} and ω_{health} varied based on food type. The attribute starting time model (M2) assumed τ_s varied based on food type. Finally, the full model (M3) assumed ω_{taste}, ω_{health}, and τ_s varied based on food type. All models were estimated using the same procedures described in the Computational model subsection of the Methods section located in the main text.

Model identifiability and posterior predictive checks

We then evaluated the identifiability of each model. For each model, a set of simulated datasets was generated reflecting the structure and parameters of the corresponding model. First, each model was fit to the empirical data using the procedures described in the Materials and Methods section of the main text, under the subsection *Computational Modeling*. Data were simulated using the empirical trial structure and the medians of the individual-level posterior parameter distributions. Each simulated dataset was fit using all four models (M0, M1, M2, and M3), regardless of the generating model. For model fitting, we employed the same Bayesian hierarchical framework that was used in previous steps. We evaluated model identifiability by determining the frequency with which the model that generated the data was also identified as the best-fitting model, based on WAIC (Watanabe, 2010) implemented in the *loo* package in R (Vehtari et al., 2017, 2024). This allowed us to assess the extent to which each model was uniquely identifiable and distinguishable from alternative models. A model was considered successfully recovered when it was the best-fitting model for the data it generated.

In addition, we performed posterior predictive checks to ensure that the models could capture key features of the observed data. We assessed the simulated choice and response time patterns of the model variants using nonparametric equivalent aligned rank tests (Wobbrock et al., 2011) implemented in the *ARTool* package in R (Kay et al., 2021). For this procedure, we calculated both the empirical and the simulated mean choice proportions and median response times for each participant across Affect Conditions and Food Types. Aligned rank tests were run separately on mean choice proportions and median response times evaluating the interaction between Group, Affect Condition, Food Type, and Source (empirical or simulated data) for each model. We subsequently ran the multi-level logistic regression models predicting trial-level choice from the interaction between Group, Affect Condition, and Food Type on the simulated data to confirm we could recover the results from the empirical data.

Parameter recovery

We conducted a parameter recoverability exercise to ensure that the values of the winning model could be accurately recovered from data that had similar qualities to the empirical data included in the current study. For this exercise, we took the mean values of the subject-level parameters (χ_fit) estimated in both the neutral and negative affect conditions and simulated data using the trial-level health and taste ratings from the empirical data used during estimation. This synthetic data was then used to fit the winning model using the procedures described previously. We extracted this new set of parameters, once again calculating the mean values of the subject-level parameters (χ_rec). We ran Pearson-correlations between χ_fit and χ_rec to assess parameter recovery.

Analysis of additional DDM parameters

Mixed-effects regression models were fit to non-decision time (τ_{ND}), boundary separation (α), and starting point bias (z), following the same estimation procedures described in the main text. As these parameters did not include food type as a factor, each participant contributed two observations (one per affect condition). Where significant or trending group by condition interactions were observed, condition effect scores (negative minus neutral) were compared between groups using two-sample Wilcoxon ranksum tests, with additional one-sample Wilcoxon signed-rank tests run within each group to assess whether the condition effect differed from zero.

Confirmatory analysis of DDM parameters

To confirm the presence of significant interactions identified in the mixed-effects regression models, we conducted difference-in-difference analyses for each DDM parameter where a significant two-way or three-way interaction was observed. For each participant, we computed two sets of difference scores. For parameters with a significant three-way interaction (Group × Condition × Food Type), we calculated the food-type effect within each condition (high-fat minus low-fat) separately for the neutral and negative affect conditions, the condition effect within each food type (negative minus neutral) separately for low-fat and high-fat foods, and an overall difference-in-difference score by subtracting the food-type effect in the neutral condition from the food-type effect in the negative condition. For parameters with a significant two-way interaction, we computed the relevant difference score corresponding to the interaction of interest. Group differences in each of these scores were assessed using Wilcoxon rank-sum tests.

Mood analysis

We analyzed the specific emotions that changed following the mood inductions. As described in the main text, mood was assessed using the Profile of Mood States (POMS; (McNair et al., 1989)). The POMS has the following subscales: Anger, Confusion, Depression, Fatigue, Tension, and Vigor.

Mood induction effectiveness

To assess the effectiveness of the mood induction in the BN group, we conducted follow-up simple effects analyses using estimated marginal means from our primary linear mixed-effects model (reported in Table S1). This model included group (HC = 0, BN = 1), condition (Neutral = 0, Negative = 1), and time (preinduction = 0, post-induction = 1) as fixed effects, all interactions, and random intercepts for participants to account for within-subject correlations.

We conducted two complementary analyses. First, we examined the group × time interaction within each condition separately to assess whether the mood induction produced significant changes in negative affect within the BN group. Second, we directly compared post-induction affect between conditions within the BN group to determine whether participants experienced meaningfully different affective states when completing the food decision task.

Effect sizes for within-subject comparisons were calculated as Cohen's d using the mean difference divided by the pooled standard deviation of the repeated measures.

Alternative symptom severity measure

Recent studies suggest that negative urgency (i.e., acting impulsively when experiencing negative affect) contributes to binge-eating pathology (Wonderlich et al., 2024). Given the restrictive food choice behavior we observed in this study, decision parameters might correlate with symptom severity measures related to restrictive behaviors outside the lab. We used linear regressions to evaluate the extent to which subject-level parameters (i.e., τ_S, ω_{taste}, ω_{health}) could predict scores on the negative urgency subscale of the UPPS-P Impulsive Behavior Scale (Lynam et al., 2006) and the restraint subscale of the Eating Disorder Examination Questionnaire (EDE-Q; (Fairburn & Beglin, 1994)).

Association between dimensions of symptom severity

To address concerns about the specificity of associations with binge eating versus other eating disorder symptoms, we examined the correlation between self-reported binge frequency and dietary restraint in our BN sample. Specifically, we assessed the relationships between the EDE-Q Restraint subscale and two measures of binge frequency over the past three months: subjective binge episodes (SBEs; episodes involving a loss of control over eating but without consuming an objectively large amount of food) and objective binge episodes (OBEs; episodes involving both loss of control and consumption of an objectively large amount of food). Given that binge frequency data are typically positively skewed (i.e., log-normally distributed), we used Spearman's rank correlation (ρ) to examine these associations, as this non-parametric approach is robust to violations of normality assumptions.

Supplementary Results

Analysis of choice patterns

We replicated the findings from the original analysis, confirming that individuals with BN were more likely than the HC group to choose the reference item (Table S9; $\beta = -0.48$, $z = -2.63$, $p = 0.009$), with the BN group selecting the reference item in approximately 68.3% of trials (95% CI: 57.0% – 77.8%) and the HC group selecting it in 45.3% of trials (95% CI: 33.9% – 58.3%). We also replicated the Group-by-Food Type interaction reported in the original analysis ($\beta = -0.27$, $z = -1.99$, $p = 0.047$). Those in HC group chose approximately 63.1% (*95% CI: 52.0% – 73.0%*) of low-fat foods and 46.0% (*95% CI: 27.8% – 65.3%*) of high-fat foods, while individuals with BN chose 53.0% (*95% CI: 42.6% – 63.1%*) of low-fat foods and 16.1% (*95% CI: 8.3% – 28.8%*) of high-fat foods. While individuals with BN were less likely to choose the presented foods over the neutral reference item in general, both groups chose high-fat foods less often than low-fat foods ($\beta = -0.62$, $z = -4.58$, $p = 4.75 \times 10^{-6}$).

Similar to the original analysis, we did not observe a significant main effect of Affect Condition ($\beta = -0.02$,$z = -0.26$, $p = 0.793$), an interaction between Affect Condition and Group ($\beta = 0.07$,$z = 0.82$, $p = 0.410$), or an interaction Affect Condition and Food Type ($\beta = -0.03$,$z = -0.62$, $p = .535$) on food choice. We also did not observe a significant three-way interaction between Affect Condition, Food Type, and Group on choice ($\beta = -0.10$, $z = -1.91$, $p = 0.056$).

In a separate multilevel regression (Table S10), we examined the relationships among food choice, taste and health ratings, BN diagnosis, and negative affect. We confirmed the original paper's finding that health ratings more strongly influenced food choice among the BN group compared to the HC group ($\beta = 0.64$, $z = 2.77$, $p = 0.006$). We additionally observed that taste ratings were less influential among the BN group compared to the HC group ($\beta = -0.65$, $z = -2.78$, $p = 0.005$).We also reproduced the original paper's finding of a significant, negative interaction between Affect Condition and taste ratings on choice, such that negative affect reduced the influence of taste ratings on choice among both groups ($\beta = -0.45$, $z = -2.57$,$p = 0.010$).

Furthermore, we examined trials with an opportunity for self-control (Table S11). Reproducing the original analyses, we found that the BN group used self-control more often than the HC group (β = 0.38, z = 2.14, p = 0.033). We did not observe a main effect of Affect Condition on self-control (β = 0.08, z = 0.70, p = 0.486), nor an interaction between Affect Condition and Group (β = 0.15, z = 1.27, p = 0.204).

We next examined RT patterns using multilevel regression models fit to trial-level RT data (Table S12). We did not observe significant main effects of Affect Condition or its interactions with other variables. An interaction between Food Type and choice on RT indicated that, across groups, rejections were relatively faster for high-fat foods than for low-fat foods, while selections were relatively slower for high-fat foods than for low-fat foods (β = 0.03, t = 2.70, p = 0.007). Additionally, we found a positive interaction between Group and choice, suggesting that the BN group was slower than the HC group to choose presented foods over the neutral reference item (β = 0.03, t = 2.44, p = 0.015).

Modeling results

Model comparison

These different model variants are described in Table S13. The winning model (M3) included terms representing the influence of Food Type on both attribute weights (ω_{taste}, ω_{health}) and attribute starting time (τ_s).

Model identifiability

The results of the model recovery exercise are summarized in Table S14. Models M0, M1, and M3 all had perfect recoverability. However, M2 had poor recoverability, as it was always mis-identified as M3.

Posterior predictive check

The data from M3 best replicated the results from the empirical data. While all models had biases in predicting choice data, these were smallest in M3. M0 (Table S15) was significantly biased in predicting choice based on Food Type (F(1,352) = 11.97, p = 0.001) and was biased in predicting response times (F (1,352) = 7.88, p = 0.005). Both M1 (Table S16) and M2 (Table S17) were similarly biased in predicting choice based on Food Type (M1: F(1,352) = 45.07, $p = 7.63 \times 10^{-11}$; M2: F(1,352) = 4.90, p = 0.028), but M3 (Table S18) was not (F(1,352) = 1.70, p = 0.193).

In the empirical data, a multilevel logistic regression found significant, negative main effects of Group and Food Type, and a negative Group × Food Type interaction (Table S9). M0 (Table S19) was able to reproduce the main effect of Group (β = −0.34, t = −2.74, p = 0.006) and the Group × Food Type interaction (β = −0.32, t = −4.34, $p = 1.41 \times 10^{-5}$), but not the main effect of Food Type (β = −0.01, t = −0.19, p = 0.848). M1 (Table S20) produced a marginal negative effect of Group (β = −0.28, t = −1.93, p = 0.053) and the negative Group × Food Type interaction (β = −0.22, t = −3.27, p = 0.001), but the significant effect of Food Type had the wrong sign (β = 0.42, t = 6.35, $p = 2.17 \times 10^{-1}$). M2 (Table S21) produced the negative Group effect (β = −0.42, t = −3.08, p = 0.002) and the negative Group × Food Type interaction (β = −0.36, t = −3.36, $p = 7.93 \times 10^{-4}$), but not the significant effect of Food Type (β = −0.16, t = −1.52, p = 0.128). Only M3 (Table S22) could reproduce all three patterns: a negative effect of Group (β = −0.36, t = −2.68, p = 0.007), a negative effect of Food Type (β = −0.36, t = −4.16, $p = 3.25 \times 10^{-5}$), and a negative interaction between the two (β = −0.27, t = −3.11, p = 0.002).

Parameter recovery

As illustrated in Figure S1, recovery was satisfactory for all parameters of M3.

Analysis of additional DDM parameters

Non-decision time

We did not observe significant effects of either Group or Affect Condition in nondecision time (τ_{ND}; Table S23). However, we found a significant Group by Affect Condition interaction (β = −0.091, t = −2.19, p = 0.034), suggesting that the BN group, compared to the HC group, had faster non-decision times following the negative affect induction versus the neutral condition. Although the two-sample Wilcoxon test comparing the condition effect between groups fell just short of significance (W = 351, p = 0.058), one-sample tests indicated that the condition effect was non-significant in HC (W = 143, p = 0.355) while trending toward significance in BN (W = 96, p = 0.075), consistent with a selective speeding of non-decision time under negative affect in BN that was not observed in HC.

Boundary separation

For the boundary separation parameter (α; Table S24), there was a significant difference between the HC and BN groups (β = −0.397, t = −2.84, p = 0.007), with the BN group exhibiting reduced response caution compared to the HC group. We found a significant negative effect of Affect Condition (β = −0.366, t = −4.26, p < 0.001), and a trending positive interaction between Group and Affect Condition (β = 0.227, t = 1.94, p = 0.058). One-sample Wilcoxon tests confirmed that the negative affect induction significantly reduced boundary separation in HC (V = 13, p < 0.001), but not in BN (V = 99, p = 0.090), though the group difference in condition effect fell just short of significance (W = 187, p = 0.098). Together, these results suggest that negative affect prompted greater response caution reductions in HC than in BN, consistent with the trending interaction, though the non-significant Wilcoxon two-sample test urges caution in this interpretation.

Starting point bias

There were no Group or Affect Condition differences in starting point bias (z; Table S25).

Confirmatory analysis of DDM parameters

Relative attribute onset (τ_S)

To confirm the significant three-way interaction (Group × Condition × Food Type), we computed difference-in-difference scores for each participant. In the neutral condition, HC showed a larger food-type effect than BN (HC: M = 0.14, BN: M = −0.15; W = 367, p = 0.021), while this group difference was absent in the negative affect condition (HC: M = 0.02, BN: M = 0.00; W = 270, p = 0.879). Groups also differed in how negative affect modulated attribute onset for low-fat foods (HC: M = −0.14, BN: M = −0.31; W = 359, p = 0.033) but not high-fat foods (HC: M = −0.20, BN: M = −0.16; W = 280, p = 0.710). The overall difference-in-difference score was significantly larger in BN than HC (HC: M = −0.10, BN: M = 0.15; W = 156, p = 0.018), confirming the presence of the three-way interaction.

Taste weights (ω_{taste})

To confirm the significant two-way interaction between group and condition, we compared groups on the condition effect within each food type. Groups differed significantly in the condition effect for low-fat foods (HC: M = −0.16, BN: M = 0.02; W = 162, p = 0.026) but not high-fat foods (HC: M = −0.13, BN: M = −0.07; W = 221, p = 0.369), confirming that the group difference in response to negative affect induction was specific to low-fat foods. To confirm the significant two-way interaction between group and food type in the negative affect condition, groups did not differ in the foodtype effect in the neutral condition (HC: M = −0.24, BN: M = −0.29; W = 292, p = 0.526), but did differ significantly in the negative affect condition (HC: M = −0.22, BN: M = −0.38; W = 372, p = 0.015), confirming that the group difference in food-type bias emerged specifically following negative affect induction.

Healthiness weights (ω_{health})

To confirm the significant two-way interaction between group and food type, we compared groups on the food-type effect within each condition. Groups differed significantly in both the neutral condition (HC: M = −0.04, BN: M = 0.24; W = 133, p = 0.004) and the negative affect condition (HC: M = 0.03, BN: M = 0.27; W = 156, p = 0.018), confirming that BN placed consistently greater weight on health information for high-fat versus low-fat foods regardless of affective state, while HC did not.

Mood analysis

Anger subscale

We found that the BN group had significantly higher scores on the Anger subscale (Table S26; β = 7.59, t = 3.16, p = 0.002). We also found a significant, positive interaction between Affect Condition and Timing (β = 5.71, t = 3.04, p = 0.003), indicating that the negative affect induction increased anger ratings across Groups.

Confusion subscale

We found that the BN group had significantly higher scores on the Confusion subscale (Table S27; β = 6.07, t = 4.75,p = 1.03 × 10^{-5}). We also found a significant, positive interaction between Affect Condition and Timing (β = 2.30, t = 2.42, p = 0.017), indicating that the negative affect induction increased confusion ratings across Groups.

Depression subscale

We found that the BN group had significantly higher scores on the Depression subscale (Table S28; β = 16.06, t = 4.63,p = 1.84 × 10^{-5}). We also found a significant, positive interaction between Affect Condition and Timing (β = 5.58, t = 2.44, p = 0.016), indicating that the negative affect induction increased depression ratings across Groups.

Fatigue subscale

We found that the BN group had significantly higher scores on the Fatigue subscale (Table S29; β = 6.04, t = 3.34,p = 0.001). No other significant contrasts emerged.

Tension subscale

We found that the BN group had significantly higher scores on the Tension subscale (Table S30; β = 8.76, t = 4.38,p = 5.05 × 10^{-5}). We also found both a significant, negative interaction between Group and Timing (β = −2.50, t = 1.67, p = 0.021) and a significant, positive interaction between Group, Affect Condition, and Timing (β = 3.82, t = 2.53, p = 0.013). Taken together, these indicate that only the BN group's tension ratings changed after the affect induction, but in a conditiondependent way: decreasing tension following the neutral affect induction and increasing tension following the negative affect induction.

Vigor subscale

We found that the BN group had significantly lower scores on the Vigor subscale (Table S31; β = −10.40, t = −5.73,p = 2.33 × 10^{-7}). We also found a significant, negative interaction between Affect Condition and Timing (β = −4.93, t = −3.58, p = 0.001), indicating that the negative affect induction decreased vigor ratings across Groups.

Effectiveness of mood induction in the BN group

In the first analysis, we examined changes in negative affect from pre- to post-induction within each condition for the BN group. In the Negative condition, individuals with bulimia nervosa demonstrated a substantial increase in negative affect from pre- to post-induction (mean difference = 20.36, SE = 4.21, t = 4.84, p < 0.0001, Cohen's d = 0.97). This large effect size indicates that the negative mood induction produced a meaningful increase in negative affect. In contrast, the Neutral condition showed no significant change in negative affect in the BN group (mean difference = 7.16, SE = 4.21, t = 1.70, p = 0.327, Cohen's d = 0.34).

In the second analysis, we directly compared post-induction negative affect between conditions within the BN group. Reported negative affect was significantly higher following the negative mood induction than after the neutral mood induction (mean difference = 17.40, SE = 4.21, t = 4.13, p = 0.0003, Cohen's d = 0.83). This large effect size represents a meaningful and statistically robust difference in affective states between conditions.

These within-group effects confirm that the negative mood induction was effective in producing increased negative affect in the BN group and resulted in significantly different affective states between the negative and neutral conditions. Critically, these findings demonstrate that BN participants completed the food decision task under meaningfully different affective states in the two conditions, supporting the interpretability of the subsequent analyses.

Alternative symptom severity measures

There were no significant relationships between negative urgency ratings decision parameters (Table S32) or restrictive behaviors (Table S33).

Associations between symptom severity measures

The correlation between EDE-Q Restraint and subjective binge episodes was small and non-significant (ρ = 0.21, S = 2045.2, p = 0.306). Similarly, the correlation between EDE-Q Restraint and objective binge episodes was near zero and non-significant (ρ = 0.05, S = 2465.7, p = 0.806). These results indicate that subjective binge frequency and dietary restraint were relatively independent dimensions of eating pathology in our sample. This dissociation supports the specificity of our primary findings: the fact that our DDM parameters were associated with binge frequency but not with dietary restraint suggests that the affect-induced changes in decision-making we observed are specifically related to loss-of-control eating behavior rather than reflecting a general correlate of dietary restraint.

Supplementary Tables

Supplementary Table S1. Linear mixed-effects regression analyzing overall negative affect across Group, Affect Condition and Timing

Predictors	B	SE	T	p
Intercept	-7.14	7.35	-0.97	0.335
Group	55.02	9.97	5.52	**<0.001**
Affect Condition	-2.43	4.49	-0.54	0.590
Timing	0.05	4.49	0.01	0.992
Group × Affect Condition	-7.69	6.09	-1.26	0.209
Group × Timing	-7.21	6.09	-1.18	0.239
Affect Condition × Timing	20.43	6.35	3.22	**0.002**
Group × Affect Condition × Timing	7.09	8.62	0.82	0.412
$N_{subject}$	46			
Observations	184			

Supplementary Table S2. Linear mixed-effects regression analyzing attribute onset (τ_s) across Group, Affect Condition, and Food Type.

Predictors	B	SE	T	P
Intercept	-0.46	0.07	-6.04	**<0.001**
Group	0.14	0.10	1.39	0.173
Affect Condition	-0.16	0.07	-2.16	**0.032**
Food Type	0.14	0.09	1.54	0.125
Group × Affect Condition	-0.23	0.10	-2.26	**0.026**
Group × Food Type	-0.29	0.12	-2.33	**0.022**
Affect Condition × Food Type	-0.12	0.09	-1.45	0.150
Group × Affect Condition × Food Type	0.28	0.12	2.36	**0.020**
$N_{subject}$	46			
Observations	184			

Supplementary Table S3. **Simple effects analyses comparing groups on attribute onset (τ_s) difference scores.**

Contrast	Estimate	SE	df	t	p
Group difference in condition effect by food type					
Condition effect for Low Fat foods	-0.23	0.10	132	-2.26	**0.026**
Condition effect for High Fat foods	0.05	0.10	132	0.51	0.614
Group differences in food type effects by condition					
Food type effects in Neutral condition	-0.29	0.13	132	-2.33	**0.022**
Food type effects in Negative condition	-0.01	0.03	132	-0.52	0.607
Food type effects within groups and condition					
Low fat vs. High fat (HC Neutral)	0.23	0.06	132	4.05	**<0.001**
Low fat vs. High fat (HC Negative)	0.29	0.05	132	5.64	**<0.001**
Low fat vs. High fat (BN Neutral)	0.22	0.06	132	3.61	**<0.001**
Low fat vs. High fat (BN Negative)	0.38	0.05	132	7.00	**<0.001**

Note. HC = healthy controls; BN = bulimia nervosa. Difference scores reflect food-type effects (high-fat minus low-fat) and condition effects (Negative minus Neutral). Bold p-values indicate p < 0.05.

Supplementary Table S4. **Linear mixed-effects regression analyzing tastiness attribute weights (ω_{taste}) across Group, Affect Condition, and Food Type**

Predictors	B	SE	T	P
Intercept	0.57	0.04	13.55	**<0.001**
Group	-0.39	0.06	-6.86	**<0.001**
Affect Condition	-0.16	0.05	-2.96	**0.004**
Food Type	-0.23	0.06	-4.05	**<0.001**
Group × Affect Condition	0.18	0.07	2.46	**0.015**
Group × Food Type	-0.06	0.08	-0.83	0.410
Affect Condition × Food Type	0.01	0.05	0.21	0.834
Group × Affect Condition × Food Type	-0.10	0.07	-1.47	0.144
$N_{subject}$	46			
Observations	184			

Supplementary Table S5. **Simple effects analyses comparing groups on tastiness attribute weight (ω_{taste}) difference scores.**

Contrast	Estimate	*SE*	*df*	*t*	*p*
Group difference by condition interaction					
Neutral vs. Negative (HC – BN)	0.13	0.06	132	1.95	0.053
Group differences within conditions					
HC vs, BN (Neutral)	0.42	0.04	44	9.68	**<0.001**
HC vs. BN (Negative)	0.30	0.06	44	5.17	**<0.001**
Condition effects within groups					
Neutral vs. Negative (HC)	0.15	0.05	132	3.20	**0.002**
Neutral vs. Negative (BN)	0.03	0.04	132	0.61	0.542
Food type effects within groups					
Low-fat versus High-fat (HC)	0.22	0.05	132	4.29	**<0.001**
Low-fat versus High-fat (BN)	0.34	0.05	132	7.12	**<0.001**

Note. HC = healthy controls; BN = bulimia nervosa. Bold p-values indicate p < 0.05..

Supplementary Table S6. **Linear mixed-effects regression analyzing healthiness attribute weights (ω_{health}) across Group, Affect Condition, and Food Type.**

Predictors	B	SE	T	P
Intercept	-0.51	0.04	-12.14	**<0.001**
Group	0.38	0.06	6.73	**<0.001**
Affect Condition	0.09	0.05	1.82	0.071
Food Type	-0.04	0.07	-0.62	0.533
Group × Affect Condition	-0.13	0.07	-1.89	0.062
Group × Food Type	0.28	0.09	3.09	**0.002**
Affect Condition × Food Type	0.07	0.08	0.87	0.384
Group × Affect Condition × Food Type	-0.04	0.11	-0.34	0.735
$N_{subject}$	46			
Observations	184			

Supplementary Table S7. **Simple effects analyses comparing groups on healthiness attribute weight (ω_{healt}) difference scores.**

Contrast	Estimate	SE	df	T	P
Group Differences within conditions					
HC vs, BN (Neutral)	-0.53	0.04	44	-12.02	**<0.001**
HC vs. BN (Negative)	-0.38	0.06	44	-6.42	**<0.001**
Condition effects within groups					
Neutral vs. Negative (HC)	-0.13	0.05	132	-2.50	**0.014**
Neutral vs. Negative (BN)	0.02	0.05	132	0.46	0.645
Food type effects within groups					
Low-fat versus High-fat (HC)	0.01	0.06	132	0.13	0.897
Low-fat versus High-fat (BN)	-0.26	0.06	132	-4.40	**<0.001**

Note. HC = healthy controls; BN = bulimia nervosa. Bold p-values indicate p < 0.05.

Supplementary Table S8. **Zero-inflated negative binomial model of symptom severity.**

Outcome	Predictors		B	SE	Z	p
Subjective Binge Episodes	**Count Model**	Intercept	-29.45	12.30	-2.40	**0.017**
		τ_s Neu LF	-0.21	0.60	-0.36	0.722
		τ_s Neu HF	1.48	1.29	1.14	0.254
		τ_s Neg LF	1.09	4.02	-0.27	0.787
		τ_s Neg HF	-46.39	16.56	-2.80	**0.005**
	Zero-Inflated Model	Intercept	-1.28	0.60	-2.12	**0.034**
		$N_{subject}$	25			
		Observations	25			
Objective Binge Episodes	**Count Model**	Intercept	5.69	4.20	1.36	0.175
		τ_s Neu LF	0.47	0.28	1.69	0.091
		τ_s Neu HF	0.16	0.59	0.27	0.790
		τ_s Neg LF	0.99	3.03	0.33	0.745
		τ_s Neg HF	0.07	4.62	0.02	0.998
	Zero-Inflated Model	Intercept	-21.27	8306.43	0.00	0.998
		$N_{subject}$	25			
		Observations	25			

Note: Neu = Neutral affect condition; Neg = Negative affect condition; LF = Low-fat food; HF = High-fat food.

Supplementary Table S9. **Logistic mixed-effects regression analyzing food choice across Affect Condition and Food Type split by Group.**

Predictors	B	SE	Z	p
Intercept	-0.29	0.18	-1.59	0.113
Group	-0.48	0.18	-2.63	**0.009**
Affect Condition	-0.02	0.09	-0.26	0.793
Food Type	-0.62	0.13	-4.58	**<0.001**
Group × Affect Condition	0.07	0.09	0.82	0.410
Group × Food Type	-0.27	0.13	-1.99	**0.047**
Affect Condition × Food Type	-0.03	0.05	-0.62	0.535
Group × Affect Condition × Food Type	-0.10	0.05	-1.91	0.056
$N_{subject}$	46			
Observations	3271			

Supplementary Table S10. **Logistic mixed-effects regression analyzing choice across Group and Affect Condition using tastiness and healthiness attribute ratings.**

Predictors	B	SE	Z	p
Intercept	-0.60	0.24	-2.46	**0.014**
Group	-0.80	0.24	-3.36	**<0.001**
Affect Condition	0.05	0.20	0.26	0.797
Taste Rating [z]	2.93	0.26	11.40	**<0.001**
Health Rating [z]	1.49	0.24	6.29	**<0.001**
Group × Affect Condition	0.24	0.19	1.21	0.225
Taste × Group	-0.65	0.23	-2.78	**0.005**

Health × Group	0.64	0.23	2.77	**0.006**
Taste × Affect Condition	-0.45	0.17	-2.57	**0.010**
Health × Affect Condition	-0.10	0.17	-0.61	0.542
Taste × Group × Affect Condition	-0.03	0.13	-0.20	0.841
Health × Group × Affect Condition	0.04	0.16	0.24	0.812
$N_{subject}$	46			
Observations	3271			

Supplementary Table S11. Logistic mixed-effects regression analyzing self-control across Group and Affect Condition.

Predictors	B	SE	Z	p
Intercept	-0.30	0.18	-1.68	0.093
Group	0.38	0.18	2.14	**0.033**
Affect Condition	0.08	0.12	0.70	0.486
Group × Affect Condition	0.15	0.12	1.27	0.204
$N_{subject}$	46			
Observations	1118			

Supplementary Table S12. Linear mixed-effects regression analyzing log-transformed response times across Group and Affect Condition using tastiness and healthiness attribute ratings.

Predictors	B	SE	Z	p
Intercept	0.53	0.04	15.10	**<0.001**
Group	-0.02	0.04	-0.70	0.487
Affect Condition	-0.02	0.02	-1.16	0.250
Food Type	-0.03	0.01	-2.07	**0.044**
Choice	0.01	0.01	0.64	0.525
Group × Affect Condition	-0.02	0.02	-0.84	0.406
Group × Food Type	-0.02	0.01	-1.17	0.248
Affect Condition × Food Type	0.01	0.01	0.64	0.527
Group × Choice	0.03	0.01	2.44	**0.015**
Affect Condition × Choice	0.00	0.01	-0.50	0.959
Food Type × Choice	0.03	0.01	2.70	**0.007**
Group × Affect Condition × Food Type	-0.01	0.01	-0.64	0.524
Group × Affect Condition × Choice	-0.01	0.01	-1.26	0.208
Group × Food Type × Choice	-0.01	0.01	-0.65	0.513
Affect Condition × Food Type × Choice	0.01	0.01	0.75	0.455
Group × Affect Condition × Food Type × Choice	-0.01	0.01	-0.60	0.548
$N_{subject}$	46			
Observations	3271			

Supplementary Table S13. Model fit metrics for alternative model specifications.

All models were estimated with the time-varying drift rate but differed in which parameters varied by Food Type.

Model	Parameters per subject	Group	WAIC	Parameters varying with Food Type
M0	7	HC	5104	NaN
		BN	6510	

M1	9	HC	4886	$\omega_{\text{health}}, \omega_{\text{taste}}$
		BN	6266	
M2	8	HC	4926	τ_s
		BN	6298	
M3	10	HC	4874	$\tau_s, \omega_{\text{health}}, \omega_{\text{taste}}$
		BN	6135	

Note. ω_{health} = health coefficient, ω_{taste} = taste coefficient, τ_s = relative starting time.

Supplementary Table S14. Confusion matrix indicating frequency with which each candidate model was selected for each simulated model

		Predicted			
	Model	M0	M1	M2	M3
Simulated	M0	1.00	0.00	0.00	0.00
	M1	0.00	1.00	0.00	0.00
	M2	0.00	0.00	0.00	1.00
	M3*	0.00	0.00	0.00	1.00

Note: * symbol indicates winning model from model comparison based on empirical data

Supplementary Table S15. Analysis of variance using mixed effects models to assess deviance of M0 predicted choice frequencies compared to empirical data

Terms	F	Df	p
Data source	18.36	1, 308	**<0.001**
Data source × Group	0.14	1, 308	0.709
Data source × Affect Condition	0.55	1, 308	0.458
Data source × Food Type	16.76	1, 308	**<0.001**
Data source × Group × Affect Condition	0.06	1, 308	0.808
Data source × Group × Food Type	1.32	1, 308	0.252
Data source × Affect Condition × Food Type	0.00	1, 308	0.964
Data source × Group × Affect Condition × Food Type	0.01	1, 308	0.919
N_{subject}	46		
Observations	368		

Supplementary Table S16. Analysis of variance using mixed effects models to assess deviance of M1 predicted choice frequencies compared to empirical data

Terms	F	Df	p
Data source	95.27	1, 308	**<0.001**
Data source × Group	4.73	1, 308	**0.030**
Data source × Affect Condition	0.10	1, 308	0.754
Data source × Food Type	67.78	1, 308	**<0.001**
Data source × Group × Affect Condition	0.89	1, 308	0.346
Data source × Group × Food Type	0.34	1, 308	0.563
Data source × Affect Condition × Food Type	0.18	1, 308	0.670
Data source × Group × Affect Condition × Food Type	0.60	1, 308	0.441
$N_{subject}$	46		
Observations	368		

Supplementary Table S17. Analysis of variance using mixed effects models to assess deviance of M2 predicted choice frequencies compared to empirical data

Terms	F	Df	p
Data source	14.08	1, 308	**<0.001**
Data source × Group	0.05	1, 308	0.827
Data source × Affect Condition	0.06	1, 308	0.803
Data source × Food Type	6.61	1, 308	**0.011**
Data source × Group × Affect Condition	0.20	1, 308	0.654
Data source × Group × Food Type	1.49	1, 308	0.223
Data source × Affect Condition × Food Type	0.27	1, 308	0.601
Data source × Group × Affect Condition × Food Type	0.05	1, 308	0.821
$N_{subject}$	46		
Observations	368		

Supplementary Table S18. Analysis of variance using mixed effects models to assess deviance of M3 predicted choice frequencies compared to empirical data

Terms	F	Df	p
Data source	8.84	1, 308	**0.003**
Data source × Group	0.00	1, 308	0.944
Data source × Affect Condition	0.92	1, 308	0.339
Data source × Food Type	1.70	1, 308	0.193
Data source × Group × Affect Condition	0.13	1, 308	0.720
Data source × Group × Food Type	0.70	1, 308	0.404
Data source × Affect Condition × Food Type	0.11	1, 308	0.741
Data source × Group × Affect Condition × Food Type	0.97	1, 308	0.326
$N_{subject}$	46		
Observations	368		

Supplementary Table S19. Logistic mixed-effects regression assessing choice predictions from Model M0.

Predictors	B	SE	Z	p
Intercept	0.29	0.12	2.30	0.021
Group	-0.34	0.12	-2.74	**0.006**
Affect Condition	-0.08	0.07	-1.15	0.251
Food Type	-0.01	0.07	-0.19	0.848
Group × Affect Condition	0.03	0.07	0.39	0.695
Group × Food Type	-0.32	0.07	-4.34	**<0.001**
Affect Condition × Food Type	-0.05	0.05	-1.01	0.312
Group × Affect Condition × Food Type	-0.06	0.05	-1.27	0.205
$N_{subject}$	46			
Observations	6542			

Supplementary Table S20. Logistic mixed-effects regression assessing choice predictions from Model M1.

Predictors	B	SE	Z	p
Intercept	0.89	0.15	6.12	**<0.001**
Group	-0.28	0.15	-1.93	0.053
Affect Condition	-0.08	0.07	-1.03	0.303
Food Type	0.42	0.07	6.35	**<0.001**
Group × Affect Condition	0.17	0.07	2.33	**0.020**
Group × Food Type	-0.22	0.07	-3.27	**0.001**
Affect Condition × Food Type	-0.04	0.04	-0.98	0.325
Group × Affect Condition × Food Type	0.02	0.04	0.60	0.552
$N_{subject}$	46			
Observations	6542			

Supplementary Table S21. Logistic mixed-effects regression assessing choice predictions from Model M2.

Predictors	B	SE	Z	p
Intercept	0.28	0.14	2.05	**0.041**
Group	-0.42	0.14	-3.08	**0.002**
Affect Condition	-0.03	0.08	-0.39	0.695
Food Type	-0.16	0.11	-1.52	0.128
Group × Affect Condition	0.00	0.08	0.01	0.993
Group × Food Type	-0.36	0.11	-3.36	**<0.001**
Affect Condition × Food Type	-0.08	0.06	-1.37	0.170
Group × Affect Condition × Food Type	-0.07	0.06	-1.30	0.195
$N_{subject}$	46			
Observations	6542			

Supplementary Table S22. **Logistic mixed-effects regression assessing choice predictions from Model M3.**

Predictors	B	SE	Z	p
Intercept	0.13	0.13	0.94	0.347
Group	-0.36	0.13	-2.68	**0.007**
Affect Condition	-0.11	0.07	-1.50	0.133
Food Type	-0.36	0.09	-4.16	**<0.001**
Group × Affect Condition	0.02	0.07	0.28	0.777
Group × Food Type	-0.27	0.09	-3.11	**0.002**
Affect Condition × Food Type	-0.09	0.04	-1.96	0.050
Group × Affect Condition × Food Type	0.03	0.04	0.58	0.562
$N_{subject}$	46			
Observations	6542			

Supplementary Table S23. **Linear mixed-effects regression assessing non-decision time (τ_{ND}) across Group and Affect Condition.**

Predictors	B	SE	T	p
Intercept	0.51	0.03	16.06	**<0.001**
Group	0.02	0.04	0.39	0.701
Affect Condition	0.02	0.03	0.74	0.464
Group × Affect Condition	-0.09	0.04	-2.19	**0.034**
$N_{subject}$	46			
Observations	92			

Supplementary Table S24. **Linear mixed-effects regression assessing boundary separation (α) across Group and Affect Condition.**

Predictors	B	SE	T	P
Intercept	3.46	0.10	33.55	**<0.001**
Group	-0.40	0.14	-2.84	**0.007**
Affect Condition	-0.37	0.09	-4.26	**<0.001**
Group × Affect Condition	0.23	0.12	1.94	0.058
$N_{subject}$	46			
Observations	92			

Supplementary Table S25. **Linear mixed-effects regression assessing starting point (z) across Group and Affect Condition.**

Predictors	B	SE	T	p
Intercept	0.41	0.02	20.13	**<0.001**
Group	-0.01	0.03	-0.48	0.634
Affect Condition	-0.02	0.02	-1.01	0.317
Group × Affect Condition	0.03	0.03	0.98	0.331
$N_{subject}$	46			
Observations	92			

Supplementary Table S26. **Linear mixed-effects regression assessing the Anger POMS subscale as a function of Group, Affect Condition, and Timing.**

Predictors	B	SE	T	p
Intercept	0.57	1.77	0.32	0.748
Group	7.59	2.40	3.16	**0.002**
Affect Condition	0.00	1.33	0.00	1.000
Timing	0.00	1.33	0.00	1.000
Group × Affect Condition	-2.28	1.80	-1.26	0.208
Group × Timing	-1.01	1.83	-0.55	0.581
Affect Condition × Timing	5.71	1.88	3.04	**0.003**
Group × Affect Condition × Timing	0.66	2.57	0.26	0.798
$N_{subject}$	46			
Observations	184			

Supplementary Table S27. **Linear mixed-effects regression assessing the Confusion POMS subscale as a function of Group, Affect Condition, and Timing.**

Predictors	B	SE	T	p
Intercept	2.99	0.94	3.17	**0.002**
Group	6.07	1.28	4.75	**<0.001**
Affect Condition	-0.74	0.68	-1.08	0.282
Timing	-0.84	0.68	-1.22	0.224
Group × Affect Condition	-0.44	0.92	-0.48	0.630
Group × Timing	1.14	0.93	1.23	0.221
Affect Condition × Timing	2.30	0.95	2.42	**0.017**
Group × Affect Condition × Timing	-0.92	1.30	-0.71	0.482
$N_{subject}$	46			
Observations	173			

Supplementary Table S28. **Linear mixed-effects regression assessing the Depression POMS subscale as a function of Group, Affect Condition, and Timing.**

Predictors	B	SE	T	p
Intercept	0.82	2.57	0.32	0.750
Group	16.06	3.47	4.63	**<0.001**
Affect Condition	-0.34	1.66	-0.21	0.836
Timing	-0.49	1.63	-0.30	0.765
Group × Affect Condition	-4.01	2.23	-1.80	0.074
Group × Timing	-0.20	2.23	-0.09	0.928
Affect Condition × Timing	5.58	2.29	2.44	**0.016**
Group × Affect Condition × Timing	3.56	3.13	1.14	0.258
$N_{subject}$	46			
Observations	174			

Supplementary Table S29. **Linear mixed-effects regression assessing the Fatigue POMS subscale as a function of Group, Affect Condition, and Timing.**

Predictors	B	SE	T	p
Intercept	3.52	1.33	2.65	**0.010**
Group	6.04	1.81	3.34	**0.001**
Affect Condition	-1.26	0.95	-1.34	0.183
Timing	0.48	0.93	0.51	0.610
Group × Affect Condition	1.82	1.27	1.43	0.154
Group × Timing	-0.98	1.28	-0.77	0.443
Affect Condition × Timing	0.74	1.33	0.56	0.578
Group × Affect Condition × Timing	-0.35	1.80	-0.20	0.845
$N_{subject}$	46			
Observations	181			

Supplementary Table S30. **Linear mixed-effects regression assessing the Tension POMS subscale as a function of Group, Affect Condition, and Timing.**

Predictors	B	SE	T	p
Intercept	2.76	1.47	1.88	0.066
Group	8.76	2.00	4.38	**<0.001**
Affect Condition	0.05	0.79	0.06	0.952
Timing	0.10	0.79	0.12	0.904
Group × Affect Condition	-1.57	1.07	-1.47	0.145
Group × Timing	-2.50	1.07	-2.34	**0.021**
Affect Condition × Timing	1.86	1.11	1.67	0.098
Group × Affect Condition × Timing	3.82	1.51	2.53	**0.013**
$N_{subject}$	46			
Observations	184			

Supplementary Table S31. **Linear mixed-effects regression assessing the Vigor POMS subscale as a function of Group, Affect Condition, and Timing.**

Predictors	B	SE	T	p
Intercept	17.52	1.33	13.14	**<0.001**
Group	-10.40	1.82	-5.73	**<0.001**
Affect Condition	0.88	0.98	0.90	0.372
Timing	-0.57	0.96	-0.59	0.554
Group × Affect Condition	0.87	1.34	0.65	0.518
Group × Timing	0.48	1.34	0.36	0.720
Affect Condition × Timing	-4.93	1.37	-3.58	**<0.001**
Group × Affect Condition × Timing	1.30	1.90	0.68	0.495
$N_{subject}$	46			
Observations	176			

Supplementary Table S32. **Linear regression models of negative urgency.**

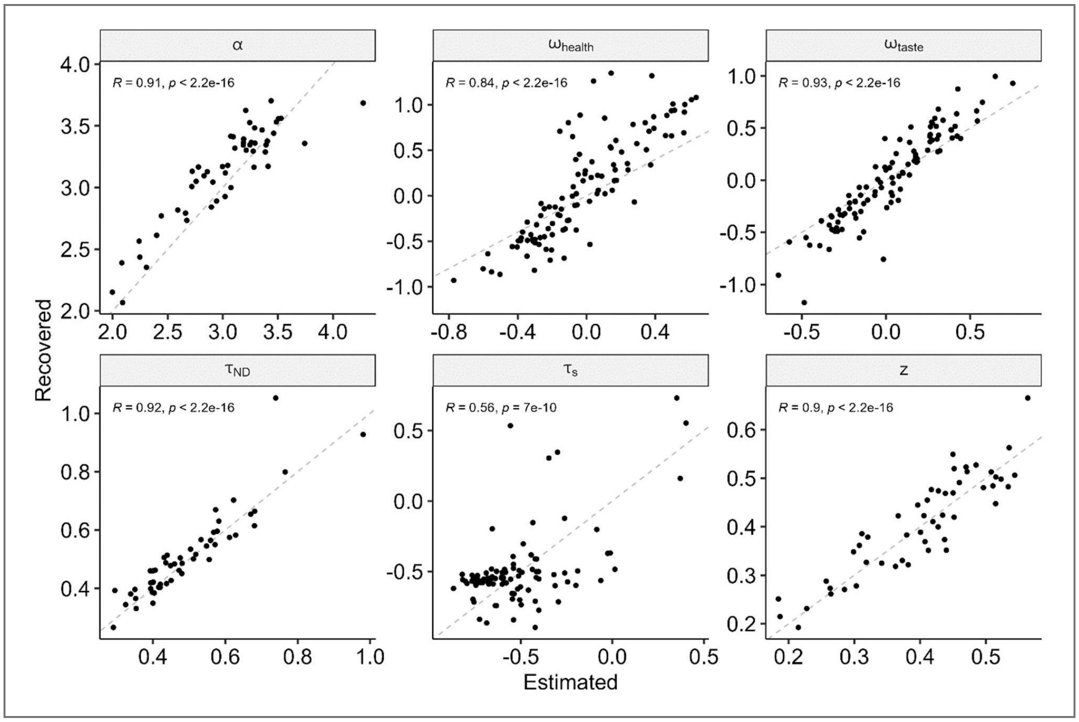
Parameter	Predictors		B	SE	t	p
Attribute onset (τ_s)	Intercept		-2.18	3.61	-0.61	0.552
	Neutral $_{LF}$		0.17	0.22	0.76	0.456
	Neutral $_{HF}$		0.13	0.47	0.28	0.779
	Negative $_{LF}$		1.12	2.52	0.45	0.661
	Negative $_{HF}$		-8.37	4.15	-2.02	0.057
	N $_{subject}$	25				
	Observations	25				
Tastiness weight (ω_{Taste})	Intercept		2.92	0.15	19.61	**<0.001**
	Neutral $_{LF}$		0.26	0.46	0.57	0.578
	Neutral $_{HF}$		0.73	0.57	1.28	0.215
	Negative $_{LF}$		-0.51	0.47	-1.08	0.291
	Negative $_{HF}$		-0.17	0.47	-0.36	0.724
	N $_{subject}$	25				
	Observations	25				
Healthiness weight (ω_{Health})	Intercept		2.86	0.11	26.01	**<0.001**
	Neutral $_{LF}$		-0.59	0.36	-1.62	0.122
	Neutral $_{HF}$		-0.42	0.31	-1.38	0.184
	Negative $_{LF}$		0.65	0.39	1.66	0.113
	Negative $_{HF}$		0.27	0.27	1.00	0.330
	N $_{subject}$	25				
	Observations	25				

Supplementary Table S33. **Linear regression models of the restraint subscale of EDE-Q.**

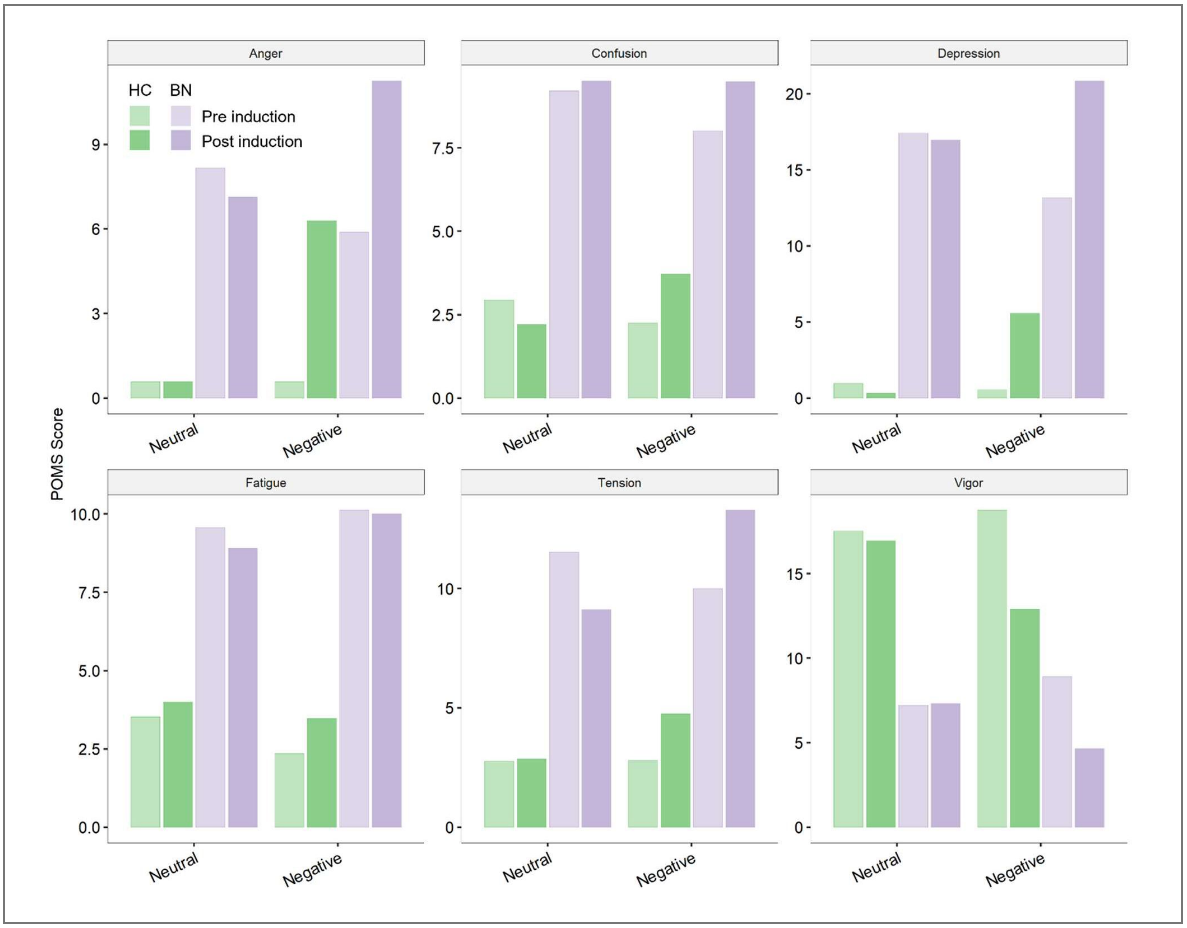
Parameter	Predictors		B	SE	t	p
Attribute onset (τ_s)	Intercept		13.81	14.00	0.99	0.336
	Neutral $_{LF}$		0.58	0.87	0.66	0.515
	Neutral $_{HF}$		1.50	1.83	0.82	0.424
	Negative $_{LF}$		1.45	9.77	0.15	0.883
	Negative $_{HF}$		11.87	16.09	0.74	0.469
	N $_{subject}$	25				
	Observations	25				
Tastiness weight (ω_{Taste})	Intercept		4.45	0.51	8.80	**<0.001**
	Neutral $_{LF}$		-2.67	1.55	-1.72	0.101
	Neutral $_{HF}$		1.46	1.93	0.76	0.457
	Negative $_{LF}$		-1.28	1.61	-0.79	0.437
	Negative $_{HF}$		-0.28	1.58	-0.17	0.863
	N $_{subject}$	25				
	Observations	25				

Healthiness weight (ω_{Health})	Intercept		3.99	0.42	9.42	<**0.001**
	Neutral $_{LF}$		1.82	1.40	1.30	0.209
	Neutral $_{HF}$		0.08	1.19	0.07	0.949
	Negative $_{LF}$		1.40	1.51	0.93	0.366
	Negative $_{HF}$		0.45	1.03	0.44	0.668
	N $_{subject}$	25				
	Observations	25				

Supplementary Figures



Supplementary Figure S1. Results from the parameter recovery exercise. *Note.* α = boundary separation; ω_{health} = health coefficient, ω_{taste} = taste coefficient, τ_{ND} = non-decision time, τ_s = relative starting time, z = starting point bias.



Supplementary Figure S2. **Effectiveness of affect induction across POMS subscales.**

Data availability

The current manuscript is a computational study, so no data have been generated for this manuscript. The code for the analyses presented in this paper is openly accessible at *https://github.com/blairshevlin/Computations_BN_Food_Choice*.

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JES: Original Study: Resources and Supervision; Current Paper: Writing - Review and Editing

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ECL: Writing - Review and Editing

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All authors reviewed and approved the final version of the manuscript.
SIGNIFICANCE STATEMENT Our study revealed that negative emotions biased individuals toward prioritizing taste over health in food choices, and this effect is exaggerated among individuals with bulimia nervosa. This heightened bias, particularly for high-fat foods, was associated with the frequency of subjective binge episodes. These findings clarify the impact of negative affect on food-related decision-making and offer implications for understanding and addressing binge eating.

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Peer reviews

Reviewer #1 (Public review):

Summary:

Using a computational modeling approach based on the Drift and Diffusion Model (DDM) introduced by Ratcliff and McKoon in 2008, the article by Shevlin and colleagues investigates whether there are differences between neutral and negative emotional states in:

(1) The timings of the integration in food choices of the perceived healthiness and tastiness of food options in individuals with bulimia nervosa and healthy participants

(2) The weighting of the perceived healthiness and tastiness of these options.

Strengths:

By looking at the mechanistic part of the decision process, the approach has potential to improve the understanding of pathological food choices.

Comments on revised version:

I went carefully through the answers of the authors to my last concerns - they answered all my points. I am grateful that they obtained consistent results with the different analyses.

https://doi.org/10.7554/eLife.105146.4.sa1

Author response:

The following is the authors' response to the previous reviews

> ***eLife Assessment***
>
> *This study makes a valuable contribution to understanding how negative affect shapes food-choice decision making in bulimia nervosa by leveraging a mechanistic drift diffusion model to quantify the weighting of tastiness and healthiness attributes. The evidence is solid, supported by a randomized crossover design and generally appropriate statistical analyses. However, the interpretability of the findings is limited by ambiguities in the affect manipulation, particularly regarding whether neutral and negative inductions yielded reliably distinct affective states at the time of task performance in the bulimia nervosa group. Consequently, session-related differences in model parameters cannot be unequivocally attributed to negative affect rather than to uncontrolled state or contextual factors, and clearer separation of affective conditions alongside analyses aligned with the paired data structure would strengthen the conclusions.*

We thank the Editor and Reviewers for their careful summary of the study's strengths and for their constructive feedback.

The eLife Assessment identified two specific limitations that qualified the strength of evidence:

(1) ambiguity regarding whether the two affect inductions yielded reliably distinct affective states in the BN group at the time of task performance, and (2) analyses that were not fully aligned with the paired data structure. We have directly addressed both concerns in this

revision. We provide explicit statistical evidence confirming that neutral and negative inductions yielded distinct affective states in the bulimia nervosa group; and we have re-analyzed all DDM parameters using updated mixed-effects regressions with an unstructured covariance matrix that appropriately accounts for the paired data structure. For completeness, we have also added the requested difference-in-difference analysis. Both approaches yielded conclusions consistent with those originally reported.

In light of these revisions, we would be grateful if the Editorial Team would consider whether the strength of evidence rating might be updated from "solid" to "convincing." All changes in the revised manuscript are marked in blue.

> ***Public Reviews:***
>
> ***Reviewer #1 (Public review):***
>
> *Summary:*
>
> *Using a computational modeling approach based on the Drift and Diffusion Model (DDM) introduced by Ratcliff and McKoon in 2008, the article by Shevlin and colleagues investigates whether there are differences between neutral and negative emotional states in:*
>
> *(1) The timings of the integration in food choices of the perceived healthiness and tastiness of food options in individuals with bulimia nervosa (BN) and healthy participants (2) The weighting of the perceived healthiness and tastiness of these options.*
>
> *Strengths:*
>
> *By looking at the mechanistic part of the decision process, the approach has potential to improve the understanding of pathological food choices.*
>
> *Weaknesses:*
>
> *I thank the authors for revising their manuscript.*
>
> *I still notice that the authors did not go through their manuscript to look for wordings refering to a prediction interpretation of their results while I already highlighted the inappropriateness of this wording in my two first rounds of reviews: e.g. there is still "we used zero-inflated negative binomial models to predict the three-month frequency" and I can find other statements like this. The design of their study does not allow such claims.*

We thank the Reviewer for identifying cases where the term “predicted” may mislead readers about the causal nature of our claims. We have made the following edits (changes are italicized):

Methods (lines 516-518): “For these exploratory analyses, we used negative binomials to test the association between parameter estimates and the three-month frequency of retrospectively reported Objective Binge Episodes (OBE) and Subjective Binge Episodes (SBE).”

Figure 5 (lines 881-882): “Affect-induced changes in information onset were associated with more frequent subjective binge episodes.

> *The authors answered my major concern regarding the experimental induction towards a negative or a neutral state before running the food decision task. My concern is: BN patients already seemed to be already in a high negative state before undergoing the neutral induction, while these patients are in a lower negative state before undergoing the negative induction. It is therefore not surprising that patients seem to report a similar level of negative state after the two inductions (according to the figure of the*

> *authors' previous article). Of note is that the additional analysis the authors ran within the BN group only provides a significant result: this result shows that there has been an induction but does not rule out that patients were in the exact same magnitude of negative state to perform the task as the figure in their previously published article suggests it. The major issue is to show that:*
>
> *(1) As compared to the neutral induction, there has been a higher variation in negative state after as compared to before the negative induction.*
>
> *(2) The magnitude of the negative state after the negative induction is higher than the magnitude of the negative state after the neutral induction.*
>
> *The first point shows that the induction worked. The second point shows that the participants are in two distinct states. Without showing the second point, it may be possible that one induction increases the negative state of participants to the same level as the one of the second induction that has not increased anything.*
>
> *Within this context, how is it possible to associate, in patients, a difference in the DDM between the two sessions to a negative state (which is one of the main focus of the article) rather than to another parameter that has not been captured? A similar situation would be in an experiment studying the consequence of stress, a stressfull induction over relaxed participants attending the lab has high chances to raise the level of stress of those participants to the same level as the one that the same participants would experience after a neutral induction when these participants attend the lab with an already high level of stress. In that case, would it be approrpiate to claim that a difference at a task performed after the induction would be related to stress while the participants would be at the same level of stress when performing the task despite the fact that the induction worked ?*
>
> *In the experiment performed by the authors, the additional analysis to perform would be a paired sample t-test (or the appropriate non-parametric test) to check whether the magnitude of negative state of BN patients was different between the negative and neutral conditions after the induction only. If not, associating the difference at the DDM with negative states in BN is highly misleading.*

We thank the Reviewer for pressing on this point, and we apologize that our previous response did not make this sufficiently explicit. We agree with the Reviewer that two things must be demonstrated: (1) that the negative induction produced a greater change in negative affect than the neutral induction, and (2) that the magnitude of post-induction negative affect was higher following the negative induction than the neutral induction. We had included the results of analyses addressing both points in the Supplementary Materials of our previous submission, but we appreciate that we had not made this clear in our response.

Regarding point (1), the mixed-effects model in Supplementary Table S1 yielded a significant Affect Condition × Timing interaction ($\beta = 20.43$, $SE = 6.35$, $t = 3.22$, $p = 0.002$), confirming that negative affect increased significantly more from pre- to post-induction in the negative condition than in the neutral condition. This is further supported by within-BN-group analyses in the Supplementary Materials: the negative affect induction produced a large, significant increase in negative affect (mean difference = 20.36, $SE = 4.21$, $t = 4.84$, $p < 0.0001$, Cohen's $d = 0.97$), whereas the neutral induction was not associated with a significant change in negative affect (mean difference = 7.16, $SE = 4.21$, $t = 1.70$, $p = 0.327$, Cohen's $d = 0.34$).

Regarding point (2), we directly compared post-induction negative affect between conditions within the BN group, as requested by the Reviewer. The magnitude of negative affect was significantly higher following the negative mood induction than after the neutral mood induction (mean difference = 17.40, $SE = 4.21$, $t = 4.13$, $p = 0.0003$, Cohen's $d = 0.83$). This large effect size confirms that participants with BN were in meaningfully distinct affective states when performing the food decision task under the two conditions.

Together, these analyses establish (1) that the induction worked as intended, and (2) that the two post-induction states were both statistically and practically distinct. We have added explicit language to the manuscript to make both of these points clear (lines: 181-185):

Critically, post-induction negative affect within the BN group was significantly higher following the negative affect induction than after the neutral affect induction (mean difference = 17.40, SE = 4.21, t = 4.13, p < 0.001, Cohen's d = 0.83; see Supplementary Materials for full details), confirming that BN participants completed the food decision task under meaningfully distinct affective states across the two sessions.

> *I read carefully the authors' answer related to mixed models: they claim that mixed models take into account correlations within their repeated data. The specification of the structure of the covariance matrix allows to control only partly for that. I notice that the authors did not specify the structure of that matrix: the article they refer to justify the appropriateness of their analyses is not adapted. The specification of the structure of the covariance matrix needs to address, in a mixed model, the difference in handling 4 repeated data per participants that cannot be paired as compared to 4 repeated data that can be paired (two per session with one before and one after the neutral or negative priming sessions, if I count right). Of note is that a covariance structure that is left free of constraint for the fit of the model does not capture appropriately the pairing of the data: it has all chances to capture the covariance in a different way. And a covariance structure that has constraints has more chances to lead to a model that cannot be estimated because of an absence of convergence of the algorithms.*
>
> *By the way, a single two-sample t-test (or a Mann-Whitney test if appropriate), and not a set of multiple paired-sample t-test as the authors suggest, would answer the goal of the authors to test for what they call the three-way interaction in their comment. This test would be performed between the two groups of participants (BN/controls) with the computation for each participant separately: (assessment after neutral induction-assessment before neutral induction)-(assessment after negative induction-assessment before negative induction). This analysis answers points 1, 2 and 4 they raise together with my point of controlling for the paired data. I would have agreed with their choice of a mixed model if they had an unbalanced dataset within each participant.*

We thank the Reviewer for this clarification, and we apologize that our previous response did not adequately distinguish between two different sets of analyses: (1) analyses of DDM parameter estimates, which involved four observations per participant (2 affect conditions × 2 food types); (2) trial-level analyses of choice and response time behavior, where each participant contributed many trials per condition and the dataset is genuinely unbalanced across participants due to trial exclusions – precisely the situation where mixed-effects models with participant-level random slopes are appropriate. The concern about covariance structure applies specifically to the DDM parameter analyses, but does not apply to our trial-level analyses.

We also want to clarify a point about the task design that may have caused confusion. The Food Choice Task was administered only once per session, after the mood induction (i.e., once after negative mood induction, and once after neutral mood induction). As detailed in Figure 1, the task was not completed pre-induction. The four observations per participant in the DDM parameter analyses therefore reflect 2 affect conditions × 2 food types assessed within each condition, not a pre/post structure. This does not change how we address the concern about covariance structure, as there is still a nested feature of interest (food type within condition), but we wanted to correct this misunderstanding explicitly.

For the DDM parameter analyses, we agree with the Reviewer that the original random effects structure did not adequately account for the paired nature of the four within-person observations.

We have addressed this in two ways.

First, we re-estimated the mixed model specifying an unstructured covariance matrix using the nlme package, which places no constraints on the correlation pattern among the four withinperson observations. We acknowledge the Reviewer's point that an unconstrained covariance matrix is not guaranteed to recover the within-session pairing structure. We explored whether a more constrained specification would be preferable. Specifically, we tested a nested random effect of affect condition within subject, which would directly encode the pairing of Low-Fat and High-Fat observations within each session. However, this model failed to converge. This is not a numerical issue but a fundamental identification problem: with only two observations per session per subject, the session-level and residual variance components cannot be separately estimated. We therefore selected the unstructured model as a more conservative option. Importantly, even if the unstructured model does not explicitly encode the pairing, it is a more general mathematical formula which would not impose incorrect constraints on the correlation structure.

Consistent with our original findings, the mixed model with an unstructured covariance matrix yielded a significant three-way interaction (Group × Condition × Food Type: $\beta = 0.28$, $SE = 0.12$, $t = 2.36$, $p = 0.020$). All simple effects analyses have been updated to reflect the models with this covariance structure, and these are reported in the updated Supplementary Tables.

Second, following the Reviewer's suggestion (adapted to the actual design structure, in which the Food Choice Task was administered once per session after the mood induction rather than before and after), we computed a difference-in-difference score for each participant's relative attribute onset parameter (τ_s) following the affect inductions: (negative condition, high-fat – negative condition, low-fat) – (neutral condition, high-fat – neutral condition, low-fat). This score directly encodes the paired structure by construction, bypassing the covariance specification problem entirely. Consistent with the Reviewer's recommendation to use a non-parametric test where appropriate, we used a Wilcoxon rank-sum test (equivalent to Mann-Whitney U) to compare these difference scores between groups. The results confirmed that BN participants showed significantly larger food-type-specific changes in τs following negative affect induction relative to HC ($W = 156$, $p = 0.018$). We then applied this approach to all other DDM parameters (i.e., ω_{taste}, ω_{health}, α, τ_{ND}, and z), and report these results alongside updated mixed-effects model results in the Supplementary Materials. The conclusions drawn from the difference-in-difference analyses were consistent with those from the mixed-effects models across all parameters.

Both approaches converge on the same conclusion and we report both sets of complementary results in the manuscript: the updated mixed-effects models address the full factorial design in a single framework, while the added difference-in-difference analyses explicitly resolve the covariance specification problem by encoding the paired structure directly into each participant’s score, as the Reviewer recommended.

> ***Reviewer #2 (Public review):***
>
> *Summary:*
>
> *Binge eating is often preceded by heightened negative affect, but the specific processes underlying this link are not well-understood. The purpose of this manuscript was to examine whether affect state (neutral or negative mood) impacts food choice decision-making processes that may increase likelihood of binge eating in individuals with*

bulimia nervosa (BN). The researchers used a randomized crossover design in women with BN (n=25) and controls (n=21), in which participants underwent a negative or neutral mood induction prior to completing a food-choice task. The researchers found that despite no differences in food choices in the negative and neutral conditions, women with BN demonstrated a stronger bias toward considering the 'tastiness' before the 'healthiness' of the food after the negative mood induction.

Strengths:

The topic is important and clinically relevant and methods are sound. The use of computational modeling to understand nuances in decision-making processes and how that might relate to eating disorder symptom severity is a strength of the study.

Weaknesses:

Sample size was relatively small, and participants were all women with BN, which limits generalizability of findings to the larger population of individuals who engage in binge eating. It is likely that the negative affect manipulation was weak and may not have been potent enough to change behavior. These limitations are adequately noted in the discussion.

We thank the reviewer for their thorough description of the strengths and weaknesses of this study.

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