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Cookie or clementine? Psychophysiological stress reactivity and recovery after eating healthy and unhealthy comfort foods

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Abstract

Many people eat unhealthy foods that are high in calories, fat, or sugar when feeling stressed, yet little is known about whether this *unhealthy comfort eating* actually comforts. Additionally, prior research has not tested whether *healthy comfort eating* of fruits and vegetables might also alleviate stress, or whether comfort eating during the stress anticipation phase versus immediately after a stressful event is more beneficial for stress relief. The present experiment tested whether unhealthy and healthy comfort eating reduce acute psychophysiological responses to a socially evaluative stressor. Participants ($N = 150$ healthy women) underwent the Trier Social Stress Test in the lab and were randomly assigned to one of five conditions according to a 2 (food type: unhealthy vs. healthy) \times 2 (eating timing: during stress anticipation vs. after the stressor) + 1 (no food control) between-subjects design. Stress outcomes included mood, cognitive appraisals, rumination, salivary cortisol, heart rate variability, and pre-ejection period. Unhealthy and healthy comfort eating did not dampen reactivity or enhance recovery of psychophysiological stress compared to control, and no differences in reactivity or recovery were found by comfort food type. Findings suggest that by replacing unhealthy comfort foods with fruits and vegetables, women will not be

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Author Contributions

Laura E. Finch: conceptualized and designed the experiment and methodology, acquired funding for the study, supervised data collection and project administration, curated data, wrote the original manuscript draft, created data visualizations, reviewed and edited the manuscript, and approved of the final manuscript.

Jenna R. Cummings: advised on and validated data analysis, reviewed and edited the manuscript, and approved of the final manuscript.

A. Janet Tomiyama: conceptualized and designed the experiment and methodology, acquired funding for the study, provided resources, reviewed and edited the manuscript, and approved of the final manuscript.

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sacrificing any stress-reducing benefits and can inherently improve the quality of their diet while avoiding potential drawbacks of unhealthy comfort eating (e.g., links with abdominal obesity).

Keywords

Stress; Eating; Comfort eating; Cortisol; Emotion; Mood

1. Introduction

Stress is ubiquitous in the United States and many people respond to stress by increasing their food intake (Rutters, Nieuwenhuizen, Lemmens, Born, & Westerterp-Plantenga, 2009), especially intake of foods high in calories, fat, or sugar. This behavior—referred to here as *unhealthy comfort eating*—is common, with approximately 39% of American adults reporting overeating or eating unhealthy foods because of stress in the past month (American Psychological Association, 2016). However, does unhealthy comfort eating actually comfort? It is crucial to understand which behaviors are effective in reducing stress, as frequent stress-induced activation of physiological allostatic systems or failure to shut off this activity after stress can chronically lead to disease (McEwen, 1998).

Findings from rodent models have demonstrated what Dallman and colleagues (2003) term a *chronic stress response network* model, wherein comfort eating reduces stress-induced activation of the hypothalamic-pituitary-adrenal (HPA) axis. For example, intake of palatable substances high in fat or sugar dampens stress-induced HPA responses of adrenocorticotrophic hormone (Foster et al., 2009; Pecoraro et al., 2004), hypothalamic corticotropin-releasing hormone mRNA expression, and corticosterone (Foster et al., 2009; Ulrich-Lai et al., 2007). Despite this preliminary evidence for stress dampening, only a few of studies have experimentally tested the acute physiological effects of comfort eating in humans, finding that dark chocolate intake dampens pro-inflammatory (Kuebler et al., 2016) and endocrine (Wirtz et al., 2014) stress responses. However, these samples did not include women and no study has yet examined effects on the stress response of the autonomic nervous system.

With regard to psychological comfort, prior studies in humans exclusively assessed the capacity of comfort eating to repair film-induced negative mood, including the outcomes of sadness (Macht & Mueller, 2007) and general negative affect (Wagner et al., 2014). Experiments assessing the impact of comfort eating on psychological stress—a construct that is related, yet distinct from mood—are absent from the literature. However, rodent models suggest that palatable food consumption inhibits stress-induced behavioral anxiety (Finger et al., 2011); for example, these rodents show greater exploration of open spaces in an elevated maze (Maniam & Morris, 2010; Prasad & Prasad, 1996). Nevertheless, it is unknown whether these findings translate to humans, given that studies in human and non-human animals differ both qualitatively and in their methodology [e.g., type and amount of food consumed, stress/negative emotion manipulation, and outcomes assessed (psychological, physiological, behavioral)].

Accordingly, the first aim of the present study was to fill these gaps as the first known experiment in humans to test for causal impacts of unhealthy comfort eating on psychological and autonomic stress responses. This is also the first experiment in women to examine effects on neuroendocrine stress responses—an investigation that is overdue given that more women than men report engaging in comfort eating (American Psychological Association, 2012; Zellner et al., 2006). In light of the aforementioned evidence for neuroendocrine and behavioral stress dampening in rodents, we hypothesized that unhealthy comfort eating would similarly buffer acute psychophysiological stress in the present, human sample, compared to eating no food at all.

However, even if unhealthy comfort eating does indeed reduce stress, it may not necessarily be a behavior that should be promoted. Comfort foods tend to be high in calories, refined sugars, and fat, and poor diet is a leading cause of morbidity and mortality (U.S. Burden of Disease Collaborators et al., 2018). Comfort eating has also been linked with greater abdominal obesity in middle-aged and older adults (Cummings et al., 2017; Tomiyama et al., 2011). Despite these potential health risks, eradicating comfort eating altogether is likely not a viable strategy, because eating is hedonically rewarding (Adam & Epel, 2007) and food cues are omnipresent in modern society (Wadden et al., 2002). Therefore, we examined a novel method for harnessing any potential benefits of comfort eating (i.e., stress reduction) without simultaneously increasing physical health risks. More specifically, the second aim of the study was to assess whether *healthy comfort eating*—eating fruits and vegetables in response to stress—might also serve to dampen psychophysiological stress responses. If so, stressed individuals could alleviate stress while also inherently consuming fewer calories, fat, and sugar compared to eating traditional, unhealthy comfort foods.

For this aim, our hypotheses were two-fold. First, we predicted that unhealthy comfort foods would reduce stress to a greater extent than healthy comfort foods, as the physiological stress dampening observed in some rodent models may be related to sugar intake (Pecoraro et al., 2004; Ulrich-Lai et al., 2007). In addition to stimulating opioid release (Adam & Epel, 2007), sugar affects a metabolic-brain-negative feedback pathway, such that sucrose inhibits stress-induced cortisol secretion in humans (Tryon et al., 2015).

However, we also expected that healthy comfort foods would dampen stress compared to eating no food. Although they typically contain less sugar than unhealthy comfort foods, fruits do contain natural sugars (e.g., fructose). Moreover, some vegetables contain levels of sugar comparable to fruits (e.g., 1 serving of carrots and strawberries each contains 6–7g of sugar). In addition, it may be the act of chewing itself that relieves stress, as chewing gum has been shown to acutely reduce anxiety, stress, and salivary cortisol (Scholey et al., 2009).

Finally, in addition to comparing these two different *types* of comfort eating, the present study examined the impact of comfort eating *timing* as the third aim. In the context of a planned, acute stressful event such as a job interview, is comfort eating most effective during the stress anticipation phase as a method for reducing stress reactivity, or after the event to hasten stress recovery? Gross' process model of emotion regulation (2002) posits that strategies used earlier in the stress process are more effective than later strategies; therefore, we expected that comfort eating would have a greater stress-buffering effect when taking

place during stress anticipation, rather than after a stressful event. It is important to understand methods for both reducing stress reactivity and hastening stress recovery, as both anticipatory stress and post-stressor rumination are linked with heightened cardiovascular and endocrinological activity, which impact disease (Brosschot et al., 2006).

2. Method

2.1 Study design

Participants underwent an acute laboratory stressor (the Trier Social Stress Test; TSST; Kirschbaum et al., 2008) and were randomly assigned to one of five experimental conditions according to a 2 (food type: healthy vs. unhealthy) \times 2 (eating timing: during stress anticipation vs. after the TSST) + 1 (no food control) design. The TSST is known to reliably induce psychological stress and cortisol responses (Kudielka et al., 2007), and thus provided an experimental paradigm fit to the experience of comfort eating as well as one where psychophysiological stress responses could be observed.

2.2 Participants

A total of 490 individuals were screened for the study, which yielded 155 eligible undergraduate women who participated for research credit in their psychology courses. Two participants completed the study but were not analyzed due to researcher error ($n = 1$) or the participant skipping ahead in the surveys ($n = 1$). In addition, three participants did not complete the study because they: consumed food during the hour prior to the laboratory visit ($n = 1$), did not wish to consume their assigned food ($n = 1$), or did not wish to be video recorded ($n = 1$). This left a total of 150 participants ($n = 30$ per condition) in the final analysis.

Power analysis was performed using G*Power software (version 3.1.9.3; Faul, Erdfelder, Buchner, & Lang, 2009). The analysis focused on identifying the appropriate sample size to detect a within-between interaction in an omnibus test comparing five experimental conditions across 3 repeated measures of psychophysiological stress (our anticipated lowest-powered repeated measures analysis). We conservatively specified a small interaction effect size $\eta_p^2 = .002$ and entered the software's default moderate correlation ($r = .5$) among repeated measures. The significance level was set at $p = .05$ and the minimum power at .80. This power analysis indicated that a total sample size of 130 was needed, and we conservatively over-recruited to achieve a final sample size of 150. As shown in Supplementary Table 1, given the observed correlations between repeated measures and the actual sample sizes available for each particular analysis, nearly all analyses should be sufficiently powered at a level greater than .80 (with two exceptions: the negative mood reactivity and recovery tests appear to be underpowered at power of .78 and .67, respectively).

2.3 Recruitment and pre-screening procedures

The University Institutional Review Board approved all study activities. Participants were recruited via the University's psychology subject pool. In online pre-screening, individuals

provided information about their demographics and health status, which determined eligibility.

Inclusion criteria included: female, aged 18 or older, and fluent in English. Only women were recruited, as a greater proportion of women than men report engaging in comfort eating (American Psychological Association, 2012). Exclusion criteria were chosen based on incompatibility with the study methods or with cortisol measurement and included: metabolic or endocrine disease, post-menopausal status, chronic asthma, history of substance abuse or eating disorder, current strict dieting, current diagnosed psychiatric condition or current major illness or injury. Women exhibiting an elevated level of depressive symptoms were also excluded (score > 23 on the Center for Epidemiological Studies Depression Scale (CES-D); Radloff, 1977), as comfort eating may not dampen psychological stress in this sub-population of young women (Finch & Tomiyama, 2015). This conservatively high CES-D cutoff score has been used in previous studies in samples of young adult women (Finch & Tomiyama, 2015; Franko et al., 2004). The most common reasons for ineligibility were elevated depressive symptoms (25%), current diagnosed psychiatric condition (9%), or history of an eating disorder (8%).

In pre-screening, individuals completed a Food Opinions Survey adapted from Wagner and colleagues (2014). There, individuals ranked their top three healthy and unhealthy foods in response to the prompt, “What foods would make you feel better if you were in a bad mood?” Participants were presented a list of 10 healthy foods—defined as fruits and vegetables—and 10 unhealthy foods (i.e., processed foods high in sugar and/or fat) to choose from. These questions were embedded among distractor questions such as, “What foods would you want if you were on-the-go?” The food lists were based on which foods were rated most highly in a pilot survey we conducted in 73 women, wherein participants rated 112 healthy and unhealthy foods and beverages on “To what extent would this food/beverage make you feel better if you were in a bad mood?” (1 = *not at all* to 7 = *very much*). Compared to the 10 selected healthy foods, the 10 unhealthy foods contained on average about 211% more calories (70 vs. 218 kcal), 1,200% more fat (0.9 vs. 11.7 g), and 62% more sugar (11.0 vs. 17.8 g) per serving, respectively.

2.4 Lab day procedure

Eligible participants were invited to complete a 2.5-hour lab visit and provided informed consent. Lab visits were conducted between 1330h and 1700h to control for daily diurnal cortisol rhythm (Posener et al., 1996). The primary lab activities are outlined in Figure 1. To ensure reliable cortisol measurement, participants were instructed not to: (1) consume caffeine in the 3 hours prior, (2) smoke or engage in any moderate to vigorous exercise in the 2 hours prior, or (3) consume food in the hour prior. The experimenter verbally confirmed with participants that they had adhered to these instructions.

Throughout the lab visit, participants were attached to wireless physiological equipment (BIOPAC Systems, Inc., Goleta, California, U.S.A.) using electrocardiography and impedance cardiography with non-invasive sensors to assess measures of autonomic activation. Prior to baseline assessments, participants were given 3 minutes to sit and relax to

become accustomed to the sensation of having physiological equipment on the body (Mendes, 2009). Then, baseline autonomic, mood, and cortisol measures were collected.

Next, three members of research staff (blinded to condition) in white lab coats informed participants that they would soon be delivering a 5-minute speech to be evaluated by a trained committee. After these staff left the room, participants randomly assigned to eat during anticipatory stress were provided with their top-rated unhealthy or healthy comfort food to consume. Alternatively, participants in the other conditions were not given food at this time, and were instead told to simply sit and wait while the experimenter prepared the next part of the study.

Participants in eating conditions were provided with their top-ranked foods to enhance ecological validity. They were always provided with 2 servings of the food (each in a separate dish) and asked to consume at least one dish of the food; however, participants who ate some food but not at least one full serving ($n = 10$) were not excluded from hypothesis testing. The fruits and vegetables in the healthy food conditions were served raw and foods were prepared in bite-sized pieces when appropriate (e.g., apples were sliced, and chocolate bars were broken into pieces).

After 5 minutes of eating or waiting, all participants completed a second mood assessment and a measure of anticipatory cognitive appraisals. Then they were given 3 minutes to prepare for their speech. Thereafter, two TSST staff members administered the 5-minute speech task, followed by a 5-minute mental arithmetic task. Both tasks were video recorded to increase TSST salience.

Immediately after the TSST, participants completed a third mood assessment. Then, participants randomly assigned to eat after the stressor were provided with their top-rated unhealthy or healthy comfort food to consume. The other conditions were not given food at this time, and were instead told to simply sit and wait while the experimenter prepared the next part of the study.

After 5 minutes of eating or waiting, all participants completed a fourth mood assessment. Then, participants were told to simply sit and rest for 3 minutes in order for resting physiology signals to be taken. However, the true purpose of this wait time was to provide participants with a chance to ruminate before completing the Modified Thoughts Questionnaire—a rumination measure.

Next, cortisol samples were collected at 15- and 25- minutes post-TSST. Then, for the remainder of the recovery period, all participants viewed a film with neutral emotional valence describing how products such as hearing aids are manufactured (Hoss & Allard, 2005). At 60 minutes post-TSST, final mood and cortisol assessments were administered. Weight and height were then measured, followed by debriefing and compensation.

2.5 Pre-questionnaire measures

2.5.1 Demographic information.—The following self-reported demographic variables were included: age, race/ethnicity, family income while growing up [assessed via income

ranges (see Table 1) that were then coded from 1–13 with higher values denoting higher income], current oral contraceptive use, and subjective social status as assessed via the MacArthur Scale of Subjective Social Status-Youth Version (Goodman et al., 2001).

2.5.2 Depressive symptoms—Depressive symptoms were assessed using the CES-D ($\alpha = .77$; Radloff, 1977; 0 = *rarely or none of the time* and 3 = *most or all of the time*). Sample items include: “I felt that people disliked me,” and “My sleep was restless.”

2.5.3 Food opinions.—Section 2.3 contains the prompts for the Food Opinions Survey (Wagner et al., 2014). For each food that they ranked, participants were also asked to give details about which flavor, brand, and/or type of the food that they preferred (e.g., Häagen-Dazs coffee ice cream). According to their randomized condition, on lab day participants were served the unhealthy or healthy food that they rated the most highly as a mood-improver (or their second-highest rated food if the first was unavailable), and the food provided was aligned as closely as possible with the idiosyncratic flavor, brand, and food type preferences indicated by each individual participant.

2.5.4 Trait emotional eating—To characterize the sample, trait-like emotional eating was measured by the 13-item Emotional Eating Subscale from the Dutch Eating Behavior Questionnaire ($\alpha = .93$; van Strien, Fritjers, Bergers, & Defares, 1986). This subscale asks participants to rate how often they have a desire to eat when experiencing different emotions (e.g., lonely, worried) from 1 = *never* to 5 = *very often*. All items were averaged to create mean scores, with higher scores indicating higher trait emotional eating.

2.6 Lab day questionnaire measures

2.6.1 Anticipatory cognitive appraisals—The Primary Appraisal Secondary Appraisal scale (Gaab et al., 2005) assessed anticipatory cognitive appraisals before the TSST tasks began. This validated, 16-item scale maps on with Transactional Stress Theory (Lazarus & Folkman, 1984). The scale assessed the cognitive appraisal constructs of threat (e.g., “This situation scares me”; $\alpha = .85$), challenge, (e.g., “This task challenges me”; $\alpha = .70$), self-efficacy (e.g., “I can think of a lot of solutions for solving this task”; $\alpha = .83$), and control expectancy (e.g., “It mainly depends on me whether the experts judge me positively”; $\alpha = .65$). These variables were computed following the example of Gaab and colleagues (2005). Threat, self-efficacy and control expectancy were negatively skewed; thus, these variables were squared to correct for normality.

2.6.2 Rumination—Post-TSST rumination was measured using The Negative Thoughts Subscale from the Modified Thoughts Questionnaire (Zoccola et al., 2008), which consisted of 14 items assessing how much participants had negative thoughts in the time since the speech task had ended. Sample items: “How often did you think about how bad your speech was?” on a 5-point scale (*never* to *very often*). Item responses were summed to create a total Negative Thoughts Subscale score ($\alpha = .94$), such that higher scores indicate greater negative thought rumination. Negative thought rumination was positively skewed; therefore, this variable was square root transformed to improve the normality of the distribution.

2.6.3 Mood state—Positive and negative mood state were assessed at five time points (see Figure 1) using the Positive and Negative Affect Schedule (PANAS; Watson et al., 1988) on a scale from 1 (*very slightly or not at all*) to 5 (*extremely*). Like previous comfort eating research (Wagner et al., 2014), we added the items “sad” and “happy” to the original list of 20 emotions and included them in their relevant subscales. Example item: “Indicate to what extent you currently feel this way,” for the item “excited,” on a scale from 1 (*very slightly or not at all*) to 5 (*extremely*). Item responses were summed to create a total score for each subscale. Across the five different time points, both positive and negative mood showed acceptable reliability (α ranges of .88-.91 and .74-.89, respectively).

2.7 Physiological measures

2.7.1 Heart rate variability and pre-ejection period—Electrocardiography and impedance cardiography captured ANS activation, including outcomes of heart rate variability (HRV) and pre-ejection period (PEP; BIOPAC Systems, Inc., Goleta, California, U.S.A.). For electrocardiography, three spot electrodes were placed, with one on participants’ lowest left rib, lowest right rib, and right collarbone. For impedance cardiography, four bioimpedance strip electrodes were placed, with two on the back of the neck and two on the lower back. All signals were recorded using AcqKnowledge 4.2 software offline (BIOPAC Systems, Inc., Goleta, California, U.S.A.), and were analyzed using MindWare software (MindWare Technologies, Ltd., Gahanna, Ohio, U.S.A.).

For HRV and PEP, seven time periods of interest were assessed: baseline (5 minutes), delivery of the TSST instructions (3 minutes), TSST speech preparation occurring immediately after some participants ate (3 minutes), TSST speech task (5 minutes), TSST math task (5 minutes), immediately post-TSST (5 minutes; recovery 1), and immediately after other participants ate (5 minutes; recovery 2). For each time period, each ANS outcome was calculated in MindWare in 1-minute epochs. Following the example of previous research, (Mendes et al., 2007), to examine the “fast response” of the ANS, the last minute of baseline and the first minute of each subsequent time period were analyzed.

HRV was assessed using time-domain estimation to compute RMSSD (the root mean square of the difference of successive R-R intervals), which is recorded in milliseconds (Mendes, 2009). Higher RMSSD indicates greater parasympathetic activation (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). PEP was also assessed because it is considered one of the purest measures of SNS activity (Mendes, 2009) and is defined as the time elapsed from the contraction of the left ventricle (i.e., the Q point on the ECG wave) to the opening of the aortic valve (i.e., the B point on the first derivative of the impedance waveform). PEP is recorded in milliseconds, and smaller values indicate greater sympathetic activity. To correct for normality, RMSSD values were log transformed and PEP values were squared.

2.7.2 Salivary cortisol—Salivary cortisol samples were collected at baseline and at 15, 25, and 60 minutes post-TSST. Immediately prior to the second cortisol sample, all participants were asked to swish their mouths with water 2–3 times, as participants in some

conditions had consumed food within 10–15 minutes of the sampling procedure. Saliva samples were frozen at -20°C .

Salivary cortisol levels were assayed at Technical University of Dresden, Germany using chemiluminescence immunoassay and single determination assays. Raw cortisol values were first log transformed to normalize the cortisol distribution. Then, following the recommendation of Adam and Kumari (2009), log transformed values found to be more than 3 standard deviations above or below the mean for each time point were winsorized. This applied to 1 of the 600 samples.

2.7.3 Anthropometry—Weight was measured using a Tanita Professional Body Composition Monitor SC-331S. Height was measured using a stadiometer and recorded to the nearest 1/8 inch. BMI was calculated using the standard formula of $\text{weight}(\text{kg})/\text{height}^2(\text{cm})$.

2.8 Statistical analysis plan

In addition to the tests reported here, the data and code for planned tests of moderation and mediation will be available online via the Open Science Framework at: <https://osf.io/j95tf/wiki/home/>.

Between-subjects analysis of variance (ANOVA) tests were used to examine main effects of condition on outcomes collected at a single time point. Mixed repeated measures ANOVA models were used to test hypotheses related to repeated measures.

To inform the selection of model covariates, bivariate correlations were computed between all outcomes and the following descriptive variables: age, race/ethnicity, income, subjective social status, and BMI. For cortisol, we also considered depressive symptoms (Stetler & Miller, 2011) and oral contraceptives (Kirschbaum et al., 1999) as potential covariates, given that they have been associated with HPA axis functioning. Any variables significantly related to an outcome were considered as covariates for that outcome (see Supplementary Table 2). Throughout, the pattern of results did not change regardless of whether covariates were included; therefore, the more parsimonious models are presented here.

For repeated measures outcomes (i.e., mood, HRV, PEP, and cortisol), we examined reactivity and recovery trajectories separately. Mood reactivity included measurements from baseline to immediately post-TSST, and mood recovery trajectories were defined as measurements from immediately post-TSST to the end of the study. Autonomic reactivity trajectories were defined as measurements from the TSST instruction period to immediately post-TSST, and autonomic recovery trajectories were defined as measurements from immediately post-TSST to immediately after the post-TSST comfort eating. Given that mean cortisol levels peaked at 15 minutes post-TSST, the cortisol reactivity trajectory was defined as all samples up to and including 15 minutes post-TSST and the cortisol recovery trajectory was defined as all samples including and after 15 minutes post-TSST.

Each repeated measures ANOVA model included effects of condition, time, and the condition by time interaction. Interaction terms were examined to test the study hypotheses.

For reactivity analyses, hypothesis testing began with 3-condition omnibus tests assessing any differences in reactivity trajectories between those who had unhealthy food during anticipatory stress, those who had healthy food during anticipatory stress, and those who had no food during anticipatory stress. To maximize power in reactivity analyses, this third “no food” group collapsed together the unhealthy-after, healthy-after, and no food groups, given that the experimenter treated them identically up to this point in the study. If significant omnibus tests emerged for reactivity, these tests were followed with pairwise tests comparing the groups that ate during stress anticipation versus those that did not. For recovery analyses, hypothesis testing began with 5-condition omnibus tests assessing any group differences in recovery trajectories. If significant omnibus tests emerged for recovery, these tests were to be followed with planned pairwise tests comparing: (1) the control group versus each of the four other groups individually; (2) those who ate healthy versus unhealthy food before the TSST; and (3) those who ate healthy versus unhealthy food after the TSST.

All analyses were conducted using SPSS software (version 24.0, IBM, USA). Statistical significance for all analyses was set at $p < .05$. Study hypotheses were pre-registered online via the Open Science Framework at: <https://osf.io/j95tf/wiki/home/>.¹

3. Results

3.1 Descriptive information

Descriptive statistics for participants' demographic characteristics and other variables of interest are presented in Table 1. The most commonly served healthy foods were strawberries ($n = 18$), grapes ($n = 9$), and bananas ($n = 7$); the most commonly served unhealthy foods were ice cream ($n = 29$), chocolate ($n = 9$), and chocolate chip cookies ($n = 7$).

3.2 Manipulation check

No significant group differences in primary outcomes were found at baseline (all $ps > .05$). Results suggest that the TSST successfully induced stress. As shown in Table 2, the psychophysiological outcomes with repeated measures significantly reacted to the stressor. Figures 2, 3, and 4 show that these reactivity trajectories were in the expected direction, such that negative mood, sympathetic activation (i.e., PEP), and cortisol increased, whereas positive mood and parasympathetic activation (i.e., HRV) decreased.

3.3 Psychological outcomes

3.3.1 Anticipatory cognitive appraisals—The omnibus between-subjects ANOVA tests comparing those that ate unhealthy foods during anticipatory stress, those that ate

¹We note the following deviations between the present manuscript and the study pre-registration. Although we had hypotheses regarding moderating and mediating variables, due to space constraints we chose to limit the scope of the manuscript to tests of main effects of comfort eating on stress responses. Nonetheless, we will upload the data and syntax for these additional tests online via the Open Science Framework at: <https://osf.io/j95tf/wiki/home/>. We note that the tests of hypothesized moderation (including testing scores on the Emotional Eating Subscale of the Dutch Eating Behavior Questionnaire as a moderator variable) and mediation yielded largely non-significant results. We limited our reported questionnaire outcomes to measures that have been published previously. Electrodermal activity was collected but not analyzed; the signals were contaminated by simultaneous impedance cardiography. We did not include respiratory rate as a covariate in autonomic analysis, given justification from the literature that it is not necessary (Denver, Reed, & Porges, 2007).

healthy foods during anticipatory stress, and those that did not eat during anticipatory stress showed no group differences in anticipatory cognitive appraisals, including threat, $F(2, 147) = 2.02, p = .14, \eta_p^2 = .027$ challenge, $F(2, 147) = 0.05, p = .95, \eta_p^2 = .001$; self-efficacy, $F(2, 147) = 2.14, p = .12, \eta_p^2 = .028$; and control expectancy, $F(2, 147) = 1.06, p = .35, \eta_p^2 = .014$.

3.3.2 Rumination—The omnibus between-subjects ANOVA test comparing all conditions revealed no differences in post-TSST negative thought rumination, $F(4, 145) = 0.09, p = .99, \eta_p^2 = .002$.

3.3.3 Mood—Figure 2 displays negative and positive mood trajectories by condition. As displayed in Table 2, negative and positive mood reactivity trajectories each did not differ when comparing those that ate unhealthy or healthy foods during anticipatory stress to those that did not eat during anticipatory stress. In addition, mood recovery trajectories each did not differ when comparing those that ate unhealthy or healthy foods after the TSST to those that never ate during the study.

3.4 Physiological outcomes

3.4 Heart rate variability and pre-ejection period—Figure 3 presents raw HRV and PEP trajectories by condition. As shown in Table 2, HRV and PEP reactivity trajectories did not differ when comparing those who ate unhealthy or healthy foods during anticipatory stress to those who did not eat during anticipatory stress. In addition, HRV and PEP recovery trajectories did not differ when comparing those that ate unhealthy or healthy foods after the TSST to those that never ate during the study.

3.4.2 Cortisol—Figure 4 presents raw cortisol trajectories by condition. As shown in Table 2, cortisol reactivity trajectories did not differ between those that ate unhealthy or healthy foods during anticipatory stress to those that did not eat during anticipatory stress. Furthermore, cortisol recovery trajectories did not differ between those that ate unhealthy or healthy foods after the TSST to those that never ate during the study.

4. Discussion

The present study fills several key gaps in the literature as the first experiment in humans to assess the effects of comfort eating on acute psychophysiological stress responses. However, given the potential drawbacks of unhealthy comfort eating, the present study also aimed to assess optimal modifications of this behavior by testing effects of comfort eating type [unhealthy (i.e., processed and high in calories, refined sugars, and fat) or healthy (i.e., fruits and vegetables)] and timing (during stress anticipation or after a stressful event). Results revealed that those who ate unhealthy or healthy food during the stress anticipation phase did not show reduced psychophysiological stress reactivity compared to those who did not eat at this time. Similarly, those who ate unhealthy or healthy food immediately after the stressor did not show hastened psychophysiological stress recovery compared to those who never ate during the study. Finally, consuming unhealthy food did not provide any benefit for

psychophysiological stress reduction compared to eating healthy food, regardless of when the comfort eating took place.

These findings are consistent with prior findings that comfort eating after a negative event does not provide any psychological mood benefits (Wagner et al., 2014). Although another prior experiment found that palatable chocolate consumption improved psychological mood after a negative event, these effects dissipated after three minutes (Macht & Mueller, 2007). Notably, these studies examined comfort eating in the context of laboratory-induced sadness (Macht & Mueller, 2007; Wagner et al., 2014), whereas the present study assessed responses to an acute laboratory stressor. However, taken together with the present results, this growing literature suggests that benefits of comfort eating for mood may be non-existent (or transient if observed at all). Thus, although many people eat foods high in sugar, fat and calories when stressed, these individuals may be giving unhealthy comfort foods “credit” for mood effects that would occur with the passage of time even without eating (Wagner et al., 2014).

Although the present findings corroborate previous research on the psychological mood effects of comfort eating, prior studies testing the physiological effects of comfort eating in rodents (Dallman et al., 2003; Foster et al., 2009; Pecoraro et al., 2004; Ulrich-Lai et al., 2007) and men (Wirtz et al., 2014) have shown that palatable food intake provides neuroendocrine stress dampening, and the present study did not show this. One explanation may be that neuroendocrine and autonomic responses to acute stressors like the TSST are often more pronounced among men than women in young adulthood (Kajantie & Phillips, 2006). It is therefore possible that detecting stress-reducing effects of comfort eating on neuroendocrine and autonomic responses may be more methodologically/statistically difficult in women than in men. Thus, future studies may benefit from using a within-subjects design with women engaging in all types and timings of comfort eating. Another potential explanation for the lack psychophysiological stress dampening may be the chronicity of the stress and/or the comfort eating. Several studies in rodents have found that repeatedly ingesting palatable substances decreases both acute (Foster et al., 2009) and chronic stress responses (Pecoraro et al., 2004; Ulrich-Lai et al., 2007). In addition, there is some evidence that self-reported engagement in “trait-like” emotional eating buffers the impact of adverse life events on chronic perceived stress (Finch & Tomiyama, 2015). However, studies in men have also demonstrated that a single instance of comfort eating reduces physiological responses to acute stress (Kuebler et al., 2016; Wirtz et al., 2014), suggesting that repeated comfort eating events and a chronic stress context are not required—at least for physiological stress dampening.

Nonetheless, when comfort eating attenuates chronic stress responses, this may be partially explained by abdominal fat. Dallman and colleagues (2003) posit in their *chronic stress response network* model that chronic comfort eating yields abdominal obesity, and the presence of abdominal fat may suppress stress-induced HPA axis responses via negative feedback. Although this complete model has not been experimentally tested in humans, some research has evidenced relationships consistent with this pathway. For example, one study found that middle-aged women with high versus low chronic stress also showed higher levels of emotional eating, greater sagittal diameter, and a blunted cortisol response to acute stress in the laboratory (Tomiyama et al., 2011). In the present study, participants were

young, healthy adults; therefore, further research should examine comfort eating effects in older samples with greater variability in abdominal obesity.

These results should be interpreted in light of study limitations. Although the experimental design provides strong internal validity, the study's ecological validity was limited in some aspects. Perhaps participants would have felt more comfortable consuming comfort food in the privacy of their own home and the laboratory setting may have inhibited comforting effects. Future experimental studies could be conducted in a laboratory setting designed to mimic a home atmosphere. Furthermore, participants were presented with two servings of food and asked to consume at least one dish/serving. A more realistic manipulation might involve ad libitum eating with no lower or upper limit on the amount consumed. In addition, the sample was predominately Asian/Asian American/Pacific Islander with low representation of African American and Hispanic groups. This is an important gap in the literature, given that compared to Whites, Hispanic and African American adults are disproportionately vulnerable to both stress (American Psychological Association, 2016) and obesity (Ogden et al, 2014), and comfort eating appears to be a behavior linking these risk factors (Tomiyama et al., 2011). Thus, future comfort eating research could benefit from including more representation from these minority groups. In addition, future research should further examine other potential biopsychosocial pathways through which observed stress-dampening effects may be functioning (see Tomiyama et al., 2015 for a review).

These limitations notwithstanding, the present study offered several methodological strengths. First, these relationships were examined in a sample of women—the sex/gender most likely to engage in comfort eating—and within the context of a gold-standard laboratory stress paradigm. Second, for most analyses the experiment was well powered to capture a small effect ($\eta_p^2 = .02$) in a complex within-between interaction analysis. However, the analyses for negative mood reactivity and recovery appear to have been underpowered and therefore, the results for those two particular tests should be interpreted with caution. Third, the study advanced the literature by providing a comprehensive, multi-system assessment of stress responses including psychological, autonomic, and neuroendocrine measures. Fourth, given that comfort food preferences vary across individuals, participants were given a food that they had ranked highly in pre-screening to enhance ecological validity.

Importantly, these findings have practical implications. Eating foods high in calories, fat, and sugar can lead to disease and premature death (U.S. Burden of Disease Collaborators et al., 2018). Although the present study findings provide further justification for the eradication of unhealthy comfort eating, comfort eating is widespread (American Psychological Association, 2016), hedonically rewarding (Adam & Epel, 2007), and triggered by cues in the “toxic” environment (Wadden et al., 2002). Accordingly, the present study introduced the concept of healthy comfort eating as an alternative. The equivalent findings between unhealthy and healthy comfort eating leave the door open for stressed women to shift their comfort eating away from unhealthy foods and toward health_S1_Reference41y ones—without any corresponding loss of stress-dampening benefits.

5. Conclusion

Even relatively small, yet regular changes to diet can have a clinically significant health impact, as meta-analytic findings indicate that all-cause mortality risk is decreased by 6% and 5% for each additional daily serving of fruits and vegetables, respectively (Wang et al., 2014). By transforming their comfort eating toward healthy comfort eating, individuals should inherently receive the benefit of improved dietary nutrition and in turn, decrease their risk of morbidity and mortality over time. As suggested by the present findings, women will not be sacrificing any stress-reducing benefits by doing so.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Highlights:

- We tested comfort eating effects on stress reactivity and recovery in women
- Results did not differ comparing unhealthy versus healthy comfort eating
- Neither type of comfort food outperformed the no-food control condition
- Unhealthy comfort eating may not be beneficial for acute stress reduction in women
- Healthy comfort eating may be a viable replacement for unhealthy comfort eating

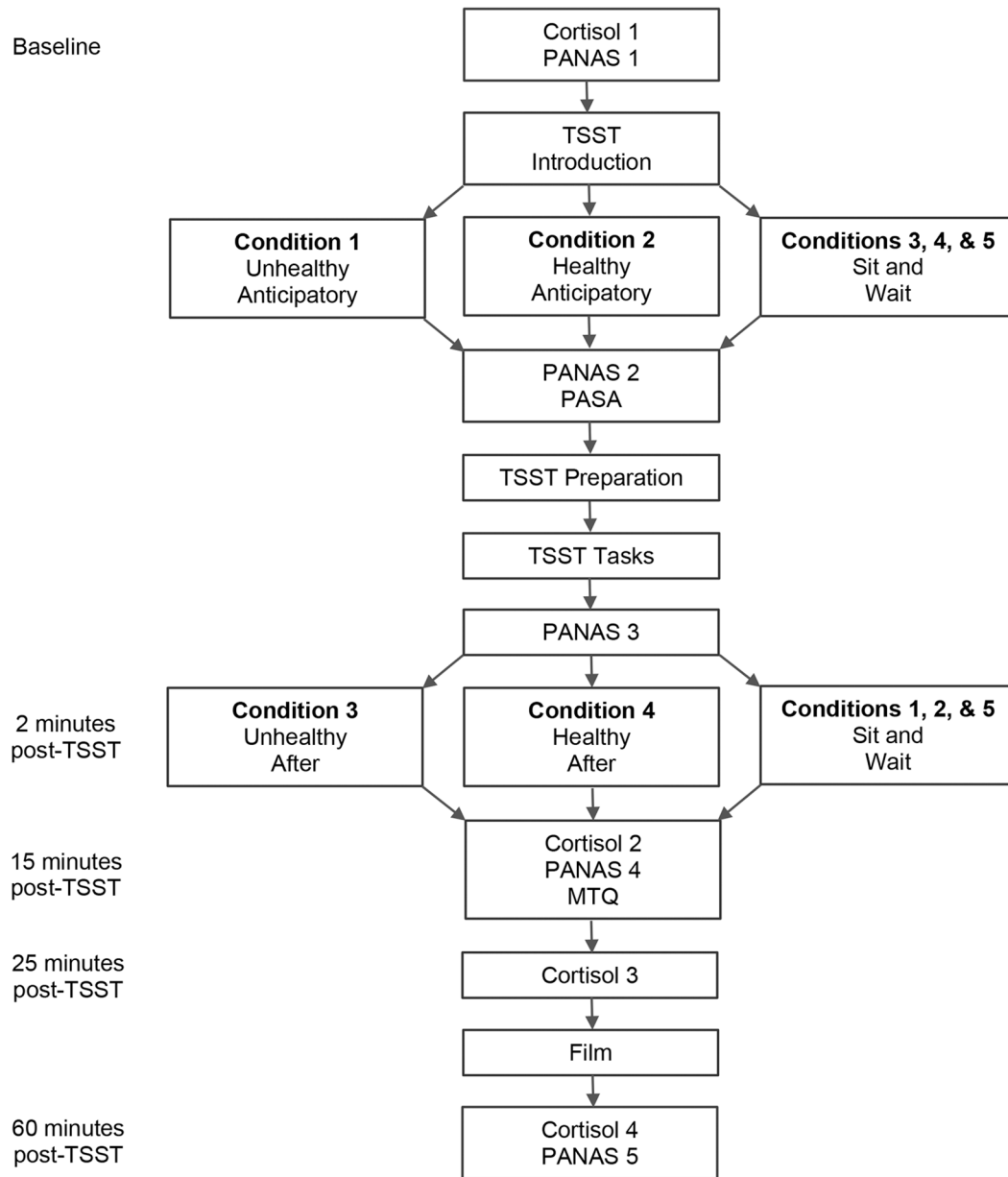


Figure 1. Overview of data collection timeline for survey and neuroendocrine outcomes. Autonomic data were collected continuously throughout the entire laboratory visit. PANAS = Positive and Negative Affect Schedule; PASA = Primary Appraisal Secondary Appraisal Scale; TSST = Trier Social Stress Test; MTQ = Modified Thoughts Questionnaire.

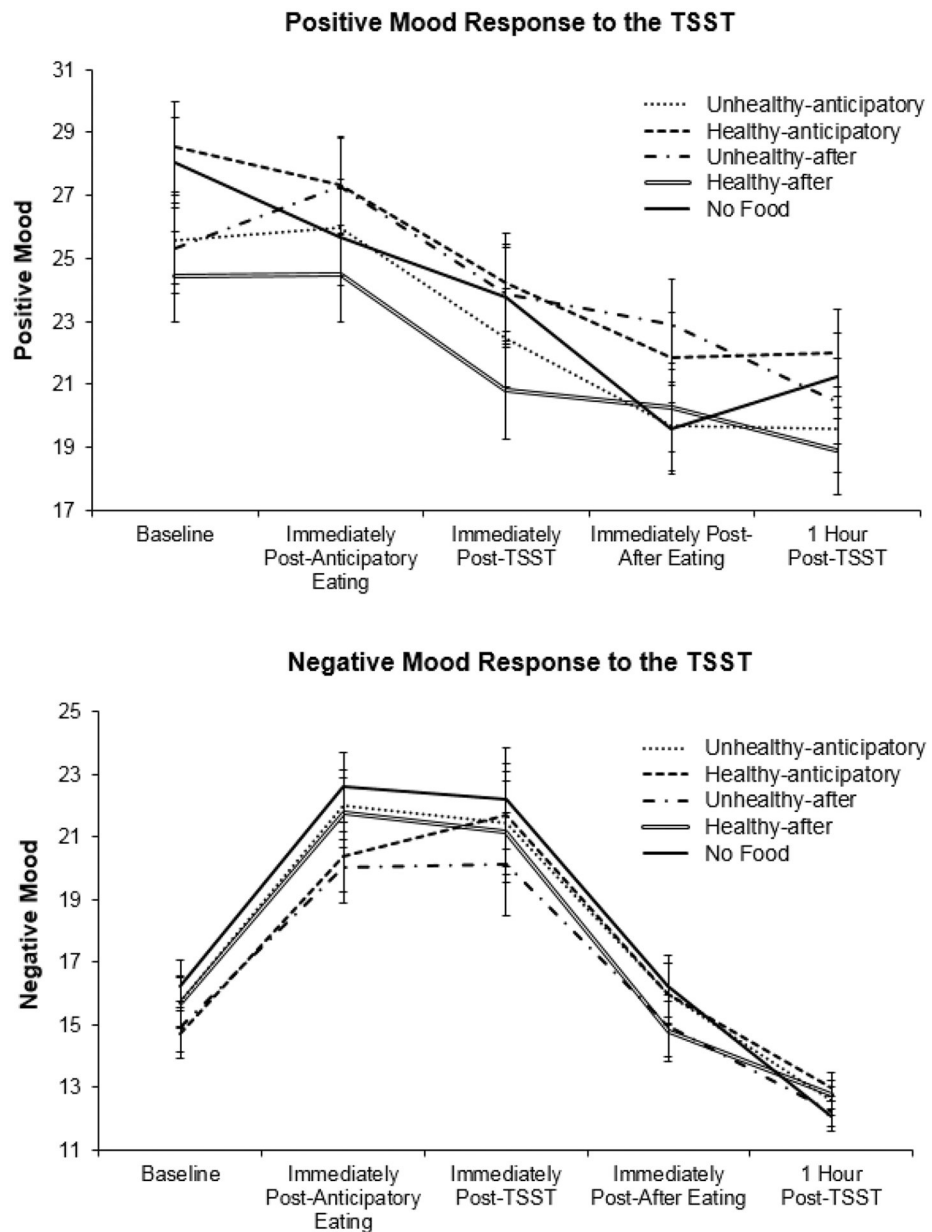


Figure 2. Positive and negative mood response to the Trier Social Stress Test (TSST). Scores were calculated from the negative mood subscale of the Positive and Negative Affect Schedule. Error bars represent standard errors.

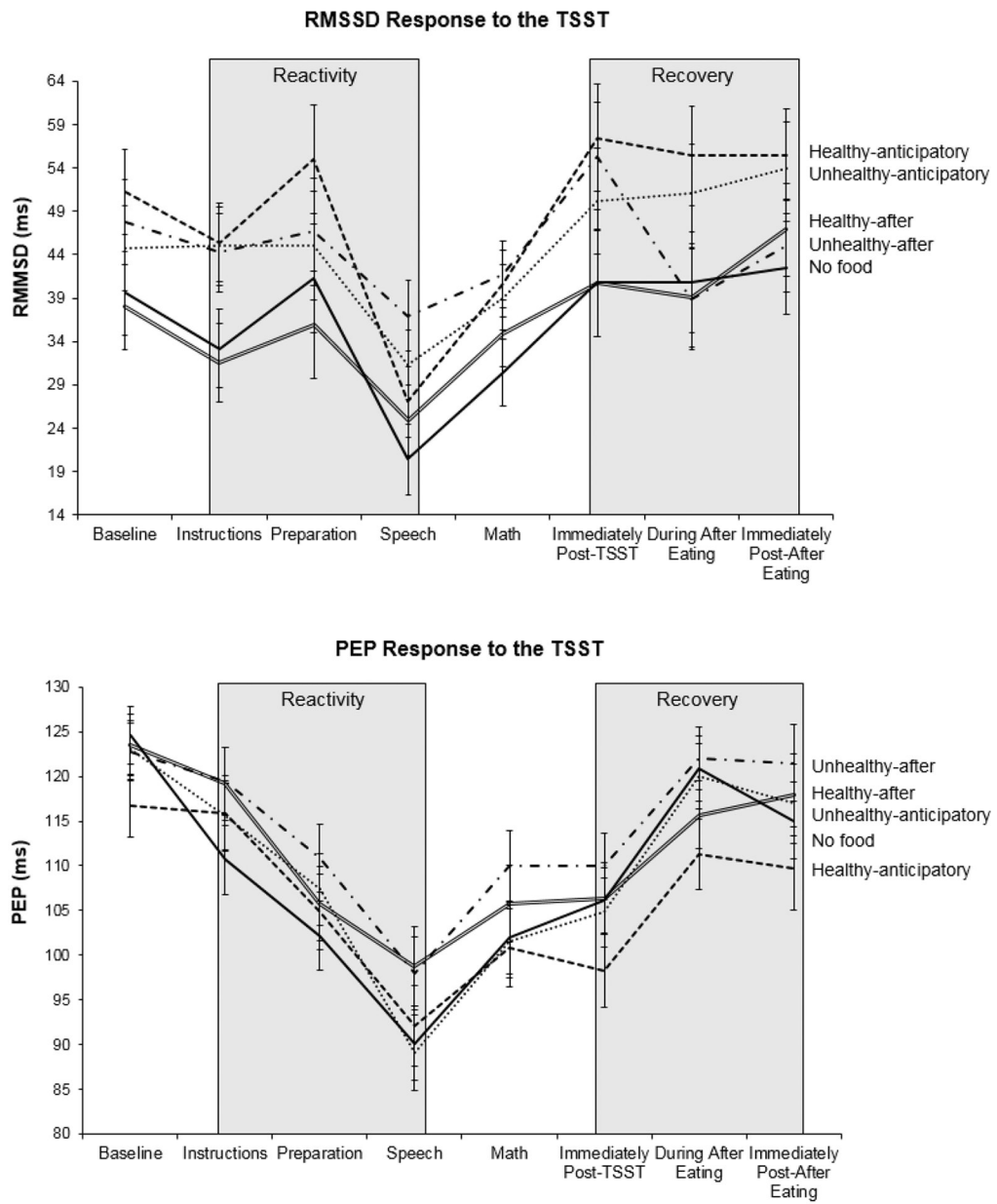


Figure 3. RMSSD and PEP responses to the Trier Social Stress Test (TSST). Raw values are presented here. Error bars represent standard errors.

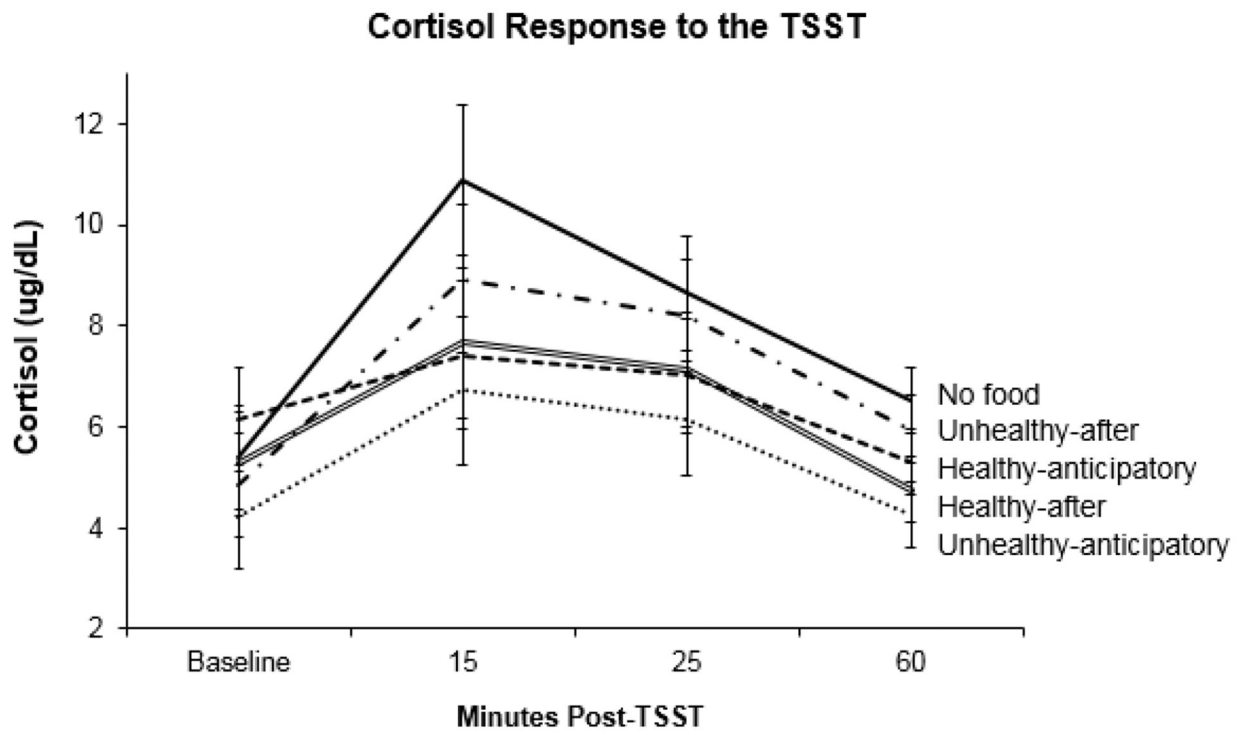


Figure 4. Cortisol response to the Trier Social Stress Test (TSST). Raw values are presented here. Error bars represent standard errors.

Table 1

Sample Demographics

Characteristic	<i>n</i>	<i>M (SD) or %</i>	Min-Max
Age	150	20.24 (2.21)	18–37
Race/ethnicity	150		
Asian, Asian American, Pacific Islander	68	45.3	
White/Anglo or European American	35	23.3	
Hispanic/Latino(a)	22	14.7	
Bi-racial	14	9.3	
Arabic/Middle Eastern	4	2.7	
Other	4	2.7	
Black/African American, Caribbean	3	2.0	
Family income			
Less than \$10,999	4	2.7	
\$10,000–\$19,999	7	4.7	
\$20,000 – \$29,999	10	6.7	
\$30,000 – \$39,999	11	7.3	
\$40,000 – \$49,999	11	7.3	
\$50,000 – \$59,999	11	7.3	
\$60,000 – \$69,999	12	8.0	
\$70,000 – \$79,999	8	5.3	
\$80,000 – \$89,999 (<i>Median</i>)	12	8.0	
\$90,000 – \$99,999	6	4.0	
\$100,000–\$124,999	20	13.3	
\$125,000 – \$149,999	8	5.3	
Over \$150,000	30	20.0	
Subjective social status	150	7.29 (1.22)	4–10
Depressive symptoms (CES–D)	150	10.53 (5.63)	0–23
Trait emotional eating (DEBQ–EE)	150	2.36 (0.81)	1.00–4.46
Body Mass Index	150	22.45 (3.48)	15.97–35.20
Underweight (<18.5)	14	9.3	
Normal weight (18.5–24.99)	107	71.3	
Overweight (25–29.99)	24	16.0	
Obese (30+)	5	3.3	

Note. CES-D = Centers for Epidemiological Studies Depression Scale; DEBQ-EE = Emotional Eating Subscale of the Dutch Eating Behavior Questionnaire.

Table 2

Tests of Psychophysiological Reactivity and Recovery by Condition

Test	<i>n</i>	<i>F</i>	<i>df</i>	<i>p</i>	η_p^2
Positive mood reactivity					
Time	150	21.83	2, 146	<.001	.230
Time x Condition (Unhealthy-anticipatory, Healthy-anticipatory, No food)	150	0.28	4,294	.891	.004
Negative mood reactivity					
Time	150	45.95	2, 146	<.001	.386
Time x Condition (Unhealthy-anticipatory, Healthy-anticipatory, No food)	150	0.40	4,294	.811	.005
RMSSD reactivity					
Time	150	42.68	2, 146	<.001	.369
Time x Condition (Unhealthy-anticipatory, Healthy-anticipatory, No food)	150	0.23	4,294	.919	.003
PEP reactivity					
Time	139	86.31	2,272	<.001	.388
Time x Condition (Unhealthy-anticipatory, Healthy-anticipatory, No food)	139	0.76	4,272	.555	.011
Cortisol reactivity					
Time	150	16.54	1, 147	<.001	.101
Time x Condition (Unhealthy-anticipatory, Healthy-anticipatory, No food)	150	0.49	2, 147	.613	.007
Positive mood recovery					
Time	150	13.28	2, 144	<.001	.156
Time x Condition (Unhealthy-anticipatory, Healthy-anticipatory, Healthy-after, Unhealthy-after, No food)	150	1.87	8,290	.065	.049
Negative mood recovery					
Time	150	77.38	2, 144	<.001	.518
Time x Condition (Unhealthy-anticipatory, Healthy-anticipatory, Healthy-after, Unhealthy-after, No food)	150	0.54	8,290	.823	.015
RMSSD recovery					
Time	150	7.68	2, 144	.001	.096
Time x Condition (Unhealthy-anticipatory, Healthy-anticipatory, Healthy-after, Unhealthy-after, No food)	150	1.33	8,290	.228	.035
PEP recovery					
Time	142	34.08	2, 136	<.001	.334
Time x Condition (Unhealthy-anticipatory, Healthy-anticipatory, Healthy-after, Unhealthy-after, No food)	142	0.60	8,274	.778	.017
Cortisol recovery					
Time	150	12.09	2, 144	<.001	.144
Time x Condition (Unhealthy-anticipatory, Healthy-anticipatory, Healthy-after, Unhealthy-after, No food)	150	0.92	8,290	.493	.025

Note. For reactivity analyses, the “No food” group collapses the control group and the “after” eating groups together, as these groups were indistinguishable at this point in the study. η_p^2 = partial eta squared (effect size)