AN EXAMINATION OF BETWEEN- AND WITHIN-SUBJECT EFFECTS OF STRESS ON EMOTIONAL EATING OVER 49 CONSECUTIVE DAYS IN WOMEN

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ABSTRACT

AN EXAMINATION OF BETWEEN- AND WITHIN-SUBJECT EFFECTS OF STRESS ON EMOTIONAL EATING OVER 49 CONSECUTIVE DAYS IN WOMEN

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Objective: Stress is associated with emotional eating (EE) in women cross-sectionally (*between-subject*). However, few studies have examined stress longitudinally limiting our understanding of how within-subject variations in stress level influence risk for EE over time and whether stress is in fact a risk factor or consequence of EE (*within-subject*). This study used an intensive, longitudinal study design to examine *between-* and *within-subject* effects of major life stress, daily stress impact, and cortisol on EE in women. **Methods:** An archival sample of 477 women aged 15-30 years recruited from the Michigan State University Twin Registry provided daily ratings of EE and stress impact for 49 consecutive days, along with self-reports of major life stress in the last 12 months and hair cortisol concentration (HCC), a longitudinal measure of cortisol secretion. Mixed linear models examined main and interactive effects of each stress variable on EE. **Results:** Both *between-* and *within-subject* analyses showed that daily stress more strongly predicted EE than major life stress. Specifically, women engaged in higher levels of EE when they experienced higher levels of daily stress impact relative to other women (*between-subject*) and their own daily stress levels (*within-subject*). There was a tendency for lower HCC to predict increased levels of EE (*between-subject*). **Discussion:** Findings confirm longitudinal associations between daily stress impact and cortisol with EE in women. Results also highlight the importance of *within-subject* shifts in a woman's stress level in her risk for EE and suggest that stress management techniques may a be useful tool for treatment.

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KEY TO ABBREVIATIONS

BE = binge eating

BMI = body mass index

EE = emotional eating

INTRODUCTION

Binge eating (BE) is characterized by repeated, intermittent bouts of overconsumption of food (typically highly palatable food that is high in sugar and/or fat) accompanied by a subjective endorsement of loss of control (American Psychiatric Association, 2013). BE is present in several eating disorders (e.g., anorexia nervosa-binge/purge [AN-BP], bulimia nervosa [BN], binge-eating disorder [BED], many forms of other specified feeding and eating disorders (OSFEDs)) and affects approximately 5% of Americans (Hudson, Hiripi, Pope, & Kessler, 2007), with much higher rates (2-10 times) in females versus males (American Psychiatric Association, 2013; Klump, Culbert, & Sisk, 2017).

BE is thought to be influenced by changes in affective states (Hawkins & Clement, 1984). Supporting this hypothesis, emotional eating (EE) (i.e., overeating in response to negative emotions; Arnow, Kenardy, & Agras, 1995) is strongly associated with BE in both clinical (Masheb & Grilo, 2006; Ricca et al., 2009) and non-clinical (Stice, Presnell, & Spangler, 2002; Tanofsky-Kraff et al., 2007; van Strien, Engels, van Leeuwe, & Snoek, 2005) populations. EE has prospectively been shown to predict BE onset (Stice et al., 2002) and is positively associated with BE severity (Ricca et al., 2009). For these reasons, EE is considered a useful dimensional construct of BE behavior (Haedt-Matt et al., 2014). EE and BE are both comorbid with other psychiatric and medical conditions, including depression, anxiety, and obesity (Braden, Musher-Eizenman, Watford, & Emley, 2018; Hudson, Hiripi, Pope, & Kessler, 2007; Lazarevich, Camacho, Velazquez-Alva, & Zepeda, 2016; Ouwens, van Strien, & van Leeuwe, 2009; van Strien et al., 2016); however, the etiology of EE and BE remain poorly understood. Given the significant negative consequences associated with BE/EE, it is important to understand their development in order to better identify at-risk individuals and develop more tailored prevention and treatment options.

Stress has repeatedly been implicated in the etiology of EE and BE (Pike et al., 2006; Rojo, Conesa, Bermudez, & Livianos, 2006; Smyth et al., 2007; Wolff, Crosby, Roberts, & Wittrock, 2000). While the current study focused on associations between stress and EE, the majority of studies have examined associations between stress and BE. Given this, findings from studies of stress on both EE and BE are discussed. Stress can be defined in terms of stress frequency or stress response. Stress frequency refers to the amount of any real (e.g., abuse) or perceived (e.g., fear of judgement) threat to an individual's general well-being that disrupts their homeostasis (i.e., steady state of optimal bodily functioning; Levine, 2005), while the stress response refers to either the psychological impact of (e.g., perceived impact of a stressor; typically assessed via self-report) or physiological reaction (e.g., cortisol response to stress) to a stressor. Oftentimes stress frequency and stress response are correlated, with higher frequencies of stress being associated with a greater degree of stress impact and stronger physiological response (Rab & Admon, 2020). While increased frequency and impact of major (e.g., death of a loved one, trauma) and acute, daily stressors (e.g., heavy traffic, argument with a coworker) are both associated with increased risk for EE/BE (Becker & Grilo, 2011; Degortes et al., 2014; Hay & Williams, 2013; Loth, van den Berg, Eisenberg, & Neumark-Sztainer, 2008; Pike et al., 2006), it is well accepted that the stress response has a greater influence on an individual's overall health and functioning than stress frequency (Hay & Williams, 2013; McEwen & Akil, 2020; Rojo et al., 2006; Woods et al., 2010).

The majority of studies examining associations between stress frequency, impact and EE/BE have examined *between-subject* effects. *Between-subject* studies typically compare major life and daily stress frequency, the psychological impact of a stressor or the physiological stress response between women who report high versus low levels of EE/BE. These *between-subject*

studies can be used to identify *for whom* stress increases risk for EE/BE. In other words, do women who exhibit higher levels of EE/BE experience more major life or daily stressors and/or exhibit greater psychological (e.g., stress impact) and physiological (e.g., cortisol) responses to stress than women with lower levels of EE/BE? Most of these studies have utilized retrospective self-reports (i.e., assessments of past stress exposure and stress impact over a certain period of time – 1 month ago, 1 year ago, over one's lifetime) of major life and acute stress to determine how exposure to and impact of stressors are associated with EE and BE.

In general, *between-subject* studies have shown that increased frequency of both major (e.g., death of a loved one, trauma) and acute, daily stress (e.g., traffic, argument with a coworker) is associated with increased risk for EE and BE behaviors (Becker & Grilo, 2011; Degortes et al., 2014; Hay & Williams, 2013; Loth et al., 2008; Pike et al., 2006). Additionally, increased perceived impact of major life (Micali et al., 2017; Rojo et al., 2006; Woods, Racine, & Klump, 2010) and acute, daily stressors (Crowther, Sanftner, Bonifazi, & Shepard, 2001; Diggins, Woods-Giscombe, & Waters, 2015; Jarvela-Reijonen et al., 2016; Kwan & Gordon, 2016; Richardson, Arsenault, Cates, & Muth, 2015; Smyth et al., 2007; Thurston, Hardin, Kamody, Herbozo, & Kaufman, 2018; Tomiyama, Dallman, & Epel, 2011) are associated with increased levels of BE (Hay & Williams, 2013; Kwan & Gordon, 2016; Thurston et al., 2018; Woods et al., 2010) and EE (Diggins et al., 2015; Jarvela-Reijonen et al., 2016; Richardson et al., 2015). It should be noted that while not all studies of major and acute, daily life stress examined both stress frequency and impact, results are consistent with the general stress literature in suggesting a stronger role for stress impact versus stress frequency in risk for EE/BE (Hay & Williams, 2013; Rojo et al., 2006; Woods et al., 2010).

Studies examining *between-subject* physiological indices of stress (e.g., basal cortisol levels, cortisol reactivity) have been more mixed in their findings. These studies are predicated on two well established findings in the general stress literature. Firstly, increased exposure to, and impact of, major life and acute, daily stressors alter physiological indices of stress and can cause increased basal cortisol levels (Godoy, Rossignoli, Delfino-Pereira, Garcia-Cairasco, & de Lima Umeoka, 2018) and increased risk for various psychological conditions (e.g., depression; de Kloet, Joëls, & Holsboer, 2005). Secondly, responses to stress exhibit an inverted U-shaped relationship with stress severity (Sapolsky, 2015). For example, at low or chronically high levels of stress frequency or impact, physiological responses to acute stress (e.g., cortisol reactivity) are low or blunted compared to moderate levels of stress frequency or impact. Because women who EE/BE tend to have experienced more major and acute, daily stressors (see above), it is possible that elevated basal cortisol levels and a blunted cortisol response to acute stress are associated with increased risk for EE and BE.

Studies examining these hypotheses have produced mixed results. For example, despite elevated cortisol levels being associated with increased hedonic value (Adam & Epel, 2007) and consumption of palatable food (Dallman et al., 2003; Gluck, 2006; Godfrey et al., 2019; la Fleur, Akana, Manalo, & Dallman, 2004; Laugero, Falcon, & Tucker, 2011; Pecoraro, Reyes, Gomez, Bhargava, & Dallman, 2004), there have been inconsistent associations reported for cortisol levels and BE in women or female animals. Three studies found that obese (Gluck, Geliebter, Hung, & Yahav, 2004; Gluck, Geliebter, & Lorence, 2004a) and non-obese (Koo-Loeb, Costello, Light, & Girdler, 2000) women who BE exhibit significantly higher levels of 24 hour urinary cortisol (i.e., urinary cortisol collected every hour for 24 hours; Koo-Loeb et al., 2000) and serum basal cortisol levels (Gluck, Geliebter, Hung, et al., 2004; Gluck, Geliebter, & Lorence,

2004a) than women who do not BE. In contrast, two studies found reduced 24 hour urinary (Lavagnino et al., 2014) and salivary cortisol levels (collected 3 times per day for two consecutive days; Larsen, van Ramshorst, van Doornen, & Geenen, 2009) in obese women with BED compared to obese women without BED. Lastly, two studies found no association between salivary cortisol and BE in obese women with BED compared to obese women without BED (Coutinho, Moreira, Spagnol, & Appolinario, 2007; Schulz, Laessle, & Hellhammer, 2011).

Although it is difficult to identify the factors that contributed to these mixed associations, it is possible that differences in age, type of tissue in which cortisol was assessed, or time of day time of cortisol collection may have contributed. The majority of studies that found positive associations between cortisol and BE examined women in young adulthood (Gluck, Geliebter, Hung, et al., 2004; Gluck, Geliebter, & Lorence, 2004a; Koo-Loeb et al., 2000), while studies that found negative or no association between cortisol and BE examined women in middle adulthood (Coutinho et al., 2007; Larsen et al., 2009; Lavagnino et al., 2014; Schulz et al., 2011). While cortisol levels are known to increase from birth into the first six months of life, decrease during early childhood, and increase again during late childhood and adolescence (Kamin & Kertes, 2017), it is uncertain how cortisol levels vary across young to middle adulthood. This can be a fruitful avenue for future research that may help to clarify these mixed associations. Additionally, the two studies that found no association between cortisol and BE used salivary cortisol (Coutinho et al., 2007; Schulz et al., 2011) while the two studies that examined serum cortisol found positive associations between cortisol and BE in women (Gluck, Geliebter, Hung, et al., 2004; Gluck, Geliebter, & Lorence, 2004a). Because cortisol is not uniformly secreted throughout the day, many studies recommend using salivary cortisol, rather than serum or urinary assays, to assess cortisol levels (Manetti et al., 2013; Odeniyi & Fasanmde, 2013;

Putignano et al., 2003; Viardot et al., 2005). However, salivary cortisol is subject to diurnal fluctuations (Parikh et al., 2018); thus, salivary cortisol only represents cortisol levels in that moment, as opposed to overall. Thus, because the two studies that found positive associations (Gluck, Geliebter, Hung, et al., 2004; Gluck, Geliebter, & Lorence, 2004a) examined cortisol at noon while the study that found no association (Schulz et al., 2011) examined cortisol in the late afternoon, diurnal fluctuations in cortisol may have contributed to the mixed associations between cortisol and BE. Clearly, additional studies are needed to clarify these methodological differences and current results; but at present, it appears that there may be positive associations between serum cortisol levels and BE in women assessed in young adulthood, but associations may vary in women assessed at other ages or using other collection (e.g., salivary) methods.

Far fewer studies of stress and BE have examined these associations on a *within-subject* level. *Within-subject* studies examine stress at an individual level to determine when and how variations in a woman's stress level influences her risk for EE/BE. These studies commonly use longitudinal measures that include daily diaries or ecological momentary assessment data that assess stress and BE multiple times per day to examine *if* and *when* stress is a risk factor for (rather than a consequence of) BE. These studies typically use "within-person" centered data that index the extent to which a women's stress levels on a particular day are higher or lower than her average stress levels (across several days) to determine how variations from baseline stress predict variations in BE.

The few *within-subject* studies that have been conducted suggest that stress is a risk factor for, and not correlate of, BE and that daily stress impact may more strongly influence BE than daily stress frequency. For example, both increased levels of daily stress frequency and impact have been found to precede BN behaviors (e.g., BE) in women (Goldschmidt et al.,

2014). While there appears to be no difference in the frequency of daily stressors (e.g., traffic, argument with a coworker) on BE versus non-BE days (Wolff et al., 2000), two studies found that women perceived daily stressors as more impactful on days when they engaged in BE (Smyth et al., 2007; Wolff et al., 2000). Only one study has examined the influence of stress on one day with BE on subsequent days; and found that daily stress impact is more strongly associated with same-day, as opposed to subsequent-day BE (Freeman & Gil, 2004). To the author's knowledge, only one *within-subject* study has examined associations between cortisol and BE and found that individuals with BED demonstrated a greater cortisol response to acute-stress in the morning compared to the afternoon (Carnell et al., 2018). This suggests that associations between physiological indices of stress and BE may vary by time of day.

Clearly, additional studies of *within-subject* effects of stress frequency, impact and physiological responses are needed. While the current findings suggest that stress may precede EE/BE and is more strongly associated with same-day EE/BE (Freeman & Gil, 2004), BE is typically followed by feelings of guilt, shame, or disgust over the binge episode (American Psychiatric Association, 2013). Therefore, it is possible that EE/BE may also contribute to stress about the EE/BE behaviors that then increases negative feelings (e.g., guilt, shame) and reinforces EE/EE as a way to cope with the stress and negative feelings. The cross-sectional nature of the current study did not allow for examination of this effect; however, additional *within-subject* studies of stress and EE/BE can help delineate stress as a risk factor for versus consequence of EE/BE. These studies can also clarify *when* deviations in stress levels most strongly associate with EE/BE (e.g., same-day versus subsequent days).

However, other gaps in the literature also remain. Studies of physiological responses to stress have produced mixed findings, potentially due to differences in participant age, tissue in

which cortisol was assessed, or time of day of cortisol collection. Additionally, because reduced hours of sleep per night (Nollet, Wisden, & Franks, 2020) has been associated with increased levels of cortisol, mixed associations between cortisol and EE/BE may also be due to sleep deprivation. Additional studies, potentially using more novel measures of cortisol secretion (e.g., hair cortisol concentrations (HCC)), that control for participant age and that include measures of sleep patterns are needed. In addition, despite *between-* and *within-subject* studies examining main effects of major life and daily stress frequency and impact, few studies have examined interactions between major life stress and daily stress frequency, or daily stress impact. Evidence suggests that *between-subject* effects of daily stress on BE are heightened in women who experience high levels of major life stress impact (Woods et al., 2010), indicating a potential sensitization to negative effects of acute, daily stress following high major life stress exposure. Thus, examining potential interactions between major life and daily stress frequency and impact is critically important for future work.

Given the above, the proposed study extends prior research on stress and EE/BE by examining main and interactive effects of major life stress, daily stress frequency, and daily stress impact on EE in an archival sample of women assessed across 49 consecutive days. Assessments of major life stress (e.g., death of a loved one) were previously collected over 49 days as well as more distal time periods (e.g., last 12 months), and daily ratings of "hassles" (e.g., traffic, argument with a coworker) were also collected. This study capitalized on the longitudinal, daily study design and conduct both *between-* and *within-subject* analyses to determine *for whom* stress increases risk for EE (*between-subject* effects), distinguish between stress as a predictor versus consequence of EE (*within-subject* effects), and elucidate *when* deviations in a woman's stress levels are most strongly associated with deviations in her EE

(same day or subsequent days) (*within-subject* effects). *Between-subject* analyses were expected to show that increased levels of major life stress, average stress frequency and impact would predict increased average levels of EE in women during the study period. In *within-subject* analyses, main and interactive effects of increased levels of daily stress frequency and impact were expected to prospectively predict increased levels of daily EE episodes (i.e., stress would be a risk factor for EE. *Within-subject* prospective effects of daily stress were expected to be stronger for same-day versus subsequent day stress-EE, as well as in women with high versus low levels of major life stress. Both *between- and within-subject* analyses are expected to show significant interactions between stress frequency and psychological impact, such that stress frequency is associated with increased EE only when levels of stress impact are also high.

In addition to examining stress frequency and impact, this study explored whether a novel measure of cortisol response (i.e., HCC) was associated with EE. Cortisol is thought to incorporate into hair via passive diffusion from the bloodstream (Stalder & Kirschbaum, 2012). Because hair grows approximately 1 cm per month (Wennig, 2000) and cortisol can remain stable in hair for up to 6 months (Kirschbaum, Tietze, Skoluda, & Dettenborn, 2009; Noppe et al., 2014), HCC provides a retrospective measure of cortisol levels over an extended time period (Stalder & Kirschbaum, 2012). In the current study, a subsample of women provided a 1.5 cm HCC sample at the end of the study that indexed cortisol secretion over the 49-days. Thus, an additional exploratory aim of this study examined *between-subject* associations between HCC and EE. No previous study has examined associations between HCC and BE or EE; consequently, these analyses were exploratory, but consistent with some past studies of cortisol levels and palatable food intake and BE (Adam & Epel, 2007; Dallman et al., 2003; Gluck, 2006; Gluck, Geliebter, Hung, et al., 2004; Gluck, Geliebter, & Lorence, 2004b; Koo-Loeb et al., 2000;

Laugero et al., 2011; Pecoraro, Reyes, Gomez, Bhargava, & Dallman, 2004) in women in young adulthood, it was expected that higher HCC would be associated with increased average EE.

METHODS

Participants

Participants included 477 female twins (aged 15-30 years old; *M* = 21.8 years; SD = 3.0) from the Michigan State University Twin Registry (MSUTR; see Burt & Klump, 2013, 2019; Klump & Burt, 2006 for MSUTR description). The MSUTR is a population-based twin registry that recruits twins in collaboration with the Michigan Department of Health and Human Services (for additional information about study recruitment, see Burt & Klump, 2013, 2019; Klump & Burt, 2006) via the use of birth records. Participants for the current project were recruited from an on-going study within the MSUTR (i.e., *A Twin Study of Exogenous Hormone Exposure and Binge Eating; EHE-BE*) that examines effects of combined oral contraceptives (COC) on disordered eating. All participants were required to meet the following criteria: 1) at least one member of the twin pair must have been taking COC for at least 3 months prior to starting the study (82.7% of participants are taking COCs, 17.3% of participants are not taking COCs); 2) for participants who are not taking COCs, menstrual cycles must be regular (between 22-32 days); 3) no psychotropic or steroid medications within past 4 weeks; 4) no pregnancy/lactation within the past 6 months; and 5) no genetic or medical conditions known to influence hormones or appetite/weight.

It is important to note that while the stress measures (described below) for this study were administered to all participants, the collection of a hair sample for HCC analysis was an optional procedure for which participants received extra compensation. In order to participate in the optional procedure, EHE-BE participants had to have hair longer than 1 inch that was free of chemical treatments to the hair roots (e.g., dying, bleaching, chemical straightening). Because of these additional criteria, the sample for the exploratory analyses (*n* = 234, 49% of the total

sample of 477 participants) is smaller than the sample for the primary analyses (n = 477). Compared to previous MSUTR studies (Burt & Klump, 2013, 2019; Klump & Burt, 2006), participants in the full sample (96.2% Non-Hispanic/Latinx, 89.3% White, 5.0% Black, 1.3% Asian, and 4.4% Multiracial) and participants who opted into the HCC study (97.4% Non-Hispanic/Latinx, 91.1% White, 3.4% Black, 0.4% Asian or Pacific Islander, and 5.5% Multiracial) had a higher percentage of participants who identified as non-Hispanic/Latinx and White. Participants who provided a hair sample were also on average, 1 year older and experienced higher levels of major life stress across their lifetime than participants who did not provide a HCC sample (p<.001; Supplemental Table 1). The two groups of participants otherwise did not differ significantly from one another on the other variables (i.e., EE, stress levels) or other key demographic variables (e.g., race/ethnicity; all p's>.05; Supplemental Table 1).

Procedures

All measures and procedures were approved by the Michigan State University Institutional Review Board. Participants provided behavioral data for 49 consecutive days. Questionnaires were completed each evening (after 5:00 pm) using an online data system or pre-printed scantrons. Additionally, all participants completed three in-person assessments occurring at the beginning of the study, halfway through the study (~ day 25), and at the end of the study (~ day 49). During these in-person assessments, each participant's eligibility was reassessed, height and weight were measured, and completed materials were collected. Hair samples for HCC were collected during the last study visit to ensure that the study captured cortisol concentrations over the 49-day study period. Between visits, staff contacted participants 1x/week to confirm continued protocol adherence and answer questions.

Measures

Daily Measures

Emotional eating (EE)

EE was assessed daily using the Emotional Eating scale of the Dutch Eating Behavior Questionnaire (DEBQ; van Strien, Frijters, Bergers, & Defares, 1986). The Emotional Eating scale assesses eating in response to negative emotions (example item: "Did you have a desire to eat when you were depressed?"); responses were made using scales from 1 (not at all) to 5 (very often). Internal consistencies for the DEBQ Emotional Eating scale were excellent in previous research (α = .93; Klump, Keel, Culbert, & Edler, 2008; Racine et al., 2012; van Strien, Frijters, Bergers, & Defares, 1986) and in the current sample (45-day average α = .90). It is important to note that eating in response to negative emotions is thought to be a core feature of BE, and the DEBQ Emotional Eating scale has demonstrated validity in differentiating among individuals with clinical and subclinical objective BE episodes (i.e., binge eating episodes characterized by an overconsumption of highly palatable food in a short period of time, accompanied by a sense of loss of control over eating; American Psychiatric Association, 2013), overweight individuals, and college students (Wardle, 1987). Furthermore, the DEBQ Emotional Eating scale is significantly and positively correlated with established measures of BE (*r*'s = .55–.69; Racine, Culbert, Larson, & Klump, 2009; van Strien et al., 1986) as well as with palatable food intake (i.e., ice cream) in a laboratory setting (van Strien, 2000). Similar to previous research (Klump et al., 2008), the instructions for the DEBQ emotional eating scale were modified with permission to ask about EE over the current day.

Daily stress

The Daily Stress Inventory (Brantley, Waggoner, Jones, & Rappaport, 1987) was used to assess daily stressors over the study period. The DSI is a 60-item, self-report questionnaire that asks participants to report whether or not they have experienced daily stressors or daily hassles over the course of the day. Items include stressors such as 'traffic difficulties', 'an argument with another person', or 'experienced bad weather'. Participants rated all events as either present or absent. For all stressors that were present, participants rated the impact of those stressors on a 7-point Likert scale that ranges from 1 = 'occurred, but was not stressful' to 7 = 'caused me to panic'. The total number of events that occurred each day was summed to create a daily stress frequency score. Similarly, the total impact for events that occurred each day was summed to create a daily impact score. The DSI has demonstrated excellent construct validity in prior research, as it is highly correlated with the frequency and intensity scales of other stress measures (e.g., the Hassles Scale; Brantley et al., 1987; Kanner, Coyne, Schaefer, & Lazarus, 1981) as well as positive associations and high convergent validity with endocrine measures of stress (e.g., urinary cortisol levels; Brantley, Dietz, McKnight, Jones, & Tulley, 1988), and good internal consistency.

Non-Daily Measures

Major life stress

The Social Readjustment Rating Schedule (SRRS; Holmes & Rahe, 1967) was used to retrospectively assess major life stress. The SRRS is a 43-item questionnaire that asks participants whether or not they have experienced any of the 43 commonly reported stressful events (e.g., death of a loved one, illness) over the course of the past year. Each item/event that occurred is then assigned a life change unit (LCU) score (ranging from 11-100) that indicates the

overall severity of the event in terms of the relative degree of adjustment necessary following exposure to each event. The item LCUs are then summed to create a total LCU score across all events. It should be noted that the LCU score for each item combines frequency and impact scores into one value, thus there are no independent LCUs or LCU total scores for frequency versus impact. A psychometric study of the SRRS has demonstrated acceptable internal consistency ($\alpha = .72$) and a very high correlation ($r = .97$) between the SRSS and the Schedule of Recent Events, another measure of major life stress (Lei & Skinner, 1980). Similar to the original SRRS, participants were asked to rate the SRRS items for the past 12 months. However, in order to assess for major life stress over the same time period as the daily stress measures and HCC, participants also completed the SRRS items for stressors that occurred over the 49-day study period. In addition, because women who do and do not EE/BE tend to differ in their stress exposure and impact across their lifetime, participants were also asked to complete the SRRS items for events occurring any time over the course of their life.

Hair cortisol concentration (HCC)

Following standard procedures (Wright et al., 2018), hair samples were obtained by cutting the hair from the posterior vertex of the scalp, as close to the scalp as possible. Because each 1 cm of hair from the scalp corresponds to cortisol secretion over the past month (Stalder & Kirschbaum, 2012), hair samples were collected during the final assessment and the first 1.5 cm of hair most proximal to the scalp were assayed for the current study to provide a retrospective index of cortisol secretion over the 49-day study period. Hair samples were wrapped in aluminum foil for protection and stored at room temperature, as previously described (Wennig, 2000), until they were shipped to the Behavioral Immunology and Endocrinology Laboratory at the University of Colorado, Denver at the Anschutz Medical Campus for analysis. Following the

procedures outlined in Hoffman, D'Anna-Hernandez, Benitez, Ross, & Laudenslager (2017), upon arrival at the lab, hair was ground and cortisol levels were measured using a commercial high sensitivity EIA kit (Salimetrics, LLC, State College, PA) that was conducted according to manufacturer's instructions as previously described in (D'Anna-Hernandez, Ross, Natvig, & Laudenslager, 2011). To calculate inter-assay coefficients of variation (CV), a pooled control of previously ground hair was extracted using the same procedures outlined above and included on each EIA plate in duplicate. Inter-assay CV for the control hair pool was 9.2% for the high hair control and 11.2% for the low hair control and intra-assay CV was 1.4%.

A growing number of studies have used HCC as a measure of cumulative cortisol secretion (see reviews Sander et al., 2020; Stalder et al., 2017; Stalder & Kirschbaum, 2012) and found high test-retest associations between repeated HCC assessments (*r*'s between 0.68-0.79; Stalder & Kirschbaum, 2012), positive associations between HCC and 30-day average salivary cortisol levels (*r*'s = 0.61, $p = .01$; Short et al., 2016) and levels of major life stress ($\beta = 0.21$, $p = .04$ for stressors such as death of a close relative, serious illness, divorce; Karlén, Ludvigsson, Frostell, Theodorsson, & Faresjö, 2011) assessed over the same time period. These data support the validity of HCC as a measure of cumulative cortisol concentration over the study period.

Hair care practices questionnaire

During the final assessment, participants completed a brief questionnaire about key hair care practices (e.g., how often they wash, color, bleach, and/or chemically straightening their hair, whether they use any scalp medication, etc.) that could influence the reliability/validity of the HCC.

Covariates

Because of the wide age range of my sample, and the fact that negative affect, body mass index (BMI), total hours of sleep per night, and income are associated with self-reported stress (Goldschmidt et al., 2014; Langer et al., 2018; Spinosa, Christiansen, Dickson, Lorenzetti, & Hardman, 2019; Tenk et al., 2018), cortisol levels (Cohen, Doyle, & Baum, n.d.; Faresjö et al., 2013; Manenschiin, van Kruysbergen, de Jong, Koper, & van Rossum, 2011; Nollet et al., 2020; Stalder & Kirschbaum, 2012; Wester et al., 2014), and BE and EE (Goldschmidt et al., 2014; Langer et al., 2018; Smyth et al., 2007; Spinosa et al., 2019), these variables were included as covariates in the *between-subject* analyses only. They were not included in the *within-subject* models, as these models examined how variability in a woman's own stress levels influence variation in her own levels of EE.

Daily ratings of negative affect were assessed via the Negative Affect Scale from the Positive and Negative Affect Schedule (Watson, Clark, & Tellegen, 1988). This scale consists of 10 items measuring the full range of daily negative emotions (e.g., distress, nervousness, irritability, fear). Participants rated the degree to which each emotion was experienced; responses were made using Likert scales from 1 (very slightly/not at all) to 5 (extremely).

Height and weight were measured using a wall-mounted ruler and digital scale during the three in-person visits (i.e., beginning, intermediate, end of study) in order to calculate BMI (i.e., weight (kilograms)/height (meters)^2). Because prior work from our lab has shown that changes in weight across a 45-day period are minimal (e.g., M = -0.20 lb change, SD = 3.39; Klump et al., 2013), the average BMI across the three study visits was calculated and used in analyses.

Income was assessed via the question '*What is the approximate average income of your parents*' with response options as 'under $20,000', '$20,000-$40,000', '$40,000-$60,000',

'$60,000-$100,000', and 'over $100,000'. Lastly, because sleep (Nollet et al., 2020) is associated with cortisol levels, total hours of sleep per night was included as a covariate in the exploratory HCC analyses. Total hours of sleep per night was assessed via the question '*How many hours of sleep did you get last night?'* with response options ranging from '0-4 hours' to 'more than 13 hours'.

Statistical Analyses

General Modeling Approach

Originally, I planned to examine *between-* and *within-subject* main and interactive effects of daily stress frequency, daily stress impact, and major life stress, including covariates on EE. However, average levels of daily stress frequency and impact were very strongly correlated (r = 0.93, p<.002), suggesting that these variables assess the same or very similar constructs in this study. Therefore, to reduce multicollinearity and because prior studies suggest that stress impact more strongly influences EE as compared to stress frequency (Hay & Williams, 2013; McEwen & Akil, 2020; Rojo et al., 2006; Woods et al., 2010), only daily stress impact was included in all *between-* and *within-subject* analyses.

Prior to analyses, daily stress impact, major life stress, HCC, BMI, negative affect, and EE were log transformed prior to analyses in order to account for positive skew. Mixed linear models (MLMs) were used to examine both *between-* and *within-subjects* associations, as MLMs can control for the non-independence of the twin data as well as the repeated measures that are examined in analyses of *within-subject*/daily effects. Primary analyses of the major life stress variables focused on major life stress in the last 12 months to maximize variability in scores, but secondary analyses explored associations between major life stress over the 49-day study period and across the lifetime with EE. Because results were nearly identical for the primary versus

secondary analyses of major life stress scores, only results from the primary analyses are discussed below. Results from the secondary analyses are included in the supplemental material only (see Supplemental Tables 2 & 3). To control for multiple comparisons, a *p*-value of .01 and the more conservative Bonferroni corrected *p*-value = .002 were used.

Primary Analyses

Between-Subject Analyses

Between-subject analyses examined whether women who reported higher levels of daily stress impact and major life stress) also reported higher levels of EE compared to women with lower stress levels. For these analyses, all daily measures (i.e., daily levels of EE, stress impact, negative affect, age) were averaged across the 49-day study period in order to obtain a mean value that indexed overall levels of each variable across the study period. Variables that were assessed at only a single-time point (i.e., major life stress, income) or were already averages (i.e., BMI) did not require this averaging, but instead were included as the total score/value. All predictor variables were standardized prior to analysis in order to reduce multi-collinearity between the main and interaction effect variables and to create a common unit of measure for each variable.

Pearson correlations were calculated first to provide initial indications of associations between the stress scores and EE. Two-level, MLMs were used to test the main as well as interactive effects of each stress variable (e.g., major life stress x daily stress impact) on EE, with participants (level 1) nested within twin pairs (level 2).

Within-Subject Analyses

Within-subject analyses examined whether variations in a woman's reported daily stress frequency and impact were associated with her levels of same-day and subsequent-day EE.

These analyses focused on the daily variables (i.e., daily stress impact, negative affect, EE) in order to examine *within-subject* effects within and across days. For these analyses, all daily variables were *within-subject* centered (i.e., each participant's daily value of each variable was subtracted from each participant's average daily score for each variable) and then standardized to determine how daily variation in each woman's stress levels relative to her average levels influenced daily changes in EE. Daily stress impact from 1 day ago was calculated by lagging the *within-subject* centered and standardized same-day daily stress variable and daily stress impact from 2 days ago was calculated by lagging the daily stress from 1 day ago variable. Because major life stress is not a daily variable, it was not *within-subject* centered and standardized; instead major life stress was *between-subject* standardized prior to being included in analyses.

Pearson correlations provided initial indications of associations between same-day and time-lagged daily stress impact and daily EE. Three-level MLM models examined how changes in daily stress frequency, daily stress impact, and covariates associated with changes in same-day and subsequent-day EE. Observations (level 1) were nested within participants (level 2), and participants were nested within twin pairs (level 3). The first set of MLMs examined predictive effects of daily stress impact including covariates on same-day EE.

Then, because chronic exposure to major life stress can sensitize women to the negative effects of daily stressors (see *Introduction*), a second series of models examined the 2-way interaction between the *between-subject* major life stress variables and the *within-subject* daily stress impact variable, controlling for the covariates.

A final set of MLMs was then conducted to examine predictive effects of daily stress impact from 1 and 2 days ago on EE. Models were conducted the same as described above (i.e.,

separate models for main and interactive effects) except that the same-day stress variables were also included in the time-lagged models with the daily stress variables from 1 and 2 days ago. This ensured that that any predictive effects of the time-lagged stress variables were above and beyond those of the same-day stress variables.

Exploratory Analyses

Exploratory analyses examined HCC and its association with *between-subject* risk for EE in the subsample of women who provided a hair sample. Because HCC is a cumulative measure of cortisol over the 49-day study period daily *within-subject* effects of cortisol levels on EE could not be examined. Analyses were identical to the *between-subject* models described above, including data preparation methods (e.g., using average EE values), the Pearson correlations examining initial associations between HCC and EE, and the MLMs examining main effects of HCC on EE. Again, because total hours of sleep has been shown to influence cortisol levels, this variable was included as a covariate, along with BMI, negative affect, age, and income. To confirm that associations between HCC and EE were unaffected by participants' hair care practices, all analyses were repeated in the 220 women (49% of the total sample) who did not exhibit any potentially confounding factors for HCC (e.g., chemical treatments).

RESULTS

Descriptive statistics

Descriptive statistics for the full sample are presented in Table 1. A wide range of EE was represented (average EE score range = 0-2.58 out of a possible range of 0-4), indicating good variability in dysregulated eating symptoms. Participants also varied considerably on indices of daily stress impact and major life stress. Average levels of daily stress impact ranged from 0.92-253.02, out of a possible 0-399. Average levels of major life stress in the last 12 months ranged from 0-668 out of a possible 0-2246. The mean and range of these scores are consistent with findings from previous population-based studies of EE (e.g., Hildebrandt et al., 2015; Klump, Keel, Burt, et al., 2013), major life stress (e.g., Woods et al., 2010), and daily stress impact (e.g., Brantley et al., 1987; Wolff et al., 2000; Woods et al., 2010) in young adults.

Between-subject analyses

Pearson correlations

As shown in Figure 1 and Supplemental Table 4, average levels of EE were strongly correlated with average levels of stress impact (r = 0.56, p<.002). However, average levels of EE were not significantly correlated with levels of major life stress in the last 12 months (r = 0.07, p = .16), suggesting that EE is more strongly associated with measures of daily stress impact compared to major life stress. Average levels of EE and daily stress impact were also strongly associated with average levels of negative affect (r's = 0.67, p<.002), reflecting the affective nature of EE and stress. Minimal associations were found between average EE levels and BMI (r = 0.05, p .35), age (r = -0.10, p = .02), and income (r = 0.10, p = .82).

Mixed linear models

Results from the MLMs corroborated the above correlations between daily stress impact and EE (Table 2) and showed that even after controlling for negative affect, BMI, age, and income, women who experienced higher levels of average daily stress impact over the 49-day study period engaged in higher levels of average EE than women with lower levels of average daily stress impact ($\beta = 0.35$, $p<.001$). While major life stress in the last 12 months did not significantly predict average levels of EE ($\beta = -0.02$, $p>.05$), there was a trend-level two-way interaction between average stress impact and major life stress in the last 12 months ($\beta = 0.11$, $p = .03$). Specifically, women who experienced high levels of major life stress in the last 12 months (defined as 1 SD above the mean) engaged in significantly higher levels of EE when they also experienced high versus low levels of average stress impact (Figure 2). In contrast, women who experienced low levels of major life stress in the last 12 months (defined as 1 SD below the mean) showed no change in average levels of EE regardless of average stress impact level (Figure 2).

Within-subject analyses

Pearson correlations

Correlations between the *within-subject* variables were similar to the correlations between the *between-subject* variables. As shown in Figure 3 and Supplemental Table 5, levels of daily EE were moderately correlated with levels of same-day stress impact ($r = 0.22$, $p <.002$). Daily EE was less strongly correlated with time-lagged measures of daily stress impact (r's = 0.06-0.06, p's<.002). Similar to the *between-subject* correlations, daily levels of EE and stress impact were significantly associated with daily levels of negative affect (r's = 0.18-0.46, p's<.002).

Mixed linear models

As shown in Table 3, increases in same-day stress impact predicted increased levels of daily EE above and beyond the effects of the covariates. Specifically, women were more likely to engage in higher levels of daily EE when they experienced higher than their average levels of daily stress impact ($\beta = 0.15$, $p<.001$). While shifts in a woman's daily stress impact significantly predicted shifts in her levels of EE two days later ($\beta = 0.03$, $p<.001$), shifts in daily stress impact more strongly predicted shifts in EE when stress impact and EE occurred on the same day (see Table 3). Counter to hypotheses, women with higher levels of major life stress in the last 12 months did not increase their daily EE levels when they experienced higher levels of daily stress impact relative to their average daily stress impact levels, either when daily stress impact occurred on the same-day or on prior days (β's <0.01, all p's>.05; see Tables 3).

Exploratory analyses - HCC

Descriptive statistics

Similar to the full sample of women, the subsample of women who provided a hair sample demonstrated a wide range of EE (average EE score range = 0-2.48, out of a possible 0-4) and considerable variability on indices of daily stress impact and major life stress (Table 1). While there is currently no standard range for HCC, participants did appear to exhibit ample variable in HCC over the study period (mean = 10.71 pg/mg, SD = 18.86 pg/mg; range = 1.82-191.2 pg/mg). This mean and range are consistent with HCC findings from other population-based samples in young adults and adults (e.g., Cieszyński, Jendrzejewski, Wiśniewski, Owczarzak, & Sworczak, 2019; Ferro & Gonzalez, 2020; García-León, Pérez-Mármol, Gonzalez-Pérez, García-Ríos, & Peralta-Ramírez, 2019; O'Brien, Meyer, Tronick, & Moore, 2017).

Pearson correlations

HCC was not significantly associated with average levels of EE in the full sample of participants who provided a hair sample (r = -0.13, p = .05; Figure 4 and Supplemental Table 6) or in the subsample of women who did not exhibit any potentially confounding factors for HCC (e.g., chemical treatments) (r = -0.15, p = .03; Figure 5). Additionally, and unexpectedly, HCC was not significantly associated with average levels of daily stress impact collected over the same time period (r = 0.01-0.02, p's>.05; Figure 4 & 5) in either sample.

Mixed linear models

While HCC did not significantly predict average levels of EE in either the full sample of women that provided a hair sample (β = -0.14, p = .007) or the subsample of women without confounding factors for HCC (e.g., no chemical hair treatments; β = -0.16, p = .003; Table 4) when using the more conservative Bonferroni corrected p-value = .002, results were significant at p<.01. These results suggest a tendency for lower HCC levels to predict increased levels of average EE after controlling for the covariates. Notably, these results are in contrast to the correlations described above. Because HCC can be influenced by a participant's BMI, age, average hours of sleep, etc., discrepancies between HCC's association with EE in the Pearson correlations versus the MLMs may be due to confounding effects of the covariates. To examine this possibility further, post-hoc partial correlations were run to examine associations between HCC and EE, controlling for the covariates. Indeed, controlling for the covariates did strengthen the association between HCC and EE in both the full sample of women (r = -0.21, p = .002) and in the subsample of women who did not exhibit any potentially confounding factors for HCC (r = -0.23, p = .001). Importantly results from the partial correlations mimic the results found in the

MLMs. Specifically, they show there is a tendency for low HCC to predict higher levels of EE in both the full sample ($p<.01$) and subsample of HCC women ($p<.01$).

DISCUSSION

This is the first longitudinal study to examine *between-* and *within-subject* effects of daily stress impact, major life stress, and HCC on EE in women. My findings indicate that daily stress is a more robust predictor of EE in women than major life stress. Specifically, women were more likely to report higher levels of EE when they experienced higher than average levels of daily stress relative to other women (*between-subject* effects) and their own daily stress levels (*within-subject* effects). Additionally, shifts in daily stress impact more strongly predicted same-day EE as compared to subsequent-day EE. Lastly, there was a tendency for women with lower HCC to report higher levels of EE (*between-subject* effects). Overall, the current study extends prior findings of stress and EE in women by distinguishing the relative influence of daily stress impact, major life stress, and cortisol on EE, identifying *for whom* stress increases risk for EE, and *when* effects of stress on EE are strongest.

Stronger predictive effects of daily stress impact compared to major life stress on EE suggest that daily stress plays a stronger role in triggering EE episodes than major life stress. This may seem surprising given the inherently more severe nature of major life stressors (e.g., death of a loved one, trauma) compared to daily stressors (e.g., traffic, negative interactions with a coworker). It is important to note, however, that major life stress may be more important for precipitating the initial onset of disordered eating behavior rather than predicting on-going EE/BE. Women are 6x more likely to develop disordered eating behaviors if they experience chronically high levels of major life stress compared to women who experience low levels of major life stress (Pike et al., 2006). Compared to women without an eating disorder, women who eventually developed an eating disorder experienced significantly higher levels of major life stress in the year preceding their disorder (Rojo et al., 2006). Of particular salience are chronic

and/or severe stressors, such as trauma (e.g., sexual, physical, and/or emotional abuse) (Backholm, Isomaa, & Birgegård, 2013; Palmisano, Innamorati, & Vanderlinden, 2016; Smyth, Heron, Wonderlich, Crosby, & Thompson, 2008; Zelkowtiz, Zerubavel, Zucker, & Copeland, 2021). Because I did not assess trauma experience in my participants, I was unable to compare associations between trauma versus daily stress and cortisol on EE. However, given the role of trauma in disordered eating etiology, this could be an important avenue for future research to better understand how different types of stress influence risk for EE in women.

The accumulation of stress impact throughout the day may also contribute to the stronger associations between daily stress and EE in my study. In fact, a recent study by Smith and colleagues (2020) showed that individuals with higher levels of perceived stress and greater stress accumulation throughout the day engaged in higher levels of BE and reported more food cravings than individuals with lower levels of perceived stress or stress accumulation (*between-subject* effects). They also found that within each day, individuals were more likely to engage in higher levels of BE following moments of greater stress accumulation (*within-subject* effects) (Smith et al., 2020). My study suggests that women who experience higher levels of major life stress in the last 12 months may experience stronger associations between daily stress impact and EE. This is an important finding that may help to better identify women at-risk for EE. However, this finding should be interpreted with caution as the finding was only at a trend-level in the within-subject models and was not observed in studies of *between-subject* effects. Replication studies will be needed to verify this finding and further elucidate the relationship between major life stress and daily stress impact on EE in women.

Originally, this study planned to examine how both daily stress frequency and impact are associated with EE, however, due to multicollinearity between these variables, I was only able to

examine associations between daily stress impact and EE. Other studies have also found multicollinearity between daily stress frequency and impact measures (Hay & Williams, 2013; McEwen & Akil, 2020; Rojo et al., 2006; Woods et al., 2010) and suggested a possible need for to assess frequency and impact via multiple measures (e.g., Perceived Stress Questionnaire, Daily Stress Inventory, Daily Hassles Scale, Daily Hassles & Uplifts Scale) to reduce the likelihood of multicollinearity between the variables and allow for better delineation of the associations between stress frequency and impact on EE. However, this option is not guaranteed to eliminate the multicollinearity between the two variables. If multicollinearity persists, it may bring into question a conceptual issue – i.e., that the two constructs are not truly separate. In other words, do women who experience a low frequency of highly impactful daily stressors engage in higher levels of EE than women who experience a high frequency of low impact daily stressors? This is a yet unexplored question in the stress and disordered eating literature. Given the inter-individual nature of the stress response, both options are possible and likely depend upon a woman's past experience with the daily stressors, level of control over the stressor, or duration between stressors, among other factors. Future studies will be needed to answer this question. If stress frequency and impact are indeed found to be separate constructs, it will also be important to examine the nature of the association between stress frequency and impact and their role in EE (e.g., does stress impact mediate or moderate associations between stress frequency and EE in women?).

Importantly, in the present study, increases in same-day stress impact more strongly predicted increases in daily levels of EE than stress impact from prior days, suggesting that stress and EE must occur in close temporal proximity in order for stress to influence EE. This finding is consistent with other daily studies showing a stronger association between increased stress and

same-day versus next-day disordered eating (Barker, Williams, & Galambos, 2006; Freeman & Gil, 2004; Smith et al., 2020). While the current study did not assess the temporal sequence of stress and EE within the day, other studies report that stress levels increase significantly in the hours leading up to BE and decrease following BE (Smyth et al., 2009). Increased levels of negative affect appear to mediate associations between increased stress impact and increased levels of BE (Goldschmidt et al., 2014; Srivastava, Lampe, Michael, Manasse, & Juarascio, 2021). Thus, changes in stress levels may be stronger for same-day rather than subsequent-day EE because of the immediate increase in negative affect - a strong prospective risk factor for EE (Klatzkin et al., 2019; Steinsbekk, Barker, Llewellyn, Fildes, & Wichstrøm, 2018). Alternatively, because same-day and time-lagged variables are often correlated with one another (r's = 0.10-0.13, p<.002 in this study), a lag structure within a model may over-specify the influence of the same-day predictors by including a sequence of lagged variables that contribute only marginally to the dependent variable, thus making it difficult to detect a significant effect of time-lagged variables above and beyond that of the same-day variables. Future studies are needed to determine if stronger associations between same-day stress and EE are due to factors such as negative affect, statistical artifacts, or both.

Moving forward, it will be important to identify the mechanisms underlying daily stress impact-EE associations. Given the interplay between stress and brain reward systems, alterations within the mesocorticolimbic system is a key candidate to consider. Acute stress increases activity in mesocorticolimbic regions (e.g., anterior cingulate cortex, nucleus accumbens) while simultaneously reducing activity in brain regions associated with inhibitory control and executive functioning (e.g., prefrontal cortex) that play an important role regulating emotions (Dixon, Thiruchselvam, Todd, & Christoff, 2017) and 'braking' reward-related behavior

(Arnsten, 2015; Baik, 2020). Following chronic stress, activity in regulatory regions becomes downregulated such that they are no longer able to actively regulate emotional responses to stress (Orem et al., 2019) or reward-related behavior following stress (Arnsten, 2015). With poor regulatory control over food intake and emotion regulation, it may be more difficult to not overeat, particularly when a woman is eating highly rewarding palatable food and experiencing high levels of stress impact. Additionally, stress interacts with mesocorticolimbic dopamine signaling to influence reward-related behavior. For example, acute stress enhances reward-induced dopaminergic signaling to brain reward regions (e.g., nucleus accumbens) (Graf et al., 2013), helping to reinforce the rewarding behavior (e.g., EE). However, chronic stress suppresses reward-induced dopamine release into the nucleus accumbens (de Kloet, Joëls, & Holsboer, 2005a; Minami et al., 2017), weakens signaling to reward reinforcing pathways, and increases signaling to reward inhibitory pathways (Francis et al., 2015). Because palatable food is inherently rewarding, chronic stress may promote the overeating of palatable food to compensate for this hypo-reward state.

Stress may also alter signals associated with general feeding to influence risk for EE in women. Of particular interest is the orexigenic neurotransmitter, ghrelin. Ghrelin, traditionally thought of as a pro-hunger hormone (Müller et al., 2015), also regulates stress (Stone, Harmatz, & Goosens, 2020) and reward (Stievenard et al., 2017). Following acute stress, ghrelin levels transiently increase (Stone et al., 2020) and can remain elevated for prolonged periods of time following chronic stress (Stone et al., 2020). Through its actions in the mesocorticolimbic reward system, ghrelin enhances dopamine release into the nucleus accumbens and increases dopamine turnover (Stievenard et al., 2017). In particular, ghrelin's actions in the ventral tegmental area (the central hub for dopamine synthesis; (Han et al., 2017) increase the firing rate of

dopaminergic neurons (Abizaid et al., 2006) and play a key role in the motivation for and consumption of palatable food (Stievenard et al., 2017). Thus, stress-induced elevations in ghrelin levels may increase risk for EE via increased dopaminergic signaling and subsequent increased motivation for palatable food reward. While little is known about how stress and ghrelin interact to influence disordered eating, ghrelin is positively associated with EE in women (Rossi et al., 2021) and has been suggested to mediate associations between early childhood trauma and BE (Rossi et al., 2021). These associations may be due to altered ghrelin feedback mechanisms (Raspopow, Abizaid, Matheson, & Anisman, 2010, 2014) that can prolong ghrelin's orexigenic and dopaminergic effects to increase palatable food consumption even after the stress is no longer present. Additional work using animal studies will be needed to examine these mechanisms more directly to better elucidate how stress influences EE in women.

Lastly, a novel finding of my study was that lower HCC predicted higher levels of EE, potentially indicative of hypoactive hypothalamic-pituitary-adrenal (HPA)-axis functioning in women who engage in higher levels of EE. These findings are consistent with prior findings showing a blunted/hypoactive cortisol response to stress in women who EE (Het, Vocks, Wolf, Herpertz, & Wolf, 2020; Het et al., 2015; Tomiyama et al., 2011; van Strien, Roelofs, & de Weerth, 2013). Of note, these findings are also in contrast to other studies reporting either a positive (Gluck, Geliebter, Hung, & Yahav, 2004; Gluck, Geliebter, & Lorence, 2004a; Koo-Loeb, Costello, Light, & Girdler, 2000) or no association (Coutinho et al., 2007; Schulz et al., 2011) between cortisol and EE/BE in women. This inconsistency may be due to diurnal fluctuations in salivary and urinary cortisol measures (Carnell et al., 2018). Because HCC provides a cumulative, longitudinal measure of cortisol that is unaffected by diurnal fluctuations in cortisol, it may provide a useful index of cortisol-EE/BE associations in women. Importantly,

however, HCC significantly predicted EE only after controlling for BMI, age, total hours of sleep per night, income, and hair hygiene practices. Additionally, while hypoactive HPA-axis functioning can develop following chronic stress exposure (Lo Sauro, Ravaldi, Cabras, Faravelli, & Ricca, 2008), my findings found no significant association between HCC and daily stress impact (r = 0.02; p>.05) assessed over the same time period (Table 4). Other studies have reported a similar lack of association between HCC and self-reported indices of stress (e.g., Braig et al., 2016; O'Brien, Tronick, & Moore, 2013; Schlotz et al., 2008; Streit et al., 2016). A misalignment in time periods over which HCC and self-reported measures assessed cortisol/stress (e.g., HCC assessed cortisol over the past 3 months and self-reported measures assessed stress in the past 1 month) is one potential reason for these lack of associations; however this was controlled for in my study. Thus, other yet unknown factors must be contributing to these lack of associations. Future studies are needed to clarify this discrepancy and replicate associations between HCC and EE.

Before concluding, it is important to note limitations of the current study. Firstly, while my goal was to examine how stress is associated with EE, it is possible that stress and EE exhibit a reciprocal relationship. In fact, despite studies reporting stress levels decreasing immediately after BE (Smyth et al., 2009), BE is often accompanied by feelings of shame, guilt, and/or disgust over the binge (American Psychiatric Association, 2013), suggesting that BE is distressing in the long-term. To test the possibility that EE is also distressing, post-hoc MLMs were conducted examining predictive effects of EE from one and two days ago on daily stress impact (see Supplemental Table 7). Results showed that while EE from two days ago did not predict daily stress impact (β = 0.02; p>.01), EE from one day ago did (β = 0.03; p<.001). These findings are intriguing given that the difficulty detecting time-lagged associations using MLMs.

Nevertheless, these findings do suggest that there may be a reciprocal relationship between EE and stress that can persist over multiple days. Because I didn't examine when in the day stress and EE occurred relative to each other, I was unable to verify if EE predicts stress impact later that same day. While some studies suggest that bulimic symptoms, including BE, predict increased levels of daily hassles (Kwan & Gordon, 2016) and negative affect (Barker et al., 2006), additional studies will be needed to better capture the temporal dynamic between EE and stress on the same-day (e.g., ecological momentary assessments studies).

Secondly, because HCC provided a cumulative measure of cortisol levels over the 49-day study period, I was only able to examine how *between-subject* changes in average cortisol levels were associated with changes in average levels of EE. However, in order to more fully understand how cortisol influences EE in women, *within-subject* studies that examine how daily variation in a woman's cortisol levels contribute to shifts in her daily levels of EE are needed. Given individual differences in cortisol reactivity to stress (Appelhans, Pagoto, Peters, & Spring, 2010; Raspopow et al., 2010; Tomiyama et al., 2011; van Strien et al., 2013) and diurnal variation in cortisol-disordered eating associations (Carnell et al., 2018), assessing *within-subject* effects of cortisol levels on EE can be difficult and may require multiple measures of cortisol levels throughout the day. Additionally, because cortisol response to stress varies as a function of stress impact (Sapolsky, 2015), *within-subject* changes in daily cortisol levels may influence daily shifts in EE differently in women with high versus low levels of stress impact. To address these challenges and improve our understanding of the individual factors that contribute to a woman's risk for EE, future studies that examine how *within-subject* changes in both daily stress impact and cortisol levels are associated with shifts in a woman's EE are needed.

Lastly, my sample identified as predominantly non-Hispanic/Latinx and White and reported high parental income. Prior studies have reported that women identifying as Black, Indigenous, or a Person of Color (BIPOC) experience significantly higher levels of stress than White women, due to increased levels of discrimination, oppression, and lower socioeconomic status, among other stressors (Hatch & Dohrenwend, 2007; Lehrer, Goosby, Dubois, Laudenslager, & Steinhardt, 2020; O'Brien et al., 2017; Pickett, McCoy, & Odetola, 2020). Therefore, it will be important to replicate this study in a more racially, ethnically, and socioeconomically diverse sample of women to determine if findings from this study apply to women from other demographic groups.

Nevertheless, my study had several strengths, including its intensive, longitudinal, daily study design, the inclusion of both self-reported and physiological indices of stress, and the examination of both *between-* and *within-subject* effects of stress on EE. Overall, the current study extends prior findings of stress and EE in women by distinguishing the relative influence of daily stress impact, major life stress, and cortisol on EE, identifying *for whom* stress increases risk for EE, and *when* shifts in stress levels most strongly influence a woman's shifts in her EE.

To build upon these findings and continue improving our understanding of how stress is associated with EE in women, future studies will be needed to address the following questions. Firstly, how are different types of stress associated with EE in women? While trauma (Backholm, Isomaa, & Birgegård, 2013; Palmisano, Innamorati, & Vanderlinden, 2016; Smyth, Heron, Wonderlich, Crosby, & Thompson, 2008; Zelkowtiz, Zerubavel, Zucker, & Copeland, 2021), interpersonal (Cain, Bardone-Cone, Abramson, Vohs, & Joiner, 2008; Goldschmidt et al., 2014; Monteleone et al., 2019), and psychosocial (Azarbad, Corsica, Hall, & Hood, 2010; Badrasawi & Zidan, 2019) stressors have been noted to be particularly salient forms of major life

and daily stress for disordered eating in women, most studies use measures that do not distinguish between the different stress types (e.g., interpersonal, psychosocial, work- and health-related stress). This limits our understanding of how certain types of stress are associated with risk for EE and if particular stress types are more likely to interact with one another to augment risk for EE in certain women compared to others. Studies that examine specific stress types would be helpful for better identifying at-risk women and can inform prevention/treatment practices to include more targeted resources for certain 'risky' stress types.

Secondly, in order to better understand how stress frequency and impact are associated with EE, studies will need to examine women who experience low levels of high impact stressors are more likely to EE than women who experience high levels of low impact events. As mentioned above, both options are plausible given the inter-individual nature of the stress response. But until this question has been addressed, it will be difficult to understand whether stress frequency and impact are truly separate constructs or not.

Lastly, more studies are needed to identify the mechanisms that underlie associations between daily stress impact and EE in women. Both human and animal studies will be necessary to answer this. Given strong associations between same-day stress impact and EE, human studies can focus on elucidating the factors that contribute to the temporal dynamics between daily stress and EE (e.g., increases in negative affect). To better understand how associations between stress, reward, and general feeding circuits influence EE, animal studies can examine how acute versus chronic stress influences dopaminergic regulation of palatable food consumption and how alterations in stress-induced ghrelin release contributes to this association. Together these studies will expand our understanding of how stress influences EE in women, help better identify at-risk women, and highlight potential mechanisms that inform prevention/treatment practices for EE.

APPENDICES

APPENDIX A

Primary Analyses

Table 1

Descriptive information for the full sample (N = 477) and HCC sample (N = 234).

Variable	FULL SAMPLE		HCC SAMPLE		Total Possible Range
	Mean (*SD*)	**Observed Range**	**Mean (*SD*)**	**Observed Range**	
Daily Variables – 49-Day Avg[a]					
Avg. emotional eating	0.34 (*0.42*)	0-2.58	0.37 (*0.47*)	0-2.48	0-4
Avg. stress impact	29.53 (*27.37*)	0.92-253.02	15.08 (*3.68*)	10.38-32.13	0-399
Avg. stress frequency	11.08 (*7.53*)	0.67-56.11	11.81 (*7.94*)	0.91-56.11	0-57
Avg. negative affect	15.26 (*3.89*)	10.38-42.37	1.37 (*0.47*)	1-3.48	0-80
Non-Daily Variables					
Major life stress in last 12 months[b]	151.79 (*217.53*)	0-668	130.46 (*107.38*)	0-668	0-2246
HCC (pg/mg)[b]	--	--	10.71 (*18.86*)	1.82-191.20	--
BMI (kg/m^2)[c]	24.63 (*5.38*)	17.06-58.12	24.63 (*5.15*)	17.06-54.08	--
Ethnicity/Race/Income	**Percent (N)**		**Percent (N)**		
Ethnicity					
Hispanic or Latinx	3.8% (18)	--	2.6% (6)	--	--
Non-Hispanic or Latinx	96.2% (460)	--	97.4% (229)	--	--
Race					
White	89.3% (427)	--	91.1% (214)	--	--
Black or African American	5.0% (24)	--	3.4% (8)	--	--
Asian	1.3% (6)	--	0.4% (1)	--	--
More than one race	4.4% (21)	--	5.1% (12)	--	--
Income					
Under $20,000	2.1% (10)	--	2.1% (5)	--	--
$20,000-$40,000	3.6% (17)	--	3.1% (7)	--	--
$40,000-$60,000	10.9% (52)	--	11.1% (25)	--	--

Table 1 (cont'd)

$60,000-$100,000	27.8% (133)	--	27.1% (61)	--	--
Over $100,000	52.3% (250)	--	56.4% (127)	--	--

Note: Avg. = average; BMI = body mass index; HCC = hair cortisol concentration; stress impact = daily stress impact; stress frequency = daily stress frequency.

[a]These values are the non-standardized means and standard deviations (SDs) for each daily variable that was collected over the 49-day collection period.

[b]These values are the non-standardized means and SDs for major life stress in the last 12 months and HCC across the 49-day study period.

[c]This value is the non-standardized mean and SD for BMI that was measured at the beginning, middle, and end of the 49-day collection period.

Table 2

Results from the between-subject MLMs examining main and interactive effects of stress variables and covariates on average levels of emotional eating (N = 477).

MAIN EFFECTS			
Variables	**β (*SD*)**	**t (df)**	***p***
Intercept	-0.07 (*0.34*)	-0.22 (206.26)	.83
Avg. stress impact	**0.35 (*0.06*)**	**5.77 (148.63)**	**<.001*** **
Avg. negative affect	**0.28 (*0.06*)**	**4.35 (218.29)**	**<.001*** **
Avg. BMI	0.02 (*0.04*)	0.55 (313.12)	.59
Age	<0.01 (*0.02*)	0.08 (209.57)	.94
Income	0.01 (*0.04*)	0.28 (62.67)	.78
Intercept	0.05 (*0.37*)	0.13 (205.01)	.90
Major life stress in the last 12 months	-0.02 (*0.05*)	-0.33 (103.59)	.75
Avg. negative affect	**0.52 (*0.06*)**	**9.40 (170.10)**	**<.001*** **
Avg. BMI	0.06 (*0.05*)	1.24 (283.52)	.22
Age	<0.01 (*0.02*)	-0.22 (*208.42*)	.83
Income	0.01 (*0.05*)	0.17 (57.95)	.87
INTERACTION EFFECTS			
	β (*SD*)	**t (df)**	***p***
Intercept	-0.09 (*0.35*)	-0.27 (200.31)	.79
Avg. stress impact	**0.39 (*0.06*)**	**6.13 (153.75)**	**<.001*** **
Major life stress in the last 12 months	-0.11 (*0.05*)	-2.22 (79.57)	.03
Avg. stress impact X major life stress in the last 12 months	0.11 (*0.05*)	2.18 (88.64)	.03
Avg. negative affect	**0.27 (*0.06*)**	**4.14 (202.83)**	**<.001*** **
Avg. BMI	0.03 (*0.04*)	0.73 (286.46)	.47
Age	<0.01 (0.02)	-0.01 (205.04)	.99
Income	0.01 (*0.04*)	0.15 (55.31)	.88

Note: BMI = body mass index; Stress impact = daily stress impact. All daily variables were averaged prior to being included in the above models. BMI was averaged across the 3 study assessments prior to being included in the above models.

*** Bonferroni corrected $p<.002$

Table 3

Results from the within-subject MLMs examining main and interactive effects of the same-day and time-lagged stress variables and covariates on daily levels of emotional eating (N = 477).

SAME-DAY DAILY STRESS IMPACT			
MAIN EFFECTS			
Variables	**β (*SD*)**	**t (df)**	***p***
Intercept	0.01 (*0.01*)	0.68 (2804.13)	.50
Same-day stress impact	**0.15 (*0.01*)**	**10.45 (329.19)**	**<.001*** **
Negative affect	**0.13 (*0.01*)**	**9.09 (415.05)**	**<.001*** **
BMI	<0.01 (*0.03*)	0.06 (112.56)	.96
INTERACTION EFFECTS			
	β (*SD*)	**t (df)**	***p***
Intercept	0.01 (*0.01*)	0.81 (2881.49)	.42
Same-day stress impact	**0.15 (*0.02*)**	**9.95 (327.49)**	**<.001*** **
Major life stress in the last 12 months	<0.01 (*0.01*)	0.18 (2933.70)	.86
Same-day stress impact X major life stress in the last 12 months	<0.01 (*0.01*)	-0.18 (249.34)	.86
Negative affect	**0.13 (*0.02*)**	**8.82 (406.50)**	**<.001*** **
BMI	0.01 (*0.01*)	0.79 (4636.52)	.43
DAILY STRESS FROM ONE DAY AGO			
MAIN EFFECTS			
	β (*SD*)	**t (df)**	***p***
Intercept	-0.02 (*0.01*)	-2.72 (3180.25)	.01
Same-day stress impact	**0.13 (*0.02*)**	**8.60 (370.53)**	**<.001*** **
Stress impact from one day ago	**0.04 (*0.01*)**	**2.68 (344.12)**	**.008** **
Negative affect	**0.13 (*0.01*)**	**9.04 (422.47)**	**<.001*** **
BMI	<0.01 (*0.01*)	0.54 (4664.91)	.59
INTERACTION EFFECTS			
	β (*SD*)	**t (df)**	***p***

Table 3 (cont'd)

Intercept	-0.02 (*0.01*)	-2.62 (3088.08)	.01
Same-day stress impact	**0.13 (*0.02*)**	**8.33 (369.94)**	**<.001*** **
Stress impact from one day ago	**0.04 (*0.01*)**	**2.77 (340.22)**	**.006** **
Major life stress in the last 12 months	<0.01 (*0.01*)	0.17 (3179.58)	.86
Same-day stress impact X major life stress in the last 12 months	0.01 (*0.02*)	0.76 (276.65)	.45
Stress impact from one day ago X major life stress in the last 12 months	<0.01 (*0.01*)	-0.33 (274.43)	.74
Negative affect	**0.13 (*0.01*)**	**8.78 (416.59)**	**<.001*** **
BMI	<0.01 (*0.01*)	0.51 (4519.89)	.61

DAILY STRESS FROM TWO DAYS AGO

MAIN EFFECTS

	β (*SD*)	**t (df)**	***p***
Intercept	**-0.03 (*0.01*)**	**-3.49 (3046.27)**	**<.001*** **
Same-day stress impact	**0.12 (*0.01*)**	**8.24 (366.51)**	**<.001*** **
Stress impact from two days ago	**0.03 (*0.01*)**	**3.00 (318.08)**	**<.001*** **
Negative affect	**0.14 (*0.02*)**	**8.83 (425.05)**	**<.001*** **
BMI	0.01 (*0.01*)	0.78 (4372.88)	.43

INTERACTION EFFECTS

	β (*SD*)	**t (df)**	***p***
Intercept	**-0.03 (*0.01*)**	**-3.40 (2946.43)**	**.001** **
Same-day stress impact	**0.12 (*0.02*)**	**8.12 (370.16)**	**<.001*** **
Stress impact from two days ago	0.03 (*0.01*)	2.42 (323.26)	.02
Major life stress in the last 12 months	<0.01 (*0.01*)	0.27 (3038.50)	.79
Same-day stress impact X major life stress in the last 12 months	0.01 (*0.01*)	0.63 (272.52)	.53

Table 3 (cont'd)

Stress impact from two days ago X major life stress in the last 12 months	<0.01 (*0.01*)	-0.17 (243.41)	.86
Negative affect	**0.13 (*0.02*)**	**8.51 (417.74)**	**<.001*** **
BMI	0.01 (*0.01*)	0.74 (4223.49)	.46

Note: BMI = body mass index; Stress impact = daily stress impact.

** $p<.01$; *** Bonferroni corrected $p<.002$

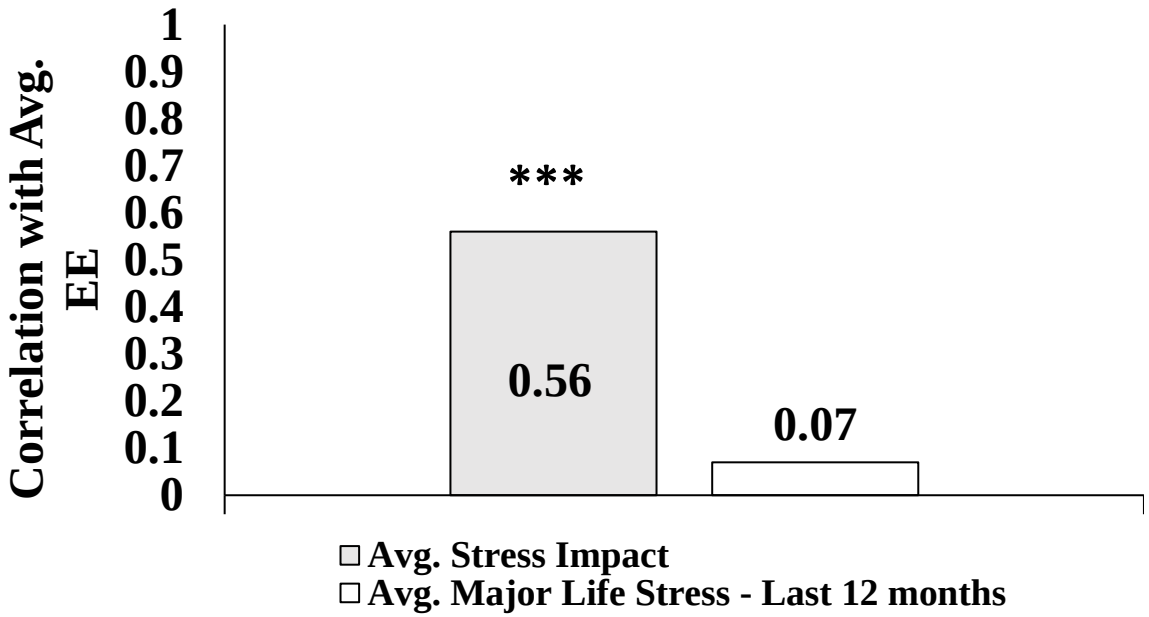

Figure 1.

Between-subject Pearson correlations for average daily stress, major life stress, and emotional eating (N = 477).

Note: ***Bonferroni corrected $p<.002$.

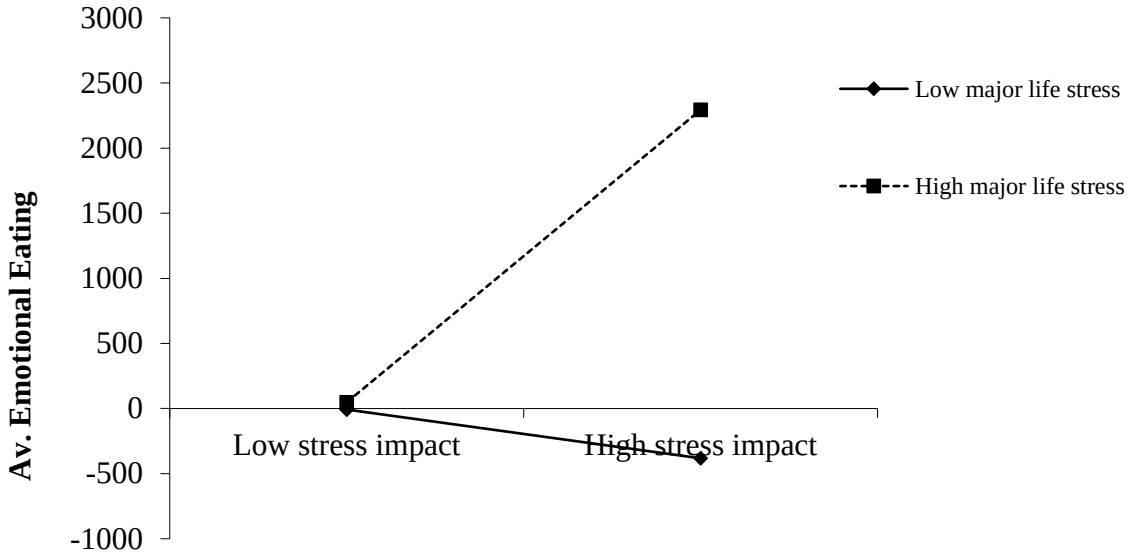

Figure 2. Two-way interaction between average stress impact and major life stress in the last 12 months. “High” and “Low” values represent 1 SD above and below the mean, respectively.

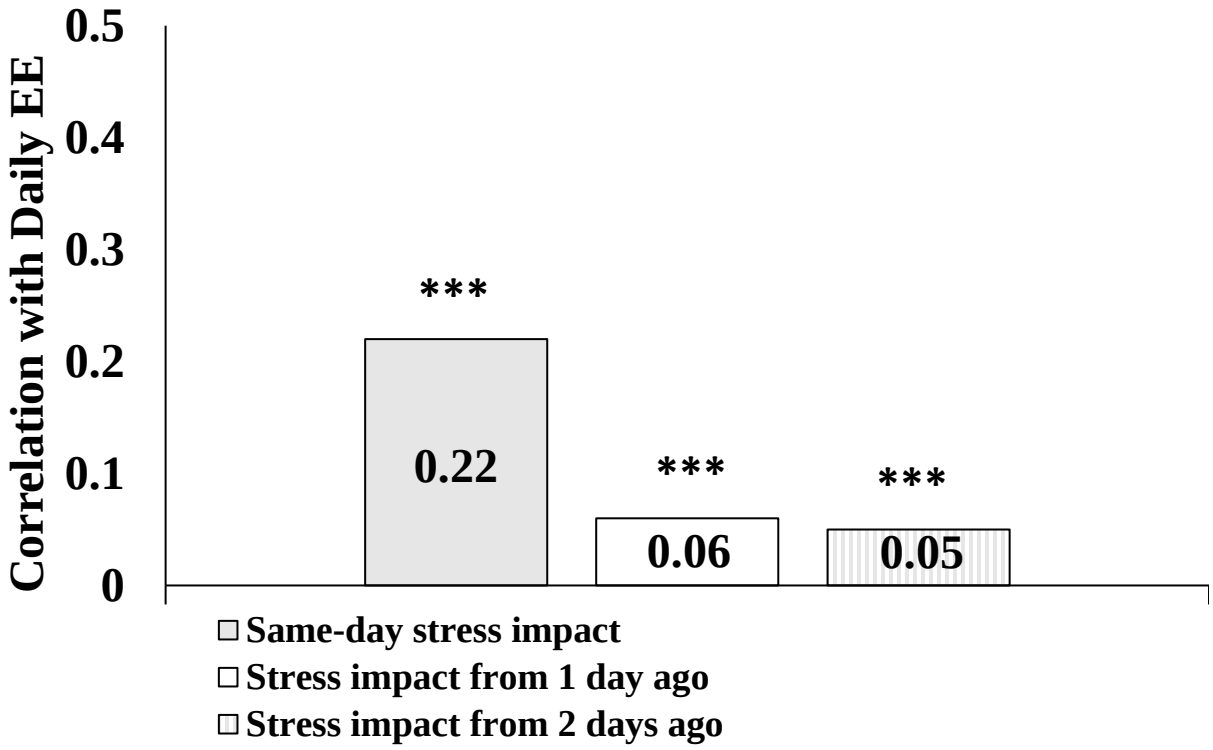

Figure 3.

Within-subject Pearson correlations for same-day and time-lagged daily stress impact, daily emotional eating, and average major life stress (N = 477). ***Bonferroni corrected $p<.002$.

APPENDIX B

Exploratory Analyses

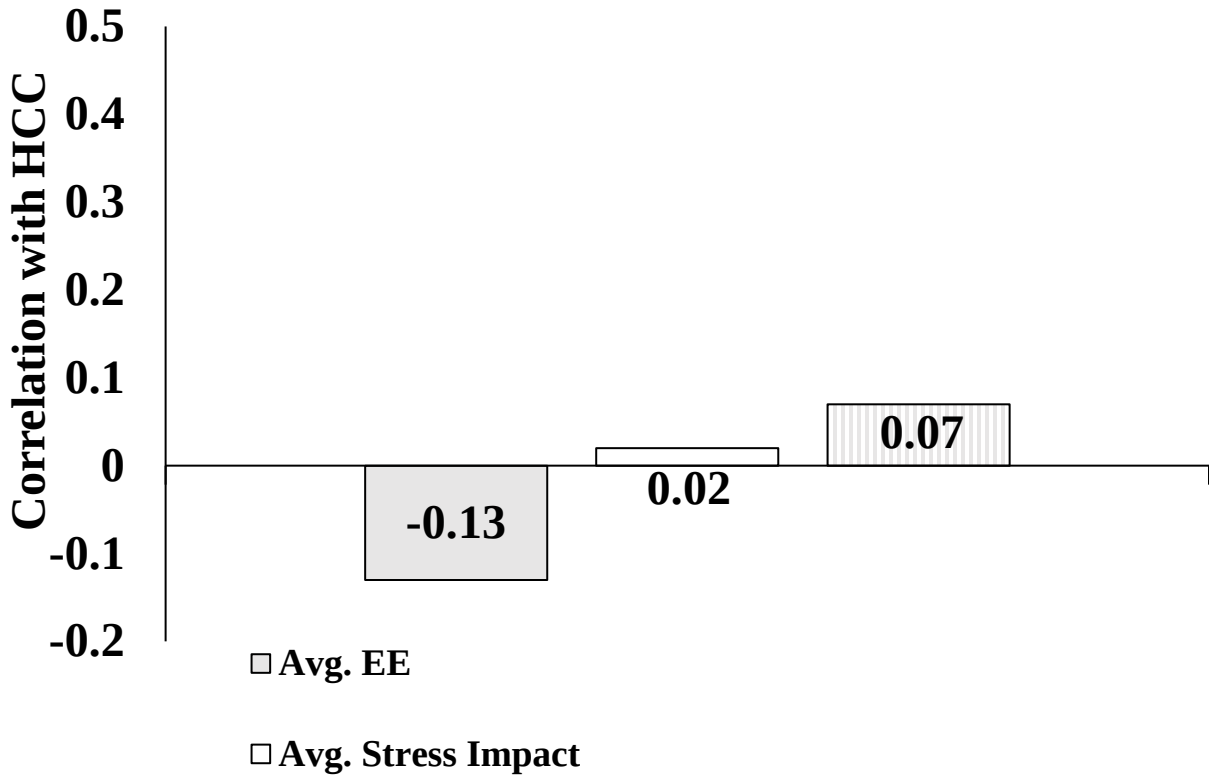

Figure 4.

Pearson correlations for hair cortisol concentration (HCC), average stress impact, major life stress in the last 12 months, and average levels of emotional eating in the full sample of HCC participants (N = 234).

Note: There were no significant correlations between HCC and either of the stress or eating variables.

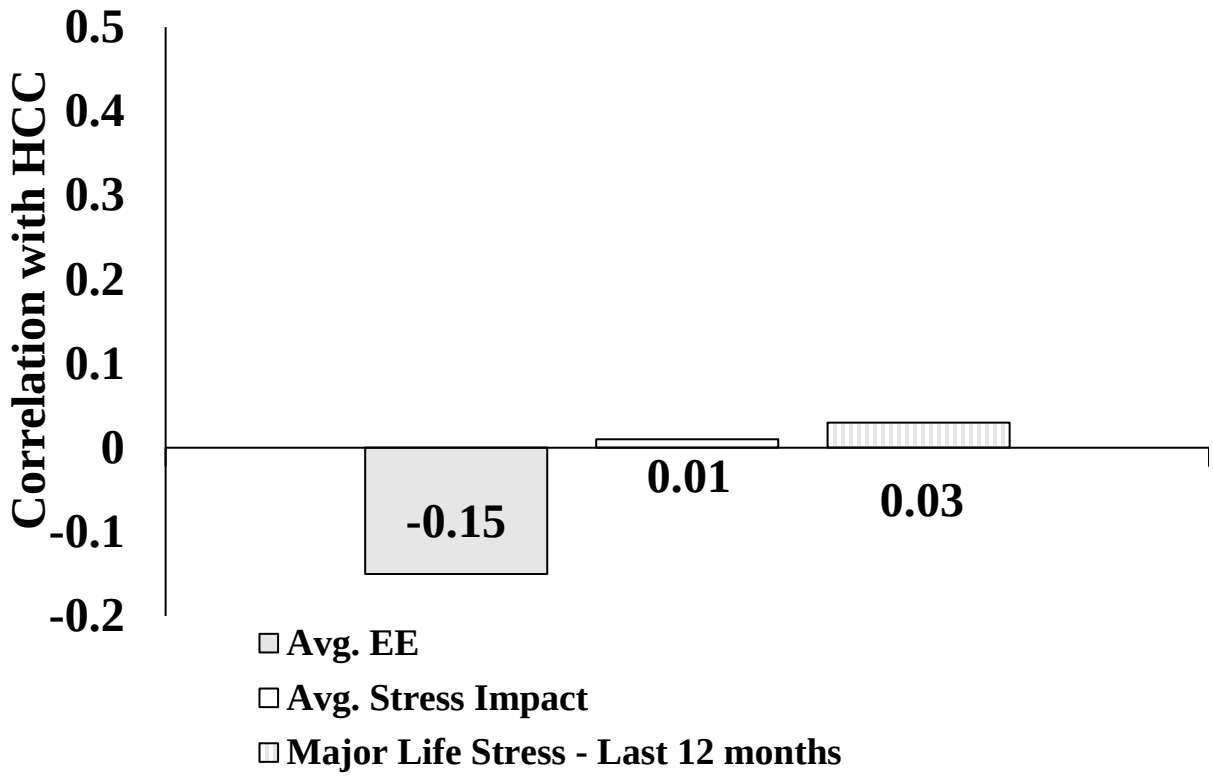

Figure 5.

Pearson correlations for hair cortisol concentration (HCC), average stress impact, major life stress in the last 12 months, and average levels of emotional eating in the subsample of HCC participants without confounding factors for HCC (N = 220).

Note: There were no significant correlations between HCC and either of the stress or eating variables.

Table 4

Results from the between-subject, exploratory MLMs examining the effects of hair cortisol concentration (HCC) and covariates on average levels of emotional eating.

Full HCC sample (*N* = 234)			
	β (*SD*)	**t (df)**	***p***
Intercept	-0.21 (*0.50*)	-0.42 (132.08)	.68
HCC	**-0.14 (*0.05*)**	**-2.70 (203.34)**	**.007****
Avg. negative affect	**0.59 (*0.07*)**	**8.81 (93.28)**	**<.001*****
Avg. BMI	0.03 (*0.05*)	0.48 (179.02)	.64
Age	0.01 (*0.02*)	0.37 (135.70)	.71
Avg. hours of sleep/night	0.01 (*0.05*)	0.10 (204.55)	.92
Income	0.03 (*0.06*)	0.47 (128.11)	.64
Subsample of women without confounding factors for HCC (*N* = 220)			
	β (*SD*)	**t (df)**	***p***
Intercept	-0.10 (*0.50*)	-0.16 (124.72)	.88
HCC	**-0.16 (*0.05*)**	**-2.97(179.83)**	**.003****
Avg. negative affect	**0.59 (*0.07*)**	**8.49 (81.79)**	**<.001*****
Avg. BMI	0.01 (*0.06*)	0.18 (28.75)	.86
Age	<0.01 (*0.02*)	0.11 (128.72)	.91
Avg. hours of sleep/night	<0.01 (*0.05*)	0.07 (177.99)	.95
Income	0.03 (*0.06*)	0.51 (119.39)	.61

Note: Avg. = average; BMI = body mass index; HCC = hair cortisol concentration.

All daily variables were averaged prior to being included in the above models. BMI was averaged across the 3 study assessments prior to being included in the above models.

** $p<.01$; *** Bonferroni corrected $p<.002$

APPENDIX C

Supplemental Analyses

Supplemental Table 1

Comparing descriptive information between participants who did and did not provide a hair sample.

	HCC sample (*N = 234*)	**Non-HCC sample (*N = 243*)**	**t-test results**	***p***
Variable	**Mean (*SD*)**	**Mean (*SD*)**	**t(*df*) = F**	
Daily variables- 49-day Avg[a]				
Avg. emotional eating	0.37 (*0.47*)	0.31 (*0.36*)	-1.60(*475*) = 8.00	.11
Avg. stress impact	30.67 (*27.01*)	28.42 (*27.71*)	-0.90(*475*) = 0.16	.37
Avg. stress frequency	11.81 (*7.94*)	10.39 (*7.06*)	-2.07(*475*) = 2.50	.04
Avg. negative affect	15.08 (*3.68*)	15.43 (*4.08*)	0.99(*475*) = 0.13	.33
Non-Daily variables				
Major life stress in the last 12 months	130.46 (*107.38*)	142.69 (*108.88*)	1.22(*460*) = 0.29	.23
Major life stress over 49 days	62.63 (*70.08*)	71.92 (*73.19*)	1.39(*460*) = 0.99	.16
Major life stress across the lifetime	109.44 (*168.48*)	194.51 (*250.94*)	4.28(*460*) = 27.94	**<.001**
BMI (kg/m^2)[b]	24.63 (*5.15*)	24.63 (*5.70*)	0.00(*399*) = 0.56	1.00
Ethnicity/Race/Income	**Percent (N)**	**Percent (N)**	**Z-test of proportions**	
Ethnicity				
Hispanic or Latinx	2.6% (6)	4.9% (12)	-0.24	.81
Non-Hispanic or Latinx	97.4% (229)	95.1% (231)	1.35	.18
Race (%, *n*)				
White	91.1% (214)	87.7% (213)	1.13	.26
Black or African American	3.4% (8)	6.6% (16)	-0.34	.73
Asian	0.4% (1)	2.1% (5)	-0.14	.89
More than one race	5.1% (12)	3.7% (9)	0.16	.87
Income				
Under $20,000	2.1% (5)	2.1% (5)	0	1.00
$20,000-$40,000	3.1% (7)	4.1% (10)	-0.11	.91

Supplemental Table 1 (cont'd)

$40,000-$60,000	11.1% (25)	11.1% (27)	0	1.00
$60,000-$100,000	27.1% (61)	30.4% (72)	-0.37	.71
Over $100,000	56.4% (127)	51.9% (123)	0.71	.48

Note: Avg. = average; BMI = body mass index; HCC = hair cortisol concentration.

[a]These values are unstandardized (i.e., non-z-scored) means and standard deviations (SD) across the 49-day collection period that index the average level of study variables on any given day.

[b]This value is the unstandardized mean and SD for BMI that was measured at the beginning, middle, and end of the 49-day collection period.

Supplemental Table 2

Results from the between-subject MLMs examining interactive effects of the daily stress variables with major life stress over the 49-day study period and across the lifetime on daily levels of emotional eating, including the covariates (N = 477).

MAIN EFFECTS			
Predictors	**β (*SD*)**	**t (df)**	***p***
Intercept	0.03 (*0.36*)	0.10 (203.40)	.93
Major life stress <u>over 49 days</u>	-0.05 (*0.04*)	-1.29 (351.50)	.20
Avg. negative affect	**0.53 (*0.05*)**	**9.75 (163.24)**	**<.001*** **
Avg. BMI	0.05 (*0.04*)	1.14 (288.41)	.26
Age	<0.01 (*0.02*)	-0.19 (206.44)	.85
Income	0.01 (*0.05*)	0.26 (57.07)	.80
Intercept	0.04 (*0.36*)	0.11 (205.78)	.91
Major life stress <u>across the lifetime</u>	-0.06 (*0.04*)	-1.32 (357.70)	.19
Avg. negative affect	**0.53 (*0.05*)**	**9.74 (160.41)**	**<.001*** **
Avg. BMI	0.05 (*0.04*)	1.23 (290.52)	.22
Age	<0.01 (*0.02*)	-0.21 (208.84)	.84
Income	0.01 (*0.05*)	0.28 (59.40)	.78
INTERACTION EFFECTS			
Predictors	**β (*SD*)**	**t (df)**	***p***
Intercept	0.04 (*0.35*)	0.12 (201.43)	.90
Avg. stress impact	**0.36 (*0.06*)**	**6.17 (346.42)**	**<.001*** **
Major life stress	-0.08 (*0.04*)	-1.82 (365.03)	.07
Avg. stress impact X major life stress <u>over 49 days</u>	0.08 (*0.04*)	1.98 (345.15)	.05
Avg. negative affect	**0.33 (*0.06*)**	**5.59 (345.13)**	**<.001*** **
Avg. BMI	0.06 (*0.05*)	1.19 (67.95)	.24
Age	<0.01 (*0.02*)	-0.18 (205.34)	.86
Income	0.03 (*0.05*)	0.68 (80.22)	.50
Intercept	-0.01 (*0.35*)	-0.04 (204.77)	.97
Avg. stress impact	**0.35 (*0.06*)**	**5.60 (154.50)**	**<.001*** **
Major life stress	-0.08 (*0.04*)	-2.07 (345.29)	.04
Avg. stress impact X major life stress <u>across the lifetime</u>	-0.04 (*0.05*)	-0.91 (196.32)	.37

Supplemental Table 2 (cont'd)

Avg. negative affect	**0.27 (*0.07*)**	**4.11 (211.79)**	**<.001*** **
Avg. BMI	0.03 (*0.04*)	0.70 (294.34)	.48
Age	<0.01 (*0.02*)	-0.16 (208.03)	.87
Income	<0.01 (*0.04*)	0.03 (202.41)	.98

Note: Avg. = average; BMI = body mass index; Stress impact = daily stress impact. All daily variables were averaged prior to being included in the above models.

*** Bonferroni corrected $p<.002$

Supplemental Table 3

Results from the within-subject MLMs examining interactive effects of the same-day and time-lagged daily stress variables with major life stress over the 49-day study period and across the lifetime on daily levels of emotional eating, including the covariates (N = 477).

SAME-DAY DAILY STRESS IMPACT			
INTERACTION EFFECTS			
Predictors	**β (*SD*)**	**t (df)**	***p***
Intercept	0.01 (*0.01*)	0.80 (2879.56)	.43
Same-day stress impact	**0.15 (*0.01*)**	**9.98 (320.06)**	**<.001*** **
Major life stress over 49 days	<0.01 (*<0.01*)	-0.04 (2870.98)	.97
Same-day stress impact X major life stress over 49 days	<0.01 (*0.01*)	-0.29 (320.64)	.77
Negative affect	**0.13 (*0.02*)**	**8.83 (406.46)**	**<.001*** **
BMI	0.01 (*0.01*)	0.79 (4637.11)	.43
Intercept	0.01 (*0.01*)	0.83 (2872.77)	.41
Same-day stress impact	**0.15 (*0.01*)**	**9.97 (315.94)**	**<.001*** **
Major life stress across the lifetime	<0.01 (*0.01*)	0.38 (2884.66)	.70
Same-day stress impact X major life stress across the lifetime	<0.01 (*0.01*)	0.27 (305.11)	.79
Negative affect	**0.13 (*0.02*)**	**8.83 (406.61)**	**<.001*** **
BMI	0.01 (*0.01*)	0.79 (4638.27)	.43
DAILY STRESS IMPACT FROM ONE DAY AGO			
INTERACTION EFFECTS			
Predictors	**β (*SD*)**	**t (df)**	***p***
Intercept	-0.02 (*0.01*)	-2.62 (3083.42)	.01
Same-day stress impact	**0.13 (*0.02*)**	**8.39 (361.99)**	**<.001*** **
Stress impact from one day ago	**0.04 (*0.01*)**	**2.82 (335.40)**	**.005** **
Major life stress over 49 days	<0.01 (*0.01*)	0.29 (3075.63)	.78

Supplemental Table 3 (cont'd)

Same-day stress impact X major life stress over 49 days	0.01 (*0.02*)	0.41 (346.57)	.68
Stress impact from one day ago X major life stress over 49 days	-0.02 (*0.01*)	-1.50 (357.20)	.14
Negative affect	**0.13 (*0.01*)**	**8.78 (416.60)**	**<.001*** **
BMI	<0.01 (*0.01*)	0.51 (4521.33)	.61
Intercept	-0.02 (*0.01*)	-2.63 (3073.31)	.01
Same-day stress impact	**0.13 (*0.02*)**	**8.43 (356.87)**	**<.001*** **
Stress impact from one day ago	**0.04 (*0.01*)**	**2.78 (332.70)**	**.006** **
Major life stress across the lifetime	<0.01 (*0.01*)	0.02 (3097.52)	.99
Same-day stress impact X major life stress across the lifetime	<0.01 (*0.02*)	0.16 (342.98)	.87
Stress impact from one day ago X major life stress across the lifetime	0.01 (*0.01*)	0.70 (338.34)	.48
Negative affect	0.13 (*0.01*)	8.77 (342.98)	.87
BMI	<0.01 (*0.01*)	0.51 (4521.98)	.61

DAILY STRESS IMPACT FROM TWO DAYS AGO

INTERACTION EFFECTS

Predictors	**β (*SD*)**	**t (df)**	***p***
Intercept	**-0.03 (*0.01*)**	**-3.42 (2940.67)**	**.001** **
Same-day stress impact	**0.13 (*0.02*)**	**8.21 (359.49)**	**<.001*** **
Stress impact from two days ago	0.03 (*0.01*)	2.38 (315.26)	.02
Major life stress over 49 days	<0.01 (*0.01*)	0.03 (2932.01)	.98
Same-day stress impact X major life stress over 49 days	<0.01 (*0.01*)	-0.13 (349.06)	.90
Stress impact from two days ago X major life stress over 49 days	0.01 (*0.01*)	0.55 (332.59)	.58
Negative affect	**0.13 (*0.02*)**	**8.50 (417.74)**	**<.001*** **
BMI	0.01 (*0.01*)	0.74 (4223.28)	.46
Intercept	**-0.03 (*0.01*)**	**-3.41 (2930.84)**	**.001** **

Supplemental Table 3 (cont'd)

Same-day stress impact	**0.13 (*0.02*)**	**8.23 (354.24)**	**<.001*** **
Stress impact from two days ago	0.03 (*0.01*)	2.43 (308.35)	.02
Major life stress across the lifetime	<0.01 (*0.01*)	0.05 (2955.46)	.96
Same-day stress impact X major life stress across the lifetime	<0.01 (*0.02*)	0.15 (344.43)	.88
Stress impact from two days ago X major life stress across the lifetime	0.01 (*0.01*)	0.97 (312.38)	.33
Negative affect	**0.13 (*0.02*)**	**8.50 (417.67)**	**<.001*** **
BMI	0.01 (*0.01*)	0.72 (4225.50)	.47

Note: BMI = body mass index; Stress impact = daily stress impact.

** $p<.01$; *** Bonferroni corrected $p<.002$

Supplemental Table 4

Between-subject Pearson correlations for average daily stress, major life stress, emotional eating, and covariates (N = 477).

	1.	2.	3.	4.	5.	6.	7.
Daily Variables							
1. Avg. emotional eating	1.00	**0.56*** **	**0.54*** **	0.07	0.05	-0.10*	0.10
2. Avg. stress impact	--	1.00	**0.67*** **	**0.33*** **	0.03	-0.12*	-0.02
3. Avg. negative affect	--	--	1.00	**0.28*** **	-0.02	**-0.24*** **	-0.04
Non-Daily Variables							
4. Avg. Major life stress in last 12 months	--	--	--	1.00	0.09	-0.05	**-0.18*** **
5. Avg. BMI	--	--	--	--	1.00	**0.31*** **	-0.09
6. Age	--	--	--	--	--	1.00	-0.10*
7. Income	--	--	--	--	--	--	1.00

Note: Avg. = average; BMI = body mass index; Stress impact = daily stress impact

All daily variables were averaged prior to being included in the above models. BMI was averaged across the 3 study assessments before being included in the above models.

*p <.05; ***Bonferroni corrected p<.002

Supplemental Table 5

Within-subject Pearson correlations for same-day and time-lagged daily stress impact, daily emotional eating, average major life stress, and covariates (N = 477).

Variables	1.	2.	3.	4.	5.
Same-day daily variables					
1. Emotional eating	1.00	**0.22*** **	**0.18*** **	**0.06*** **	**0.05*** **
2. Same-day stress impact	--	1.00	**0.46*** **	**0.13*** **	**0.10*** **
3. Negative affect	--	--	1.00	**0.07*** **	**0.05*** **
Time-lagged daily stress variables					
4. Stress impact 1 day ago	--	--	--	1.00	**0.79*** **
5. Stress impact 2 days ago	--	--	--	--	1.00

Note: All variables were averaged prior to being included in analyses.

***Bonferroni corrected $p<.002$.

Supplemental Table 6

Pearson correlations for hair cortisol concentration (HCC), emotional eating, and covariates in the full sample of HCC participants (N = 234) and subsample of HCC participants without confounding factors for HCC (N = 220).

Full sample (N = 234)	1.	2.	3.	4.	5.	6.	7.	8.	9.
1. HCC	1.00	-0.13	0.02	0.07	0.06	-0.05	0.05	<0.01	0.09
2. Avg. emotional eating	--	1.00	**0.58*** **	0.11	**0.62*** **	0.10	-0.12	0.03	<0.01
3. Avg. stress impact	--	--	1.00	**0.32*** **	**0.75*** **	0.13*	-0.14*	-0.07	-0.01
4. Major life stress in the last 12 months	--	--	--	1.00	**0.22** **	0.16*	0.13*	<0.01	0.09
5. Avg. negative affect	--	--	--	--	1.00	0.08	**-0.24*** **	<0.01	0.01
6. Avg. BMI	--	--	--	--	--	1.00	**0.28*** **	-0.09	-0.12
7. Age	--	--	--	--	--	--	1.00	-0.14*	**-0.26** **
8. Avg. hours of sleep/night	--	--	--	--	--	--	--	1.00	0.11
9. Income	--	--	--	--	--	--	--	--	1.00
Subsample of women without confounding factors for HCC (N = 220)	1.	2.	3.	4.	5.	6.	7.	8.	9.

Supplemental Table 6 (cont'd)

1.	HCC	1.00	-0.15*	0.01	0.03	0.06	-0.06	0.04	<0.01	0.10
2.	Avg. emotional eating	--	1.00	**0.58***	0.02	**0.62***	0.08	-0.15*	0.03	0.01
3.	Avg. stress impact	--	--	1.00	**0.26***	**0.76***	0.13	-0.15*	-0.08	-0.01
4.	Major life stress in the last 12 months	--	--	--	1.00	0.10	0.12	0.05	0.13	0.05
5.	Avg. negative affect	--	--	--	--	1.00	0.08	**-0.26***	<0.01	0.01
6.	Avg. BMI	--	--	--	--	--	1.00	**0.27***	-0.09	-0.11
7.	Age	--	--	--	--	--	--	1.00	-0.13	**-0.24****
8.	Avg. hours of sleep/night	--	--	--	--	--	--	--	1.00	-0.13
9.	Income	--	--	--	--	--	--	--	--	1.00

Note: Avg. = average; BMI = body mass index; HCC = hair cortisol concentration.

All daily variables were averaged prior to being included in the above correlations.

BMI was averaged across the 3 study assessments prior to being included in the above correlations.

*p <.05; **p <.01; *** Bonferroni corrected p<.002.

Supplemental Table 7

Results from the post-hoc within-subject MLMs examining predictive effects of emotional eating from one and two days ago on daily stress impact, including the covariates (N = 477).

EMOTIONAL EATING FROM 1 DAY AGO			
Predictors	**β (*SD*)**	**t (df)**	***p***
Intercept	-0.02 (*0.01*)	-2.58 (2987.48)	.01
Same-day emotional eating	**0.09 (*0.01*)**	**8.73 (288.74)**	**<.001*** **
Emotional eating from one day ago	**0.03 (*0.01*)**	**3.92 (263.40)**	**<.001*** **
Avg. negative affect	**0.42 (*0.01*)**	**35.22 (292.04)**	**<.001*** **
Avg. BMI	0.01 (*0.01*)	0.83 (273.23)	.41
EMOTIONAL EATING FROM 2 DAYS AGO			
Predictors	**β (*SD*)**	**t (df)**	***p***
Intercept	**-0.03 (*0.01*)**	**-4.08 (2861.87)**	**<.001*** **
Same-day emotional eating	**0.09 (*0.01*)**	**7.96 (284.70)**	**<.001*** **
Emotional eating from two days ago	0.02 (*0.01*)	2.39 (261.92)	.02
Avg. negative affect	**0.42 (*0.01*)**	**33.82 (296.87)**	**<.001*** **
Avg. BMI	0.02 (*0.01*)	1.57 (272.89)	.12

Note: Avg. = average; BMI = body mass index. All daily variables were averaged prior to being included in the above models.

*** Bonferroni corrected $p<.002$

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