Adult adiposity linked to relationship hostility for low cortisol reactors

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Author Note: SPSS syntax for all analyses can be found at <https://osf.io/zq6tr/>. Data from this study have been published elsewhere; however, none of the findings published elsewhere are related to adult adiposity, cortisol reactivity, or snacking behavior. This research is available upon request.

Abstract

Past research on the relation between hostility in intimate relationships and adiposity has yielded mixed findings. The present study investigated whether the association between relationship hostility and adiposity is moderated by people's biological reactions to couple conflict. Cohabiting adult couples ($N = 117$ couples) engaged in two conflict interactions, before and after which salivary cortisol levels were measured. Results revealed an association between relationship hostility and adiposity, but this association was concentrated among people with relatively low levels of cortisol reactivity to couple conflict. Results are interpreted in light of research demonstrating that cortisol reactivity can become blunted over time in response to repeated stressors. These results provide precision to etiological models of obesity by identifying cortisol reactivity as a factor that moderates the association between relationship hostility and adiposity.

Key words: aggression, conflict, dyadic/couple data, marriage and close relationships, obesity

Adult Adiposity Linked to Relationship Hostility for Low Cortisol Reactors

The prevalence of clinically significant levels of body fat or adiposity—a body mass index (BMI) ≥ 30 is considered obese (Ogden, Carroll, Kit, & Flegal, 2014)—among adults is a significant health concern. Approximately 13% of adults in the world are obese (World Health Organization, 2016), with this rate notably higher in the United States (34.9%; Ogden et al., 2014). The costs of obesity in the U.S., both financially (approximately \$150 billion annually; Finkelstein, Trogdon, Cohen, & Dietz, 2009) and in terms of human life (an estimated 1 in 5 deaths; Masters et al., 2013), are dramatic and have resulted in the recognition of obesity as a major public health issue (U.S. Department of Health and Human Services, 2001).

Scholars agree that numerous factors—such as genetics (Albuquerque, Stice, Rodriguez-Lopez, Manco, & Nobrega, 2015), physical activity (Conn, Hafdahl, Phillips, Ruppar, & Chase, 2014), and diet (Swinburn, Caterson, Seidell, & James, 2004)—contribute to adiposity. In addition, factors related to social environments, such as chronic social stress (Scott, Melhorn, & Sakai, 2012), are increasingly implicated. Among social factors, those embedded in intimate relationships have gained recent attention, given their documented relations with other health outcomes (Kiecolt-Glaser & Newton, 2001; Robles, Slatcher, Trombello, & McGinn, 2014) and the commonality of intimate relationships in the lives of adults (U.S. Census Bureau, 2014).

In the present investigation, we investigate the role of relationship hostility, a class of behaviors within intimate relationships that includes both physical (e.g., hitting; slapping) and emotional (e.g., humiliation; withholding money or identification) forms of aggression or intimate partner violence (IPV) that are directed at one's partner, as well as "garden variety" noxious behaviors (e.g., yelling). Relationship hostility is both common (Slep & O'Leary, 2005) and impactful. The experience of hostility within relationships is associated with poor physical

(e.g., cardiovascular disease; Breiding, Black, & Ryan, 2008) and psychological health outcomes (e.g., depression; Choi & Marks, 2008). Additional factors embedded in intimate relationships, such as relationship satisfaction and distress, are also associated with adiposity and other health outcomes (e.g., Whisman, Uebelacker, & Settles, 2010); however, in this research, we focus specifically on the role of hostility.

To date, the literature on the relation between relationship hostility and adiposity has yielded mixed findings. Some studies indicate that there is a positive relation (e.g., Davies, Lehman, Perry, & McCall-Hosenfeld, 2016), while others have found no relation (e.g., Breiding et al., 2008; Dichter, Cerulli, & Bossarte, 2011) or even a negative relation (e.g., Ferreira et al., 2015). Such disparate findings may occur because relationship hostility does not have the same impact on adiposity for all individuals. The identification of factors that moderate the association of hostility and adiposity would help increase the precision of etiological models of adiposity. Biological reactivity to stress may be one such moderator.

The association between relationship hostility and adiposity may be stronger for those who are more biologically reactive to partner conflict. This hypothesis is based on the differential susceptibility to environment model of Boyce, Ellis, and colleagues (Boyce, 2016; Boyce & Ellis, 2005). According to this model, neurobiological susceptibility to the environment alters the impact of environments on physical and psychological health. The more biologically sensitive individuals are to their context—marked by sympathetic nervous system (SNS) or hypothalamic-pituitary-adrenal (HPA) reactivity to stressors—the more they are thought to be influenced by those environments. For individuals who are more sensitive or reactive to their context, supportive environments are thought to lead to positive outcomes, while adverse environments yield negative ones. Environmental factors are thought to have less impact on

individuals who are less biologically stress-reactive. Although most research on this phenomenon has been conducted in children (for a review, see Boyce, 2016), adult research has begun to yield supportive findings. For example, Lorber, Erlanger, and Slep (2013) found that males' IPV more strongly predicted women's affective functioning and alcohol problems in women with greater cardiovascular reactivity to laboratory stressors.

If relationship hostility negatively affects the adiposity of those who are most biologically reactive to couple conflict, what mechanisms might be involved? One likely mediator is eating behavior—in particular, high-calorie snacking. Experiencing hostility within one's relationship can increase perceived stress, anger, and depression (Lorber et al., 2013; Vaeth, Ramisetty-Mikler, & Caetano, 2010), and such negative affect and associated physiological changes, such as elevations in cortisol, may in turn cause increases in eating in an attempt to downregulate that negative affect (Epel, Lapidus, McEwen, & Brownell, 2001; Groesz et al., 2012). An increase in emotional eating is especially problematic for weight gain because it is often marked by an increase in the consumption of snacks (versus meals) and comfort foods: those high in calories, fat, and/or sugar (Epel et al., 2001; Oliver & Wardle, 1999). Therefore, there is reason to suspect that those who react strongly to couple conflict will also exhibit increases in high-calorie snacking. Additionally, because snacking is more likely than planned meals to be influenced by stress (Cleobury & Tapper, 2014; Oliver & Wardle, 1999), relationship hostility is more likely to impact obesity through snacking behavior than through food consumption during meals. To our knowledge, dietary behaviors have not yet been studied as mechanisms linking relationship hostility to adiposity.

The Present Research

We aimed to clarify the relation between relationship hostility and adiposity by extending previous research in three fundamental ways. First, in the search for factors that amplify or attenuate the relationship hostility—adiposity relation, we examined how it might be moderated by people's biological reactions to couple conflict. Specifically, we were interested in biological reactions that occur when people perceive events as negative or stressful; the type of responding one might expect after an interpersonal stressor, such as couple conflict. To capture this type of biological sensitivity, we measured cortisol—a product of HPA axis activation that is a glucocorticoid and helps the body mobilize energy to cope with stressors (Kovacs & Ojeda, 2012). Changes in cortisol are typically observed in response to situations that are perceived as stressful and that elicit negative affect and social threat (Lovallo & Thomas, 2000), and hundreds of studies have demonstrated acute changes in cortisol from exposure to short-term social stressors (Dickerson & Kemeny, 2004). Second, we tested a possible mediator of the association of relationship hostility and adiposity by examining eating behavior likely to lead to weight gain, specifically high-calorie, non-nutrient dense snacking. Finally, we explored the moderating role of gender, extending the vast majority of research in this domain which utilizes samples of women only.

We studied a community sample of cohabiting couples to test the hypothesis that relationship hostility is positively associated with adiposity (Hypothesis 1) and that this association is mediated by high-calorie, non-nutrient dense snacking (“obesogenic snacking”; Hypothesis 2). We further hypothesized that the relationship hostility—adiposity association would be moderated by biological reactivity to couple conflict, as measured by a marker of HPA axis reactivity, salivary cortisol (Hypothesis 3). In addition to the moderational and mediational

hypotheses, we tested the hypothesis that the indirect effect of relationship hostility on adiposity via obesogenic snacking would be moderated by cortisol reactivity (Hypothesis 4; i.e., moderated mediation). We explored gender differences in all of these associations.

Method

Participants

Participants were 117 heterosexual married couples (male age: $M = 44.25$, $SD = 4.93$; female age: $M = 42.81$, $SD = 4.59$) living in the suburbs of a large city in the Northeastern United States. Female and male participants identified themselves, respectively, as White (96.2%, 94.6%), Black (2.8%, 2.7%), Asian (.90%, .90%), and multiracial (0%, 1.8%); 5.3% of females and 2.6% of males indicated they were Latino of any race. Their median family income was \$110,000. According to 2010 US Census data for the participants' county of residence, 80.8% of the population is White, 7.4% is Black, 3.4% is Asian, and 16.5% is Latino of any race; median family income is \$87,187.

The couples in this study participated in a previous study, for which they were recruited via telephone using random digit dialing of landline phones. A sampling firm provided an oversample of high-minority represented areas. To be included in the original study, couples must have been married or living together for at least one year; both partners read and spoke English. One member of the couple needed to be a biological parent to a child in the 4- to 8-year-old range, who was living with the parent. The original sample of 399 couples was invited via mail and telephone to participate in the present study. Couples in the present study comprised 29% of the original sample and did not significantly differ from the rest of the sample on demographic and family functioning measures (reported in Lorber, Slep, Heyman, Xu,

Dasanayake, & Wolff, 2014). All couples who indicated an interest in the study participated in the study.

Procedure

Couples came to the laboratory for a 2.5-hour session, starting between the hours of 8:00 am and 8:00 pm, with all procedures approved by the Institutional Review Boards at New York University and Stony Brook University. After providing consent, participants completed health questionnaires in separate rooms from their partners to ensure anonymity. Following the questionnaires, participants engaged in two video-recorded conflict interactions (described in detail below). Family aggression questionnaires, as well as additional questionnaires (e.g., on diet), were completed in separate rooms after the conflict interactions. Finally, participants' weight and height were measured. Participants completed additional procedures that are not the focus of the present paper and, thus, not reported on further here. Couples were compensated \$150 for their participation.

Measures

Adiposity. Participants' body mass index (BMI) was computed from height and weight measured in the laboratory session. Participants were asked to remove their shoes and heavy clothing before measurements were taken. BMI was calculated by dividing weight (in kilograms) by the squared value of height (in meters). Although BMI is an imperfect measure of adiposity because it does not discriminate between body fat and excess muscle or lean body mass, it is highly correlated with more direct measures of adiposity (Strain & Zumoff, 1992) and less expensive and invasive than other measures to obtain. For reference, 40.2% of the men and 30.8% of women in the present sample had BMIs exceeding 30, the most common threshold for

obesity; these rates are fairly comparable to the prevalence of obesity among American adults (Ogden et al., 2014).

Obesogenic snacking. To assess obesogenic snacking (i.e., snacking habits that are typically associated with weight gain), participants were asked to indicate the frequency with which they snack between meals (0 = *never*; 1 = *rarely*; 2 = *sometimes*; 3 = *often*; 4 = *always*) and to list up to three of their regular snacks. A trained research assistant then coded the snacks for their potential to lead to weight gain, placing foods into one of five categories, with higher numbers reflecting a greater potential to lead to weight gain: 1 = *low calorie, nutrient dense* (e.g., vegetables and fruits); 2 = *low calorie, non-nutrient dense* (e.g., rice cakes and pretzels); 3 = *medium calorie, nutrient dense* (e.g., low-fat or non-fat yogurt and oatmeal); 4 = *high calorie, nutrient dense* (e.g., peanuts, meat, and fish); 5 = *high calorie, non-nutrient dense* (e.g., cookies and white bread). Nutrient dense foods at the same calorie level are less likely to lead to weight gain than those that are non-nutrient dense because they are more filling and thus, less likely to be followed by hunger and the consumption of additional food (see Weigle et al., 2005). A second trained research assistant coded 27% of snack entries using the same method, and interrater agreement was excellent (92%; $\kappa = .90$). Entries were excluded from further analysis if there was not enough information for categorization (5.6% of total snacks listed). Using the categorizations provided by the first research assistant, we computed an average score for each participant, reflecting the potential of their regular snacks to lead to weight gain. We then multiplied this average by the frequency with which participants reported snacking to compute a measure of obesogenic snacking.

Relationship hostility.

Emotional and physical aggression. Participants completed the Partner Emotional (e.g., humiliation, withholding money or identification; 9 items) and Physical (e.g., grabbing, hitting, kicking; 14 items) Aggression subscales of the Family Maltreatment Measure (FM; Foran, Slep, Heyman, & United States Air Force Family Advocacy Research Program, 2011). Participants rated the frequency of their perpetration of aggressive actions toward their partners, as well as their victimization (physical aggression only) by their partners' actions, on a 6-point scale ranging from 0 = *never* to 5 = *more than 10 times* in the last 12 months. We calculated average physical and emotional aggression scores for analysis using the maximum of each participant's perpetration of (self-reported) and victimization by (partner-reported) physical aggression and victimization by emotional aggression. The associations (Spearman ρ) between participants' self-reported perpetration scores and partner-reported victimization scores were .49 for male and .53 for female physical aggression, $ps < .001$.

FM items are built to range from less to more extremely aggressive tactics, rather than sampling the aggression construct with a number of related items. Individuals who exhibit less extreme forms of aggression (e.g., slapping; insulting or swearing at) most often do not exhibit more extreme forms of aggression (e.g., choking; withholding money or identification), although such behaviors clearly all fall within the domains of physical and emotional aggression, respectively. Thus, Cronbach's α is not reported per Shortt, Capaldi, Kim, and Owen (2006).

Observed hostility. Prior to the two 7-minute conflict interactions, couples identified areas of conflict in their relationships on a questionnaire that listed common areas of relationship conflict. They were also allowed to write in topics of conflict. Participants indicated whether they had raised each issue with their partner in the last year. For each item that had been raised, they rated the importance of their partner making the indicated change (using a 6-point scale

ranging from 1 = *not important* to 6 = *very important*). For each partner, research assistants then identified the topic with the highest importance rating that had been discussed in the last year, selecting randomly among ties if necessary. Once a topic had been identified for each partner, participants were instructed to discuss each conflict for seven minutes in a manner they might at home. The order of the discussions involving female-identified and male-identified topics was counterbalanced.

Research assistants, who were blind to the study hypotheses, coded the two conflict interactions for observed hostility (e.g., angry affect, criticism, and combativeness) using the Rapid Marital Interaction Coding System (RMICS; Heyman, 2004). In over 20 studies, this coding system has been shown to have discriminative, convergent, predictive, and construct validity (see Heyman, 2004, for a review). Hostility and ten other behaviors that are not a present focus were coded as present/absent at each speaker turn. A randomly selected 25% of the interactions were coded by a master rater, and interrater agreement between the master rater and second coder across all 11 codes was good (95%; $\kappa = .61$). We calculated hostility scores as the percentage of each person's speaker turns that were scored with the hostility code and then averaged across the two interactions, Spearman $\rho = .29$ ($p = .002$) for male and $.23$ ($p = .017$) for female hostility.

Composite for analysis. We standardized participants' scores on the emotional aggression, physical aggression, and observed hostility measures and computed the average of the three scores to obtain a measure of hostility that reflects the overall degree to which partner-directed hostility existed within each relationship ($\alpha = .73$). We created this couple-level hostility measure because we anticipated that relationship hostility would be distressing to couples regardless of who perpetrated it (see Williams & Frieze, 2005). Furthermore, couple hostility is a

highly dyadic phenomenon (i.e., perpetration and victimization are highly correlated; Capaldi & Crosby, 1997; Lorber & O’Leary, 2012) and, in our data, the composite measures of hostility were highly correlated within couples, $r(117) = .73, p < .001$.

HPA reactivity. Each participant provided two saliva samples for a measure of cortisol reactivity to the conflict interactions. The baseline sample was taken after participants had given informed consent and completed a brief questionnaire (approximately 30 minutes after arrival at the lab) and prior to the two conflict interactions. The second sample was taken, on average, 20.77 minutes ($SD = 4.40$) following the start of the first conflict interaction and, on average, 3.66 minutes ($SD = 3.99$) following the end of the second conflict interaction. A stimulated saliva/passive drool methodology was used in which participants were first instructed to chew on a piece of Parafilm M(R). After 3 minutes, participants tilted their heads forward and passively drooled into 50ml collection tubes. Up to 5ml of the saliva was then pipetted into a storage tube that was immediately placed on ice and transferred to a -70°C freezer where it remained until it was shipped on dry ice to an external laboratory (The Behavioral Immunology and Endocrinology Laboratory of the University of Colorado at Denver Health Sciences Center) for assay using a Salimetrics (State College, PA) Enzyme Immunoassay kits for cortisol ($\mu\text{g/dl}$) analysis. The intra-assay coefficient of variance was less than 5%, and the inter-assay coefficient of variance was less than 10%. We treated four participants’ cortisol levels as missing because their levels at one or both time points were more than four SD s from the sample M (see Diamond, Hicks, & Otter-Henderson, 2008).

To ensure a representative sample of adults from a community population, we did not exclude participants on the basis of physical or psychological health conditions, as is sometimes done when measuring cortisol (see Dickerson, 2011). Instead, we obtained extensive self-reports

from participants regarding variables that may impact HPA activity (see Granger, Johnson, Szanton, Out, & Schumann, 2012): age (Otte et al., 2005), current illness (Dickerson, 2011), food consumption (in the past two hours, past four hours, and earlier that day; Holl, Fehm, Voigt, & Teller, 1984), medications taken (in the past two hours, earlier that day, yesterday, and in the past week; Granger, Hibbel, Fortunato, & Kapelewski, 2009), and days since last menstrual period (for premenopausal women only; Kirschbaum et al., 1999).

We then coded food and medications for their potential impact on cortisol levels and summed the impact of individual items to obtain quantitative measures of the degree to which participants' food and medications were likely to impact cortisol. To accomplish this, we conducted a literature review to determine the influence of foods and medications on cortisol. We coded for the impact of food on cortisol as 0 (no effect or cannot be sure due to conflicting results or lack of research; e.g., vegetables), .5 (very small excitatory effect; e.g., cheese), 1 (small excitatory effect; e.g., grains), 2 (medium excitatory effect; e.g., eggs), or 3 (large excitatory effect; e.g., meat). Medications were coded as -1 (inhibitory effect), 0 (no effect or cannot be sure due to conflicting results or lack of research), or 1 (excitatory effect). For example, for their effects on cortisol, corticosteroids were coded as -1, proton pump inhibitors as 0, and serotonin reuptake inhibitors as 1.

Next, we conducted individual regression analyses predicting baseline and post-conflict cortisol levels from the variables listed above, along with time of day and a quadratic term for time of day given diurnal variations in cortisol (Dickerson & Kemeny, 2004). Time of day, food eaten in the past four hours, food eaten earlier that day, and days since last menstruation had significant impacts on baseline cortisol, post-conflict cortisol, or both (see Table 1 for correlations between these variables and cortisol measurements). Given these results, we

regressed cortisol slopes (post-conflict levels minus baseline levels divided by the amount of time elapsed between measurements) on these variables. We then saved the residuals for use as the reactivity variables for analysis. All results are consistent when using a) residuals from a model where baseline and post-conflict cortisol levels are predicted only from a linear term for time of day, b) residuals from a model where we regressed cortisol difference scores (instead of slopes) on the influential variables, and c) residuals from a model where we regressed post-conflict cortisol on the influential variables and baseline cortisol.

Analytic Framework

Because participants were nested within couples, all analyses were conducted using Generalized Estimating Equations (GEE). GEE is ideal for these analyses because it allows adjustment for non-independence and is robust to violations of assumptions of normality (Wang, 2014), such as those found in positively skewed hostile behaviors and BMI. The level 1 predictors included obesogenic snacking, cortisol reactivity, and gender; the level 2 predictor is relationship hostility.

Because all couples were heterosexual, we conducted models where dyad members were treated as distinguishable by including the main effect of gender (coded as -1 = female and 1 = male) and interactions between all fixed effects and gender (Kenny, Kashy, & Cook, 2006). Except for gender, all predictors were centered at their means. When exploring interactions with gender, in our simple effects tests, we recoded the gender of interest to be zero in the analysis so that other effects in the model refer to that gender (see Aiken & West, 1991). Using this method, observations that have missing values of the dependent variable are not used in estimating the covariance parameters. We specified an exchangeable working correlation matrix for the

residuals. Rates of missing data are as follows: adiposity (2.6%), relationship hostility (0%), obesogenic snacking (1.3%), and cortisol reactivity (17.1%).

Results

Descriptive statistics for study variables are reported in Table 2.

Adiposity in Relation to Relationship Hostility

To test the association of adiposity with relationship hostility, we estimated a model in which BMI was regressed on relationship hostility. In contrast to Hypothesis 1, the association between relationship hostility and adiposity was nonsignificant ($b = 1.37$, $SE = 0.92$, Wald $\chi^2(1) = 2.22$, $p = .14$, 95% CI: -0.43, 3.16). We also tested a model in which BMI was regressed on relationship hostility, gender, and a Relationship Hostility $\times$ Gender interaction term. The association between relationship hostility and adiposity was not moderated by gender ($b = -0.15$, $SE = 0.56$, Wald $\chi^2(1) = 0.08$, $p = .78$, 95% CI: -1.24, 0.94).

Obesogenic Snacking as a Mediator of the Relationship Hostility—Adiposity Association

Given that relationship hostility and BMI were not significantly associated, there was no basis for examining mediators between relationship hostility and BMI. Thus, Hypothesis 2—that obesogenic snacking would mediate the relation between relationship hostility and BMI—was not supported. We further observed that obesogenic snacking was not predicted by relationship hostility in a model predicting obesogenic snacking from relationship hostility ($b = 0.14$, $SE = 0.21$, Wald $\chi^2(1) = 0.43$, $p = .51$, 95% CI: -0.27, 0.55). We also tested a model in which obesogenic snacking was regressed on relationship hostility, gender, and a Relationship Hostility $\times$ Gender interaction term. The association between relationship hostility and obesogenic snacking was not moderated by gender ($b = -0.15$, $SE = 0.23$, Wald $\chi^2(1) = 0.46$, $p = .50$, 95% CI: -0.60, 0.29). Moreover, obesogenic snacking did not predict BMI in a model predicting BMI

from obesogenic snacking ($b = 0.17$, $SE = 0.12$, Wald $\chi^2(1) = 2.07$, $p = .15$, 95% CI: -0.06, 0.40).

In a model predicting BMI from obesogenic snacking, gender, and an Obesogenic Snacking $\times$ Gender interaction term, the association between obesogenic snacking and BMI was not moderated by gender ($b = -0.11$, $SE = 0.10$, Wald $\chi^2(1) = 1.22$, $p = .27$, 95% CI: -0.29, 0.08).

HPA Reactivity as a Moderator of the Relationship Hostility—Adiposity Association

We regressed BMI on relationship hostility, cortisol reactivity, and a Relationship Hostility $\times$ Cortisol Reactivity interaction term to test Hypothesis 3. The Relationship Hostility $\times$ Cortisol Reactivity interaction was significant ($b = -3820.06$, $SE = 1127.94$, Wald $\chi^2(1) = 11.47$, $p = .001$, 95% CI: -6030.79, -1609.33). To examine whether this relation was moderated by gender, we regressed BMI on relationship hostility, cortisol reactivity, and gender, as well as Relationship Hostility $\times$ Gender, Cortisol Reactivity $\times$ Gender, Relationship Hostility $\times$ Cortisol Reactivity, and Relationship Hostility $\times$ Cortisol Reactivity $\times$ Gender interaction terms. There was a significant Relationship Hostility $\times$ Cortisol Reactivity $\times$ Gender interaction ($b = 1778.95$, $SE = 761.34$, Wald $\chi^2(1) = 5.46$, $p = .019$, 95% CI: 286.75, 3271.15). Follow-up tests, reported below, indicated that the Relationship Hostility $\times$ Cortisol Reactivity interaction was significant in women but not in men. However, the nature of the interaction was the opposite of what had been predicted: higher levels of relationship hostility were positively associated with adiposity for low cortisol reactors, but not high cortisol reactors.¹

Females. Results for females indicated that cortisol reactivity moderated the association between relationship hostility and BMI ($b = -5266.70$, $SE = 1161.91$, Wald $\chi^2(1) = 20.55$, $p < .001$, 95% CI: -7544.01, -2989.39). The relationship hostility—BMI association for females is plotted at ± 1 *SD* above/below the *M* of cortisol reactivity in the top panel of Figure 1. To explore this interaction further, we tested whether the relationship hostility—BMI association

was significant at the 10th through 90th percentiles of cortisol reactivity (Table 3). The association between relationship hostility and BMI became more positive at lower levels of cortisol reactivity. The positive relationship hostility—BMI association became significant at the 35th percentile of cortisol reactivity, which corresponds to a decrease in cortisol from baseline to post-interaction.

Males. Results for males indicated that cortisol reactivity did not moderate the relationship hostility-BMI association ($b = -1708.80$, $SE = 1133.18$, Wald $\chi^2(1) = 2.27$, $p = .13$, 95% CI: -3929.80, 512.19).

Moderated Mediation Involving HPA Reactivity and Obesogenic Snacking in Relation to Adiposity

As a follow-up to the significant Relationship Hostility $\times$ Cortisol Reactivity $\times$ Gender interaction in relation to BMI described above, we next evaluated whether the relation between relationship hostility and BMI was mediated by obesogenic snacking but was concentrated among women who exhibited lower levels of cortisol reactivity. We tested this possibility by regressing obesogenic snacking on relationship hostility, cortisol reactivity, and gender, as well as Relationship Hostility $\times$ Gender, Cortisol Reactivity $\times$ Gender, Relationship Hostility $\times$ Cortisol Reactivity, and Relationship Hostility $\times$ Cortisol Reactivity $\times$ Gender interaction terms. Given the fact that Relationship Hostility $\times$ Cortisol Reactivity $\times$ Gender interaction was not significant ($b = -125.48$, $SE = 497.91$, Wald $\chi^2(1) = 0.06$, $p = .80$, 95% CI: -1101.38, 850.41) in predicting obesogenic snacking (the mediator), moderated mediation (Hypothesis 4) was not supported.

Discussion

In the present study, we found support for the hypothesized association of relationship hostility and adiposity, but this association was conditional on HPA reactivity. The association of relationship hostility and BMI was concentrated among people with relatively low levels of cortisol reactivity to couple conflict, and there is some evidence – based on exploratory analyses – to suggest that this effect is stronger for women than for men. Contrary to our hypotheses, we did not find evidence that snacking behavior mediated the relationship hostility-adiposity relation.

Based on the differential susceptibility to environment model (Boyce, 2015), we predicted that the relationship hostility—adiposity relation would be stronger at higher levels of cortisol reactivity. Yet our findings suggest that it may be dampened cortisol reactivity to couple conflict that increases people's susceptibility to hostility within their relationships. These results may be interpreted based on research demonstrating that a dampening of cortisol reactivity to stress can occur over time in response to repeated stressors (e.g., Elzinga, et al., 2008; Gunnar & Vasquez, 2001). Although cortisol typically rises in response to short-term stressors (Dickerson & Kemeny, 2004), repeated experiences of stress over time can lead to blunted cortisol reactions. Blunted reactivity to repeated stress is a plausible consequence of experiencing relationship hostility due to the well-documented and substantial stability of hostility within relationships (e.g., Lorber & O'Leary, 2012; Shortt et al., 2006). That is, unless the relationship is new, hostility experienced in the present has very likely been experienced in the past as well. It is accordingly possible that people with highly hostile relationships who presently respond to couple conflict with blunted cortisol reactivity are those whose cortisol reactivity has become downregulated over time in response to the stress of persistent hostility. For these people, the

stress that current couple conflict generates may be greater given the long-term nature of it, and thus, it may have a stronger impact on eating behavior and adiposity.

Alternatively, it is possible that people who show high cortisol reactivity to couple conflict are better at adapting to this stressor. Given that cortisol is released to help the body cope with stressors (McEwen & Wingfield, 2003), high cortisol reactors may be successfully coping with the challenges of couple conflict. In contrast, low cortisol reactors may need to engage in unhealthy behaviors, like overeating or heavy drinking, to cope with the stressor. This hypothesis is supported by recent work showing that childhood maltreatment is associated with greater externalizing problems, but only among those low in cortisol reactivity to a stressor (Hagan, Rubinov, Mistler, & Luecken, 2014; see also Johnson and Gans, 2016).

Another possibility is that adiposity itself generates blunted cortisol reactivity. This may occur if people who are exposed to persistent hostility eat more comfort foods over time in response to the stress of this hostility (Dallman, Pecoraro, & la Fleur, 2005; Epel et al., 2001; Oliver & Wardle, 1999). These comfort foods may then lead to greater abdominal fat, which can cause blunted cortisol reactivity (Pecoraro, Reyes, Gomez, Bhargava, & Dallman, 2004; Tomiyama, Dallman, & Epel, 2011). Future research examining longitudinal changes in cortisol reactivity in response to relationship hostility, and its relations over time to adiposity, could more thoroughly test this possibility.

The present results suggest that relationship hostility for low cortisol reactors has stronger implications for women's adiposity than for men's. We view this effect with caution given the influence of outliers on this effect, as well as the fact that it was the result of exploratory analyses. If low cortisol reactivity to couple conflict in women exposed to highly hostile relationships is, as we suggest above, the result of experiencing repeated stress over time, our

results may be consistent with literature showing that couple conflict has a greater physiological impact on women than men (Kiecolt-Glaser & Newton, 2001), perhaps due to women's higher trait communion (versus agency; Skitka & Maslach, 1996) and self-representations emphasizing relational interdependence (Guimond, Chatard, Martinot, Crisp, & Redersdorff, 2006). If women consistently experience couple conflict as more stressful than men do, it may be that women's cortisol responding to couple conflict in highly hostile relationships is more likely to become blunted over time than men's. We note that this rationale is speculative and that future research examining the causes and consequences of blunted cortisol reactivity in response to couple conflict are needed to test these hypotheses.

The association of relationship hostility and adiposity among women who were low cortisol reactors (e.g., women who exhibited cortisol reactivity to partner conflict at one *SD* below the mean) was clinically significant. To illustrate via extrapolations from the regression results, their predicted BMI was 33.83—a value well into the clinical obesity range (> 30)—if they experienced relationship hostility at one *SD* above the mean. Therefore, high levels of relationship hostility for women who are low cortisol reactors raises the risk of frank obesity. Moreover, predicted BMI for low cortisol reactive women crossed the threshold for obesity when hostility within their relationship was only slightly greater (3 percent of a *SD*) than the mean, indicating that relationship hostility need not be extreme for it to be associated with obesity in low HPA-reactive women. Of course these findings are relative to the mean adiposity of the population, which varies by such factors as age, race, and ethnicity (Flegal, Carroll, Ogden, & Curtin, 2010). However, the present findings suggest that the combination of average to high levels of relationship hostility and below average cortisol reactivity to couple conflict places predominantly White suburban mothers in their 30s and 40s at elevated risk of obesity. We

further note that adiposity is a continuously distributed characteristic. The threshold for clinically significant obesity is debatable (Mascie-Taylor & Goto, 2007) and should probably be viewed as heuristic. Thus we do not wish to suggest that combinations of relationship hostility and cortisol reactivity that place women at risk of elevated BMIs that fall short of 30 are clinically irrelevant.

In addition, several limitations of our snacking measure should be noted. First, research has demonstrated that people high in adiposity tend to underreport consuming foods high in fats or carbohydrates, and such underreporting may have occurred in our sample (e.g., Tooze et al., 2004). Second, we theorized that individuals particularly sensitive to relationship hostility would attempt to cope with the stress of relationship hostility by eating more comfort foods. However, our measure of snacking did not capture foods specifically eaten in response to conflict or stress. It is possible that the type of snacks one eats regularly are not related to relationship hostility but that snacks eaten specifically in response to relationship hostility are. Future research should address this possibility by measuring the types of foods consumed specifically in response to couple conflict.

We note that our sample was composed predominantly of White, affluent adults in their 30s and 40s who owned landline phones, and the generalizability of our findings to other populations is not assured. We also note that our measure of adiposity—BMI—does not distinguish between central adiposity and more peripheral adiposity (Stevens, McClain, & Truesdale, 2008), which can be important in predicting health outcomes. However, BMI is still widely used to assess the risk of disease, given its high correlation with other measures of adiposity, ease of measurement, and test-retest reliability (Ellis, 2001; Strain & Zumoff, 1992). Additionally, this study was conducted at a wide range of times throughout the day, which is not ideal for cortisol collection. However, time of day did not significantly predict cortisol

responding to the couple conflict, suggesting that cortisol reactivity was not affected by being collected at an earlier versus later time of day. Finally, the tests of interactions involving gender were exploratory, thus it will be important to confirm the gender specificity of the results in future research.

Despite the limitations of this study, its findings lend increased precision to etiological models of adiposity, suggesting that relationship hostility may only play a role in contributing to adiposity for people who exhibit low HPA reactivity to relationship hostility. Future research examining the over-time progression and behavioral consequences of HPA responding to relationship hostility may help develop interventions to prevent particularly high levels of adiposity in these people.

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Footnotes

¹ Upon reviewers' requests, we conducted two additional sets of analyses. In the first, we examined the influence of outliers in this analysis given that there are several participants in this sample with very high BMIs (see Table 2 for descriptive statistics). We conducted the above analyses while filtering out participants with a BMI greater than two *SDs* above the mean (resulting in a BMI cutoff of 41.28). In an analysis predicting BMI from relationship hostility, cortisol reactivity, and a Relationship Hostility $\times$ Cortisol Reactivity interaction term, the Relationship Hostility $\times$ Cortisol Reactivity interaction followed the same pattern and was marginally significant ($b = -1921.17$, $SE = 1129.44$, Wald $\chi^2(1) = 2.89$, $p = .089$, 95% CI: -4134.83, 292.49). As in the main text, to examine whether this relation was moderated by gender, we regressed BMI on relationship hostility, cortisol reactivity, and gender, as well as Relationship Hostility $\times$ Gender, Cortisol Reactivity $\times$ Gender, Relationship Hostility $\times$ Cortisol Reactivity, and Relationship Hostility $\times$ Cortisol Reactivity $\times$ Gender interaction terms. The Relationship Hostility $\times$ Cortisol Reactivity $\times$ Gender interaction was no longer significant ($b = 439.75$, $SE = 1279.85$, Wald $\chi^2(1) = 0.12$, $p = .73$, 95% CI: -2068.71, 2948.21). Therefore, we view the gender difference with caution.

In the second set of analyses, we examined the possibility that a mediation model would better fit these data than a moderation model. To do this, we tested the following pathways: a) hostility to blunted reactivity and b) blunted reactivity to adiposity. The path from hostility to blunted reactivity was non-significant, ($b = -0.00006$, $SE = 0.00006$, Wald $\chi^2(1) = 0.80$, $p = .37$, 95% CI: -0.0002, 0.00007). The path from blunted reactivity to adiposity was also non-significant, ($b = -912.31$, $SE = 703.45$, Wald $\chi^2(1) = 1.68$, $p = .20$, 95% CI: -2291.05, 466.44). Therefore, we determined the moderation model to be a better fit of the data.

Table 1

Correlations between Cortisol Measurements and Variables Used for Cortisol Residuals

	Baseline Cortisol	Post-Conflict Cortisol
1. Linear term for time of day	-.40**	-.31**
2. Quadratic term for time of day	-.41**	-.31**
3. Food eaten in the past four hours	.17*	.16*
4. Food eaten earlier that day	-.11	.13 [†]
5. Days since last menstruation	-.30*	-.05

Note. [†] $p < .10$. * $p < .05$. ** $p < .001$.

Table 2

Descriptive Statistics for Study Variables by Gender

Variable	Females				Males			
	<i>M</i>	<i>SD</i>	Min.	Max.	<i>M</i>	<i>SD</i>	Min.	Max.
BMI	28.41	7.11	18.30	51.59	29.25	5.22	19.74	51.76
Obesogenic snacking	7.84	3.53	0.00	16.00	8.08	3.72	0.00	20.00
Couple-level relationship hostility	-0.01	0.74	-0.75	4.30	-0.01	0.74	-0.75	4.30
Person-level emotional aggression	0.28	0.43	0.00	2.22	0.23	0.42	0.00	2.11
Person-level physical aggression	0.06	0.25	0.00	1.93	0.04	0.17	0.00	1.29
Person-level observed hostility	6.60	7.64	0.00	35.94	4.66	7.35	0.00	34.85
Cortisol slopes	-0.0002	0.001	-0.003	0.002	-0.0003	0.001	-0.003	0.002

Note. BMI is body mass index. Cortisol reactivity values are presented in their raw form (i.e., without adjustments for time of day and food).

Table 3

Association of Relationship Hostility and BMI for Women at Varying Levels of Cortisol Reactivity

Cortisol reactivity			Effect of relationship hostility on BMI		
Percentile	Raw value	z-score	<i>b</i>	<i>SE</i>	<i>p</i>
10 th	-.0013	-1.39	6.63	1.30	< .001
20 th	-.0007	-0.55	4.00	1.03	< .001
30 th	-.0004	-0.21	2.42	1.00	0.016
40 th	-.0003	-0.05	1.60	1.03	0.122
50 th	-.0002	0.08	1.14	1.06	0.284
60 th	-.0001	0.24	0.31	1.14	0.783
70 th	.0000	0.40	-0.21	1.20	0.860
80 th	.0002	0.60	-0.74	1.27	0.561
90 th	.0006	1.13	-2.85	1.60	0.076

Figure 1. Associations between relationship hostility and BMI at low and high values of cortisol reactivity (+1/-1 SD from mean). $^{***}p < .001$.