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Adult Adiposity Linked to Relationship Hostility for Low-Cortisol Reactors

Katherine R. Thorson, Michael F. Lorber, Amy M. Smith Slep, and Richard E. Heyman
New York University

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.

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

The prevalence among adults of clinically significant levels of body fat, or adiposity, is a significant health concern (Ogden, Carroll, Kit, & Flegal, 2014). Approximately 13% of adults in the world are obese (i.e., they have a body-mass index [BMI] $\geq$ 30; 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 United States, both financially (approximately $150 billion annually; Finkelstein, Trogdon, Cohen, & Dietz, 2009) and in terms of human life (an estimated one in five deaths; Masters et al., 2013), are dramatic and have resulted in the recognition of obesity as a major public health issue (United States Department of Health and Human Services, Public Health Service, Office of the Surgeon General, 2001).

Scholars agree that numerous factors—such as genetics (Albuquerque, Stice, Rodríquez-López, Manco, & Nóbrega, 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), have increasingly been 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 study, we investigated 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) 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 association 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), and others have found no relation (e.g., Breiding et al., 2008; Dichter, Cerulli, & Bossarte, 2011) or 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 (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 contexts, marked by sympathetic nervous system (SNS)

Katherine R. Thorson, Department of Psychology, New York University; Michael F. Lorber, Amy M. Smith Slep, and Richard E. Heyman, Department of Cariology and Comprehensive Care, New York University.

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Correspondence concerning this article should be addressed to Katherine R. Thorson, Department of Psychology, New York University, 6 Washington Place, New York, NY 10003. E-mail: katherine.thorson@nyu.edu

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 contexts, supportive environments are thought to lead to positive outcomes, whereas 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 also begun to yield supportive findings. For example, Lorber, Erlanger, and Slep (2013) found that men's IPV more strongly predicted women's affective functioning and alcohol problems in those 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 (vs. 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 people who react strongly to couple conflict also exhibit increases in high-calorie snacking. In addition, 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 glucocorticoid product of HPA-axis activation that helps the body mobilize energy to cope with stressors (Kovacs & Ojeda, 2012). Changes in cortisol are often observed in response to situations that are perceived as stressful and that elicit negative affect and social threat (Lovallo & Thomas, 2000). 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 between relationship hostility and adiposity by examining eating behavior likely to lead to weight gain, specifically, high-calorie, nonnutrient-dense snacking. Finally, we explored the moderating role of gender, extending the vast majority of research in this domain, which has studied samples of women only.

We studied a community sample of cohabiting couples to test the hypotheses that relationship hostility is positively associated with adiposity (Hypothesis 1) and that this association is mediated by high-calorie, nonnutrient-dense snacking (i.e., 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 by way of obesogenic snacking would be moderated by cortisol reactivity (i.e., moderated mediation; Hypothesis 4). We explored gender differences in all of these associations.

Method

Participants

Participants were 117 heterosexual married couples (men's average age = 44.25 years, *SD* = 4.93; women's average age = 42.81 years, *SD* = 4.59) living in the suburbs of a large city in northeastern United States. Women and men 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 women and 2.6% of men indicated they were Latino of any race. Their median family income was $110,000. According to 2010 United States Census data for the participants' county of residence, 80.8% of the population was White, 7.4% Black, 3.4% Asian, and 16.5% Latino of any race; median family income was $87,187.

The couples in this study participated in an earlier study, for which they were recruited by 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 a year, with both partners able to read and speak 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 et al., 2014). All couples who indicated an interest participated in the study.

Procedure

Couples came to the laboratory for a 2.5-hr session, starting between 8:00 a.m. and 8:00 p.m., 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 and other 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 were not the

focus of the present paper, and, thus, they were not reported any further here. Couples were compensated $150 for their participation.

Measures

Adiposity. Participants' BMI was computed from height and weight measured during 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 snacked 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, nonnutrient-dense* (e.g., rice cakes and pretzels); 3 = *medium-calorie, nutrient-dense* (e.g., low-fat or nonfat yogurt and oatmeal); 4 = *high-calorie, nutrient-dense* (e.g., peanuts, meat, and fish); 5 = *high-calorie, nonnutrient-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 denser in nonnutrients because they are more filling, 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 aggression and physical aggression. Participants completed the Partner Emotional Aggression (e.g., humiliation, withholding money or identification; nine items) and Physical Aggression (e.g., grabbing, hitting, kicking; 14 items) subscales of the Family Maltreatment Measure (FM; Foran, Slep, & Heyman, 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 each participant's maximum perpetration of (self-reported) and victimization by (partner-reported) physical aggression and victimization by emotional aggression. The associations (Spearman's 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 (Foran et al., 2011) 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. Before the two 7-min 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 partners in the past year. For each item that had been raised, they rated the importance of their partner making the indicated changes (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 past year, selecting randomly among ties, if necessary. Once a topic had been identified for each partner, participants were instructed to discuss each conflict for 7 min as if they had been in their homes. The order of the discussions involving female-identified and male-identified topics was counterbalanced.

Research assistants 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 10 other behaviors that were 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 reflected 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, relationship 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 min after arrival at the lab) and prior to the two conflict interactions. The second sample was taken, on average, 20.77 min (SD = 4.40) following the start of the first conflict interaction and, on average, 3.66 min (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) Bemis Company, Wisconsin. After 3 min, participants tilted their heads forward and passively drooled into 50-ml collection tubes. Up to 5 ml 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 Salimetrics (State College, PA) enzyme immunoassay kits for cortisol (μg/dl) analysis. The intraassay coefficient of variance was less than 5%, and the interassay 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 4 SDs 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), namely age (Otte et al., 2005), current illness (Dickerson, 2011), food consumption (over the past 2 hours, 4 hours, and earlier that day; Holl, Fehm, Voigt, & Teller, 1984), medications taken (in the past 2 hours, earlier that day, yesterday, and over the past week; Granger, Hibel, Fortunato, & Kapelewski, 2009), and days since last menstrual period (for premenopausal women only; Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 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 postconflict 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 4 hours, food eaten earlier that day, and days since last menstruation had significant impacts on baseline cortisol, postconflict cortisol, or both (see Table 1 for correlations between these variables and cortisol measurements). Given these results, we regressed cortisol slopes (postconflict 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 we used (a) residuals from a model in which baseline and postconflict cortisol levels were predicted only from a linear term for time of day, (b) residuals from a model in which we regressed cortisol difference scores (instead of slopes) on the influential variables, and (c) residuals from a model in which we regressed postconflict cortisol on the influential variables and baseline cortisol.

Table 1
Correlations Between Cortisol Measurements and Variables Used for Cortisol Residuals

Variable	Baseline cortisol	Postconflict 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 4 hours	.17*	.16*
4. Food eaten earlier that day	−.11	.13†
5. Days since last menstruation	−.30*	−.05

† $p < .10$. * $p < .05$. ** $p < .001$.

Analytic Framework

Because participants were nested within couples, all analyses were conducted using generalized estimating equations (GEEs). GEEs are ideal for these analyses because they allow adjustment for nonindependence and are 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 was relationship hostility.

Because all couples were heterosexual, we conducted models in which 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 0 in the analysis so that other effects in the model referred to that gender (see Aiken & West, 1991). Observations that had missing values of the dependent variable were not used in estimating the covariance parameters. We specified an exchangeable working correlation matrix for the residuals. Rates of missing data were as follows: adiposity (2.6%), relationship hostility (0%), obesogenic snacking (1.3%), and cortisol reactivity (17.1%).

Results

Descriptive statistics for the 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

Table 2
Descriptive Statistics for Study Variables by Gender

Variable	Women				Men			
	M	*SD*	Min	Max	*M*	*SD*	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	.00	16.00	8.08	3.72	.00	20.00
Couple-level relationship hostility	−.01	.74	−.75	4.30	−.01	.74	−.75	4.30
Person-level emotional aggression	.28	.43	.00	2.22	.23	.42	.00	2.11
Person-level physical aggression	.06	.25	.00	1.93	.04	.17	.00	1.29
Person-level observed hostility	6.60	7.61	.00	35.94	4.66	7.35	.00	34.85
Cortisol slopes	−.0002	.001	−.003	.002	−.0003	.001	−.003	.002

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

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 × 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 $\chi^2(1) = 0.45$, $p = .50$, 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 × 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 × 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 × Cortisol Reactivity interaction term to test Hypothesis 3. The Relationship Hostility × 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 × Gender, Cortisol Reactivity × Gender, Relationship Hostility × Cortisol Reactivity, and Relationship Hostility × Cortisol Reactivity × Gender interaction terms. There was a significant Relationship Hostility × Cortisol Reactivity × 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 × 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]

Women. Results for women 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 women is plotted at ±1 *SD* above/below the mean 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 (see Table 3). The association between

[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 2 *SD*s 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 × Cortisol Reactivity interaction term, the Relationship Hostility × 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 × Gender, Cortisol Reactivity × Gender, Relationship Hostility × Cortisol Reactivity, and Relationship Hostility × Cortisol Reactivity × Gender interaction terms. The Relationship Hostility × Cortisol Reactivity × 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 cortisol reactivity and (b) cortisol reactivity to adiposity. The path from hostility to cortisol reactivity was nonsignificant, $b = -0.00006$, $SE = 0.00006$, Wald $\chi^2(1) = 0.80$, $p = .37$, 95% CI [−0.0002, 0.00007]. The path from cortisol reactivity to adiposity was also nonsignificant, $b = -912.31$, $SE = 703.45$, 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 to the data.

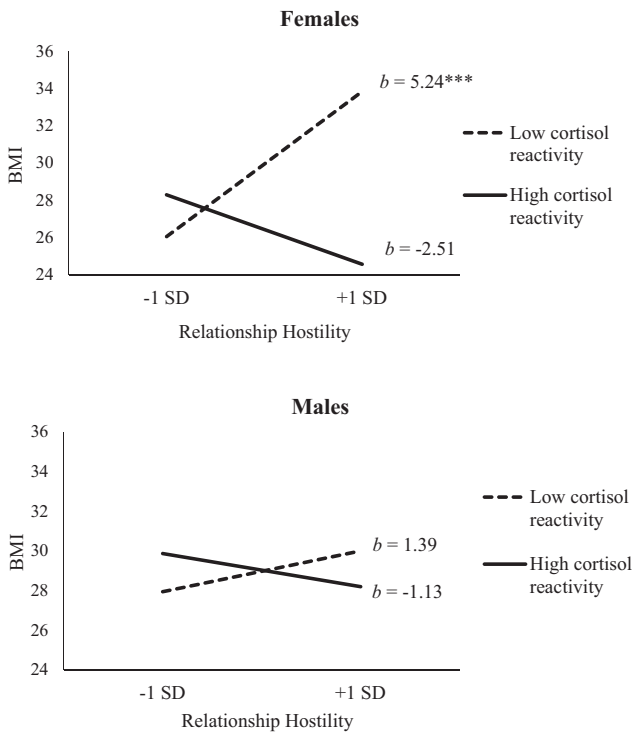

Figure 1. Associations between relationship hostility and BMI at low and high values of cortisol reactivity (±1 *SD* from the mean). *** $p < .001$.

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 postinteraction.

Men. Results for men 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 × Cortisol Reactivity × 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 × Gender, Cortisol Reactivity × Gender, Relationship Hostility × Cortisol Reactivity, and Relationship Hostility × Cortisol Reactivity × Gender interaction terms. Given that the Relationship Hostility × Cortisol Reactivity × 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 BMI (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 the environment model (Boyce, 2016), we predicted that the relationship hostility–adiposity relation would be stronger at higher levels of cortisol reactivity. Instead, 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, as hostility within relationships is substantially stable (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. Accordingly, it is possible that people with severely hostile relationships who presently respond to couple conflict with blunted cortisol reactivity have downregulated cortisol reactivity over time in response to the stress of persistent hostility. For these people, the stress that current couple conflict generates may be even greater, given the long-term nature of it, and thus, it may have a stronger impact on 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,

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	*b*	*SE*	*p*
10th	−.0013	−1.39	6.63	1.30	<.001
20th	−.0007	−.55	4.00	1.03	<.001
30th	−.0004	−.21	2.42	1.00	.016
40th	−.0003	−.05	1.60	1.03	.122
50th	−.0002	.08	1.14	1.06	.284
60th	−.0001	.24	.31	1.14	.783
70th	.0000	.40	−.21	1.20	.860
80th	.0002	.60	−.74	1.27	.561
90th	.0006	1.13	−2.85	1.60	.076

Note. BMI = body mass index.

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, Roubinov, Mistler, & Luecken, 2014; see also Johnson & Gans, 2016).

Another possibility is that adiposity itself generates blunted cortisol reactivity, which 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.

Our 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 a result of women's higher trait-communion (vs. trait-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 very 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 1 *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—if they experienced relationship hostility at 1 *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 relationships was only slightly greater (3% of an *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 lead to BMIs falling short of 30 are clinically irrelevant, only that such combinations place women at risk for elevated BMIs.

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 fat or carbohydrates, which 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 food. However, our measure of snacking did not capture foods specifically eaten in response to conflict or stress. It is possible that the type of snack one eats regularly is not related to relationship hostility, but that the type of snack eaten specifically in response to relationship hostility is. Future researchers should address this possibility by measuring the type of food 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 telephones, 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). In addition, 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 responses to couple conflict, suggesting that cortisol reactivity was not affected by collection time. Finally, the tests of interactions involving gender were exploratory, thus it is important that future researchers confirm the gender specificity of our results.

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 overtime 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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