

Novel Links Between Troubled Marriages and Appetite Regulation: Marital Distress, Ghrelin, and Diet Quality

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Abstract

Distressed marriages enhance risk for health problems; appetite dysregulation is one potential mechanistic pathway. Research suggests that ghrelin and leptin, appetite-relevant hormones connected to shorter and longer-term energy balance, may differentially affect people with a higher versus lower body mass index (BMI). During this double-blind randomized crossover study, both members of a couple ($N = 86$ participants) ate a standardized meal at the beginning of two visits. Observational recordings of a marital conflict assessed marital distress. Ghrelin and leptin were sampled premeal and postmeal at 2, 4, and 7 hr. Diet quality was measured using the USDA 24-Hour Multiple-Pass Approach. People in more distressed marriages had higher postmeal ghrelin (but not leptin) and a poorer quality diet than those in less distressed marriages, but only among participants with a lower BMI. These effects were consistent for both spouses. Ghrelin and diet quality may link marital distress to its corresponding negative health effects.

Keywords

marriage, diet, ghrelin, hunger, obesity, interpersonal relationships

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Being married often confers health benefits. For example, married people have lower premature all-cause mortality rates and fewer chronic health conditions than their non-married counterparts (Johnson, Backlund, Sorlie, & Loveless, 2000; Schoenborn, 2004). However, marriage is not universally health-beneficial. For instance, people in more distressed marriages reported worse overall health than those in less distressed marriages (Robles, Slatcher, Trombello, & McGinn, 2014). Distressed marriages also enhance risk for a variety of health problems such as cardiovascular disease, the metabolic syndrome, and premature mortality (Orth-Gomér et al., 2000; Robles et al., 2014; Troxel, Matthews, Gallo, & Kuller, 2005).

Appetite dysregulation is one potential pathway through which marital distress may influence health.

Humans have a basic need for close and caring relationships (Baumeister & Leary, 1995). This need to belong likely has evolutionary roots; the probability of survival was largely dependent on people reciprocally investing in each other's welfare. Ultimately, this evolved into a fundamental need to form close interpersonal relationships. Accordingly, marital distress threatens this basic need and should lead people to try and restore their sense of social inclusion, thus satisfying their need to belong (Pickett & Gardner, 2005). One novel possibility is

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that feeling hungry and eating in response to marital distress may allow people to feel socially reconnected. In fact, recent research demonstrated that eating comfort food caused people to spontaneously think about their relationships, and thinking about comfort food decreased loneliness (Troisi & Gabriel, 2011). Consequently, feeling hungry in response to marital distress may serve a social function, insofar as it provides people with an opportunity to satisfy their need to belong. People may also eat in response to marital distress in an attempt to alleviate negative affect (Adam & Epel, 2007).

Although feeling hungry in response to marital distress may have a social or emotional function, it likely also has negative longer-term health implications. A person's appetite and eating behavior are strongly linked to obesity (Arora & Anubhuti, 2006), which contributes to a host of medical problems, including cardiovascular disease, the metabolic syndrome, and premature mortality (Billington et al., 2000). Furthermore, a person's diet is linked to his or her risk for the metabolic syndrome and cardiovascular disease, among other health problems (Esposito et al., 2004; Hu & Willett, 2002).

Two appetite-relevant hormones, ghrelin and leptin, provide a window into hunger and eating behavior. Ghrelin, an appetite-stimulating hormone, is primarily secreted in the stomach (Klok, Jakobsdottir, & Drent, 2007). Ghrelin promotes food consumption and its production is largely dependent on food intake. For instance, people felt hungrier and consumed more food when they received a ghrelin injection compared with saline (Wren et al., 2001). Furthermore, ghrelin consistently increases before eating and drops after a meal (Cummings et al., 2001). In contrast, leptin, an appetite-suppressing hormone, inhibits food intake in cooperation with other peptides (Klok et al., 2007). Fat cells are a main source of leptin, and leptin production is limited under conditions of energy deprivation. The influence of both ghrelin and leptin on food intake is largely mediated by the hypothalamus. Whereas ghrelin has relatively immediate effects on a person's appetite, leptin is more important for longer-term energy balance (Klok et al., 2007).

Obesity may alter the links among marital distress, ghrelin, and leptin, one unexplored possibility. Obese people had lower fasting and postprandial (i.e., post-meal) ghrelin than those who were normal weight (Carlson, Turpin, Wiebke, Hunt, & Adams, 2009; Klok et al., 2007). In addition, ghrelin was linked to higher caloric intake and hedonic eating (i.e., eating for pleasure) among overweight people, but not among obese people (Buss et al., 2014). Leptin levels are also altered among obese people. For instance, obese people have higher serum leptin and greater leptin gene expression than normal weight people, potentially because adipose tissue is a primary source of leptin production (Considine

et al., 1996; Klok et al., 2007). Obesity is related to cellular leptin resistance, suggesting that obese people do not respond to leptin in the same way as their healthy weight counterparts (Myers, Leibel, Seeley, & Schwartz, 2010). Considered together, prior research suggests that both appetite-relevant hormones are dysregulated among obese individuals, either in terms of production, their effect on food consumption, or both. Accordingly, marital distress may be differentially linked to ghrelin and leptin among obese (body mass index; BMI ≥ 30.00) compared with nonobese individuals (BMI < 30.00).

Based on this rationale, we examined whether marital distress would be related to postprandial ghrelin and leptin for people with a lower BMI, but not among people with a higher BMI. We utilized observational data from two marital disagreements, providing behavioral marital distress data. Examining both ghrelin and leptin allowed us to test appetite-relevant hormones with more immediate versus longer-term implications for energy balance, respectively. Multiple 24-hr food recalls provided diet quality data, along with macro- and micro-nutrient information. We were particularly interested in the intake of sodium, saturated fat, and cholesterol because these are three components of a diet that may signify a less healthy eating pattern. In addition, higher levels of sodium, saturated fat, and cholesterol are related to cardiovascular disease incidence, and marital distress is a reliable risk factor for this disease (Hu & Willett, 2002; Orth-Gomér et al., 2000; Robles et al., 2014).

Method

Participants

Couples ($N = 43$ couples, 86 participants) were recruited through print and web-based announcements for a parent study about immune responses to fast-food-type-meals. Sample size was determined based on criteria for the parent study. Interested individuals completed an online screening questionnaire and an in-person screening visit. Couples were ineligible if they were not married or were married for less than 3 years. Individuals were ineligible if they or their partner had significant visual, auditory, or cognitive impairments, or any notable chronic health problems. We also excluded people if they or their partner smoked, abused alcohol/drugs, were diabetic (HbA1C > 6.5) or anemic, or used any prescription medication except birth control pills ($n = 5$) and levothyroxine ($n = 3$). A total of 350 interested individuals were excluded because either they or their spouse did not meet our stringent health criteria. Due to the nature of the parent study, we prioritized recruitment of heavier sedentary people and less happy couples. Participants fit our exercise criteria if they engaged in a maximum of 2 hr of

Table 1. Study Sample Characteristics

Characteristic	Category	Entire sample	Men	Women
		Number (%) or <i>M</i> (<i>SD</i>)	Number (%) or <i>M</i> (<i>SD</i>)	Number (%) or <i>M</i> (<i>SD</i>)
Race	White	70 (81)	35 (81)	35 (81)
	Black	16 (19)	8 (19)	8 (19)
	Other	0 (0)	0 (0)	0 (0)
Education	High school or below	12 (14)	7 (16)	5 (12)
	Some college/college graduate	37 (43)	19 (44)	18 (42)
	Graduate/professional training	37 (43)	17 (40)	20 (46)
BMI (kg/m ²)	Normal weight (<25.00)	10 (12)	3 (7)	7 (16)
	Overweight (25.00–29.99)	19 (22)	10 (23)	9 (21)
	Obese (≥30.00)	57 (66)	30 (70)	27 (63)
BMI (kg/m ²)	N/A	32.07 (5.83)	31.96 (5.06)	32.17 (6.58)
Age, years	N/A	38.22 (8.18)	39.25 (9.17)	37.19 (7.00)

Note: *N* = 43 couples, 86 individuals. Percentages reflect the proportion of participants within their respective group.

vigorous activity per week for BMI < 24.99 (normal weight) and 5 hr per week for BMI > 25 (overweight or obese). Participants' average age was 38.22 years (*SD* = 8.18, range = 24–61), and they were primarily White (81%). All couples were married, and the average marriage duration was 11.49 years (*SD* = 6.64). Additional sample characteristics for the entire sample and broken down by sex are listed in Table 1.

Study procedure

Participants completed two full-day study visits at the Clinical Research Center (CRC), a hospital research unit. During this double-blind randomized crossover study, couples ate a high saturated fat meal at the beginning of one visit and a high oleic sunflower oil meal at the beginning of the other. These meals were chosen due to the nature of the parent study. Visits were spaced 1 to 25 weeks apart, and the meal order was randomized between visits.

Couples were told to avoid alcohol use within 1 day prior and strenuous physical activity within 2 days prior to both of their study visits. Participants were also instructed to stop taking aspirin, vitamins (except multivitamins), antioxidants, and any other dietary supplements for 7 days prior to each admission. On the day before each visit, participants received three standardized meals from the CRC's metabolic kitchen, reducing any variability associated with recent food intake.

At each admission, both members of a couple arrived at 7:30 a.m. after fasting for 12 hr, and a catheter was inserted into each person's arm. Following a short relaxation period, each member of the couple had 20 min to eat the high saturated fat or high oleic sunflower oil meal; the husband and wife received the same meal and both were required to eat the entire meal. Couples also

engaged in a marital problem discussion on the morning of each visit, as described later. Ghrelin and leptin were sampled before the meal and postmeal at 2, 4, and 7 hr. Self-reported hunger was measured before the meal, immediately after the meal, and then postmeal at 2, 4, and 7 hr. A person's typical diet was assessed with three multipass 24-hr dietary recall interviews. This research was approved by the Ohio State University (OSU) Institutional Review Board; participants provided written informed consent before participating.

Standardized prestudy meals

Equations from the Dietary Reference Intakes were used to determine total kcal requirements for each participant based on age, height, weight, and physical activity (Food and Nutrition Board, 2002). Macronutrient targets (as percentage of total energy) for the prestudy meals were 54.9 + 2.68% carbohydrate, 27.6 + 2.13% fat, and 17.6 + 0.95% protein. Participants ate their last meal no later than 7:30 p.m. the night before admission to the CRC; the dinner was light and low in fat (Lairon, Lopez-Miranda, & Williams, 2007). Compliance was good: Participants consumed 91.18 ± 8.62% of their prestudy meals.

Research meals

Both the high saturated fat and the high oleic sunflower oil meals included eggs, turkey sausage, biscuits, and gravy for a total of 930 kcals, with 60 grams of fat, 59 grams of carbohydrates, and 36 grams of protein (percentage of total kcals = 60, 25, 15, respectively). In line with prior research (Poppitt et al., 2008), the saturated to unsaturated fatty acid ratio varied between the meals; the high saturated fat meal contained 16.84 g palmitic and 13.5 g oleic oil (ratio = 1.93), compared with 8.64 g

palmitic and 31.21 g oleic oil for the high oleic sunflower oil meal (ratio = 0.67). The composition of the research meals was based on the parent study; some human studies have suggested that high saturated fat meals may fuel fatigue-inducing inflammatory responses, although others have not found these effects (Herieka & Erridge, 2014).

Questionnaires and interviews

Self-reported hunger was measured multiple times at each visit with a scale that was modeled after prior research (Flint, Raben, Blundell, & Astrup, 2000). Participants were asked to rate how they felt at the current moment. The items were “How hungry are you?”, “How strong is your desire to eat?”, “How full do you feel?” (reversed), and “How satiated do you feel with the amount you have eaten? (reversed).” The scale demonstrated excellent reliability ($\alpha = .94$).

To assess a participant’s typical diet, we administered three 24-hr dietary recalls over the phone. We used the USDA 24-Hour Multiple Pass Approach to assess overall food intake and macro- and micro-nutrient consumption (Moshfegh et al., 2008). Participants provided a detailed report of the type, quantity, and preparation/cooking method for all food and beverages they consumed in the past 24 hr. Typical dietary intake was calculated by averaging across the three interviews, which included two weekdays and one weekend day. We used the dietary data to calculate the Alternate Healthy Eating Index (aHED), a summary measure of diet quality that takes into account eight aspects of a person’s diet (fruit, vegetables, nuts and soy, total fiber, ratio of polyunsaturated to saturated fat, trans fat as a percentage of total calories, servings of alcohol, and ratio of white meat to red meat), plus an optional multivitamin component that was not used in the current study (McCullough et al., 2002). Higher scores reflect a better quality diet. Software from the Nutrition Data Systems for Research allowed us to examine dietary intake of specific macro- and micro-nutrients.

The mood disorder modules of the Structured Clinical Interview for the Diagnostic and Statistical Manual of Mental Disorders (4th ed.; *DSM-IV*) Axis I Disorders–Nonpatient version (SCID-NP) measured current syndromal depression. The SCID-NP is designed for rapid and valid diagnoses by clinically trained interviewers (First, Spitzer, Gibbon, & Williams, 1997). The SCID-NP was included to account for potential relationships between marital distress and syndromal depression (Robles et al., 2014). Participants’ syndromal depression diagnosis was assessed during their in-person screening visit.

Participants completed the Pittsburgh Sleep Quality Index (PSQI) at their first visit, assessing sleep quality over the past month via a combination of subjective sleep

quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medications, and daytime dysfunction (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). The PSQI has good internal consistency and can distinguish between people with and without sleep disturbances, indicating acceptable discriminant validity (Buysse et al., 1989). Participants were also asked how many hours they slept the night before each visit. The sleep measures provided a way to assess the links among marital distress, ghrelin, and leptin independent of sleep, which can influence both hormones (Taheri, Lin, Austin, Young, & Mignot, 2004).

Participants reported their age. They also had their height and weight measured at their screening visit to calculate BMI.

Marital problem discussion

Hostile marital behavior predicts couples’ physiological changes more reliably than self-reports (Kiecolt-Glaser & Newton, 2001). To obtain behavioral data, an experimenter first conducted a 10- to 20-min interview to identify the best discussion topics for a marital disagreement discussion. These topics were selected from an inventory each spouse completed about their relationship problems (Kiecolt-Glaser et al., 2005). Couples were then asked to discuss and try to resolve one or more marital issues that the experimenter judged to be the most conflict-producing (e.g., money, communication, or in-laws). The research team remained out of sight while videotaping the subsequent 20-min problem discussion.

Marital interaction tapes were coded using the Rapid Marital Interaction Coding System (RMICS), which discriminates well between distressed and nondistressed couples (Heyman, 2004). Distressed marriages are characterized by negative affect, conflictual communication, and poor listening skills (Kiecolt-Glaser & Newton, 2001). Accordingly, the composite index summed four RMICS codes: psychological abuse (e.g., disgust, contempt, belligerence, as well as nonverbal behaviors like glowering), distress-maintaining attributions (e.g., “You’re only being nice so I’ll have sex with you tonight” or “You were being mean on purpose”), hostility (e.g., criticism, hostile voice tone, or rolling the eyes dramatically), and withdrawal (behaviors that suggest pulling back from the interaction or not listening).

Holley and Gilford’s *G* was used to quantify interrater agreement for the RMICS marital distress composite (Xu & Lorber, in press). Interrater agreement was high, with a *G* index of .88. Composite behavior scores were highly correlated across visits (Spearman $r = .77, p < .0001$) and within couples (Spearman $r = 0.81, p < .0001$); the dyad’s behavior sum was averaged across visits, reflecting marital distress.

Ghrelin and leptin assay

All blood samples for a participant were collected via a catheter, frozen after collection, and analyzed within the same assay run. Determinations for leptin and total ghrelin were made using the respective RIA kit per kit instructions with the following exception (Millipore Corporation, St. Charles, MO). Ethylenediaminetetraacetic acid (EDTA) plasma is specified in the ghrelin kit instructions. We only had EDTA plasma available for a small subset of participants. Following a conversation with the kit manufacturer, we conducted a pilot study to assess potential differences between EDTA and heparin plasma ghrelin levels. No significant differences were detected across plasma types, and thus heparin plasma samples were used for all participants. For ghrelin, the intraassay coefficient of variation was 6.4% and interassay coefficient of variation was 16.3%; sensitivity was 0.09 ng/ml. For leptin, the intraassay coefficient of variation was 4.2% and the interassay coefficient of variation was 4.5%; sensitivity was 0.5 ng/ml.

Data analytic strategy

Data preparation. One couple did not return for their second visit, and thus only their Visit 1 data were utilized. There was sporadic missingness in outcomes measured across the day, but the analysis method (mixed models) allowed subjects with these occasionally missing measurements to be included in analyses. The distributions of the ghrelin and leptin data were checked for normality and the presence of outliers. Participants whose values were more than 4 standard deviations from the sample mean were dropped from the corresponding analyses; 13 out of 669 samples (~2%) were dropped for ghrelin and 8 out of 661 samples (~1%) were dropped for leptin. The data for both ghrelin and leptin were highly skewed. Accordingly, each measure was \log_{10} transformed prior to analyses. We had two types of data available: (a) postprandial data that varied across visits and time and (b) dietary data that varied across participants, but not across visits or time. Accordingly, we structured our analyses to reflect these differences.

Postprandial data analyses. Linear mixed models were utilized to account for the correlations within couples and subjects. All models were analyzed with SPSS version 19.0 (IBM, New York, NY) using independent random effects for couple and subject. The random couple effect accounted for dependency between husbands and wives and the random subject statement accounted for the repeated effects of meal (high saturated fat versus high oleic sunflower oil) and also across sample times (measured in hours postmeal). Degrees of freedom were

calculated using the Satterthwaite approximation, which can produce noninteger degrees of freedom.

We investigated whether the combination of marital distress and BMI predicted postprandial ghrelin, leptin, and hunger. Specifically, we tested whether marital distress (continuous), BMI (continuous), time (continuous), and the three-way marital distress by BMI by time interaction (and all corresponding two-way interactions) predicted postprandial ghrelin, leptin, and hunger, controlling for premeal levels. None of the three-way interactions involving time were significant or marginally significant. Due to the complexity of the analytic model and our limited sample size, the higher order interactions involving time were dropped from the analyses. Significant marital distress by BMI interactions were decomposed in two different ways. First, following published recommendations (Aiken & West, 1991), simple slopes tests examined the effect of marital distress for people with a lower versus higher BMI (computed at ± 1 *SD* from the mean, corresponding to 26.24 and 37.90 for lower and higher BMI, respectively). Second, we targeted specific points along the BMI continuum to determine the BMI cutoff that differentiated a significant versus nonsignificant link between marital distress and each outcome. This second set of follow-up analyses provided additional information about whether the link between marital distress and each outcome was evident for healthy weight, overweight, or obese people. For all of these primary analyses, we investigated a model with no covariates except for baseline levels of the corresponding outcome.

We conducted a series of ancillary analyses using the postprandial data. First, we tested whether the results remained the same after adding potential confounds that were selected based on their theoretical and empirical relationships to marital distress, hunger, and ghrelin (Robles et al., 2014; Taheri et al., 2004). The adjusted models included meal type, age, sex, and total number of hours slept the night before each visit. We planned to include syndromal depression as a covariate; only 2 participants met criteria for a current diagnosis, and thus it was omitted from the analyses. In a second set of ancillary analyses, we tested whether sex or meal type moderated any of our effects.

Dietary data analyses. Linear mixed models were utilized to account for the correlations within couples. The random couple effect accounted for dependency between husbands and wives. We conducted two sets of dietary analyses. For both sets of analyses, we examined whether marital distress predicted participants' dietary intake.

For the first set of dietary analyses, we conducted a set of linear mixed models examining whether the combination of marital distress and BMI predicted women's typical diet in terms of quantity of foods consumed. Specifically,

Table 2. Summary of the Unadjusted Models With Marital Distress by BMI Interaction Predicting Each Outcome

Outcome	Predictor: Marital distress by BMI interaction			
	<i>F</i>	<i>df</i> numerator	<i>df</i> denominator	<i>p</i>
Ghrelin (log ₁₀ ; pg/ml)	6.63	1	65.76	.012
Leptin (log ₁₀ ; pg/ml)	0.09	1	74.82	.771
Self-reported hunger	0.22	1	76.59	.637
aHEI	7.18	1	35.92	.011
Total amount of food (g)	8.28	1	60.50	.006
Calories (kcal)	2.07	1	77.17	.154
Fatty acids (g)	2.51	1	78.30	.117
Saturated fatty acids (g)	3.81	1	78.23	.054
Monounsaturated fatty acids (g)	1.54	1	74.65	.219
Polyunsaturated fatty acids (g)	0.37	1	81.00	.545
Carbohydrates (g)	0.09	1	70/95	.768
Protein (g)	4.48	1	67.97	.038
Cholesterol (mg)	6.85	1	76.24	.011
Sodium (mg)	4.34	1	75.13	.041

Note: *N* = 43 couples, 86 participants. These analyses reflect the unadjusted models (except baseline data) reported in the primary analyses. The ghrelin, leptin, and self-reported hunger analyses included baseline levels of the corresponding outcome as a predictor.

we tested whether marital distress (continuous) and BMI (continuous) predicted aHEI scores, total grams of food consumed, calories, protein, cholesterol, carbohydrates, and sodium. We also examined total fat consumed, and a breakdown of fatty acids into their unhealthier (saturated fatty acids [SFAs]) and healthier (monounsaturated fatty acids [MFAs] and polyunsaturated fatty acids [PUFAs]) forms (Mensink & Katan, 1992). Significant marital distress by BMI interactions were decomposed in two different ways. First, following published recommendations (Aiken & West, 1991), simple slopes tests examined the effect of marital distress for people with a lower versus higher BMI. This first set of follow-up analyses were utilized for all of the dietary outcomes. Second, we targeted specific points along the BMI continuum to determine the BMI cutoff that differentiated a significant versus nonsignificant link between marital distress and diet. This second set of follow-up analyses were only used in the model predicting aHEI scores to reduce the total number of analyses conducted. There were no covariates included in these primary analyses.

In another set of dietary analyses, we conducted a series of linear mixed models testing whether marital distress predicted the percentage of calories due to three macronutrients: fat, carbohydrates, and protein. These additional analyses allowed us to investigate whether marital distress was linked to the overall quantity of macronutrients people were consuming (which would be supported by Dietary Analyses 1) versus the type of macronutrients people were consuming (which would be supported by Ancillary Analyses 2).

We conducted a series of ancillary analyses using the dietary data. First, we tested whether the results remained the same after adding age, sex, and sleep. We used the PSQI as a measure of sleep quality, measuring longer-term sleep quality. In a second set of ancillary analyses, we tested whether sex moderated any of our effects.

Results

Postprandial results

As expected, ghrelin levels rose throughout the day after breakfast, $b = 0.009$, $t(83) = 5.67$, $p < .001$. In addition, there was a significant two-way marital distress by BMI interaction predicting postprandial ghrelin levels, controlling for premeal ghrelin, $F(1, 65.76) = 6.63$, $p = .012$ (see Table 2). As shown in Figure 1, among participants with a lower BMI, those who were in more distressed marriages had higher postprandial ghrelin than those who were in less distressed marriages, $b = 0.0007$, $t(57.60) = 2.36$, $p = .021$. On the other hand, marital distress was unrelated to postprandial ghrelin among people with a higher BMI, $b = -0.0002$, $t(50.24) = -0.90$, $p = .374$. A second set of follow-up analyses determined that the link between marital distress and postprandial ghrelin was significant for participants with a BMI of 28.00, $b = 0.0005$, $t(50.40) = 2.15$, $p = .037$, marginally significant for a BMI of 29.00 and 30.00, $b = 0.0005$, $t(45.76) = 1.97$, $p = .055$ and $b = 0.0004$, $t(41.34) = 1.74$, $p = .090$, and nonsignificant for a BMI of 31.00, $b = 0.0003$, $t(37.71) = 1.46$, $p = .154$.

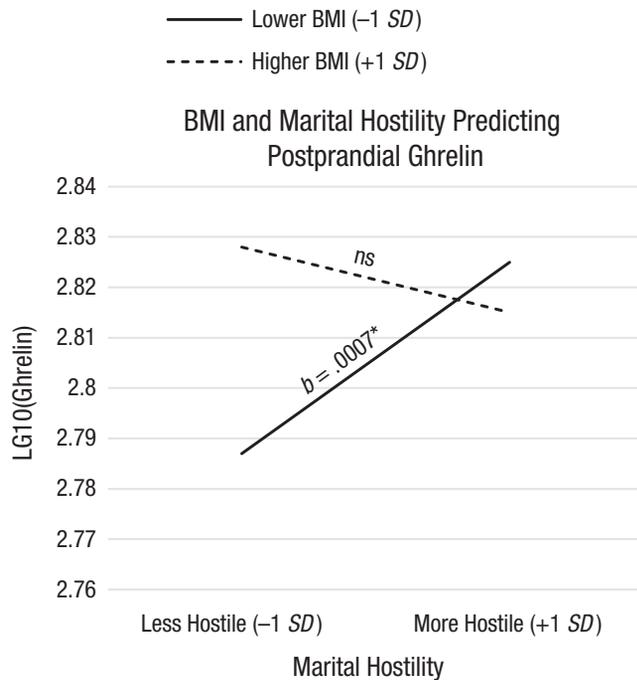


Fig. 1. The BMI and marital distress interaction predicting postprandial ghrelin. The values depicted are the estimated marginal means obtained from a model that included baseline ghrelin levels, marital distress (continuous), BMI (continuous), and the marital distress by BMI interaction. Marital distress and BMI were both plotted at 1 standard deviation above and below their respective means. This corresponded to 26.24 and 37.90 for lower and higher BMI respectively. * $p < .05$.

The link between marital distress and ghrelin was significant for people with a lower BMI. To understand the magnitude of this effect, we estimated the percentage difference (mean 1 – mean 2 / average of both means) and standardized difference (mean 1 – mean 2 / standard deviation) in ghrelin between those lower and higher in marital distress. We used the estimated marginal means at one standard deviation above and below the average of marital distress and converted the log transformed ghrelin values back to their raw form. Among participants with a lower BMI, those in more distressed marriages (+1 *SD*) had 8.74% more ghrelin than those in less distressed marriages (–1 *SD*). The standardized difference for ghrelin between lower and higher marital distress among people with a lower BMI was .28.

Next, we added the covariates we selected a priori; the two-way marital distress by BMI interaction remained significant with meal type, sex, age, and sleep added to the models. Finally, we tested whether meal type or sex moderated our effects. Neither of the three-way interactions involving meal type or sex were significant (all p values $> .629$), demonstrating that the effects of marital distress and BMI on postprandial ghrelin were the same across both meal types and for both genders.

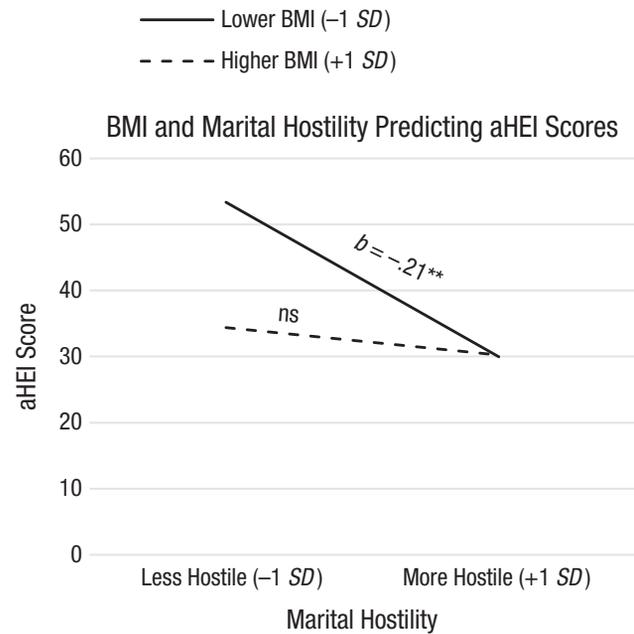


Fig. 2. The BMI and marital distress interaction predicting aHEI scores. The values depicted are the estimated marginal means obtained from a model that included marital distress (continuous), BMI (continuous), and the marital distress by BMI interaction. Marital distress and BMI were both plotted at 1 standard deviation above and below their respective means. This corresponded to 26.24 and 37.90 for lower and higher BMI respectively. aHEI = Alternate Healthy Eating Index; higher numbers reflect a better quality diet. * $p < .01$.

Consistent with the ghrelin data, self-reported hunger rose throughout the day after breakfast, $b = 0.82$, $t(85) = 28.36$, $p < .001$. However, leptin levels also increased after breakfast, $b = 0.02$, $t(76) = 9.23$, $p < .001$. Furthermore, the two-way marital distress by BMI interaction predicting postprandial leptin and self-reported hunger were nonsignificant in both the unadjusted and adjusted models, all p values $> .636$. None of the interactions involving meal type or sex were significant, all p values $> .228$.

Dietary results

There was a significant two-way marital distress by BMI interaction predicting aHEI scores, $F(1, 35.92) = 7.18$, $p = .011$. See Table 2 for a summary of the dietary results. As shown in Figure 2, among participants with a lower BMI, those who were in more distressed marriages had a poorer quality diet than those who were in less distressed marriages, $b = -0.21$, $t(47.75) = -3.32$, $p = .002$. On the other hand, marital distress was unrelated to diet quality among participants with a higher BMI, $b = -0.04$, $t(49.72) = -0.56$, $p = .562$. A second set of follow-up analyses determined that the link between marital distress and diet quality was significant for participants with

a BMI of 32.00, $b = -0.12$, $t(29.45) = -2.23$, $p = .033$, marginally significant for a BMI of 33.00, $b = -0.11$, $t(30.35) = -1.95$, $p = .060$, and nonsignificant for a BMI of 34.00, $b = -0.09$, $t(32.47) = -1.65$, $p = .108$.

The link between marital distress and the aHEI was significant for people with a lower BMI. To understand the magnitude of this effect, we estimated the percentage difference and standardized difference in aHEI scores between those lower and higher in marital distress. We used the estimated marginal means at one standard deviation above and below the average of marital distress. Among participants with a lower BMI, those in more distressed marriages (+1 *SD*) had a diet that was 56.12% worse than those in less distressed marriages (-1 *SD*). The standardized difference for aHEI scores between lower and higher marital distress among people with a lower BMI was 2.19.

To investigate diet quality further, we first examined overall quantities of specific macro- and micro- nutrients. The two-way marital distress by BMI interaction was a significant predictor of total grams of food consumed, $F(1, 60.50) = 8.28$, $p = .006$, and protein, $F(1, 67.97) = 4.48$, $p = .038$, cholesterol, $F(1, 76.24) = 6.85$, $p = .011$, and sodium, $F(1, 75.13) = 4.34$, $p = .041$, intake. Participants with a lower BMI who were in more distressed marriages consumed more total grams of food, $b = 14.78$, $t(67.12) = 2.97$, $p = .004$, protein, $b = 0.32$, $t(69.91) = 2.01$, $p = .049$, cholesterol, $b = 1.71$, $t(69.83) = 2.36$, $p = .021$, and sodium, $b = 21.06$, $t(70.79) = 2.50$, $p = .015$, than their counterparts who were in less distressed marriages. On the other hand, marital distress was unrelated to total grams of food, protein, cholesterol, and sodium among participants with a higher BMI, all p values $> .311$.

The BMI by marital distress interaction was not a significant predictor of caloric intake, $F(1, 77.17) = 2.07$, $p = .154$, carbohydrates, $F(1, 70.95) = 0.09$, $p = .768$, or total fat consumed, $F(1, 78.30) = 2.51$, $p = .117$. However, the two-way interaction was a significant predictor of the unhealthier form of fat, SFAs, $F(1, 78.23) = 3.81$, $p = .054$. On the other hand, the marital distress by BMI interaction was unrelated to the healthier forms of fat, MUFAs, $F(1, 74.65) = 1.54$, $p = .219$, and PUFAs, $F(1, 81.00) = 0.37$, $p = .545$. Participants with a lower BMI who were in more distressed marriages consumed more SFAs than their counterparts who were in less distressed marriages, $b = 0.12$, $t(70.03) = 1.86$, $p = .067$, although this effect was marginal. However, marital distress was unrelated to SFA intake among participants with a higher BMI, $b = -0.04$, $t(62.03) = -0.70$, $p = .486$.

In another set of dietary analyses, we examined whether the combination of marital distress and BMI predicted the percentage of calories due to three macronutrients: carbohydrates, protein, and fat. The two-way marital distress by BMI interaction was a significant

predictor of percentage of calories due to protein, $F(1, 66.30) = 3.97$, $p = .050$. Participants with a lower BMI who were in more distressed marriages consumed a larger percentage of their calories due to protein than their counterparts who were in less distressed marriages, $b = 0.05$, $t(69.56) = 2.33$, $p = .023$. On the other hand, marital distress was unrelated to percentage of calories due to protein among participants with a higher BMI, $p = .941$. The marital distress by BMI interaction was a marginally significant predictor of percentage of calories due to carbohydrates, $F(1, 68.78) = 3.71$, $p = .058$. However, follow-up tests were nonsignificant for both people with lower and higher BMIs (p values $> .188$). In addition, the marital distress by BMI interaction did not predict percentage of calories due to total fat, SFAs, MFAs, and PUFAs (all p values $> .181$).

Next, we added the covariates we selected a priori; the patterns remained the same when we added sex, age, and sleep to the models with the following exceptions. The models predicting total protein consumption ($p = .123$) and percentage of calories due to protein ($p = .120$) changed from significant to nonsignificant. The marital distress by BMI interaction predicting SFAs changed from nonsignificant to marginally significant, $F(1, 76.70) = 3.55$, $p = .063$. Participants with a lower BMI who were in more distressed marriages consumed a larger percentage of their calories due to SFAs than their counterparts who were in less distressed marriages, $b = 0.04$, $t(65.18) = 2.42$, $p = .018$. On the other hand, marital distress was unrelated to percentage of calories due to SFAs among participants with a higher BMI ($p = .979$).

Finally, we tested whether sex moderated our effects. None of the three-way marital distress by BMI by sex interactions were significant (all p values $> .159$), demonstrating that the dietary results were consistent for men and women.

Discussion

People in more distressed marriages had higher postprandial ghrelin levels compared with those in less distressed marriages, but only among participants with a lower BMI. Marital distress and postprandial ghrelin were unrelated for participants with a higher BMI, consistent with prior research about the differential effects of ghrelin among obese people (Buss et al., 2014). Furthermore, marital distress was related to postprandial ghrelin for participants with a lower BMI after they ate a high saturated fat and after they ate a high sunflower oil meal, and the results were consistent for both men and women.

Postprandial ghrelin, but not leptin, was related to marital distress, raising important questions about the specificity of the links among marital distress, appetite regulation, and dietary patterns. Ghrelin has relatively

immediate effects on a person's appetite, whereas leptin is more important for longer-term energy balance (Klok et al., 2007). Accordingly, the current data suggest that marital distress may be linked to a hormone that is implicated in shorter-term rather than longer-term energy balance. This possibility raises a number of important questions for future research about acute versus chronic marital distress and shorter versus longer-term energy balance.

The present research demonstrated that people in more distressed marriages had a poorer quality diet than people in less distressed marriages, as indexed by the alternative healthy eating index, but only among participants with a lower BMI. As shown in Figure 2, both participants with a higher BMI and participants in a less distressed marriage with a lower BMI had an aHEI score around 30.00. On the other hand, people in more distressed marriages with a lower BMI had an aHEI score around 53. To aid in interpreting these scores, we drew on aHEI scores calculated from 71,495 women in the Nurses' Health Study and 41,029 men in the Health Professionals Follow-Up Study (Chiuve et al., 2012; McCullough et al., 2002). The average aHEI scores in these studies were 45.00 (± 11.1) for men and 38.4 (± 10.3) for women. The average BMI across both samples was around 25.00. These data suggest that participants in the current study who were less distressed with a lower BMI had a diet that was healthier than average. Simultaneously, people who were in more distressed marriages with a lower BMI had a diet that was less healthy than average.

The current study also demonstrated that, among people with a lower BMI, those in more distressed marriages consumed more total grams of food, and more, cholesterol, sodium, and SFAs (but not MUFAs or PUFAs) than their less distressed counterparts. In addition, the percentage of calories in a person's diet due to carbohydrates and SFAs was marginally higher among people in more distressed marriages (although the latter effect was only true in the adjusted models). The percentage of calories in a person's diet due to protein was also higher among people in more distressed marriages, but only in the unadjusted models. Accordingly, people in distressed marriages were eating more macronutrients, and partially altering the proportion of their diet that came from these macronutrients. Consistent with the ghrelin findings, these dietary results were only evident among people with a lower BMI; diet quality and food consumption were unrelated to marital distress among those with a higher BMI. Interestingly, among participants with a lower BMI, marital distress was related to increased consumption of sodium, cholesterol and SFAs; all three elevate risk for cardiovascular disease (Mensink & Katan, 1992). However, marital distress was unrelated to MUFAs and PUFAs, which may reduce heart disease risk (Mensink

& Katan, 1992). According to a meta-analysis, the most health-beneficial profile of fatty acid consumption may be to consume MUFAs and PUFAs instead of SFAs, while keeping total fat the same (Mensink & Katan, 1992). The results of the present study correspond to this healthier profile. Consistent with the ghrelin data, the link between marital distress and diet quality was the same for men and women.

In all of the analyses, the effect of marital distress was tested at one standard deviation above and below the average BMI in the current sample, which corresponded to 26.24 and 37.90, respectively. Furthermore, an additional set of follow-up tests determined that marital distress was significantly related to postprandial ghrelin for people with a BMI of 28, but it was no longer significantly related for a BMI of 31. Similarly, marital distress was related to poorer overall diet quality for people with a BMI of 32, but not for a BMI of 34. Accordingly, the present study suggests that marital distress is linked to ghrelin and diet quality for healthy weight and most overweight people (BMI < 30.00), but not for many obese people (BMI \geq 30).

Marital distress is strongly linked to poor health, including an elevated risk of the metabolic syndrome and cardiovascular disease (Orth-Gomér et al., 2000; Robles et al., 2014). One important question is understanding the mechanisms that link marital distress to poor health; appetite dysregulation is one promising avenue. The current data demonstrated that marital distress is linked to postprandial ghrelin and a poor quality diet among people with a lower (but not higher) BMI. A poor quality diet is linked to a variety of health problems, including cardiovascular disease (Esposito et al., 2004; Hu & Willett, 2002), and a person's appetite and eating behavior are strongly linked to obesity (Arora & Anubhuti, 2006), a risk factor for a host of medical problems (Billington et al., 2000). Prior research in combination with the current results raise an interesting possibility for future research. Specifically, marital distress may be linked to weight gain and other health problems among people with a lower BMI because it affects ghrelin and diet quality. Investigating this provocative hypothesis would require a combination of experimental and longitudinal study designs linking marital distress, appetite-relevant hormones, and weight changes over time, one important direction of inquiry.

Another critical avenue for additional research is understanding why marital distress is linked to appetite dysregulation and diet quality. Mental health and health behaviors may contribute to the link between marital distress and diet. For instance, people in more distressed marriages have poorer sleep quality than those in less distressed marriages (El-Sheikh, Kelly, & Rauer, in press); sleep is linked to appetite regulation (Taheri et al., 2004).

The current study used stringent selection criteria that excluded people with major medical problems. In addition, ancillary analyses demonstrated that our results were unchanged after accounting for participants' age, gender, and sleep quality. Accordingly, the present data suggest that marital distress is linked to appetite dysregulation and diet quality independent of health behaviors and comorbidities.

There are a number of potential psychological explanations for the links between marital distress and appetite dysregulation. One possibility is that people eat to decrease negative affect or increase positive affect. According to the Reward Based Stress Eating model, people eat calorically dense food because consumption activates reward circuitry in the brain (Adam & Epel, 2007). For example, growing evidence demonstrates that the opioid and dopaminergic systems are integrally involved in food reward and food addiction processes (Cota, Tschöp, Horvath, & Levine, 2006). Furthermore, people often believe that eating will alleviate negative affect, a perspective that can lead to disordered eating (Hayaki, 2009).

Another related psychological explanation is that feeling hungry and eating in response to marital distress may help people feel socially reconnected. The need for social connection is fundamental to human nature. Consequently, experiencing marital distress should motivate people to try and bond with others to restore their sense of belonging. Furthermore, eating and social connection are intricately linked. For example, eating was a highly social activity throughout human evolution (Wrangham, 2010), and today meals are frequently eaten with other people. In fact, people often look to cues from others to regulate their own food intake (Herman, Roth, & Polivy, 2003), and people model the eating behavior of others more when they also desire an affiliation with those people (Cruwys, Bevelander, & Hermans, *in press*). Other researchers have theorized that preparing and eating comfort food reminds people of their emotional bonds with significant others (Locher, Yoels, Maurer, & van Ells, 2005). Consistent with this idea, eating comfort food caused people to spontaneously think about their relationships, and simply thinking about comfort food decreased loneliness (Troisi & Gabriel, 2011). Consequently, people may feel hungrier when they experience marital distress because they have either implicitly or explicitly learned that eating helps them feel socially connected or provides them with an opportunity for social connection.

One important caveat is that the current study's link between marital distress and ghrelin was attenuated among obese people. These data suggest that any links between eating and mood or social connection may be diminished among people with a higher BMI. Obesity

puts people at risk for decreased self-control in weight-relevant contexts, social stigmatization, and depression (Luppino et al., 2010; Major, Eliezer, & Rieck, 2012). Accordingly, implicit or explicit associations linking eating to social connection may be attenuated or eliminated among obese people, and thus their appetite may not be stimulated following marital distress. Based on this explanation, marital distress would not alter an obese person's appetite because the experience of marital distress is psychologically different for that person in terms of their strategies for social reconnection. Investigating the associations among hunger, eating, and social connection among obese and nonobese people will provide important insight into this possible mechanistic explanation.

There are a number of plausible physiological pathways linking marital distress to appetite dysregulation and food intake. Sympathetic nervous system activity is one promising candidate. For example, marital distress was related to markers of sympathetic nervous system activation (Kiecolt-Glaser, Glaser, Cacioppo, & Malarkey, 1998), and direct stimulation of the sympathetic nervous system in rats elevated ghrelin levels (Mundinger, 2006). The rodent and human literatures about social stressors suggest that sympathetic nervous system activity may be altered among obese people, providing a potential explanation for the current study results. Specifically, a rodent study demonstrated that a β -adrenergic antagonist attenuated the effects of chronic social defeat on leptin production, another appetite-relevant hormone (Chuang et al., 2010). However, the β -adrenergic receptor is less sensitive to stimulation among obese compared with nonobese people (Van Baak, 2001). Based on this rationale, marital distress would not alter an obese person's appetite because the β -adrenergic receptor is less responsive to marital distress, resulting in less ghrelin production.

Research examining the links among marital distress, ghrelin, and hunger is in its infancy, particularly among humans, and mechanistic pathways have not been clearly delineated. The current research suggests that a person's appetite is only linked to marital distress among people with a lower BMI. There are psychological and physiological explanations that may explain these effects, as described earlier. Further investigating the proposed explanations and other alternatives are an important target for future research.

Another interesting possible avenue of exploration is examining whether habitual diet affects ghrelin levels among people with a lower BMI. Ghrelin levels largely depend on recent macronutrient consumption (Koliaki et al., 2010), suggesting an important role for eating behavior in ghrelin levels. Accordingly, marital distress may elevate ghrelin and alter food consumption, which may then have differential effects on postmeal ghrelin,

suggesting a potential cyclical pattern. In the current study, all participants received standardized meals the day before their visit and also ate a standardized breakfast the morning of their visit, limiting the effect of habitual diet on ghrelin. Examination of ghrelin and eating behavior in a naturalistic setting could provide new insight into this intriguing possibility.

The sample was primarily White, one limitation of this study. Accordingly, researchers may gain additional insight by investigating more diverse samples. The current sample size was relatively small, another limitation. However, couples were assessed multiple times throughout the day on two separate visits, providing repeated measures for every participant. Ghrelin levels and self-reported hunger increased over time after breakfast, consistent with participants' restriction from eating the rest of the day. Leptin is typically lower when people are hungry or are fasting (Klok et al., 2007). Contrary to expectations, leptin levels actually rose throughout the day, potentially providing an explanation for the nonsignificant marital distress results. Accordingly, additional studies assessing postmeal leptin will provide insight into the consistency of the leptin findings across different samples and populations. Finally, there are no published cutoffs that delineate the clinical significance of how ghrelin alterations are linked to weight gain or other health problems. Indeed, many other commonly used physiological markers (e.g., interleukin-6, cortisol, etc.) are also lacking clear guidelines about clinical significance, particularly within ranges that would be obtained in healthier populations. Importantly, the ghrelin results are mirrored by participants own self-reports about their diet quality. Accordingly, we have consistent results across two very different but related measures. An important avenue for appetite-regulation research is to examine how the magnitude of change in appetite-relevant hormones is linked to eating behavior and potential weight gain over time.

In sum, people in more distressed marriages had higher levels of postprandial ghrelin and a poorer quality diet than those in less distressed marriages, but only among participants with a lower BMI. Marital distress was unrelated to postprandial ghrelin and diet quality among participants with a higher BMI. These data suggest that ghrelin, an appetite-regulating hormone with relatively immediate effects on energy balance, and diet quality may ultimately link marital distress to its corresponding negative health effects.

Author Contributions

L. M. Jaremka and M. E. Lindgren participated in data collection. M. A. Belury, R. R. Andridge, D. Habash, W. B. Malarkey, and J. K. Kiecolt-Glaser contributed significantly to the design of the study. L. M. Jaremka, R. R. Andridge, M. E. Lindgren, and J. K. Kiecolt-Glaser developed the study hypotheses or helped con-

duct data analyses. All authors helped write or revise the manuscript, and everyone approved the manuscript for publication.

Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

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References

- Adam, T. C., & Epel, E. S. (2007). Stress, eating and the reward system. *Physiology & Behavior, 91*, 449–458. <http://doi.org/10.1016/j.physbeh.2007.04.011>
- Aiken, L. S., & West, S. G. (1991). *Multiple regression: Testing and interpreting interactions*. Newbury Park, CA: Sage.
- Arora, S., & Anubhuti. (2006). Role of neuropeptides in appetite regulation and obesity: A review. *Neuropeptides, 40*, 375–401. <http://doi.org/10.1016/j.npep.2006.07.001>
- Baumeister, R. F., & Leary, M. R. (1995). The need to belong: Desire for interpersonal attachments as a fundamental human motivation. *Psychological Bulletin, 117*(3), 497–529. <http://doi.org/10.1037/0033-2909.117.3.497>
- Billington, C. J., Epstein, L. H., Goodwin, N. J., Hill, J. O., Pi-Sunyer, F. X., Rolls, B. J., . . . Harrison, B. (2000). Overweight, obesity, and health risk. *Archives of Internal Medicine, 160*, 898–904.
- Buss, J., Havel, P. J., Epel, E., Lin, J., Blackburn, E., & Daubenmier, J. (2014). Associations of ghrelin with eating behaviors, stress, metabolic factors, and telomere length among overweight and obese women: Preliminary evidence of attenuated ghrelin effects in obesity? *Appetite, 76*, 84–94. <http://doi.org/10.1016/j.appet.2014.01.011>
- Buysse, D. J., Reynolds, C. F., III, Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatry Research, 28*, 193–213.
- Carlson, J. J., Turpin, A. A., Wiebke, G., Hunt, S. C., & Adams, T. D. (2009). Pre- and post-prandial appetite hormone levels in normal weight and severely obese women. *Nutrition & Metabolism, 6*, 32. <http://doi.org/10.1186/1743-7075-6-32>
- Chiuve, S. E., Fung, T. T., Rimm, E. B., Hu, F. B., McCullough, M. L., Wang, M., . . . Willett, W. C. (2012). Alternative dietary indices both strongly predict risk of chronic disease. *Journal of Nutrition, 142*, 1009–1018. <http://doi.org/10.3945/jn.111.157222>
- Chuang, J.-C., Krishnan, V., Yu, H. G., Mason, B., Cui, H., Wilkinson, M. B., . . . Lutter, M. (2010). A β 3-adrenergic-leptin-melanocortin circuit regulates behavioral and metabolic changes induced by chronic stress. *Biological Psychiatry, 67*, 1075–1082. <http://doi.org/10.1016/j.biopsych.2009.12.003>

- Considine, R. V., Sinha, M. K., Heiman, M. L., Kriauciunas, A., Stephens, T. W., Nyce, M. R., . . . Bauer, T. L. (1996). Serum immunoreactive-leptin concentrations in normal-weight and obese humans. *New England Journal of Medicine*, *334*, 292–295. <http://doi.org/10.1056/NEJM199602013340503>
- Cota, D., Tschöp, M. H., Horvath, T. L., & Levine, A. S. (2006). Cannabinoids, opioids and eating behavior: The molecular face of hedonism? *Brain Research Reviews*, *51*, 85–107. <http://doi.org/10.1016/j.brainresrev.2005.10.004>
- Cruwys, T., Bevelander, K. E., & Hermans, R. C. J. (in press). Social modeling of eating: A review of when and why social influence affects food intake and choice. *Appetite*. <http://doi.org/10.1016/j.appet.2014.08.035>
- Cummings, D. E., Purnell, J. Q., Frayo, R. S., Schmidova, K., Wisse, B. E., & Weigle, D. S. (2001). A preprandial rise in plasma ghrelin levels suggests a role in meal initiation in humans. *Diabetes*, *50*, 1714–1719. <http://doi.org/10.2337/diabetes.50.8.1714>
- El-Sheikh, M., Kelly, R., & Rauer, A. (in press). Quick to berate, slow to sleep: Interpartner psychological conflict, mental health, and sleep. *Health Psychology*. <http://doi.org/10.1037/a0031786>
- Esposito, K., Marfella, R., Ciotola, M., Di Palo, C., Giugliano, F., Giugliano, G., . . . Giugliano, D. (2004). Effect of a Mediterranean-style diet on endothelial dysfunction and markers of vascular inflammation in the metabolic syndrome: A randomized trial. *JAMA*, *292*, 1440–1446. <http://doi.org/10.1001/jama.292.12.1440>
- First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1997). *User's guide for the Structured Clinical Interview for DSM-IV Axis I Disorders: SCID-1 clinician version*. Washington, DC: American Psychiatric Press.
- Flint, A., Raben, A., Blundell, J. E., & Astrup, A. (2000). Reproducibility, power and validity of visual analogue scales in assessment of appetite sensations in single test meal studies. *International Journal of Obesity & Related Metabolic Disorders*, *24*, 38–48.
- Food and Nutrition Board. (2002, October 5). *Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids*. Retrieved from <http://www.iom.edu/Reports/2002/Dietary-Reference-Intakes-for-Energy-Carbohydrate-Fiber-Fat-Fatty-Acids-Cholesterol-Protein-and-Amino-Acids.aspx>
- Hayaki, J. (2009). Negative reinforcement eating expectancies, emotion dysregulation, and symptoms of bulimia nervosa. *International Journal of Eating Disorders*, *42*, 552–556. <http://doi.org/10.1002/eat.20646>
- Herieka, M., & Erridge, C. (2014). High-fat meal induced postprandial inflammation. *Molecular Nutrition & Food Research*, *58*, 136–146. <http://doi.org/10.1002/mnfr.201300104>
- Herman, C. P., Roth, D. A., & Polivy, J. (2003). Effects of the presence of others on food intake: A normative interpretation. *Psychological Bulletin*, *129*, 873–886. <http://doi.org/10.1037/0033-2909.129.6.873>
- Heyman, R. E. (2004). Rapid Marital Interaction Coding System (RMICS). In P. Kerig & D. Baucom (Eds.), *Couple observational coding systems* (pp. 67–94). Mahwah, NJ: Erlbaum.
- Hu, F. B., & Willett, W. C. (2002). Optimal diets for prevention of coronary heart disease. *JAMA*, *288*, 2569–2578.
- Johnson, N. J., Backlund, E., Sorlie, P. D., & Loveless, C. A. (2000). Marital status and mortality: The National Longitudinal Mortality Study. *Annals of Epidemiology*, *10*, 224–238. [http://doi.org/10.1016/S1047-2797\(99\)00052-6](http://doi.org/10.1016/S1047-2797(99)00052-6)
- Kiecolt-Glaser, J. K., Glaser, R., Cacioppo, J. T., & Malarkey, W. B. (1998). Marital stress: Immunologic, neuroendocrine, and autonomic correlates. *Annals of the New York Academy of Sciences*, *840*, 656–663. <http://doi.org/10.1111/j.1749-6632.1998.tb09604.x>
- Kiecolt-Glaser, J. K., Loving, T. J., Stowell, J. R., Malarkey, W. B., Lemeshow, S., Dickinson, S. L., & Glaser, R. (2005). Hostile marital interactions, proinflammatory cytokine production, and wound healing. *Archives of General Psychiatry*, *62*, 1377–1384.
- Kiecolt-Glaser, J. K., & Newton, T. L. (2001). Marriage and health: His and hers. *Psychological Bulletin*, *127*, 472–503.
- Klok, M. D., Jakobsdottir, S., & Drent, M. L. (2007). The role of leptin and ghrelin in the regulation of food intake and body weight in humans: A review. *Obesity Reviews*, *8*, 21–34. <http://doi.org/10.1111/j.1467-789X.2006.00270.x>
- Koliaki, C., Kokkinos, A., Tentolouris, N., & Katsilambros, N. (2010). The effect of ingested macronutrients on postprandial ghrelin response: A critical review of existing literature data. *International Journal of Peptides*, *2010*, e710852. <http://doi.org/10.1155/2010/710852>
- Lairon, D., Lopez-Miranda, J., & Williams, C. (2007). Methodology for studying postprandial lipid metabolism. *European Journal of Clinical Nutrition*, *61*, 1145–1161. <http://doi.org/10.1038/sj.ejcn.1602749>
- Leary, M. R., & Cox, C. B. (2008). Belongingness motivation: A mainspring of social action. In W. L. Gardner & J. Y. Shah (Eds.), *Handbook of motivation science* (pp. 27–40). New York, NY: Guilford.
- Locher, J. L., Yoels, W. C., Maurer, D., & van Ells, J. (2005). Comfort foods: An exploratory journey into the social and emotional significance of food. *Food and Foodways*, *13*, 273–297. <http://doi.org/10.1080/07409710500334509>
- Luppino, F. S., de Wit, L. M., Bouvy, P. F., Stijnen, T., Cuijpers, P., Penninx, B. W. J. H., & Zitman, F. G. (2010). Overweight, obesity, and depression: A systematic review and meta-analysis of longitudinal studies. *Archives of General Psychiatry*, *67*, 220–229. <http://doi.org/10.1001/archgenpsychiatry.2010.2>
- Major, B., Eliezer, D., & Rieck, H. (2012). The psychological weight of weight stigma. *Social Psychological and Personality Science*, *3*, 651–658. <http://doi.org/10.1177/1948550611434400>
- Maslow, A. H. (1968). *Toward a psychology of being* (2nd ed.). Oxford, England: D. Van Nostrand.
- McCullough, M. L., Feskanich, D., Stampfer, M. J., Giovannucci, E. L., Rimm, E. B., Hu, F. B., . . . Willett, W. C. (2002). Diet quality and major chronic disease risk in men and women: Moving toward improved dietary guidance. *American Journal of Clinical Nutrition*, *76*, 1261–1271.
- Mensink, R., & Katan, M. (1992). Effect of dietary fatty-acids on serum-lipids and lipoproteins: A metaanalysis of 27 trials. *Arteriosclerosis and Thrombosis*, *12*, 911–919.
- Moshfegh, A. J., Rhodes, D. G., Baer, D. J., Murayi, T., Clemens, J. C., Rumpler, W. V., . . . Cleveland, L. E. (2008). The US Department of Agriculture automated multiple-pass method

- reduces bias in the collection of energy intakes. *American Journal of Clinical Nutrition*, 88, 324–332.
- Mundinger, T. O. (2006). Direct stimulation of ghrelin secretion by sympathetic nerves. *Endocrinology*, 147, 2893–2901. <http://doi.org/10.1210/en.2005-1182>
- Myers, M. G., Leibel, R. L., Seeley, R. J., & Schwartz, M. W. (2010). Obesity and leptin resistance: Distinguishing cause from effect. *Trends in Endocrinology and Metabolism*, 21, 643–651. <http://doi.org/10.1016/j.tem.2010.08.002>
- Orth-Gomér, K., Wamala, S. P., Horsten, M., Schenck-Gustafsson, K., Schneiderman, N., & Mittleman, M. A. (2000). Marital stress worsens prognosis in women with coronary heart disease: The Stockholm Female Coronary Risk Study. *JAMA*, 284, 3008–3014.
- Pickett, C. L., & Gardner, W. L. (2005). The social monitoring system: Enhanced sensitivity to social cues as an adaptive response to social exclusion. In K. D. Williams, J. Forgas, & W. von Hippel (Eds.), *The social outcast: Ostracism, social exclusion, rejection, and bullying* (pp. 213–225). New York, NY: Psychology Press.
- Poppitt, S. D., Keogh, G. F., Lithander, F. E., Wang, Y., Mulvey, T. B., Chan, Y.-K., . . . Cooper, G. J. S. (2008). Postprandial response of adiponectin, interleukin-6, tumor necrosis factor- α , and C-reactive protein to a high-fat dietary load. *Nutrition*, 24, 322–329. <http://doi.org/10.1016/j.nut.2007.12.012>
- Robles, T. F., Slatcher, R. B., Trombello, J. M., & McGinn, M. M. (2014). Marital quality and health: A meta-analytic review. *Psychological Bulletin*, 140, 140–187. <http://doi.org/10.1037/a0031859>
- Schoenborn, C. A. (2004). Marital status and health: United States, 1999–2002. *Advance Data*, 351, 1–32.
- Taheri, S., Lin, L., Austin, D., Young, T., & Mignot, E. (2004). Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. *PLoS Medicine*, 1, e62. <http://doi.org/10.1371/journal.pmed.0010062>
- Troisi, J. D., & Gabriel, S. (2011). Chicken soup really is good for the soul: “Comfort food” fulfills the need to belong. *Psychological Science*, 22, 747–753. <http://doi.org/10.1177/0956797611407931>
- Troxel, W. M., Matthews, K. A., Gallo, L. C., & Kuller, L. H. (2005). Marital quality and occurrence of the metabolic syndrome in women. *Archives of Internal Medicine*, 165, 1022–1027. <http://doi.org/10.1001/archinte.165.9.1022>
- Van Baak, M. A. (2001). The peripheral sympathetic nervous system in human obesity. *Obesity Reviews*, 2, 3–14. <http://doi.org/10.1046/j.1467-789x.2001.00010.x>
- Wrangham, R. (2010). *Catching fire: How cooking made us human*. New York, NY: Basic Books.
- Wren, A. M., Seal, L. J., Cohen, M. A., Brynes, A. E., Frost, G. S., Murphy, K. G., . . . Bloom, S. R. (2001). Ghrelin enhances appetite and increases food intake in humans. *Journal of Clinical Endocrinology & Metabolism*, 86, 5992–5992. <http://doi.org/10.1210/jc.86.12.5992>
- Xu, S., & Lorber, M. F. (in press). Interrater agreement statistics with skewed data: Evaluation of alternatives to Cohen’s kappa. *Journal of Consulting and Clinical Psychology*. <http://doi.org/10.1037/a0037489>