Leptin concentrations in response to acute stress predict subsequent intake of comfort foods

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ABSTRACT

Both animals and humans show a tendency toward eating more "comfort food" (high fat, sweet food) after acute stress. Such stress eating may be contributing to the obesity epidemic, and it is important to understand the underlying psychobiological mechanisms. Prior investigations have studied what makes individuals eat *more* after stress; this study investigates what might make individuals eat *less*. Leptin has been shown to increase following a laboratory stressor, and is known to regulate satiety. This study examined whether leptin reactivity accounts for individual differences in stress eating. To test this, we exposed forty women to standardized acute psychological laboratory stress (Trier Social Stress Test) while blood was sampled repeatedly for measurements of plasma leptin. We then measured food intake after the stressor. Increasing leptin during the stressor predicted lower intake of comfort food. These initial findings suggest that acute changes in leptin may be one of the factors modulating down the consumption of comfort food following stress.

1. Introduction

Coincident with increased prevalence and severity of psychological stress in the general population [1,2], the prevalence of overweight and obesity has reached epidemic levels [3]. Understanding the connections between stress and obesity are critically important. Psychological stress is an increasingly well-established factor implicated in the development of obesity [4]. A key mechanism through which stress likely leads to weight gain and obesity is *stress-induced eating*. Stress-induced eating of palatable foods is conserved across species. Both humans [5–7] and animals [8] have been documented to increase their food intake following stress or negative emotion, even if the organism is not hungry [9,10]. Further, the type of food eaten tends to be high in sugar or fat, or both [11–13], commonly referred to as "comfort food."

Various psychological and other factors predispose people to stress-induced eating. For example, being female, overweight, or scoring high in dietary restraint (a measure of chronic dieting attempts) puts one at risk for increased food intake in response to a psychological laboratory stressor [5,14,15]. As for what *physiological* factors underlie stress-induced eating, prior research has focused on the stress-responsive hypothalamic–pituitary–adrenocortical (HPA) axis and glucocorticoids. In one study, for example, subjects who responded with the greatest amount of cortisol in response to a laboratory stressor, compared to a session with no stressor, consumed the greatest amount of high fat, sugary food [12]. Furthermore, glucocorticoid administration studies in humans and rodents have documented increased food intake as well as a shift in preference toward sweet and fatty foods [16–18]. Although some studies suggest that glucocorticoids directly increase preference of sweet and high fat food [17,18], stress affects the brain and bodily systems through multiple pathways [12,19,20] and accumulating evidence exists suggesting the role of other neuropeptide systems in modulating consumption during stress towards sweet and fatty foods [8,9,12].

Although understanding what triggers increases in eating after stress is important, it is equally important to understand what might protect an individual from stress eating. That is, we must also

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identify what physiological factors might underlie lower food consumption following stress.

One possible modulator of stress eating is leptin, the protein product of the *ob* gene [21–23]. Leptin circulates in the bloodstream, reflects the amount of fat stores, recent energy balance and dietary macronutrient composition [24–27]. Leptin provides a signal to the hypothalamus of body fat reserves and recent energy intake [21–23,28,29], and appears to act as a long-term hormonal signal in the regulation of energy homeostasis [30–33]. Leptin contributes to body weight regulation by affecting both feeding behavior and energy expenditure. In rodents, leptin upregulates thermogenesis in brown adipose tissue. Leptin also exerts effects within the hypothalamus, regulating homeostatic food intake [30–32], and in the ventral tegmental area, reducing dopamine neurotransmission and extinguishing the reward value of food [34]. Paradoxically, obese humans tend to have higher levels of leptin, suggesting a state of leptin resistance [21,22,31,32].

Circulating leptin concentrations increase 4–6 h after meals in response to nutrient-induced insulin secretion [27,35]. However, leptin may also be acutely responsive to psychological stress [12,36]. Brydon et al. [19] exposed subjects to psychological stress and measured plasma leptin concentrations at baseline, immediately after the stressor, and 45 min post-stressor. They found a small but significant increase of leptin, which peaked at 45 min following exposure to the stressor and was not correlated with cortisol response to the same task.

These stress-related changes in leptin may also modulate stress-induced eating. One study has examined leptin, stress, and eating together [37] and reported that higher circulating serum leptin concentrations over one day were related to less overall food consumption following stress compared with days on which the subjects were not exposed to stress. This study suggests that *tonic* serum concentrations of leptin may be linked to decreased stress-induced eating.

A remaining question is whether *acute changes* in leptin under conditions of stress might affect subsequent food intake, or acute stress eating. In the current study, we tested whether leptin modulates stress eating by exposing women to a standardized laboratory stressor, discreetly measuring their food choices and amount eaten following the stressor, and measuring leptin throughout the stress session. Given the prior research implicating leptin in decreased food consumption, we hypothesized that leptin responses to stress would be negatively associated with comfort food consumption. We also measured cortisol to assess whether leptin effects were independent of, or associated with, the known effects of cortisol on stress-induced eating behavior.

2. Materials and methods

2.1. Subjects

Sixty-three healthy, non-smoking, post-menopausal women aged 50 to 80 years were recruited throughout the San Francisco Bay area through flyers posted within the University of California, San Francisco (UCSF) campuses and hospitals and the community as well as advertisements in local newspapers and radio stations. The sample was homogeneous in terms of being all women, postmenopausal, and of a limited age range, which reduces variance in these factors which can alter leptin levels [21,23,28,29,38–41] and eating behavior [15,42–44]. To capture subjects with a wide range of psychosocial stress, the sample was comprised of caregivers of family members with dementia as well as BMI- and age-matched control subjects. Forty of these subjects took part in the laboratory stress session. Exclusion criteria also included the presence of metabolic or endocrine disease such as diabetes, substance abuse, medications known to affect hormones (e.g. glucocorticoids), current major injuries or illness, and current bipolar or eating disorders.

2.2. Procedures

All procedures were approved by the UCSF Committee on Human Research and all subjects provided written informed consent. Subjects were screened for eligibility over the telephone and in person. To confirm eligibility, fasting laboratory tests ensured normal liver, kidney and thyroid function, and glucose levels.

Subjects completed two visits at the UCSF Clinical and Translational Science Institute Clinical Research Center (CTSI-CRC). They were asked to refrain from physical activity and consuming alcohol or caffeine after midnight prior to their first appointment. During the first visit a fasting blood sample was collected shortly after arrival to assess fasting leptin levels. All subjects' blood was collected between 0730 h and 0830 h to minimize any potential impact of the diurnal pattern of leptin [45]. Body weight, height, and waist and hip circumferences were also assessed. Subjects were then scheduled to return a week later. Questionnaires assessing sociodemographic variables and individual difference measures (see measures below) were handed out to the subjects to be completed at home and returned during the second visit.

At the second visit, subjects were provided with a standardized lunch at noon to equalize the amount of food intake prior to the experimental session. The caloric content of every subject's lunch was identical and was prepared by the CTSI-CRC metabolic kitchen. An indwelling forearm catheter was inserted and subjects rested for 1 h, followed by a brief questionnaire during which current negative affect and current hunger were assessed. Next, a modified Trier Social Stress Test (TSST) [46] was used to expose subjects to a standardized psychosocial stressor and assess circulating leptin responses to stress. Three blood samples were collected throughout the stress task and recovery period for measurement of leptin: 0 min (baseline), 50 min after the onset of the stressor, and 90 min after the onset of the stressor. Salivary samples to measure cortisol were taken at 0, 15, 20, 30, 50, and 90 min post-stressor onset [46]. All subjects were tested individually between the hours of 1415 h and 1715 h to limit the diurnal variation in HPA axis activity and leptin levels. Immediately following the laboratory stressor, negative affect and hunger were again assessed. Then subjects were moved to a break room with a snack buffet for a 30-min period during which food intake was covertly measured.

2.3. Stress manipulation

The modified TSST was a 50-min session beginning with four 5-min stressful periods (1) introduction to two "trained evaluators" and receiving instructions for the task, (2) a preparation period in which subjects were asked to prepare a 5-min speech on their "personal strengths and weaknesses," (3) delivering the speech, and (4) a challenging serial subtraction task. The evaluators were trained confederates who kept neutral facial expressions during the task performance and used a set of standardized comments to increase stress and ensure that each subject experienced the task as demanding. The tasks were followed by 30 min of sitting quietly. This test has been shown to reliably result in a short-lived increase of psychological stress with physiological manifestations (i.e., cortisol secretion) and has been used in several previous studies examining the role of stress in eating behavior [9,12].

2.4. Snacking session

During the snacking session (approximately 30 min after the stress session and 3 h since their last meal) subjects were left alone in a room with leisure reading material for 30 min and also had access to an array of snacks from which they could choose and eat as much

as they wanted. Subjects were not required to but merely invited to eat, and were not aware that their food intake was being measured.

To assess food choice with regard to sweet, salty, low-fat, and high-fat foods, four categories of snack choices were presented on a large platter, without individual packaging (see Table 1). Each serving of different food items was separately weighed to the nearest 0.1 g before and after the subject had left the room to assess how much they consumed. If subjects did not eat the food items in the laboratory but took them home, their data were not analyzed. This study design is a common paradigm to assess food choices in humans after laboratory stressors [10,12,15].

2.5. Measures

2.5.1. Body mass index (BMI)

Body weight was measured on a digital scale with subjects in light clothing without shoes. Height was measured to the nearest of 0.1 cm using a Harpenden stadiometer. BMI was calculated as weight in kilograms divided by height in meters squared.

2.5.2. Leptin

Plasma leptin samples during the stressor were obtained from blood samples collected via the inserted indwelling catheter. Blood samples were centrifuged, aliquoted and stored in polypropylene vials at $-80\,^\circ$C until analysis. Samples were assayed with a radioimmunoassay kit using a ^{125}I-human leptin tracer and human leptin standards from Linco Research, Inc. (St. Charles, MO, USA) in Dr. Havel's laboratory (UC Davis, CA, USA). The intra- and interassay variations of the assay were 6.6% and 12.0% respectively. Changes of circulating leptin during the stress session were calculated according to the area-under-the-curve with respect to increase (AUCi) formula according to the recommendations of Pruessner et al. [47] to calculate time-dependent leptin secretion. In other words, we examined leptin secretion over the test period controlling for baseline leptin values, and label this throughout as "leptin reactivity."

2.5.3. Cortisol

Salivary cortisol strongly reflects levels of serum cortisol and indexes the amount of free or biologically viable cortisol [48]. To collect salivary samples, subjects were asked to drool passively through a straw into a tube, which then were kept on ice and then frozen at $-20\,^\circ$C degrees. Samples were sent for batch assay to Dr. Kirschbaum's laboratory (Dresden, Germany). Salivary cortisol was assayed with a chemiluminescence immunoassay using a commercial kit (IBL; Hamburg, Germany). The intra-assay CV was 2.9% for high levels and 7.7% for low levels. The inter-assay CV was 5.7% for high levels and 9.1% for low levels. The sensitivity lower limit was 0.006 μg/dl. As with leptin, we calculated cortisol secretion during the stress session using AUCi.

2.5.4. Sociodemographic, individual difference, and psychological measures

Information on age and ethnicity was obtained by self-report from the subjects. The number of lifetime diets (resulting in at least a five pound loss of weight) and currently being on a diet (yes/no) were assessed in the questionnaire packet the participants completed at home between the first and second visits. Prior to and immediately after the stressor, ratings of negative affect were obtained as part of a manipulation check using six negative emotions taken from the Affect Balance Scale [49]. The items were answered on a 4-point Likert-type scale with $0=$ "not at all" and $4=$ "a great deal." A total score was obtained as sum of the items (Cronbach's $\alpha=.79$). Ratings of current subjective feelings of hunger were assessed on a 4-point Likert-type scale ($0=$not hungry at all, $4=$a great deal), prior to and after the stressor. To disguise our interest in subjects' specific food intake, the item assessing current hunger was interspersed among the mood ratings.

2.5.5. Food intake

Food intake was quantified as the weight of each food item consumed in grams. To obtain a measure for each food category, the sum of the weight of consumed food items within each category was calculated. Both total consumption and category-specific (i.e. high fat, high sugar) consumption were used as the primary dependent measures.

2.6. Statistics

Statistical analyses were performed using SPSS for Windows 15.0. (SPSS Inc., Chicago, IL, USA). Pearson correlation coefficients were calculated to test the association of outcome variables with potential confounding variables. Paired sample t-tests were computed to perform a manipulation check to confirm that the stressor increased cortisol and ratings of negative affect. The Wilcoxon Ranked Sum Test was used to test whether subjects´ subjective ratings of hunger differed significantly pre- and post-stressor. A one-way Repeated Measure Analysis of Variance was used to investigate whether leptin levels at each timepoint during the stressor differed significantly from each other. Partial correlations were calculated to investigate the relationship between cortisol and leptin reactivity controlling for potential confounding variables and to determine whether leptin reactivity significantly predicted the amount of food consumed. All leptin analyses controlled for BMI.

3. Results

3.1. Subject characteristics

Forty women completed the laboratory stressor session with complete leptin data. One subject had a fasting leptin concentration greater than four standard deviations above the mean and was excluded from statistical analysis, leaving 39 women. Ten women did not undergo the eating session, leaving 29 women for whom complete data were available. The average age was 62 years ($SD=6.33$; range: 51–79) and the average BMI was 26.2 and ranged from lean to obese ($SD=5.3$; range: 17.7–37.5). In terms of BMI categories, 3.3% were "underweight," 40% were "normal" weight, 33.3% were "overweight," 16.7% were "obese," and 6.7% were "morbidly obese." The majority of subjects self-identified as white (85%), with the remaining 13% Asian/Pacific Islander/Native American, and 3% African American.

Table 1
Food types and amounts served for post-stressor eating behavior.

	Sweet	Salty
High fat	Chocolate chip cookies (114 g)	Cheese (cheddar, Swiss, provolone) (80 g)
	Chocolate chip ice cream (500 g)	Potato chips (36 g)
Low fat	Fruit (grapes, apples) (500 g)	Pretzels (36 g)
	Animal crackers (38 g)	

Note: Gram values correspond to two pre-packaged serving sizes of each food category.

The women in this sample had a mean fasting leptin concentration of 18.8 ng/ml (SD = 14.6; range 2.5–55.0 ng/ml), which is within the normal physiological range of leptin levels [41]. The majority of the subjects (82%) reported to not be on a diet currently. Of the individuals on a diet, 50% were normal weight, 33.3% were overweight, and 16.7% were obese. The inclusion of caregivers and control subjects resulted in a moderate range of perceived stress scores of 0 to 27 out of a maximum of 30. Being a caregiver had no effect on any of the analyses and thus the two groups were combined.

3.2. Preliminary analyses and manipulation check

Q-Q plots indicated leptin and cortisol values at all time points were not normally distributed, and this was corrected via a natural log transformation. As expected, fasting leptin concentrations were positively correlated with BMI ($r = .88$, $p < .001$). No significant correlation was found between fasting leptin levels and age ($r = .16$, $p = .32$) or current dieting ($r = -.05$, $p = .75$).

The stress manipulation was successful—comparison of group means revealed that salivary cortisol levels increased from baseline, reached peak levels 30 min after the onset of the stressor and returned to baseline levels by 90 min after the onset of the stressor. Further, a t-test showed cortisol levels at peak were significantly higher than cortisol levels at baseline before the onset of the stressor, indicating that the stress manipulation elicited the intended stress reactivity response ($t = 8.91$, $p < .001$). In addition, subjects reported a significant increase in negative affect from baseline to the post-stress session ($t = 7.30$, $p < .001$) (see Table 2).

3.3. Food intake

Characteristics of the distribution of food intake measures are summarized in Table 3. In general, subjects ate more sweet food than salty food (78% vs. 22%), and more high fat sweet food (34%) than high fat salty food (17%); while few ate low fat salty food (4%; see Table 3 for details). Hunger ratings at baseline (before the stressor) did not correlate with any measures of type or quantity of food intake (all $p > .05$). This lack of correlation, and that each subject ate a standardized meal before the stressor, suggests that the snacking was non-homeostatic—not related to caloric deprivation. Hunger ratings remained stable during stress (no changes from pre to post-stress task, $U = 0.00$, $p = .99$).

Table 2
Descriptive statistics of Physiological and psychological changes during lab stressor.

	Baseline	30 min post-stressor	50 min post-stressor	90 min post-stressor
Physiological measures				
Cortisol (μg/dl)				
Mean	4.77	7.65	6.70	4.91
Standard deviation	2.35	4.70	4.20	2.32
Minimum–maximum	2.40–10.54	1.95–21.59	0.89–18.73	0.98–11.71
Leptin (ng/ml)				
Mean	15.4		15.7	16.1
Standard deviation	10.8		11.4	11.4
Minimum–maximum	3.3–40.2		3.4–41.5	3.4–43.8
Psychological measures				
Negative affect				
Mean	5.40	7.60		
Standard deviation	3.80	6.30		
Minimum–maximum	0–12	0–22		
Subjective hunger ratings				
Mean	0.16	0.16		
Standard deviation	0.70	0.70		
Minimum–maximum	0–4	0–4		

Table 3
Characteristics of distribution of food intake during the snacking session.

Food type consumed (g)	Mean	SD	Min	Max	% eaten
Total amount of food eaten	265.4	146.7	0	641.2	100%
Total sweet	211.0	130.8	0	542.2	78%
High fat sweet food	107.2	98.1	0	300.0	34%
Low fat sweet food	103.9	65.3	0	289.0	44%
Total salty	54.4	38.7	0	124.0	22%
High fat salty	46.1	36.9	0	122.0	18%
Low fat salty	8.3	10.0	0	34.0	4%

3.4. Leptin during stress

Characteristics of the distribution of circulating leptin concentrations during the stressor and during recovery are displayed in Table 2.

We first examined the relationship of leptin during stress to potential confounding variables including fasting plasma leptin concentrations, BMI, age, current dieting, and baseline hunger ratings. Leptin levels at all three time points (baseline, 50 min, and 90 min post-stressor) during the stressor were significantly correlated with fasting leptin concentrations ($r = .96$, .98. and .97, respectively, all $p < .001$), and BMI ($r = .86$, .85, and .85, respectively, all $p < .001$). No significant correlation was found between age, current dieting and baseline hunger ratings and leptin levels at all three time points (all $p > .05$).

Across the entire sample, there were mean increases of leptin concentrations from baseline to 50 min post-stressor onset ($M = 0.30$, $SD = 1.64$) and from 50 min to 90 min post-stressor onset ($M = 0.40$, $SD = 1.80$). However, neither of these increases was statistically significant ($p > .05$). Subjects showed great variation in leptin reactivity post-stress, ranging from -5.48 to $+4.30$ ng/ml. Leptin reactivity was not significantly correlated with cortisol reactivity controlling for the potential confounding variables, BMI and age (all $p > .05$). Further, leptin reactivity was not significantly correlated with BMI, fasting leptin levels, age, baseline hunger ratings or current dieting (all $p > .05$).

3.5. Leptin reactivity during stress session and intake of high fat/high sugar foods

Next we tested whether leptin reactivity was related to less intake of high fat/high sugar "comfort" foods. The average AUCi of natural-logged leptin was 1.05 ($SD = 5.55$) and ranged from -13.99 to 12.51. Leptin reactivity across the test session was negatively related to high fat sweet food intake ($r = -.40$, $p < .05$). Leptin reactivity was not related to intake in any of the other food categories (see Table 4).

3.6. Leptin reactivity and individual differences in stress and dieting

Because leptin reactivity was related to consumption of high fat/high sugar foods, we conducted analyses to examine factors that might potentially contribute to individual differences in leptin reactivity. Specifically, we examined leptin reactivity in relation to two variables that, based on prior literature, might contribute to differences in leptin reactivity: the perceived stressfulness of the task [19] and the number of episodes of dieting the participants engaged in throughout adulthood [22,29,50]. Neither leptin responses during nor after stress were significantly associated with these variables (all $p > .05$).

4. Discussion

Stress-induced eating behavior likely plays an important role in the current obesity epidemic [36]. Although the underlying physiological mechanisms are largely unknown, preliminary initial evidence implicates leptin in the phenomenon of stress-induced

Table 4
Correlations between grams of food eaten of each food category and leptin reactivity.

	Leptin reactivity	p
High fat sweet	**−.40**	**.03**
Low fat sweet	.33	.08
High fat salty	−.03	.89
Low fat salty	.26	.17
Total food	−.11	.59

eating [36]. The present study investigated whether leptin may be one of the physiological factors that modulate stress-induced eating behavior, which is the characteristic shift in food preference towards high fat and high sugar foods following exposure to stress. We found that leptin increases from baseline in response to acute stress were significantly related to lower consumption of high fat, sweet foods—"comfort" foods. This association between leptin reactivity and subsequent food intake appeared to be specific for high fat sweet food intake and no significant relationship was found between leptin reactivity and any other food category. However, we note that there was a smaller marginal relationship between leptin reactivity with greater intake of low fat sweet food, and the potential distinction between the two patterns should be re-examined in replicative research. Studies on leptin and salty food consumption is scarce. Our findings appear in line with results reported by Kawai and colleagues [52] who administered leptin into lean mice and found changes in taste nerve responses to sweet but not sour, salty, or bitter substances. More work needs to be done to delineate the effects of leptin on non-sweet substances. However, in studies of rodents [8] and humans [12,13], stress appears to predominantly affect sweet food intake (rather than chow or salty foods, respectively). Sweet in the presence of high fat may be a particularly relevant outcome measure in this specific context of stress eating.

Moreover, in the present study morning fasting leptin levels did not predict food intake following the stressor, underscoring the relevance of leptin *reactivity* as a novel correlate and potentially important predictor of stress eating, especially since this marker was found to be independent of BMI and fasting leptin concentrations.

Our result differs from the pattern of findings reported by Appelhans [37], who found that higher daily leptin concentrations were related to less total food consumption following stress but not sweet high fat food intake specifically as found in the present study. This may have been a consequence of the different leptin measures the two studies used. Rather than measuring diurnal circulating leptin values, we used leptin measures during stress, again emphasizing leptin reactivity as a potentially more relevant marker implicated in stress eating. As leptin is an energy homeostatic hormone that reduces food intake by acting centrally within the hypothalamus and the ventral tegmental area, extinguishing the reward value of food, less high fat, sweet food intake following an acute increase in leptin during stressful periods in the present study might be a consequence of a dampened comforting effect of these foods in stressful situations [30,31,51].

At the group level, exposure to the psychological laboratory stressor did not significantly increase mean leptin levels during or after the stressor. This is in contrast with Brydon et al. [19] who, with a larger sample, reported a small but significant increase in leptin levels following a similar laboratory psychosocial stressor. Our inability to replicate these findings may in part be due to a smaller sample size and a higher average age of the participants in the present study, or the wide range of leptin responses in our study, with half of the participants showing an increase of leptin during stress and others showing a decrease in leptin levels during stress. Our study suggests that a pattern whereby leptin does not increase during stress may lead to greater high fat, sweet food intake, and this underscores the importance of further studying the factors that determine whether leptin increases or decreases acutely

following stress. In this initial study, we appear to have ruled out the perceived stressfulness of the task and the number of episodes of previous dieting as possible influencing variables predicting acute leptin changes during stress.

There was no correlation between leptin and cortisol reactivity, which is in line with findings of by Brydon et al. [19]. Cortisol (in combination with insulin) has been implicated in prior studies of stress eating [53] but our data suggest that any leptin effect on food intake may be an independent process that is not driven by cortisol. This could be even more precisely pinpointed in future studies by examining stressors that do not engage the HPA axis, such as noise exposure [54], and linking leptin changes to eating without any concomitant increases of cortisol.

A number of limitations of our findings warrant mention. As this study was conducted solely in postmenopausal women and in a laboratory setting, these results cannot readily be generalized to other populations or to other less-controlled situations. Moreover, our sample was small, which reduced both our power and generalizability. We cannot rule out the possibility that subjects may have been aware that their food intake was being monitored. We demonstrate correlation, which does not infer directionality or mechanism. This study did not include an unstressed control group, which leaves the possibility that the reported pattern of leptin change is unrelated to stress and may have occurred under restful conditions over the afternoon in some subjects. Further, our inter-assay variation was larger than our observed effect (12%), suggesting that future studies should examine changes of circulating leptin in response to acute stress and compare them with leptin responses during a control session on a separate day to rule out that the observed pattern occurred by chance. We did not have measures of sympathetic nervous system (SNS) activity in this study. The SNS is activated by stress and is a regulator of leptin production [55] (and vice-versa; leptin is a potent stimulator of the SNS [56]), and future studies are needed to determine whether the SNS might drive or modulate the effects seen here. Ghrelin, a peptide that has been shown recently to mediate stress eating in mice [57], similarly was not measured, and might also be a key component driving stress eating, either alone or in concert with leptin, in humans.

4.1. Summary

This study is significant in that it documented the heterogeneity of leptin responses to acute stress. Increases of circulating leptin concentrations during a psychological laboratory stress task predicted less consumption of high fat, high sugar foods but not other types of foods. This suggests that leptin reactivity may be implicated in reducing stress-induced eating behavior. The relationship between leptin activity and stress-induced eating behavior appear to be independent of cortisol reactivity.

We speculate that leptin may be acting as a modulator of stress eating. When an individual has an adaptable, flexible allostatic stress response that is sensitive enough to upregulate leptin secretion in response to stress, that individual may not fall prey to the drive to consume comfort food. When the system does not respond, meaning that leptin reactivity is low or absent, comfort food eating may be more easily triggered. In sum, this study implicates circulating leptin reactivity in potentially dampening the known shift in food preference to high fat, sweet food following exposure to stress, and points towards its potential as an independent modulator of stress eating. Leptin responses to acute stress show a complex pattern, of which the exact nature, cause and underlying mechanisms remain to be determined.

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