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Stress may add bite to appetite in women: a laboratory study of stress-induced cortisol and eating behavior

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Abstract

To date, there are few known predictors of stress-induced eating. The purpose of this study was to identify whether physiological and psychological variables are related to eating after stress. Specifically, we hypothesized that high cortisol reactivity in response to stress may lead to eating after stress, given the relations between cortisol with both psychological stress and mechanisms affecting hunger. To test this, we exposed fifty-nine healthy pre-menopausal women to both a stress session and a control session on different days. High cortisol reactors consumed more calories on the stress day compared to low reactors, but ate similar amounts on the control day. In terms of taste preferences, high reactors ate significantly more sweet food across days. Increases in negative mood in response to the stressors were also significantly related to greater food consumption. These results suggest that psychophysiological response to stress may influence subsequent eating behavior. Over time, these alterations could impact both weight and health. © 2000 Elsevier Science Ltd. All rights reserved.

Keywords: Stress; Cortisol; Eating; Taste; Mood; Dieting

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1. Introduction

Emotional arousal has been associated with both increased or decreased food intake and weight (Stone and Brownell, 1994; Willenbring et al., 1986), but little is known about the mechanisms that determine the direction of change. Understanding predictors of stress-induced eating is important, as stress can trigger relapses of both obesity (Rand and Stunkard, 1978) and bulimic episodes (Lingswiler et al., 1989). One's physical response to stress may help explain why some tend to eat while others lose their appetite after stress. The purpose of this study was to identify whether stress reactivity, both biological and psychological responses, distinguish stress over-eaters from under-eaters. Specifically, we hypothesized that high cortisol reactivity may lead to eating in response to stress, given the relations between cortisol with both stress and mechanisms affecting hunger.

Cortisol clearly plays an important role in energy regulation, increasing available energy through gluconeogenesis and lipolysis. In animals prone to obesity (either genetically or by brain lesions), glucocorticoids lead to hyperphagia and weight gain, and are necessary for the expression of their obesity (Bray, 1985). Adrenalectomy and glucocorticoid receptor antagonists prevent or reverse obesity (Okada et al., 1992), whereas administering corticosterone leads to increased appetite for sucrose (Bell et al., 2000), hyperphagia, and weight gain (Flatt, 1989).

In humans, stress-induced cortisol may also play a role in obesity. There are a few demonstrations that cortisol affects eating in humans. In cancer patients, prednisolone significantly increased appetite, compared to a control group (Willox et al., 1984). In healthy men, administering cortisol for four days led to slightly increased energy expenditure but dramatically increased food intake (Tataranni et al., 1996). Given these associations between exogenously administered cortisol, food intake, and obesity from animal and human studies, we predicted that endogenous cortisol release, stimulated by stress, may help explain stress-induced eating.

Macronutrient selection may also be altered by stress. Women tend to prefer high fat or sweet foods when moderately stressed (Grunberg and Straub, 1992; Klein et al., 1996). There is much evidence that adrenal steroids influence macronutrient selection, by increasing appetite for carbohydrates, primarily, and for fat, and regulating the timing of eating in rodents (McEwen et al., 1993; Tempel and Leibowitz, 1994). We wanted to test similar associations between cortisol and food intake in humans. We predicted that those who secreted more cortisol in response to stress would tend to eat more calories, as well as choose sweet or high fat food.

2. Method

2.1. Study participants

Fifty nine healthy pre-menopausal women aged 30 to 45 years were recruited as part of a study on habituation to stress and body shape. We followed exclusion criteria to eliminate women with factors that would affect cortisol reactivity, such

as oral contraceptive use and menopause. Other exclusion criteria were current smoking (and having smoked 10 or more cigarettes a day within the last two years), regular alcohol (more than seven drinks a week) or medication use, major depression, eating disorders, and endocrine or metabolic disorders. To assess the presence of any of these exclusion criteria, we relied on self-report in a phone interview. In addition, when they came to our office, we again discussed the exclusion criteria. To better assess the presence of depression or an eating disorder, we administered the BDI and the EAT (described below) during the eligibility visit. Seven women scored in the potentially clinical range on the EAT (>20), and were excluded from analyses.

Eating behavior can be affected by many factors, including dieting, age and gender. Young people tend to eat more than older people, and women tend to report more stress-induced eating than men (Greeno and Wing, 1994). Given the many factors affecting both cortisol and eating behavior, we focused on pre-menopausal women, and excluded younger women (under 30 years).

2.2. Procedure

Women were exposed to four consecutive days of three-hour laboratory sessions, starting between 4.00 and 5.30 pm each day. Women began the sessions within the first five days of the follicular stage of their menstrual cycle. The first three sessions were stressful sessions, and the fourth was a rest or control session. Only the data from the first stress session and the control session were used, as we were interested in exposure to a novel stressor, rather than habituation to stress. Participants were instructed to eat a snack one hour before coming into the laboratory, and to refrain from further food and drink (excluding water) for the remainder of the hour before the session. They filled out a behavioral questionnaire, assessing the timing of eating and drinking before each session to confirm that they followed directions.

Salivary cortisol samples were collected at the same time intervals throughout each session, during a half hour baseline period (at 15 and 30 minutes), before stress (45"), during stress (60", 70"), at the cessation of stress (90"), and two recovery samples 30 and 60 minutes after stress. During the three stress sessions, participants were exposed to the same psychosocial challenges, an adapted version of the Trier Social Stress Test (Kirschbaum et al., 1993). Participants were exposed to 45 minutes of stress, including performing three challenging tasks, designed to be stressful by giving unrealistic time constraints to meet the expected goals: (1) visuospatial puzzles, (2) serial subtraction of a prime number from a high number, and (3) deliverance of a videotaped speech, with a supposed research committee evaluating her behind a one-way mirror. For the rest session, they sat quietly, reading and listening to music.

After the stressors, (and on the rest day, after the reading), participants were given a basket of snacks, and left in the room with the snacks for half an hour, with leisure reading material. Participants were not pressured to eat but merely invited to eat. Participants were not aware that we were studying their food intake. To account for individual preferences for sweet versus salty snacks, and high versus low fat food,

four snack choices were provided, in standard pre-packaged serving sizes. The snacks included two higher fat sweet and salty snacks — chocolate granola bars (39 grams) and potato chips (28 grams) and two low-fat sweet and salty snacks — flavored sweetened rice cakes (30 grams) and salty pretzels (28 grams). One serving size of each type of food was presented in a basket, four servings total. Each woman was told she could request additional servings, but very few did.

After participants left the laboratory, the amount of each snack eaten was assessed. Each serving was weighed both before and after the session to assess the amount of the serving eaten. This amount of the serving eaten was rounded off to the nearest quarter of the serving. The macronutrient content of each snack was determined using the nutritional labels. The total caloric intake for each participant was calculated. Because of the large difference in calories between high fat and low fat food, caloric consumption was not analyzed by food type. Rather, when analyzing macronutrient selection, we assessed amount of servings eaten rather than calories.

2.3. Measures

Mood reactivity was measured using the three negative affect scales of the Profile of Mood States (POMS) (McNair et al., 1981), including depression/dejection, anger/hostility, and tension/anxiety. For each scale, a “residual gains” was calculated, to assess changes in mood from time 1 to time 2 (i.e., saving residual after regressing time 1 mood from time 2 mood). The three residual gain scores from each subscale were averaged. Higher scores represent increases in negative mood.

Dietary restraint was measured using the Eating Attitudes Test (Garner et al., 1982). This measure included subscales for dietary restraint, bulimic symptoms, and oral control. The oral control scale was not internally consistent for this sample so it was not used (Cronbach’s $\alpha < 0.70$). The dietary restraint scale was used as it is relevant to stress-induced eating.

Salivary cortisol strongly reflects levels of serum cortisol (Kirschbaum and Hellhammer, 1989). Saliva samples were collected with salivettes (Sarstedt, Rommelsdorf, Germany), plastic vials with cotton dental rolls inside, and frozen until laboratory analysis. They were assayed with a radioimmunoassay by the Yale Medical School Clinical Research Center (CRC) core laboratory, using a commercial kit (Diagnostic Products Corporation, Los Angeles, CA). Intra-assay coefficients of variation were 4.8% for low concentrations and 5.1% for high concentrations of salivary cortisol. The inter-assay coefficient of variation was 4%.

“Cortisol reactivity” refers to total cortisol output on the stress day, calculated as area under the curve (AUC, in $\mu\text{g}/\text{dl} \cdot \text{minutes}$). Two women had out of range cortisol levels. In order to include them in the analyses, we used a non-parametric analysis of reactivity; Reactivity was assessed by a median split of AUC, categorizing women into high (above $26.9 \mu\text{g}/\text{dl} \cdot \text{min}$, $n=25$) and low (below or equal to $26.9 \mu\text{g}/\text{dl} \cdot \text{min}$, $n=23$) reactor groups.

2.4. Analytic technique

We analyzed eating behavior in two separate ANOVAs, one for actual calories consumed and one for food type preferred. We used a repeated measures ANCOVA, examining reactivity group (high and low) and time (stress and control days), controlling for both body mass index and dieting behavior. We examined main effects, two-way interactions with reactivity, and 3-way interactions with time. To assess direct relations between calories with mood and dieting, we performed correlations.

Given that obese women may be qualitatively different in their eating and cortisol reactivity, we first compared them to lean women on these measures. Women were categorized into two weight groups, average weight (BMI < 25, $n=30$), and an overweight group (BMI > 25, $n=23$). They were similar in both stress reactivity (lean women's AUC=25.5, SE=1.6, vs. obese women's AUC=28.3, SE=2.3) and amount eaten after stress (for lean, average servings=1.5, SE=1.8, for overweight, average servings=1.4, SE=0.22). There were no correlations between BMI and amount eaten. BMI group was entered into the ANCOVA of calories consumed, to see if there was a main effect of BMI or interactions between BMI and reactivity. There were no effects of BMI group. Therefore, overweight and lean women were analyzed together, and BMI was simply used as a covariate.

3. Results

3.1. Descriptive data

The sample was an average of 36 years old (SE=0.70, range=30 to 46). Forty four percent were single. They had an average of 16 years of education (SE=22, range 12 to 17 years). Their average annual household income was \$35,537 (SE=\$3334). The average BMI was 25.4 (SE=0.62, range 19 to 39.6). The average score on the EAT, summing across the three subscales, was 10.2, SD=7.7, which is similar to a comparison group of healthy female university students (mean=9.9, SD=9.2), and lower than bulimic and anorexic samples (36.1, SD=17) (Garner et al., 1982). Scores on the dieting and bulimia subscales are presented in Table 1. These scores were slightly lower than those of a female comparison group of university students, showing less dieting and bingeing behavior in the current sample, and much lower levels than a sample of eating disordered patients. Although the average EAT scores were

Table 1
Means of eating attitudes of sample

	<i>M</i> (SE)	Range
Dieting	5.32 (0.72)	0 to 25
Bulimia	0.13 (0.04)	0 to 1.80
Total scale	10.20 (1.00)	2 to 39

normal, there was nevertheless a wide range of scores on dieting, so dieting is controlled for in analyses of amount eaten. As previously stated, seven women scored above 20 on the EAT, which could indicate disordered eating, and were excluded. These women were similar in BMI but ate marginally significantly less than the rest of the sample (only 1.0 serving vs. 1.5 serving on the stress day, and only 0.66 vs. 1.4 on the rest day).

3.2. Manipulation check of stress

A manipulation check on mood change during the stressor confirmed an increase in negative mood. This was due to a selective increase in anxiety, as there was no average change in depression or anger. On the rest day, there were significant decreases in all three negative moods (See Table 2). Cortisol (measured as AUC) was significantly higher on the stress day ($M=28.6$, $SE=1.7$) than the rest day [$M=22.6$, $SE=1.5$, $t(54)=3.1$, $P<0.01$]. The actual cortisol levels during the stress and rest day are shown in Fig. 1. The cortisol levels of high and low reactors during stress are shown in Fig. 2.

3.3. Dieting and mood

We expected negative mood to relate to greater consumption, and dieting to lower consumption. We first examined partial correlations between average negative mood with calories consumed on each day, controlling for dieting and BMI. Increases in average negative mood during the stress session were related to food intake after stressors ($r=0.32$, $P<0.05$), whereas increases in average negative mood during the control session were not significantly related to intake ($r=0.24$, $P=0.13$).

Dieting was not related to consumption after stress ($r=-0.14$, $P=0.37$), but was significantly related to lower consumption after resting on the control day ($r=-0.32$, $P<0.05$). These correlations were unchanged when controlling for BMI.

Table 2
Negative mood reactivity

	Pre-stress <i>M</i> (SE)	Post-stress <i>M</i> (SE)	Paired <i>t</i> -tests
Stress day			
Anxiety	0.84 (0.08)	1.33 (0.12)	$t(59)=-5.0$, $P=0.0001$
Anger	0.40 (0.07)	0.40 (0.08)	$t(59)=-0.1$, $P=0.96$
Depression	0.50 (0.10)	0.46 (0.09)	$t(59)=0.5$, $P=0.50$
Control day			
Anxiety	0.41 (0.05)	0.25 (0.04)	$t(59)=4.6$, $P<0.0001$
Anger	0.17 (0.05)	0.09 (0.03)	$t(59)=2.2$, $P<0.05$
Depression	0.24 (0.05)	0.18 (0.05)	$t(59)=2.2$, $P<0.05$

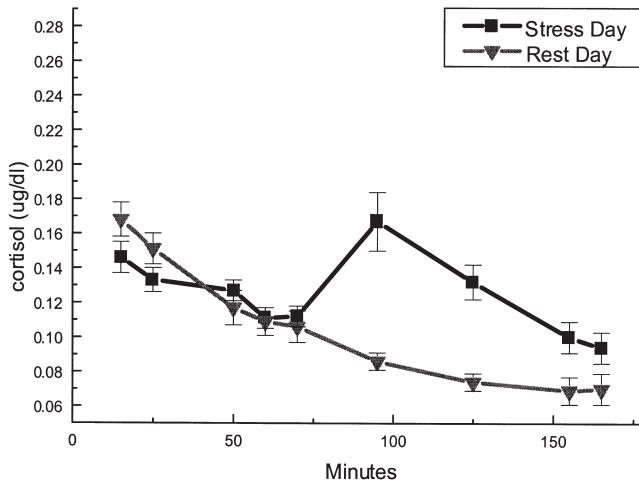


Fig. 1. Cortisol profiles on stress and control sessions across sample.

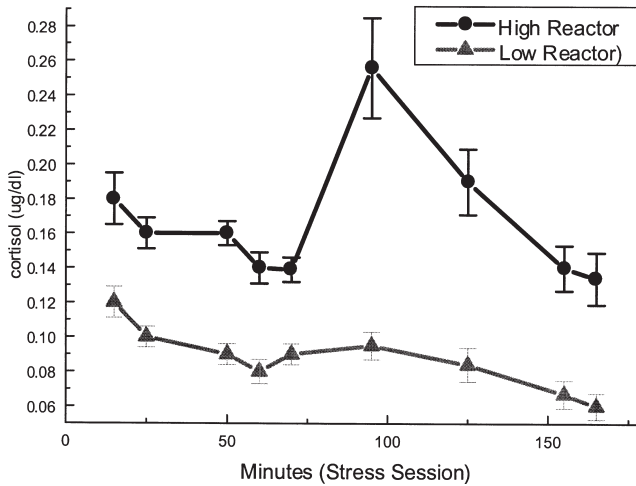


Fig. 2. Cortisol reactivity profiles of high and low reactors during stress session.

3.4. Description of food intake

The average amount of calories eaten after the stressor was 169 kcal (SE=19). The average amount of calories eaten on the rest day was similar ($M=170$ kcal, SE=19). The raw values for servings of each type of food consumed on each day

is shown in Table 3. There were no differences in food selection based on day. There was no significant preference for high or low fat food on either day across the sample, although there was a preference for sweet food across days, described below.

From these descriptive results, it appears there was no effect of the stress manipulation on eating for the sample as a whole. However, once we examine the individual difference variable of cortisol reactivity, we observe that high and low reactors had different eating behavior on the stress day, as shown below. The correlation between amount eaten on stress and control days across the sample was 0.53 ($P < 0.0001$). This correlation reflects a moderate degree of stability in eating behavior across the two sessions while at the same time shows there were individual differences in amount eaten between the days

3.5. Calories consumed

First we tested the effect of reactivity on total calories consumed (across all food types), in a 2 (time: stress and control day) \times 2 (reactivity: high and low cortisol reactors) ANCOVA, controlling for BMI and dieting. There was a significant interaction between time and reactivity [$F(1,42)=4.9$, $P < 0.03$]. Examination of the means showed that on the stress day, high reactors consumed more calories (calories $M=216.3$, $SE=29$) than low reactors (calories $M=137.3$, $SE=31.8$). On the control day, however, high reactors consumed similar amounts (calories $M=176.7$, $SE=27$) as low reactors (calories $M=187.2$, $SE=29.9$) (Fig. 3). When number of servings was examined instead of calories, a similar interaction emerged between reactivity and time, showing that high reactors ate significantly more on the stress day but not on the rest day, compared to low reactors.

We also examined partial correlations between cortisol measures (AUC, change from baseline to immediately after stress and to 30 minutes after stress) and total

Table 3

Adjusted \dagger means of serving sizes eaten for each food type on stress and control days by reactor group

		Stress day, amount of serving M (SE)		Control day, amount of serving M (SE)	
		High reactors	Low reactors	High reactors	Low Reactors
Sweet food	High fat	0.54 (0.09) ^a	0.23 (0.10) ^b	0.47 (0.10)	0.30 (0.10)
	Low fat	0.51 (0.10)	0.50 (0.11)	0.51 (0.10)	0.30 (0.11)
Total sweet food		1.07 (0.13)	0.76 (0.13)	0.95 (0.14)	0.67 (0.15)
Salty food	High fat	0.39 (0.09)	0.17 (0.10)	0.25 (0.10)	0.45 (0.11)
	Low fat	0.28 (0.10)	0.40 (0.11)	0.13 (0.08) ^b	0.46 (0.09) ^a
Total salty food		0.65 (0.13)	0.49 (0.14)	0.37 (0.11) ^b	0.83 (0.12) ^a
Total servings eaten		1.75 (0.21)	1.27 (0.22)	1.36 (0.19)	1.51 (0.21)

\dagger Means are adjusted for BMI and dieting. Means marked with ^a are significantly greater than means marked with a ^b.

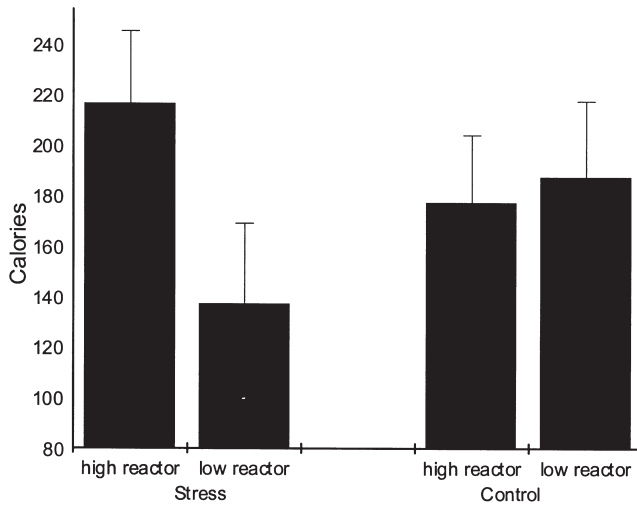


Fig. 3. Raw mean calories consumed by reactivity group on stress and control days.

calories consumed, controlling for dieting and BMI. Total calories consumed on the stress day was significantly related to change in cortisol after stress ($r=0.30$, $P<0.05$) but weakly related to AUC ($r=0.25$, $P=0.09$) and not related to change in cortisol 30 minutes after stress ($r=0.17$, ns). Total calories consumed on rest day was not related to any measures of cortisol.

3.6. Food preferences

Next, to examine food type preferences, we examined the effects of reactivity on preference (preferences for taste class and fat content) on each day while controlling for BMI and dieting. Due to the large difference in calories of high versus low fat food, we analyzed food preferences based on *servings* of each type of food, rather than caloric content. We used a 2 (fat level: high fat or low fat) \times 2 (taste: sweet or salty) \times 2 (reactivity: high versus low reactor) \times 2 (time: stress or control) repeated measures ANCOVA, controlling for BMI and dieting.

The adjusted means for average servings of each type of food consumed by reactor group and day is shown in Table 3. There was a preference for sweet food across days. Across groups, participants consumed significantly more sweet food on average [$F(1,42)=5.4$, $P<0.03$]. There was a two-way interaction between taste class and reactivity, showing that high reactors especially preferred sweet food across days [$F(1,42)=10.9$, $P<0.01$]. On average across days, high reactors ate 2.03 sweet servings total (SE=0.33), whereas low reactors ate 1.37 sweet servings total (SE=0.33), $P=0.05$. There was no group difference in salty food intake across days.

In addition, there was a marginally significant 3-way interaction between taste class, reactivity, and time [$F(1,44)=3.9$, $P=0.055$]. Examination of the means for each type of food eaten showed that on the stress day, high reactors ate significantly

more sweet high fat food than low reactors, and groups were similar in amount of sweet low fat food. On the stress day, high reactors ate on average 0.54 of high fat sweet food servings (121.8 calories of sweet food, SE=16.0), whereas low reactors ate 0.23 of high fat sweet food servings (72.9 calories of sweet food, SE=16.7; $P<0.05$). Unexpectedly, we found that high reactors also ate significantly less salty food — specifically the low fat food — than low reactors on the rest day (See Table 3 for adjusted means). Although the pattern of means suggests a four-way interaction between reactivity, taste class and fat level with time, we did not find this interaction to be significant, but also lacked the statistical power. Therefore, these findings will be interpreted with great caution.

4. Discussion

Stress-induced cortisol reactivity was related to greater caloric intake after exposure to a novel laboratory stressor. Women who were high cortisol reactors to stress ate more food than low reactors while recovering from stress. On the rest day, however, high reactors tended to eat less and low reactors tended to eat more, eliminating the difference between groups. Further, the high reactors tended to consume more sweet foods than low reactors, across days. Cortisol reactivity may be a marker for vulnerability to stress induced eating, and thus may help to explain who eats more versus who eats less after stress.

Self-reported increases in negative mood (“mood reactivity”) during the stressor were also significantly positively related to caloric consumption, whereas mood reactivity on the control day was not related to consumption that day. We should note that mood reactivity was not related to cortisol reactivity, a disassociation common in many laboratory stress studies (e.g., Buske-Kirschbaum et al., 1997). We view cortisol reactivity and mood as two somewhat independent indices of stress reactivity, and found that both were related to eating after stress, but not after rest.

These results suggest that psychophysiological response to stress influences subsequent eating behavior. The current results mirror those of a similar study of college undergraduate women (Epel et al., 2000). In both studies, we found that during a control day, dieting was related to eating less, whereas on a stress day, cortisol reactivity predicted eating more. In the current study, we were able to examine food type, and observed that reactivity was related to greater intake of sweet food. Multiple comparisons of food type consumed by reactivity group revealed further differences; of the sweet food consumed after stress, the high reactors consumed significantly more high fat food, which was as predicted. In addition, on the rest day, high reactors decreased and low reactors increased their consumption of salty food to such an extent that the difference in salty food intake became significant, which was not predicted. Findings from these multiple comparisons may be due to chance and should be interpreted with caution.

It is unknown exactly how cortisol may affect eating behavior, although some propose it has direct effects on appetite (Tataranni et al., 1996). When we examined cortisol as a continuous variable, there was only a weak correlation between cortisol

and calories eaten. It is likely that cortisol reflects or modulates other stress responsive factors, such as leptin, neuropeptide Y, or cytokines, that more directly affect appetite, rather than having direct effects itself. Stress affects every bodily system, and thus there are multiple and complex pathways through which stress can affect eating. Exposure to stress increases neuropeptide Y (Zukowska-Grojec, 1995), which can increase appetite (Morley, 1987). Further, the stress-sensitive adrenal steroids, specifically, modulate neurotransmitters that affect appetite, such as B-noradrenergic systems, neuropeptide Y and galanin (McEwen et al., 1993). Cortisol may also blunt taste threshold sensitivity (Fehm-Wolfsdorf et al., 1989; Henkin, 1970). Henkin (1970) has observed that people with Cushings, who have excessive cortisol levels, tend to add extra salt and sugar to their food. The current results are consistent with Sapolsky's explanation of stress effects on eating, where eating is thought to be suppressed during stress, due to anorectic effects of CRH, and increased during recovery from stress, due to appetite stimulating effects of residual cortisol (Sapolsky, 1998).

Eating is a complex and multidetermined behavior, especially in humans. Glucocorticoids may be more strongly related to eating behavior in animals, as several rodent studies have found that manipulating corticosterone clearly affects eating and weight. For example, corticosterone replacement in adrenalectomized mice, especially at levels comparable to that secreted during stress, overrides the effect of a leptin infusion and stimulates eating and weight gain (Solano and Jacobson, 1999). Recently, Dallman and colleagues have found that high levels of corticosterone stimulate sucrose consumption in adrenalectomized rats (Bell et al., 2000). To the limited extent that we can make a comparison, our finding of increased sucrose consumption among high cortisol reactors is consistent with their experimental findings.

We also consider a compelling alternative explanation to the observed results; in rats, prolonged high fat diets can elevate both basal and reactive levels of corticosterone (Tannenbaum et al., 1997). In this sample, there may have been pre-existing differences in high fat intake, which in turn could influence HPA axis activity. For example, women who eat high fat diets may have subsequently shown both greater cortisol reactivity and greater preference for sweet foods. Nevertheless, in this sample, we found no relationships between *basal* cortisol with food intake in the laboratory.

Although our primary interest was eating response after a novel acute stressor, we also measured eating behavior after the subsequent two stress sessions as well. There were no relationships between cortisol reactivity with eating behavior on the additional two stress sessions. When exposed to the repeated laboratory sessions, with snacks afterwards each day, cognitive factors such as expectation of the type of foods offered and dietary restraint may have influenced food intake. It is also possible that eating response to novel stress may not generalize to eating responses to familiar stressors. Several studies have shown that people under chronic stress tend to gain weight over time (Greeno et al., 1998), which may be due to both stress-related endocrine changes, as well as coping behaviors. Lastly, it is possible that participants' eating behavior between sessions was quite different from that in the

laboratory after the session (e.g., those who decreased their eating in the lab made up for it when at home). It is also unclear whether eating in the laboratory generalizes to eating in other environments. Clearly, further research is needed to examine the time-course of relations between stress, stress reactivity, and eating, in more naturalistic contexts.

In summary, the current results suggest that stress reactivity may increase consumption of food after an acute stressor, although this may hold only for a laboratory situation. These relationships were not moderated by level of obesity or dietary restraint. It is possible that women more vulnerable to stress, in their mood responses and cortisol reactivity, may be at particular risk of stress-induced eating and weight gain. Relationships between psychological stress, hormones, and eating deserve further exploration, and this study is merely a first step in that direction.

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