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What is eating you? Stress and the Drive to Eat

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Abstract

Non-human animal studies demonstrate relationships between stress and selective intake of palatable food. In humans, exposure to laboratory stressors and self-reported stress are associated with greater food intake. Large studies have yet to examine chronic stress exposure and eating behavior. The current study assessed the relationship between stress (perceived and chronic), drive to eat, and reported food frequency intake (nutritious food vs. palatable non-nutritious food) in women ranging from normal weight to obese ($N = 457$). Greater reported stress, both exposure and perception, was associated with indices of greater drive to eat— including feelings of disinhibited eating, binge eating, hunger, and more ineffective attempts to control eating (rigid restraint; r's from .11 to .36, p's < .05). These data suggest that stress exposure may lead to a stronger drive to eat and may be one factor promoting excessive weight gain. Relationships between stress and eating behavior are of importance to public health given the concurrent increase in reported stress and obesity rates.

Keywords

Stress; drive to eat; restraint; disinhibition; binge eating

A recent national survey found that nearly 50% of people report feeling greater stress now than five years ago and 43% report using food to cope with stress (APA, 2007). The high prevalence of stress-related eating may contribute to the increasing prevalence of overweight and obesity in the US, where 72.3% of adult men and 64.1% of adult women are overweight or obese (Flegal, Carroll, Ogden, & Curtin, 2010). Women report significantly more stress-related eating than men (Greeno & Wing, 1994), especially of high fat and high sugar foods (Wansink, Cheney, & Chan, 2003). Here we examine whether women who report greater life stress (stress perception or stress exposure) also report a greater drive to eat and consumption of palatable food.

Relationship between Stress and Eating

Non-human animal studies show that chronic stress exposure increases consumption of palatable food (Dallman et al., 2003; Wilson et al., 2008). Following laboratory exposure to ego threats, people exhibiting high negative affect or greater cortisol reactivity eat more sweet and high fat food (Epel, Lapidus, McEwen, & Brownell, 2001; Rutters, Nieuwenhuizen, Lemmens, Born, & Westerterp-Plantenga, 2009). Outside the laboratory, when faced with more daily stressors, people with high cortisol reactivity report greater snack food intake (Newman, O'Connor, & Conner, 2006).

Experiencing drive to eat, in the absence of true caloric need, is common, but there are large individual differences in the strength of the drive to eat (Greeno and Wing, 1994). Type of stressor, restraint and disinhibition differentially impact amount eaten both in the laboratory and in the real world. For example, only people who scored high on both disinhibition and restraint ate more following a negative mood induction (Yeomans & Coughlan, 2009). While an anticipated electric shock did not increase eating in people who scored high on restraint, ego threats significantly increased eating in high restraint people (Heatherton, Herman, & Polivy, 1991). In another study, people who endorsed restrained or emotional eating consumed equivalent amounts of high and low fat foods following both an ego-threatening and a neutral task (Wallis & Hetherington, 2009).

Dietary restraint involves efforts to control food intake for the purpose of weight loss or maintenance. However, people endorsing higher levels of dietary restraint show little difference in calorie intake compared to people with low restraint, or in food intake when unobtrusively observed in the laboratory (Stice, Sysko, Roberto, & Allison, 2010) and naturalistically (Stice, Fisher, & Lowe, 2004). Some types of dietary restraint may be more effective than others. Researchers have differentiated flexible restraint (which may prevent excessive consumption of palatable non-nutritious food) versus rigid restraint (which may be ineffective and eventually lead to overeating). Using the Eating Inventory (Stunkard & Messick, 1985) people higher on flexible restraint, compared to those higher on rigid restraint, displayed less overeating in the laboratory following a milkshake preload (Westenhoefer, Broeckmann, Munch & Pudel, 1994). People who maintain rigid rules around food appear less attentive to physiological cues of hunger and satiety, leading to overeating after a preload (Heatherton, Polivy, & Herman, 1989). Lastly, among people with binge eating disorder undergoing behavioral treatment, increases in flexible but not rigid restraint predicted both abstinence from binging and weight loss (Blomquist & Grilo, 2010).

Although strong drive to eat and 'the brake' on drive—high levels of restraint --are important factors determining food intake there has been little empirical work examining their relationship to stress, and specifically whether stress may increase drive to eat as well as be related to ineffective efforts at dietary restraint. The drive to eat is an understudied factor underlying obesity. Stress may impair inhibition and is known to trigger relapse of unwanted behaviors such as smoking (Shiffman, Paty, Gnys, Kassel, & Hickox, 1996).

Stress can result in feeling out of control while eating. For example, for people actively trying to restrain food intake, the depletion of resources caused by stressors may further impair inhibitory control, thus increasing the likelihood of overeating (Westenhoefer et al., 1994).

Current study

To determine the impact of stress exposure on eating behavior—as well as on rigid and flexible restraint—the current study tested cross-sectional associations between perceived stress, stressor exposure, and indices of drive to eat in a large sample of community women. We hypothesized that women who reported greater life stress would report greater drive to eat (hunger, disinhibition), greater intake of palatable food high in fat and sugar, and greater levels of rigid (vs. flexible) restraint.

Method

Participants

Women (N = 561) were recruited from tNorthern California via advertisements, email listserves, and community flyers to participate in an online study on "Women's Health." Our focus was on normal weight, overweight and obese women; women who reported Body Mass Index (BMI) less than 18 were excluded from our analyses (n = 20). Eighty-four people did not report relevant study variables leaving a final sample of 457 with Mage = 28.50 (SD = 6.75; range: 20.00–56.00) and MBMI = 24.20 (SD = 4.87; range: 18.51–50.11). Participants identified as 59.2% Caucasian, 17.3% Asian-American, 5.6% Latino, 7.1% African American, 2.6% Indian, 1.1 % Native American, and 6.7% other.

Procedures and Measures

Participants, after providing informed consent online, completed an online survey that included questions on age, weight, stress, and eating behavior. Participants were not compensated for participation. The protocol was approved by the UCSF IRB.

Sociodemographics: To measure education, participants responded on a 1 to 7 scale from some high school to a completed doctoral degree. Self-reported annual household income data was collected from participants.

Anthropometrics—Self-reported height and weight was used to calculate body mass index (BMI=Kg/M), a proxy measure of body fat recommended by the World Health Organization as a universal criterion of adult weight (WHO, 1998). BMI values of ≥25 and ≥30 were used as criteria to indicate overweight and obesity, respectively.

Perceived stress—The Perceived Stress Scale (Cohen & Williamson, 1988), a 10-item scale assesses unpredictability, uncontrollability, and overloading., including items such as "In the last month, how often have you been upset over something that happened unexpectedly," and "In the last month, how often have you been angered because of things that were outside of your control?" Responses were averaged to create an indicator of perceived stress (α = .87; mean range: 0.00–3.70). The current study mean of 1.68 is comparable to a national sample of women from 2009 (M = 1.6; Cohen & Janicki-Deverts, in press).

***Chronic* Stressor Exposure—**The Social Stress Index was used to assess multiple domains of chronic stress, including 51 items measuring stressors associated with work, family, and relationships (Wheaton 1994). Endorsed stressors, such as "The place you live is

too noisy or too polluted," were added to create a stress index that ranged from 0 to 51. Reliability was $\alpha = .90$ (range: 0–51) in the current study, indicating high inter-item reliability, showing that stressors tend to cluster together.

Restraint, disinhibition, and hunger—The Eating Inventory (Stunkard & Messick, 1985) was used to assess cognitive restraint, disinhibition, and susceptibility to hunger. We used the 21 item measure of restraint ($\alpha = .83$), the 16 item measure of disinhibition (e.g. "I usually eat too much at social occasions like parties or picnics"; $\alpha = .84$) and the 14 item measure of hunger ($\alpha = .78$). To determine the utility in identifying different types of restraint, we created flexible and rigid restraint subscales based on Westenhoefer et al (1994). In the current study, four items were added to the flexible scale and eight items were added to the rigid restraint scale to fully encompass restraint, resulting in an 11 item measure of flexible restraint ($\alpha = .73$), and a 15 item measure of rigid restraint (e.g., "I avoid some foods on principle even though I like them"; $\alpha = .75$). We also examined the original scale of restraint with the additional items, creating a 33 item measure of restraint (overall restraint, $\alpha = .86$). The flexible restraint and rigid restraint scales were significantly intercorrelated but not totally redundant with each other ($r = .581$, $p < .001$).

Binge eating—The Binge Eating Scale (Gormally, Black, Daston, & Rardin, 1982) was used to assess behavioral manifestations and feelings surrounding a binge episode. Items examined frequency of eating when bored, when guilty, ability to control eating urges, as well as preoccupation with eating. We removed 5 items assessing non-eating behavior (e.g., body dissatisfaction) because our interest was binge eating. In the current study, the 11 remaining items were summed and averaged ($\alpha = .85$).

Food frequency—The frequency of intake of particular food items was measured by a questionnaire adapted from Wardle and colleagues (Potischman et al., 1999). Participants were presented with food categories and asked to rate "How often do you eat the following foods?" on a 6-item Likert scale from "never" to "more than once a day." The questionnaire included items such as "burgers, pizza and hot dogs" and "whole grain bread, pasta, and brown rice." Answers were averaged within categories delineated by a factor analysis that found two reliable factors: palatable non-nutritious food, and nutritous food. The palatable non-nutritious food scale included four food groupings: chips; burgers (pizza, hotdogs); fried foods; and soda (regular soda, sweetened drinks; $\alpha = .71$; $M = 2.08$, $SD = .63$; range: 1–5). The nutritious food scale included four food groups: legumes (e.g., peas); vegetables (e.g., salad); fruit; and whole grains ($\alpha = .71$; $M = 3.86$, $SD = .86$; range: 1.25–5.75).

Analysis Strategy

SPSS, Version 17 was used. Partial correlations examined the relationships between stress exposure and perceived stress on eating patterns, controlling for BMI, education, income, and age. Analyses of variance were used for group comparisons. Significance was determined to occur *at* $p < .05$. See Table 1 for descriptive data and correlations between all variables. When examining the relationship between stress and the two types of restraint we controlled for one while examining the other in order to look at unique effects.

Results

Perceived Stress

Partial correlations, controlling for age, BMI, income, and education, found that perceived stress was related to drive to eat. Specifically, increased perceived stress was related to reported higher palatable non-nutritious food intake ($r = .154$, $p = .001$), and there was a significant negative association with nutritious food intake ($r = -.096$, $p = .040$).

Additionally, perceived stress was related to an increased lack of control over eating ($r = .321$, $p < .001$), greater hunger ($r = .327$, $p < .001$), and more frequent binge eating ($r = .362$, $p < .001$). Although perceived stress was not related to the original version of the restraint subscale ($r = -.068$, $p = .146$) or overall restraint that included additional items ($r = -.007$, $p = .887$), it was related to the sub category of rigid restraint. Controlling for flexible restraint, perceived stress was related to greater levels of rigid restraint ($r = .106$, $p = .023$). Controlling for rigid restraint, perceived stress was negatively related to flexible restraint ($r = -.094$, $p = .045$).

Stress Exposure

Similarly, stress exposure was related to drive to eat indices, controlling for age, BMI, income, and education. Stress exposure was related to reported higher palatable non-nutritious food intake ($r = .165$, $p = .0001$), but not with nutritious food intake ($r = -.071$, $p = .129$). Further, increased stress exposure was related to lack of control over eating ($r = .233$, $p < .001$), hunger ($r = .254$, $p < .001$), and binge eating ($r = .289$, $p < .001$). As with perceived stress, stress exposure was not related to the original scale of restraint ($r = -.068$, $p = .149$) or overall restraint using additional items ($r = .010$, $p = .833$), but was related to the sub-categories. Controlling for flexible restraint, stress exposure related to greater levels of rigid restraint ($r = .167$, $p < .001$). Conversely, controlling for rigid restraint, stress exposure was related to lower flexible restraint ($r = -.135$, $p = .004$).

BMI status and stress

Finally, we explored a counter hypothesis that overweight may induce greater stress and drive to eat. To test this, we compared normal weight, overweight, and obese participants on our two stress measures. A one-way analysis of variance found no differences based on BMI status on perceived stress [$F(456) = 2.150$, $p = .143$] or on stress exposure [$F(456) = .331$, $p = .565$].

Discussion

This is the first study to test, in a large sample, associations between stress exposure and indices of increased drive to eat. As hypothesized, stress was related with the drive to eat as measured by reported disinhibited eating, binge eating, and intake of palatable non-nutritious food. Additionally, stress was related to increased rigid restraint—the form of restraint more frequently associated with overeating. Those reporting greater stress, regardless of whether it was perceptions of stress, or presence of more objective exposures, also reported greater drive to eat across several indices (disinhibition, hunger, and binge eating) and more frequent palatable non-nutritious food consumption (e.g., chips, hamburgers, and soda). Results were remarkably consistent across both types of stress, perceived stress and the chronic stressor exposures, despite the limited overlap between measures (only 25% shared variance).

This study supports previous findings linking stress with greater palatable or comfort food intake. Perceived stress was associated with significantly decreased healthy eating (e.g., vegetables and whole grain foods), possibly as a result of the increased intake of highly palatable food. In non-human animal models, palatable non-nutritious food has a calming effect on the HPA axis stress response (e.g., Warne, 2009). Humans tend to eat more in a laboratory setting following an acute stressor than in a control session, if high in cortisol reactivity and negative affect (Epel et al., 2001). Sugar and fat target the brain similar to opiates and are often sought during times of stress (see Cota, Tschop, Horvath, Levine, 2006, for review; Oliver, Wardle, Gibson, 2000; Newman et al., 2006). Food is an inexpensive resource for providing relief; these highly palatable, low nutrient dense foods

can offer short term pleasure and relief from discomfort (Dallman, Pecoraro, & la Fluer, 2005). The results of the current study appear consistent with previous experimental and non-human animal research on the stress-related eating relationship.

The empirical separation of restraint into flexible and rigid subscales (Westenhoefer, 1991) proved to be useful in this study as well. It is striking that the total restraint scale, was not related to either stress measure, and we may have concluded null findings if we had not separated out rigid and flexible restraint. Although the subscales are related to each other, each subscale provided unique variance and related to stress measures in opposite directions. Greater stress exposure (controlling for age, BMI, socioeconomic status, and flexible restraint), accounted for significantly higher rigid restraint. Greater exposure to chronic stress affects brain regulation of emotions and impulses, downregulating activity in the prefrontal cortex, reducing executive control, and upregulating activity in the amygdala and hypothalamus (Epel, Tomiyama, & Dallman, 2011). Greater perceived stress can promote less conscious control over volitional behavior and greater hedonic drive via impairment of prefrontal cortex (Arnsten, 2009). Lack of control over situations in one's life could lead to the desperate but ineffective attempts to control eating such as deprivation from a particular food followed by later overeating.

Directionality cannot be determined from the analyses. For example, overweight may lead to both increased stress and increased eating. To examine this counter hypothesis, we tested potential stress differences based on BMI status. The relationship was not supported, in alignment with a recent meta-analysis finding no consistent cross sectional relationship between stress and adiposity, although the longitudinal studies showed an effect of stress on weight (Wardle, Chida, Gibson, Whitaker, & Steptoe, 2010). Another hypothesis is that genetic factors may predispose one to both excessive weight and stress. Such relationships could be mediated by differences in the glucocorticoid receptor gene. Some studies have found that certain variants are associated with both greater cortisol reactivity to stress and greater body mass index (Rosmond et al, 2000; Kumsta et al, 2007).

There are limitations to this study, including the relatively small effect size, a community sample of premenopausal who don't necessarily well represent the highest levels of stress, and potential problems inherent in internet data collection. The small observed effect sizes, from .106 to .362, indicate that the real world significance of these relationships still needs to be determined. In addition, while this sample's endorsement of mild to moderate levels of stress is equivalent to a national sample of women in 2009 (Cohen & Janicki-Deverts, in press), we were unable to detect the potential relationship between people endorsing high stress and frequency of unhealthy food intake. Although there are advantages to internet data collection including reduced social desirability in self-report (Nosek, Banaji, & Greenwald, 2002), internet data collection is vulnerable to haphazard answers and rapid responding.

This study tests the concept of a stress-related drive to eat in a large sample, in a preliminary fashion, and represents the tip of the iceberg in understanding the role of drive to eat. Our study is consistent with findings from known neural networks linking stress to a drive for dense calories (Dallman et al., 2003; Chandler-Laney et al., 2007). It underscores the need for studies more directly examining the relationship between stress and vulnerability to obesity, with increased drive to eat serving as a potential mediator. This question has become increasingly important as more basic research is uncovering the central role of reward circuitry and 'wanting' palatable food in compulsive overeating (Corbin, Avena, Boggiano, 2011; Davis et al, 2011; Finlayson et al, 2007). Future human research could incorporate real-time ecological momentary assessment paradigms to determine directionality in the relationships between stress, restraint and palatable food intake to inform weight loss treatment studies.

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Table 1

Descriptives (Means, SD) and Pearson Correlations ^

	Mean	*SD*	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.
Covariates														
1. Age	28.51	6.75	.20**	.12**	19**	.02	.10*	.01	−.06	−.09	−.06	−.02	−.04	−.11*
2. BMI	24.22	4.88		−.07	−.07	.10*	.10*	17**	−.12**	.16**	.33**	.19**	−.0	.29**
3. Education	4.52	1.23			.12*	−.09	−.17**	−.16**	.11*	−.01	−.04	−.07	−.01	−.07
4. Income	54,931	48,066				−.15**	−.16**	−.05	−.14**	.01	−.03	.08	.09	−.04
Stress Measures														
5. Perceived Stress Scale	1.68	.68					49**	.15**	−.10*	.33**	32**	.11*	−.09+	.36**
6. Stress exposure	17.06	8.32						.17**	−.07	.25**	.23**	.17**	−.14**	.29**
Food frequency														
7. Palatable Non-Nutritious Food	2.08	.63							−.31**	.12**	.08	−.11**	−.22**	.06
8. Nutritious Food	3.86	.86								−.02	.03	.00	.15**	.01
Eating Behaviors														
9. Hunger	5.24	3.33									.61**	.25**	−.11*	.65**
10. Disinhibition	6.77	3.96										.44**	.04	.82**
11. Rigid restraint	5.45	3.26											.58**	43**
12. Flexible restraint	5.49	2.61												.03
13. Binge eating (*n*=387)	9.95	5.98												

Note. N = 457 unless otherwise noted.

^ Correlations are zero order except the partial correlations (shaded). The partial correlations between stress and drive to eat indices control for age, BMI, education, and income. The partial correlations between stress and rigid restraint also control for flexible restraint and correlations between stress and flexible restraint control for rigid restraint.

+ $p < .10$.

* $p < .05$.

** $p < .01$.