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Torres, Susan and Nowson, Caryl 2007-11, Relationship between stress, eating behavior and obesity, *Nutrition*, vol. 23, no. 11-12, pp. 887-894.

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Relationship between stress, eating behaviour and obesity

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Abbreviated title: Stress and eating behavior

Word count for manuscript: 6153

Number of figures: 2

Number of tables: 2

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Abstract

Stress is thought to influence human eating behavior, and has been examined in animal and human studies. Our understanding of the stress-eating relationship is confounded by limitations inherent in the study designs; however, we can make some tentative conclusions which support the notion that stress can influence eating patterns in humans. Stress appears to alter overall food intake in two ways, resulting in under- or over-eating, which may be influenced by stressor severity. Chronic life stress seems to be associated with a greater preference for energy and nutrient dense foods, namely those that are high in sugar and fat. Evidence from longitudinal studies suggests chronic life stress may be causally linked to weight gain, with a greater effect seen in men. Stress-induced eating may be one factor contributing to the development of obesity. Future studies which measure biological markers of stress will assist our understanding of the physiological mechanism underlying the stress-eating relationship, and how stress might be linked to neurotransmitters and hormones which control appetite.

Key words: Stress, Eating behavior, Sucrose, Fat, Rat, Weight gain, Obesity

Introduction

A complex array of internal and external factors influences appetite and consequently the amount and types of food consumed by humans. Internal factors include physiological mechanisms which regulate appetite, with hormones such as neuropeptide Y stimulating food intake [1] and leptin reducing food intake [2]. Many external factors can also influence food intake and include environmental factors (eg economic, food availability) [3], social factors (eg influence of others) [4], and the palatability of foods [4]. It is a commonly held belief that stress can alter eating patterns [5]. When an acute stress is experienced, such as a threat to personal safety, there is an instant physiological response, the "flight or fight" response [6], which results in the suppression of appetite [7]. Exposure to chronic psychological stressors, for example job pressures, are one of many mental health disorders that contributes to the global burden of disease [8]. For many, the typical response to these chronic stressful situations is not to avoid food but maybe to seek out and consume energy dense foods [9, 10]. Obesity is a global epidemic and is increasing at an alarming rate, and can be attributed to a myriad of genetic and environmental factors [11]. If stress causes some individuals to consume food in excess of requirements, then this may culminate in weight gain and obesity.

Our aim is to review the evidence from both animal and human studies on the effect of acute and chronic stress on eating behaviour, and how stress-induced eating may contribute to the development of obesity. Firstly, we describe how stress can alter total food intake. Secondly, we discuss how stress can promote consumption of nutrient dense foods, specifically preference for sweet foods, which has been the recent focus of our research work. Thirdly, we present evidence to support the hypothesis that stress-induced eating may result

in future weight gain and ultimately obesity. Finally, we discuss how the physiological

responses to stress may interact with processes involved in appetite regulation.

Stress response

Stress can be defined as "the generalized, non-specific response of the body to any factor that

overwhelms, or threatens to overwhelm, the body's compensatory abilities to maintain

homeostasis" [6]. The following stressors can induce a stress response: physical stressors

(trauma, surgery, intense heat or cold); chemical stressors (reduced oxygen supply, acid-base

imbalance); physiological stressors (heavy exercise, hemorrhagic shock, pain); psychological

or emotional stressors (anxiety, fear, sorrow); and social stressors (personal conflicts, change

in life style) [6]. Stressors can be short-term (acute stress) or occur on a daily basis (chronic

stress). Reactions to stressors have been suggested to be of several types [12], and those of

most importance are the "active fight/flight" pattern (sympathetic adrenal medullary system)

and the "passive" pattern (pituitary-adrenal cortical system involving the hypothalamic-

pituitary-adrenal (HPA) axis) (Fig. 1). Activation of the sympathetic adrenal medullary

system, with release of catecholamines (adrenaline and noradrenaline), is typical during

periods of acute stress [13]. Hyper-activation of the HPA axis, with release of corticosteroids

(cortisol), has been associated with individuals who are chronically stressed [14].

Furthermore, it has been proposed that a hyperactive HPA axis may be programmed during

the prenatal period as a result of foetal growth retardation [15]. Responses to acute or

chronic stress can lead to physiological changes which include slowed gastric emptying [16],

elevation of blood pressure, increase in heart rate, mobilisation of energy stores, and decrease

in blood flow to non-essential organs, for example the digestive system, kidneys and skin

[13]. Hormones released in response to stress can specifically affect appetite. Noradrenaline

[17] and corticotropin-releasing hormone (CRH) [18] have been reported to suppress appetite

during stress, whereas cortisol is known to stimulate appetite during recovery from stress [18]. Anxiety, depression, uneasiness, anger, apathy and alienation are emotions that commonly accompany chronic stress [13]. The responses to acute or chronic stress also include a number of modifying behaviours such alcohol consumption [19], smoking [20] and eating [5].

Stress can alter food consumption- the effect of stressor severity

Greeno and Wing have outlined the individual-difference model, which suggests there are two ways in which stress may influence eating, resulting in either eating or not eating [21]. These opposing responses may be explained by the severity of stress that is encountered.

Animal studies

Animal studies provide a convenient way to measure the effect of stress on food intake. Rats can be subjected to different stressors in a controlled laboratory setting with close monitoring of food intake. In addition, there is evidence that animal models can provide valuable information about the interplay between stress and psychological/emotional processes that drive humans to eat [22, 23].

The severity of the stressor seems to influence food intake in the rat model. Immobilization, a severe stressor, consistently reduced ordinary food (rat chow) intake in rats when administered chronically [24-27] and even acutely [27]. Moderate stressors (restraint, noise stress) administered chronically have also been reported to reduce ordinary food intake [26, 28-30]. This stress-induced inhibition of feeding behavior may have a physiological basis. As CRF levels increase in response to stress, food intake decreases [31]. Furthermore, this

was confirmed by a study in rats which found CRF inhibited the hyperphagia induced by neuropeptide Y [32].

A mild stressor (tail pinch) increased intake of sweetened condensed milk in 2 studies [33, 34] but did not alter intake of chow [35], and another mild stressor (handling) in rats had no effect on chow intake [26]. Therefore, mild stressors appear to have no effect on food intake when the foods available have limited hedonic characteristics, but may increase food consumption when the foods offered are highly palatable. This is supported by studies in rats which have demonstrated that exposure to a cafeteria diet (highly palatable, high-fat diet) causes a greater increase in calorie intake and body weight when compared to rats offered ordinary chow [36, 37].

Human studies

Human studies have also found both decreased and increased eating in response to stress and this may also be related to the severity of the stressor. A retrospective survey of United States Marine's food intake during combat provided an opportunity to examine the effect of a severely stressful situation on eating behavior [38]. During the first day of combat, 68% of marines reported eating less than usual. The main reason for eating less was lack of time, followed by fear, which included being nervous, tense and scared. In a prospective study, 158 male and female subjects completed 84 daily records of stress [39]. Subjects reported both a decrease and increase in eating in response to stress; however, subjects were more likely to report eating less. Furthermore, the likelihood of eating less increased as the severity of the stressor increased. The effects of self-reported stress on eating behavior were examined in 212 students [40]. Approximately equal number of subjects reported eating

more (42%) and eating less (38%), but no information on the influence of stressor severity was reported. The effect of a major stressful event (school exam) on food consumption was investigated in 225 male and female high school students [41]. Total energy intake was significantly greater on the exam day when compared to the stress free day (2225 versus 2074 kcal, (9.1 versus 8.5 MJ)) respectively. In another study, the effect of a mild stressor (stress-inducing film) on food consumption was assessed and overall, the male stress group consumed significantly less food compared with the male control group (99 versus 242 kcal (406 kj versus 992 kj)), respectively [42]. However, there was no difference in food consumption between the female groups indicating that gender may influence eating behavior following mild stress.

Limitations and conclusions

Therefore, in animal studies, severe stress appears to lead to decreased food intake and milder forms of stress either have no effect or lead to increased food intake. In humans, stress appears to both increase and decrease food intake, although it is difficult to determine if stressor severity has a role, as there are few human studies. There are a number of limitations inherent in these human studies and include the use of imprecise methods to measure dietary intake [39, 40]. Also, in the laboratory study, prior eating was not controlled which may have influenced the amount of food consumed during the stress and control conditions [42]. An alternative explanation for why energy intake is higher during periods of life stress [41] may be unrelated to the stress, but rather due to insufficient time to purchase and prepare foods and the increased use of convenience foods, which are typically energy dense [43].

Stress, nutrient dense foods and weight gain

Obesity is a global epidemic which results from energy imbalance, with energy intake exceeding energy expenditure over a long period [11]. The causes of obesity are both diverse and complex, and can be attributed to physiological, environmental and genetic factors [11]. Specifically, weight gain and the development of obesity have been attributed to lifestyle factors, with the early work of Kaplan and Kaplan [44] suggesting that one of the contributing factors to obesity may be due to stress-induced eating, with a greater preference for nutrient dense foods, particularly those that are high in sugar and fat.

Animal studies

Studies in rats, which have investigated the effect of stress on absolute levels of sweet fluid/food intake, have yielded mixed findings. Eleven published papers were examined (reporting 14 separate studies) which used stressors of varying duration and severity, and a variety of sweet fluids and foods (Table 1). Five studies reported an increase [35, 45-47], 3 reported no change [45, 48, 49] and 6 reported a decrease in sweet fluid/food intake [50-54].

An early study with rats provided convincing evidence that stress may contribute to weight gain, as chronic tail pinch (mild stressor) in the presence of sweetened milk induced hyperphagia and led to weight gain [33]. However, when rats were subjected to chronic tail pinch and then given ordinary rat chow, the weight of stressed rats was less then that of unstressed rats [55]. This difference between these 2 studies may have been due to the type of foods offered to the rats: sweetened milk (very palatable) versus ordinary rat chow in the second study. The effect of acute, restraint (moderate stressor) was investigated in rats prone to diet-induced obesity fed either low fat chow (low energy) or medium fat chow (high

energy) [56]. Compared to unstressed controls, stressed rats fed low fat chow gained less, whereas stressed rats fed high fat chow gained more.

Conclusions

It is difficult to make strong conclusions about the effect of stress on absolute levels of sweet food/fluid intake in the rat model. With regards to the effect of stress on subsequent weight gain, this appears to be dependent on the nutrient composition of the available food, with greater weight gain seen with high energy diets. This has similarities to human studies, such that individuals fed higher energy density ad lib diets increased their total energy intake [43].

Human studies- stress and nutrient density

The effect of stress on the intake of fat and sugar has been investigated in humans. In a large cross-sectional study with 12,110 individuals, greater perceived stress was positively associated with a higher fat diet [57]. Individuals in situations of greater perceived workload and perceived stress report increases in total energy and fat intake compared with periods of low workload and low perceived stress [58]. The prevalence of obesity is greater in African Americans compared with European Americans [59], and obesity has been positively associated with life stress in African American women [60]. Furthermore, African Americans appear to have a greater desire for intense sweet tastes and greater perceived life stress compared with European Americans [9]. This desire for intense sweet tastes may translate into selection and consumption of energy and nutrient dense foods, and may be a factor contributing to the greater prevalence of obesity in African Americans.

There have been few studies in humans that have tested the effect of stress, induced in a laboratory setting, on the selection of foods. Men and women subjected to an acute stressor

(preparation of a speech) consumed similar amounts of high- and low-fat sweet foods after stress compared with a control group [10]. In a study with 59 pre-menopausal women, 45 minutes of stress (visuospatial puzzles, serial subtraction of a prime number from a high number and delivery of a videotaped speech) did not alter consumption of a range of foods, including high- and low-fat sweet foods, compared with the non-stressed condition [61]. They found that high cortisol reactors (defined as the increase from baseline to stress levels of salivary cortisol) consumed significantly more calories (220 versus 140 kcal, (902 kj versus 574 kj)) and more high-fat sweet foods on the stress day compared with low reactors, but consumed similar amounts on the control day. There is evidence to suggest cortisol, a marker of HPA axis activity, may affect the regulation of appetite via neuropeptide Y and leptin [62]. Increases in cortisol seem to be followed by elevated secretion of neuropeptide Y and blunting of the inhibitory arm of food intake, the leptin system. The overall effect may be an increase in food intake.

Limitations and conclusions

When individuals respond to stress by eating more, anecdotal evidence suggests the foods selected are typically high in sugar and fat. Although there are few human studies published in this area, there is some evidence that chronic life stress is associated with a high fat diet and greater preference for sweet foods. However, there are limitations in the studies examined including the use of a less than optimal method to measure stress levels [57] and small sample sizes [58]. Furthermore, we may not see an effect on preference for nutrient dense foods in laboratory studies as the stress induced in this artificial environment may not be sufficiently stressful to alter eating behaviour.

Human studies- stress and weight gain

In a large cross-sectional study among Finnish public sector workers, which included 37,161 women and 8649 men, there was a weak association between work stress and body mass index (BMI) (range 23-27 kg/m^2) [63]. Stress-related eating (defined as trying to make oneself feel better by eating or drinking in a stressful situation) was significantly associated with obesity, but only in women and not men [64]. There may be a gender specific response to stress where women are more likely to use food to deal with stress, whereas men are more likely to use other oral behaviours such as alcohol consumption [19] or smoking [20] as strategies to cope with stress.

If stress-induced eating is contributing to the development of obesity, then it would be expected that obese individuals would consume more food in response to stress compared with lean individuals. The results from 5 studies are summarised in Table 2. Two studies measured the effect of life stress [65, 66] and the remaining 3 studies induced stress in a laboratory setting [67-69]. Two of these studies reported an increase in food consumption in obese individuals [65, 67]; however, only one of these corrected for body weight [65]. It would be expected that obese individuals would consume larger amounts of food to maintain weight. Usual food consumption and perceived stress (pleasant, neutral or unpleasant) was recorded by normal weight and overweight women over 4 m [66]. In overweight women, total food intake was not associated with the daily level of stress, whereas normal weight women ate the most on pleasant days. Therefore, there is only 1 study which found food intake increased with chronic stress in obese individuals.

In a longitudinal case-control study [70], children who had shown an increment in relative weight of 15% between the ages of 7 to 10 y were selected and matched with a control group with the same relative weight at age seven. The authors found a significant relationship between level of psychosocial stress and increased relative weight over this 3 y period. The 'fetal origins hypothesis' argues that prenatal growth retardation is associated with adult cardiovascular and metabolic disorders [71, 72]. There is a growing body of evidence from animal and human studies that suggests prenatal growth retardation may program a persistently hyperactive HPA axis in the foetus which continues into childhood and even the adult years [15], and this may influence eating behavior. Furthermore, this programming may be gender specific with prenatal growth retardation influencing adrenocortical responses to stress in boys and basal adrenocortical activity in girls [73]. Cortisol secretion, a marker of HPA-axis activity, is also known to be elevated in obese individuals [74]. Three studies in adults have investigated the effect of stress on future weight gain. Elevated levels of stress at baseline predicted a weight gain of 10 kg over a period of 6 y in men but not in women [75]. The Whitehall II study, a large prospective study in both men and women, found that work stress increased the likelihood of weight gain over 5 y in those with a higher BMI, but was likely to predict weight loss in lean individuals, and this observed bidirectional effect of work stress and BMI was only seen in men and not women [76]. The effect of stressful life events over a 6 m period on change in BMI was studied in men and women who were classified as high (respond to stress by eating more) or low (respond to stress by eating less) emotional eaters [77]. Only male, high emotional eaters who reported greater than 3 stressful life events had an increase in weight within 6 m. These 3 studies collectively suggest higher levels of stress increase the likelihood of weight gain, with a greater effect seen in men as compared to women. Recently, 'night eating syndrome', which is characterised by morning anorexia and

night time hyperphagia [78], has been associated with disturbances in the HPA-axis [79] and positively associated with BMI [80].

Obesity is associated with comorbidities such as coronary heart disease (CHD), type 2 diabetes, hypertension and dyslipidemia, and the additional presence of abdominal obesity (central location of body fat) confers an even greater risk for CHD and type 2 diabetes [81]. Stress reactions have been linked with the development of abdominal obesity. It has been proposed that repeated activation of the HPA axis by stress, with the elevation of cortisol that follows, leads to activation of adipose tissue lipoprotein lipase and then accumulation of abdominal fat mass [62]. Stress-induced cortisol secretion has been found to be greater among men [82] and women [83] with abdominal obesity, but is it not clear if the obesity is responsible for the higher levels of cortisol with stress or if the higher level of cortisol is driving the obesity.

Limitations and conclusions

We can conclude from cross sectional studies that stress is positively associated with body weight, although these findings need to be interpreted cautiously as it is based on self reported weight and height which can cause bias [63]. In humans subjected to laboratory or life stress, only 1 study out of 5 reported, which measured the effect of life stress, actually demonstrated a significant increase in food consumption in obese individuals. We may not see an effect in laboratory studies that induce acute stress, as individuals may not be sufficiently stressed in this artificial environment when compared to stress that is experienced in the real world. Furthermore, laboratory studies can only measure the effect of acute stress rather than chronic stress, which may have a greater effect on eating behaviour. We can also conclude from longitudinal studies that there appears to be an association between chronic

life stress and future weight gain, with a greater effect seen in men as compared to women, although the reason for this difference is not clear. When examining the relationship between life stress and future weight gain, it is important to consider both sides of the energy balance equation: energy intake and energy expenditure. In all the studies cited energy intake or energy expenditure was not measured, so it is not known if the weight gain reported was due to increased energy intake or decreased physical activity levels. With regards to the relationship between stress and physical activity levels, 2 studies have reported no association [84, 85], and 1 study demonstrated that physical activity levels decreased from baseline to a stress period [86]. It may be the case that chronic life stress is contributing to this observed weight gain, and data on dietary intake and physical activity levels need to be collected to determine which is the major factor.

Conclusions

Our understanding of the stress-eating relationship is confounded by limitations inherent in the study designs. Many studies have measured the effect of acute stress on eating behavior in a laboratory setting, which allows close monitoring of food intake. Laboratory studies are limited to the testing of acute stressors, rather than chronic stressors, the latter possibly having a greater effect on eating behavior. Longitudinal studies can investigate the effect of chronic life stress on eating behavior; however, accurate dietary data can be difficult to collect over long periods.

Despite the limitations discussed, we can make some broad conclusions that support the idea that stress can influence food intake. The studies examined revealed that stress can lead to decreased and increased eating, which may be related to stressor severity, such that in

animals a severe stress results in a lower intake and in humans the response is variable. There is some evidence to suggest elevated stress levels are associated with a greater desire for hedonic, highly palatable foods which are energy dense. This may contribute to excess energy intakes and weight gain, which is supported by longitudinal studies which suggest there is an association between chronic life stress and future weight gain.

Stress appears to have an effect on eating behavior in humans, and the responses which predominate during acute and chronic stress are summarised in Fig 2. Responses to acute stress are associated with physiological changes that might be expected to reduce food intake in the short term, for instance slowed gastric emptying and shunting of blood from the gastrointestinal tract to muscles. In other situations chronic stress elicits a more passive response driven by the HPA axis, with increases in cortisol which may entice people to consume hedonic, energy dense foods and potentially lead to unwanted weight gain and obesity. Cortisol may also contribute to the accumulation of abdominal fat mass. It is important to clarify the underlying physiological mechanism whereby stress leads to over consumption of food. Most of the studies examined in this review have quantified stress using only subjective measures. Some physiological measure of stress could be incorporated into future studies to measure levels of cortisol and catecholamines. Animal studies could identify any links between stress hormones and neurotransmitters involved in appetite regulation, which would assist in our understanding of the mechanism and possibly lead to preventative treatments.

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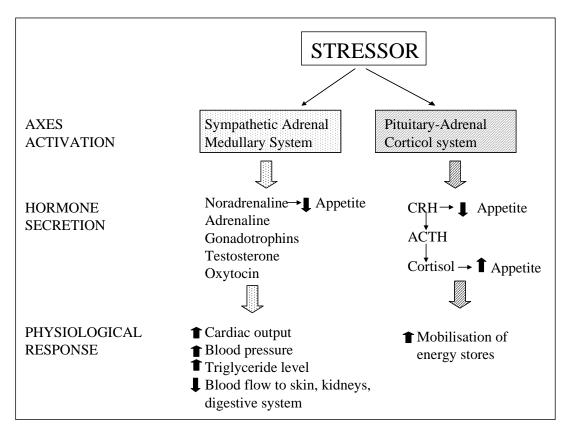
Figure 1. Physiological response to stress. CRH, corticotropin-releasing hormone;
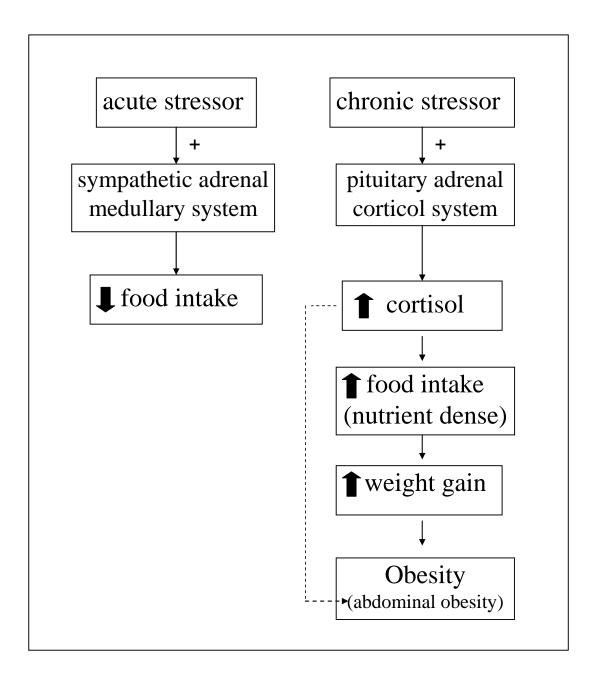
ACTH, adrenocorticotropic hormone

Figure 2. Predominant response pathways to acute and chronic stress.

Table 1

Intake of sweetened food/fluid in rats during stress

Study	Stressor	Stressor severity	Stressor duration	Food/fluid	Number stressed	Number unstressed	Intake during stress compared to unstressed condition	Weight
Bertiere [35]	Tail pinch	Mild	Acute	Sweetened milk	12	12	Increased[a]	
				Sucrose solution	12	12	Increased[a]	
Dess [47]	Tail shock	Severe	Acute	Sucrose solution	11	11	Increased[b]	
Ely [45]	Restraint	Moderate	Chronic	Froot loops®	15	16	Increased	
Silvera [46]	Restraint	Moderate	Chronic	Froot loops®	9-12	9-12	Increased	
Ely [45]	Restraint	Moderate	Acute	Froot loops®	11	11	Unchanged	
Kant [49]	Foot shock	Moderate	Chronic	Sucrose pellets	12	12	Unchanged	
Matthews [48]	Range	Mild	Chronic	Sucrose solution	16	16	Unchanged	Correction

								for body weight changes
Baker [51]	Range	Mild	Chronic	Sucrose solution	9	10	Decreased	No reported change in body weight between groups
Gronli [52]	Range	Mild	Chronic	Sucrose solution	20	20	Decreased	No reported change in body weight between groups
Papp [53]	Range	Mild	Chronic	Sucrose solution	8	8	Decreased	No body

								weights recorded
Wang [50]	Restraint	Moderate	Acute	Mixed carbohydrate diet	60[c]		Decreased	
Wang [50]	Restraint	Moderate	Chronic	Mixed carbohydrate diet	60[c]		Decreased	No body weights recorded
Willner [54]	Range	Mild	Chronic	Sucrose solution	10	10	Decreased	No body weights recorded

[a]Own control

[b] Intake measured after the conclusion of stress

[c]Total number

Table 2. Stress-induced eating in obese versus normal weight subjects

Study	Stressor	Stressor duration	Food/fluid	Timing of eating	Obese n	Intake compared to control condition	Normal weight n.	Intake compared to control condition	Weight
Slochower [65]	Life stress (college exam)	Chronic	M&M candy	During stress	23	Increased	14	Unchanged	
Pine [67]	Threat of electric shock	Acute	Peanuts	During stress	80	Increased	80	Unchanged	No correction for body weight
Reznick [68]	Intelligence	Acute	Chocolate	During stress	32	Unchanged	32	Unchanged	

	test, performance linked to threat of shock							
Ruderman [69]	Impressing strange man	Acute	Ice-cream	After stress	41	Decreased	42	Unchanged
Rosenfeld [87]	Self reported life stress (pleasant, neutral, unpleasant)	Chronic	Self-reported food intake	120 days	37	No association	37	Most food consumed on pleasant days