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Research report

Stress eating and health. Findings from MIDUS, a national study of US adults ☆

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ABSTRACT

The epidemic of obesity and its related chronic diseases has provoked interest in the predictors of eating behavior. Eating in response to stress has been extensively examined, but currently unclear is whether stress eating is associated with obesity and morbidity. We tested whether self-reported stress eating was associated with worse glucose metabolism among nondiabetic adults as well as with increased odds of prediabetes and diabetes. Further, we investigated whether these relationships were mediated by central fat distribution. Participants were 1138 adults (937 without diabetes) in the Midlife in the US study (MIDUS II). Glucose metabolism was characterized by fasting glucose, insulin, insulin resistance (HOMA-IR), glycosylated hemoglobin (HbA_{1c}), prediabetes, and diabetes status. Multivariate-adjusted analyses showed that stress eating was associated with significantly higher nondiabetic levels of glucose, insulin, insulin resistance, and HbA_{1c} as well as higher odds of prediabetes or diabetes. Relationships between stress eating and all outcomes were no longer statistically significant once waist circumference was added to the models, suggesting that it mediates such relationships. Findings add to the growing literature on the relationships among psychosocial factors, obesity, and chronic disease by documenting associations between stress eating and objectively measured health outcomes in a national sample of adults. The findings have important implications for interventive targets related to obesity and chronic disease, namely, strategies to modify the tendency to use food as a coping response to stress.

Introduction

Obesity is the most critical factor in the development of metabolic disease, with contemporary environments frequently described as obesogenic, or promoting obesity via abundant availability of energy-dense food accompanied by decline in physical activity (Chaput, Klingenberg, Astrup, & Sjodin, 2011; Swinburn, Egger, & Raza, 1999). More than 1/3 of American adults were obese in 2009–2010 (Ogden, Carroll, Kit, & Flegal, 2012); 25.8 million Americans have diabetes and 79 million have prediabetes (Centers for Disease Control, 2011). Traditionally, research has focused on diet and physical activity as cornerstones of obesity prevention and treatment, although it is clear that these factors leave considerable variance unexplained. Investigators have thus concentrated on identifying factors that extend the concept of energy balance by describing pathways to energy imbalance. Studies on triggers of eating behaviors document that a negative energy balance is a sufficient, but not necessary condition for initiating an eating episode (Del Parigi, 2010). What matters as well for initiation of an eating episode are complex interactions among emotional, cognitive, and cultural factors (Del Parigi, 2010).

Eating in excess of metabolic needs is, in fact, the leading contributor to weight gain, obesity, and subsequent morbidity. The link between stress and eating has received significant attention, given the considerable overlap between the physiological systems that regulate food intake and that mediate the stress response (Tannenbaum, Anisman, & Abizaid, 2010). During stress, the hypothalamic–pituitary–adrenal (HPA) axis is activated to prepare the organism for fight or fight; that is, mounting a defensive response

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depends on available energy. Thus, the HPA axis initiates a cascade of physiological adaptations such as the release of glucose into the bloodstream, thereby suppressing hunger (Gold & Chrousos, 2002). Emotional eaters, however, do not show the typical response of eating less during stress (Gold & Chrousos, 2002). Instead, they eat the same amount, or more, during stress (Oliver, Wardle, & Gibson, 2000; van Strien & Ouwens, 2003). It has been suggested that people use "comfort food", meaning food high in sugar and fat, in an effort to reduce activity in the chronic stress-response network with its attendant anxiety. Intake of comfort food is thought to alleviate stress by reducing HPA axis activity and promoting the activation of brain circuits involved in reward-seeking behavior (Dallman et al., 2003), thereby further reinforcing feeding behavior (Dallman, 2010).

Self-reported stress levels have been increasing over time (Cohen & Janicki-Deverts, 2012). In addition, acute and chronic stressors have been linked to energy-dense food intake, weight gain, obesity and glucoregulation (Bjorntorp, 2001; Block, He, Zaslavsky, Ding, & Ayanian, 2009; Eriksson et al., 2008; Groesz et al., 2012; Heraclides, Chandola, Witte, & Brunner, 2009; Laitinen, Ek, & Sovio, 2002; Ng & Jeffery, 2003). A recent national survey documented that 39% of people overeat, or increase consumption of energy-dense foods in response to stress (American Psychological Association, 2012). Nonetheless, it remains unclear whether eating in response to stress has metabolic consequences such as dysregulated glycemic control and abdominal obesity. One study looked at the relationship between self-reported stress eating and metabolic syndrome in medical students and found that stress eaters showed significant increases in weight and insulin during an exam period compared to students who reported eating less during stress (Epel et al., 2004).

To our knowledge, no study has examined relationships among stress eating, obesity, and metabolic disease in a national sample. The overarching goal of this investigation was to assess whether eating in response to stress was associated with glucoregulation. Further, we evaluated the role of waist circumference as a potential mediator of the relationship between stress eating and glucoregulation. The underlying rationales were first, that stress activation has been implicated in the pathogenesis of abdominal obesity (Bjorntorp, 2001; Dallman, Pecoraro, & la Fleur, 2005), and second, that waist circumference is a powerful predictor of diabetes (Klein et al., 2007). The specific hypotheses of our study therefore were:

Hypothesis 1 (H1). (a) Eating in response to stress will be associated with worse nondiabetic levels of fasting glucose, insulin, insulin resistance, and HbA_{1c}. (b) Waist circumference will mediate the relationships between using food in response to stress and fasting glucose, insulin, insulin resistance, and HbA_{1c}.

Hypothesis 2 (H2). (a) Extending the prior hypotheses to disease progression, eating in response to stress will be associated with higher odds of prediabetes in the nondiabetic subsample as well as diabetes in the full analytical sample. (b) Waist circumference will mediate the relationships between using food in response to stress and higher odds of prediabetes in the nondiabetic subsample and diabetes in the full analytical sample.

Materials and methods

Data

Data are from the Midlife in the US II (MIDUS II) study, a longitudinal follow-up of the original MIDUS sample ($N = 7108$). Begun in 1995/96, the overarching objective of MIDUS was to investigate the role of behavioral, psychological, and social factors in physical and mental health. All eligible participants were non-institutionalized, English-speaking adults in the coterminous United States, initially 25–74 years of age. Approximately 9–10 years later, respondents were re-contacted and invited to participate in MIDUS II. The longitudinal retention rate was 75%, adjusted for mortality (Radler & Ryff, 2010). One objective of MIDUS II was to extend the scientific scope of the study by adding comprehensive biological assessments on a subsample of respondents who had completed a phone interview and self-administered questionnaires. Forty-three percent of the invited MIDUS II respondents participated in the biological data collection. The majority (87.8%) of African American respondents came from a city-specific sample from Milwaukee, Wisconsin, which was implemented to increase participation of African Americans in the biological data collection, given its close proximity to one of the clinic sites. This full biomarker sample was not significantly different from the main MIDUS sample on age, sex, race, marital status, or income variables, although participants were significantly more educated than the main sample (Love, Seeman, Weinstein, & Ryff, 2010).

The current analyses used data from the biological sample of MIDUS II and included 1255 participants ages 34–84 ($M = 54.52$, $SD = 11.71$), more than half of whom (57%) were female. Only respondents who identified as White or African American were included in the current study because small sample sizes precluded the inclusion of other minority groups. After excluding 117 cases due to partially missing data on any variable in the analysis, or to a race other than black or white, 1138 participants had complete data. Table 1 includes descriptive information for all variables in the analyses.

Measures

Glucoregulation was indexed using three primary biological measures (fasting glucose, fasting insulin, and HbA_{1c}) and three composite measures (insulin resistance, prediabetes, diabetes). Fasting glucose, insulin, and HbA_{1c} samples were obtained during an overnight stay in a General Clinical Research Center (GCRC). Fasting glucose was measured via an enzymatic assay photometrically on an automated analyzer (Roche Modular Analytics P). Fasting insulin was measured with an ADVIA Centaur Insulin assay, performed on a Siemens Advia Centaur analyzer. The HbA_{1c} assay was a colorimetric total-hemoglobin determination combined with an immunoturbidometric HbA_{1c} assay, carried out using a Cobas Integra Systems instrument (Roche Diagnostics) (Wolf, Lang, & Zander, 1984). The first composite measure (insulin resistance) was calculated using the Homeostasis Model Assessment (HOMA-IR) formula that incorporates both glucose and insulin to describe the interplay between them (Matthews et al., 1985).

The other composites were dichotomous categorizations that used criteria from the American Diabetes Association to define presence of prediabetes or diabetes (2013). Specifically, nondiabetic individuals were classified as "prediabetic" if their HbA_{1c} was between 5.7% and 6.5% or their glucose was between 100 and 126 mg/dl. In the full analytical sample, diabetes status was coded positive if HbA_{1c} exceeded 6.5%, fasting glucose exceeded 126 mg/dl, or participant reported taking anti-diabetic medications. Using these criteria, 201 were coded as diabetic. All primary and composite measures are reliable and widely used in clinical practice to predict risk for disease (Ausk, Boyko, & Ioannou, 2010; Balkau et al., 1998; Bloomgarden, 2011a; Bloomgarden, 2011b; Parekh, Lin, Hayes, Albu, & Lu-Yao, 2010; Skriver, Borch-Johnsen, Lauritzen, & Sandbaek, 2010). Central fat distribution was indexed by waist circumference (WC), measured by a GCRC staff member around the abdomen just above the hip bone.

Respondents were asked to indicate how they "usually experience a stressful event," two options of which were "I eat more of

Table 1
Means (and SDs) or proportions for all measures stratified by stress eating and diabetes status.

	Nondiabetic only		Diabetic	
	High stress eaters (n = 202)	Low stress eaters (n = 735)	High stress eaters (n = 46)	Low stress eaters (n = 155)
Glucose (mg/dl)	96.4 (8.8)	94.7 (9.2)	143.9 (66.3)	130.7 (44.8)
Insulin (uIU/mL)	14.2 (11.5)	11.3 (9.4)	23.7 (33.6)	19.5 (17.1)
Insulin resistance	3.5 (3.0)	2.7 (2.5)	7.4 (7.0)	6.4 (6.7)
$HbA1_c$ (%)	5.8 (.4)	5.7 (.4)	7.8 (2.0)	7.6 (2.0)
Age (years)	51.4 (10.5)	54.5 (11.9)	55.7 (11.8)	59.2 (11.2)
Race (1 = white)	.83	.86	.67	.62
Gender (1 = male)	.24	.48	.24	.52
Education				
HS or less	.27	.26	.30	.36
Some college	.27	.29	.37	.30
College graduate	.46	.45	.33	.34
Income (1000×)	70.7 (61.8)	74.4 (58.9)	49.1 (51.7)	59.3 (55.3)
Waist (inches)	39.0 (6.0)	37.2 (5.9)	43.9 (6.9)	42.0 (6.6)

Note: High stress eating group consists of people who answered the questions the two questions about food quantity and preference with "a lot" or "a medium amount." Low stress eating group consists of people who answered the questions with "only a little" and "not at all." Stress eating was dichotomized into high and low stress eating groups for descriptive analyses only; a continuous measure of stress eating was used in all regression analyses.

my favorite foods to make myself feel better" and "I eat more than I usually do." Responses ranged from 1 = *a lot* to 4 = *not at all*. Responses to the two items were reverse coded and summed so that higher scores indicated greater use of food in response to stress. The correlation between the two items was .81 in the nondiabetic subsample and .80 in the full analytical sample.

All models were multivariate-adjusted for relevant covariates. Age, household income, and education (12 categories ranged from no school to completion of a professional degree) were treated as continuous variables. Race (black or white) and gender (male or female) were categorical variables.

Data analysis

Hierarchical multiple regression models were used to predict waist circumference and glycemic control indices (fasting glucose, insulin, insulin resistance, and HbA_{1c}). Fasting insulin and HOMA-IR were log-transformed to achieve normal distributions. Binary logistic regression models were used to predict prediabetes and diabetes status. Age and income were rescaled in the regression models such that one unit in the age variable was equal to 10 years and one unit in the income variable was equal to 10,000 dollars. We employed Baron and Kenny's (1986) criteria for testing waist circumference as a mediator. Additionally, we estimated percent explained (PE), a summary measure of the indirect effect that clarifies the extent to which the effect of stress eating on the different indicators of glucose metabolism was mediated by WC.

Preliminary analyses tested for gender, age, and race differences in the associations between using food to cope and glucoregulation by estimating a model with three two-way interaction terms created by multiplying combinations of gender, age, and race by stress eating. Since none of the interactions was significant, the entire sample was analyzed as one group. All models included all covariates (age, gender, race, education, and household income).

Results

Descriptive statistics are presented in Table 1.

H1. Stress eating will predict higher levels of glucose, insulin, insulin resistance, and HbA_{1c} and these effects will be mediated by waist circumference.

Model 1 (see Table 2) displays estimates with respect to H1. Consistent with H1, multivariate-adjusted models confirmed that stress eating was associated with higher glucose (b = .67, p < .001), log-transformed insulin (b = .08, p < .001), log-transformed insulin resistance (b = .08, p < .001), and HbA_{1c} (b = .02, p < .01). Stress eating was linked with waist circumference in the nondiabetic subsample (b = .91, p < .001) and waist circumference was associated with all measures of glucoregulation (p < .001 for all) in multivariate-adjusted models. In contrast to Model 1 estimates, coefficients in Model 1.1—which added waist circumference—indicated that the associations between stress eating and glucose, log-transformed insulin, log-transformed insulin resistance, and HbA_{1c} were reduced in size and no longer significant (p ranged from .1 to .2), thus demonstrating that WC mediated the relationships between stress eating and all measures of glucoregulation. Using PE, we found that waist circumference mediated a significant part of the effect of stress eating on glucose (61% PE), log-transformed insulin (81% PE), log-transformed insulin resistance (79% PE), and HbA_{1c} (56% PE).

H2. Stress eating will predict prediabetes and diabetes and these effects will be mediated by waist circumference.

Model 2 (see Table 3) displays results for the logistic regression models documenting that stress eating was associated with prediabetes (OR = 1.09, p < .05) in the nondiabetic sample and diabetes (OR = 1.15, p < .002) in the full analytical sample. Stress eating was linked with waist circumference in the full sample (p < .001) and waist circumference was associated with all measures of glucoregulation (p < .001 for all). In contrast to Model 2 estimates, once waist circumference was added, stress eating was no longer a significant predictor of prediabetes (p = .7) or diabetes status (p = .3) (See Model 2.1), thus providing supportive evidence that higher waist circumference mediates the relationship between stress eating and glucoregulation.

Discussion and conclusions

The objective of this study was to examine relationships between stress eating and clinically significant measures of metabolic health. The "flight or fight" response to stress causes release of glucose in the bloodstream, which is known to suppress appetite. However, for many people, the response to stressful situations is not to avoid eating, but to consume high volumes of energy-dense comfort foods. Using continuous measures of fasting glucose, insulin, insulin resistance, and HbA_{1c}, we documented that stress eating was associated with worse glycemic control and prediabetes status among nondiabetic adults as well as diabetes in the full sample. Importantly, waist circumference was found to mediate

Table 2
Linear regression results for stress eating, waist circumference, and nondiabetic glucoregulation ($N = 937$).

	Glucose		Insulin		Insulin resistance		$HbA1_c$	
	b (s.e.)	*p*	*b (s.e.)*	*p*	*b (s.e.)*	*p*	*b (s.e.)*	*p*
Model 1								
Stress eating	.67 (.16)	.000	.08 (0.1)	.000	.08 (.01)	.000	.02 (.01)	.004
Age	.38 (.26)	ns	−.02 (.02)	ns	−.01 (.02)	ns	.09 (.01)	.000
Race	−1.02 (.87)	ns	−.08 (.06)	ns	−.09 (.07)	ns	−.14 (.03)	.000
Gender	4.27 (.60)	.000	.19 (.04)	.000	.24 (.05)	.000	−.05 (.02)	.04
Income	.03 (.05)	ns	.001 (.004)	ns	.001 (.004)	ns	.001 (.002)	ns
Education	−.33 (.13)	.008	−.03 (.01)	.001	−.03 (.01)	.001	−.01 (.01)	ns
Adjusted R^2	.06		.06		.07		.10	
Model 1.1								
Stress eating	.26 (.16)	ns	.01 (.01)	ns	.02 (.01)	ns	.01 (.01)	ns
Age	.17 (.25)	ns	−.05 (.02)	.002	−.05 (.02)	.006	.09 (.01)	.000
Race	−.65 (.84)	ns	−.03 (.05)	ns	−.03 (.06)	ns	−.13 (.03)	.000
Gender	1.87 (.65)	.004	−.16 (.04)	.000	−.14 (.04)	.001	−.11 (.03)	.000
Income	.05 (.05)	ns	.003 (.003)	ns	.004 (.004)	ns	.001 (.002)	ns
Education	−.26 (.12)	.036	−.02 (.01)	.014	−.02 (.01)	.009	−.003 (.01)	ns
Waist circumference	.45 (.05)	.000	.07 (.003)	.009	.07 (.004)	.000	.01 (.002)	.001
Adjusted R^2	.13		.34		.34		.13	

Note: Unstandardized coefficients are shown. Insulin and insulin resistance are log-transformed. The age and income variables have been rescaled: one unit in the rescaled age variable is equal to 10 years and one unit in the rescaled income variable is equal to 10,000 dollars.

Table 3
Estimated odds ratios for the associations between stress eating, prediabetes, and diabetes.

	Pre-diabetes			Diabetes		
	OR	95% CI	*p*	OR	95% CI	*p*
Model 2						
Stress eating	1.09	1.01–1.17	.030	1.15	1.05–1.25	.002
Age	1.4	1.24–1.58	.000	1.61	1.39–1.86	.000
Race	2.19	1.47–3.27	.000	4.33	2.92–6.43	.000
Gender	.62	.47–.83	.001	.71	.51–1.00	.047
Income	1	.98–1.03	ns	1	.96–1.03	ns
Education	.95	.90–1.01	ns	.96	.90–1.03	ns
Model 2.1						
Stress eating	1.02	.94–1.10	ns	1.05	.96–1.16	ns
Age	1.37	1.21–1.55	.000	1.58	1.36–1.85	.000
Race	2.12	1.41–3.18	.000	3.81	2.52–5.77	.000
Gender	.91	.67–1.25	ns	1.17	.80–1.69	ns
Income	1.01	.98–1.03	ns	1	.96–1.03	ns
Education	.96	.91–1.02	ns	.98	.92–1.06	ns
Waist Circumference	1.08	1.05–1.11	.000	1.12	1.09–1.16	.000
Valid *N*	937			1138		

Note: the age and income variables have been rescaled: one unit in the rescaled age variable is equal to 10 years and one unit in the rescaled income variable is equal to 10,000 dollars.

these relationships. Taken together, these results confirm and extend results from a smaller experimental study (Epel et al., 2004), and suggest that stress eating is associated with objectively measured glucoregulation outcomes and specific diabetic morbidities, with the effects occurring primarily through central adiposity.

Traditional weight loss interventions often focus on diet and exercise, and although they may result in temporary weight loss, recurrent weight gain is common. A meta-analysis of weight loss interventions documented that 5 years post intervention three-fourths of the lost weight was regained (Anderson, Konz, Frederich, & Wood, 2001). One reason for relapse may be that traditional interventions do not target the root cause of overeating and inactivity. Using food to cope with stress may be one such underlying cause that is a potential target of intervention, as the response may be modifiable. If so, it could offer an important step toward incorporating characteristics of the individual in obesity treatment programs (National Institutes of Health, 1998). Stressors are thought to activate a neural stress-response network that promotes emotional activity and degrades executive function, resulting in employment of formed habits rather than cognitive resources (Dallman, 2010). Coping with stress by eating palatable foods may thus reduce anxiety and perceived stress, while further reinforcing the feeding habit. To reduce such stress-induced eating, it is important to identify alternative strategies that promote cognitive, goal-directed responses to stress. For example, little is known about the relationships between coping with food and problem- and emotion-focused coping skills (Carver, Scheier, & Weintraub, 1989). If comfort feeding is the default response and no active behavioral or emotional strategies are available to deal with the stressor, it is easy to understand how stressors and stress eating reinforce each other, with consequences for promoting obesity and chronic disease.

Mindfulness-based training (Kristeller & Wolever, 2011) may be relevant, given the focus on guided practices to address responses to different emotional states as well as awareness of hunger and satiety cues and related links to conscious food choices. Emerging findings from mindfulness-based interventions document improvements in weight, eating habits, and mental health (Alberts, Mulkens, Smeets, & Thewissen, 2010; Alberts, Thewissen, & Raes, 2012; Dalen et al., 2010; Daubenmier et al., 2011). Importantly, empowering people to cultivate awareness of emotional triggers and eating patterns may also promote self-acceptance (Kristeller & Wolever, 2011), which converges with the idea that effective motivational strategies for health promotion must focus on adopting health behaviors without referencing body weight (Puhl, Peterson, & Luedicke, 2012).

Several limitations of the present study need to be acknowledged. The absence of longitudinal data on stress eating and glucose metabolism limits the strength of the conclusions regarding the directionality of described relationships. However, previous prospective research linking stress eating to metabolic dysregulation (Epel et al., 2004) makes it less likely that poor glycemic control was the precipitating event that promoted stress eating. Another limitation is that many of the African–American respondents were drawn from a city-specific oversample (i.e., Milwaukee) implemented to increase participation of Blacks in the MIDUS biomarker project. Thus, they are not nationally representative. It should be noted, however, that the findings were not driven by racial factors, nor were there differences in the pattern of associations between white and black participants. It is not clear if the

results generalize to other ethnicities. Our analyses also did not examine the varieties of stress that may be precursors to stress eating, for which MIDUS has multiple indicators (e.g., job stress, care giving stress, daily stress, perceived discrimination, work/family conflict). Other individual difference variables, such as personality characteristics (e.g., neuroticism) may add further precision in identifying those most susceptible to stress eating. Finally, our analyses are modeled to capture influences on type 2 diabetes, but we did not have information on whether participants in the diabetes category had type 1 or type 2 diabetes. Given that approximately 90–95% of people with diabetes have type 2 diabetes (American Diabetes Association, 2013), our results are not significantly affected by this imprecision. Despite these caveats, the findings that stress eating was associated with increased waist circumference and worse glucoregulation helps advance understanding of the psychosocial underpinnings of obesity and glucoregulation. Continuing to elucidate the various processes that underlie energy imbalance and behavioral responses to it is critical for developing effective preventive and interventive efforts related to obesity and its disease sequelae.

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