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Associations of Weight Stigma With Cortisol and Oxidative Stress Independent of Adiposity

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Abstract

Objective—Weight discrimination is associated with increased risk of obesity. The mechanism of this relationship is unknown, but being overweight is a highly stigmatized condition and may be a source of chronic stress that contributes to the development and pathophysiology of obesity. The objective of this study was to test whether weight stigma is associated with physiological risk factors linked to stress and obesity, including hypercortisolism and oxidative stress, independent of adiposity.

Method—We examined the frequency of experiencing situations involving weight stigma and consciousness of weight stigma in relation to hypothalamic–pituitary–adrenal axis activity and oxidative stress (F_2-isoprostanes) in 45 healthy overweight to obese women.

Results—Independent of abdominal fat, weight stigma was significantly related to measures of cortisol (including salivary measures of cortisol awakening response and serum morning levels) as well as higher levels of oxidative stress. Perceived stress mediated the relationship between weight stigma consciousness and the cortisol awakening response.

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Conclusion—These preliminary findings show that weight stigma is associated with greater biochemical stress, independent of level of adiposity. It is possible that weight stigma may contribute to poor health underlying some forms of obesity.

Keywords

social stigma; weight stigma; oxidative stress; cortisol; stress; obesity; cortisol awakening response

The rise in obesity prevalence in the United States has elicited a heated dialogue across government, medical, and academic institutions, centering on the negative consequences of an obese society, such as chronic health conditions, shorter life expectancy (Fontaine, Redden, Wang, Westfall, & Allison, 2003), and economic costs (Hammond & Levine, 2010). The framing of obesity as a health threat and social burden creates fertile ground for stigma to develop. Discrimination rates based on weight are similar to those based on race and gender, and higher in some instances, such as those involving interpersonal mistreatment (Puhl & Heuer, 2009).

Could exposure to weight stigma have negative health consequences? In a nationally representative study (Sutin & Terracciano, 2013), weight discrimination was associated with a 2.5-fold increased risk of becoming obese over 4 years. Weight stigma could contribute to the development and/or pathophysiology of obesity independent of adiposity by initiating stress processes, as social–evaluative threat triggers hypothalamic–pituitary–adrenocortical (HPA) axis activation (Dickerson & Kemeny, 2004). Hypercortisolism drives overeating, increased adiposity, and is implicated in conditions comorbid with obesity, including Type 2 diabetes and cardiovascular disease (Muennig, 2008). Weight stigma is associated with higher cardiovascular reactivity (Major, Eliezer, & Reick, 2012) and modulates the relationship between central adiposity and glycemic control (Tsenkova, Carr, Schoeller, & Ryff, 2011). Our first aim, therefore, was to test whether weight stigma relates to increased cortisol indices. Secondly, stigma processes may affect disease risk factors in addition to cortisol. Racial stigma has been associated with increased oxidative stress (Szanton et al., 2012), a pathogenic mechanism underlying numerous disease states comorbid with obesity (Basu, 2008). Increased stress-related hormones, including cortisol, may disrupt the activity of antioxidant enzymes (Patel et al., 2002). Our second aim, therefore, was to provide an initial test of whether weight stigma relates to oxidative stress.

Method

The parent study of the current study was a randomized waitlist-controlled trial of a mindfulness-based intervention for stress eating (Daubenmier et al., 2011). The current analyses include all 47 women from the parent study who completed the measures below, all collected prior to randomization. The University of California, San Francisco, Committee on Human Research approved all procedures, and all participants provided written informed consent. Eligibility criteria were: body mass index (BMI) of 25–40 kg/m^2; premenopausal; no history of diabetes, cardiovascular disease, or active endocrinologic disorder; not pregnant or less than 1 year postpartum; no prior/current meditation or yoga practice; not

currently on a diet plan; no current self-reported eating disorder, alcohol, nicotine, or drug addiction; and not taking opiates, steroids, or antipsychotic medications.

Eligible participants underwent two in-lab baseline assessments, which included dual-energy x-ray absorptiometry (DEXA), and responded to questionnaires at home. Nursing staff measured height and weight and conducted blood draws. Participants took home saliva kits and collected samples across 4 days at awakening, 30 min post awakening, and across 3 days hourly between 1:00 and 4:00 p.m., and bedtime. Participants then returned to submit their saliva samples and provided fasting blood samples.

Measures

See supplemental materials online for methodologic details.

Adiposity—Nursing staff conducted weight and height measurements, and DEXA assessed abdominal and total body fat.

Weight stigma—To capture both exposure to and consciousness of weight stigma, we used two scales and also created a composite score. The Stigmatizing Situations Inventory (Myers & Rosen, 1999) measures how often respondents experienced 50 specific weight-stigmatizing situations. Cronbach's alpha was .94. The Stigma Consciousness Scale (Pinel, 1999), which measures consciousness of stigma due to race/gender/sexual orientation, was adapted for weight stigma. Cronbach's alpha was .80. To capture the total experience of weight stigma, we calculated a sum score of the two measures' *z* scores. The Cronbach's alpha of this composite measure was .95.

Cortisol—Salivary free cortisol samples were collected using standard diurnal cortisol sampling protocols. We derived three salivary cortisol indices: (a) total daily secretion using the area-under-the-curve-g formula (Pruessner, Kirschbaum, Meinlschmidt, & Hellhammer, 2003), (b) cortisol awakening response (CAR), by subtracting the waking sample from the 30-min sample, and (c) diurnal slope, the bedtime value subtracted from the waking value (lower numbers indicate steeper slope). Serum cortisol concentrations were measured from fasting morning blood samples.

Oxidative stress—F_2-isoprostane levels assessed systemic *in vivo* oxidant stress status (Milne et al., 2007). Blood was collected in 10 ml ethylenediaminetetraacetic acid tubes, centrifuged, divided into 2-ml aliquots, and stored immediately at −80°C.

Covariates—Self-reported income, self-reported education, and global perceived stress, as measured by the Perceived Stress Scale (Cohen, Kamarck, & Mermelstein, 1983), were included in analyses as possible confounding variables.

Results

Descriptive statistics appear in Table 1. See supplemental materials online for detailed results. To isolate the association of weight stigma with cortisol and oxidative stress, multivariate regression analyses modeled the relation between predictor and outcome

variables controlling for abdominal adiposity, because abdominal fat in particular has been linked to cortisol levels— both high (Champaneri et al., 2013) and low (Steptoe, Kunz-Ebrecht, Brydon, & Wardle, 2004)—and oxidative stress (Pou et al., 2007). Oxidative stress analyses additionally controlled for age (Jacob, Hooten, Trzeciak, & Evans, 2013). Analyses adjusted for the potential confounds of income, education, and global perceived stress when any were significantly related to either stigma or outcomes. Perceived stress, however, may be a mediator rather than a confound, such that weight stigma may contribute to greater perceptions of perceived stress, which in turn may increase cortisol levels, consistent with the model put forth by Dickerson and Kemeny (2004). When perceived stress was related to any outcome measure, therefore, we also tested mediation models using the INDIRECT macro available from Preacher and Hayes (2008).

After adjusting for confounds, the composite measure of weight stigma was positively related to morning serum cortisol levels and F_2-isoprostanes (see Table 2). Weight stigma frequency was positively related to morning serum cortisol levels and CAR. Weight stigma consciousness was positively related to morning serum cortisol levels and F_2-isoprostanes. Furthermore, perceived stress mediated the relationship between weight stigma consciousness and CAR. These effects were similar among those in the lowest quartile of BMI (<27.7, see supplemental Figure S1) as in the rest of the sample.

Any effect of experiencing stigmatizing events may be dependent on having higher stigma consciousness (Kaiser, Vick, & Major, 2006). However, no Frequency × Consciousness moderation models revealed significant interactions (see supplemental Results).

Discussion

We observed significant relationships between weight stigma and markers of HPA activation and oxidative stress. Even after accounting for adiposity, weight stigma was positively related to two independent indices of cortisol output (from a blood draw and CAR on separate days) and F_2-isoprostane levels, an indicator of oxidative stress. Specifically, the *frequency* of experiencing weight stigma was positively related to both morning cortisol indices, and *consciousness* of weight stigma was positively related to oxidative stress in addition to morning cortisol levels. When combined, the composite measure of weight stigma was positively related to both morning cortisol levels and oxidative stress. We found that abdominal adiposity was associated with cortisol and oxidative stress, as found in prior studies (e.g., Steptoe et al., 2004; Pou et al., 2007); however, weight stigma was associated with morning serum cortisol and oxidative stress above and beyond that accounted for by abdominal or total adiposity. Oxidative stress unfolds slowly and is proximal to disease outcomes, suggesting that weight stigma may contribute to the development of chronic disease. Indeed, our sample had higher levels of F_2-isoprostanes than healthy normal adults, suggesting that both adiposity and perceptions of stigma may increase oxidative stress levels among overweight and obese women (60 vs. 35 pg/ml; Milne et al., 2007). Taken together, these findings are consistent with a novel idea that, in addition to the well-recognized health effects of adiposity, living with weight stigma *in itself* may contribute to obesity pathophysiology.

Our findings are somewhat consistent with Szanton et al. (2012), who found positive associations between racial discrimination and oxidative stress, although they used only a measure of *frequency* of experiencing stigma. We also found a significant positive association between frequency of experiences with weight stigma and oxidative stress but not after controlling for adiposity. In the case of weight stigma, it may be more difficult to disentangle the effects of excess adiposity on the relation between frequency of stigmatizing events and oxidative stress. Greater adiposity may influence both exposure to stigmatizing events and oxidative stress, thereby accounting for the relation between frequency of stigmatizing experiences and oxidative stress. Interestingly, exposure to stigmatizing situations but not consciousness of weight stigma was related to total adiposity (see supplemental Table S1), lending some support to this alternative hypothesis. However, the standardized coefficients for stigmatizing events were on par with those of stigma consciousness, which remained significant after controlling for adiposity (Table 1), suggesting weight stigma frequency may have also been significantly related to oxidative stress given more power to detect such a relationship, such as a larger sample.

Weight stigma frequency and consciousness were moderately correlated here, as in other studies (e.g., Bunn, Solomon, Miller, & Forehand, 2007). Stigma consciousness can represent a form of vigilance or prejudice expectations that direct attention toward threatening cues that could be then classified as a stigmatizing event (Kaiser et al., 2006). However, our interaction analyses indicated that the effect of stigmatizing events was not dependent on stigma consciousness. Instead, weight stigma consciousness was associated with higher global perceived stress, which in turn mediated the relationship between stigma consciousness and CAR.

The following limitations qualify these findings. Our sample size was small and, therefore, all findings should be considered preliminary. The study was cross-sectional, which precludes statements about causality. Although reverse causation (oxidative stress causing weight stigma) is unlikely, some third variable, such as depression (Golden, 2007), rejection sensitivity (Slavich, O'Donovan, Epel, & Kemeny, 2010), or menstrual phase (Roca et al., 2003), may explain these findings. The study contained only women, and stigma processes may operate differently in overweight/obese women, members of two groups that experience discrimination, versus overweight/obese men, members of one.

Our findings, if replicated, have important implications. We found the same pattern of associations of weight stigma with HPA activity and oxidative stress indices in those at the lower and higher BMI ranges in our sample (see supplemental Figure S1). These findings are consistent with the notion that weight stigma may not only be harmful for obese individuals, but in fact may also affect women who are nearer to what is considered "normal" weight. In other words, it is likely the self-perception or appraisal of weight rather than objective BMI that is most relevant in the weight stigma domain. Weight stigma, therefore, may need to be treated in addition to obesity. Some public health and policy efforts have used weight stigma as a strategy to motivate individuals to lose weight, such as Georgia's controversial Strong 4 Life campaign that uses images of overweight children with stigmatizing messages (Teegardin, 2012). Our results imply that such policies may be

ineffective, if not counterproductive. As we treat obesity and its comorbidities, it may be important not to stigmatize the very patients we wish to treat.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Descriptive Statistics

Variables	% or *M*	*SD*	Minimum	Maximum
Age (years)	40.89	7.32	21.00	50.00
Body mass index (kg/m^2)	31.35	4.76	24.89	42.62
Abdominal fat (g)	2,119.47	651.93	999.00	3,610.00
Race				
White	61.70			
Asian/Pacific Islander	19.10			
Hispanic/Latino	14.90			
Other	4.30			
Income	5.36	1.61	1	7
Education	1.85	0.98	1	4
Perceived Stress Scale	1.90	0.59	0.40	3.5
Weight stigma				
Composite (z-score)	0.01	0.44	−0.64	1.14
Stigmatizing situations	89.00	33.36	52.00	193.00
Stigma consciousness	40.27	7.58	26.00	57.00
Cortisol				
Morning serum (μg/dl)	9.97	1.48	4.18	22.20
Daily total AUC (nmol/L)	9.03	1.42	4.39	27.11
Awakening response (nmol/L)	8.50	1.84	1.82	22.65
Diurnal slope (nmol/L)	−14.59	5.58	−5.81	−28.97
F_2-isoprostanes (ng/ml)	0.06	0.03	0.03	0.20

Note. Stigma variables represent sum scores. Q-Q plots indicated cortisol and F_2-isoprostane values were non-normal and were natural-log-transformed. One participant with a body mass index (BMI) higher than our inclusion criterion of 40 kg/m^2 was included due to a discrepancy between self-reported BMI during the eligibility screening and BMI measured in the lab.

AUC = area under the curve. Income categories were represented as 1 = Under $25,000, 2 = $25,000–$34,999, 3 = $35,000–$49,999, 4 = $50,000–$74,999, 5 = $75,000–$99,999, 6 = $100,000–$149,999, and 7 = Above $150,000. Educational attainment categories were represented as highest level of education completed, with 1 = Less than 12 years, 2 = High school graduate, 3 = Some college or technical school, 4 = AA degree, 5 = Bachelor's degree, and 6 = Advanced degree.

Table 2

Multiple Regression Results of Weight Stigma and Outcome Measures Adjusting for Abdominal Fat and Other Confounds

Variable	Predictor	β	b	*SE*	95% CI
Composite weight stigma: frequency + consciousness					
Cortisol					
Morning serum (*n* = 44)	Composite	0.39*	0.35*	0.15	[0.05, 0.65]
	Abdominal fat	−0.35*	−2E^{-4}*	9E^{-5}	[−4E^{-4}, −2E^{-5}]
	Income	−0.06	−0.02	0.04	[−0.09, 0.06]
	Global stress	−0.11	−0.07	0.11	[−0.29, 0.14]
Daily total (AUC) (*n* = 37)	Composite	0.35	0.30	0.15	[−0.01, 0.61]
	Abdominal fat	−0.17	0.00	0.00	[0.00, 0.00]
	Income	−0.16	−0.04	0.04	[−0.12, 0.04]
	Global stress	0.23	0.13	0.10	[−0.06, 0.32]
Awakening response (*n* = 41)	Composite	0.31	5.46	2.82	[−0.24, 11.17]
	Abdominal fat	−0.07	−0.001	0.002	[−0.01, 0.003
	Income	0.15	0.74	0.74	[−0.77, 2.25]
	Global stress	0.33*	4.48*	2.02	[0.39, 8.57]
Diurnal slope (*n* = 40)	Composite	0.14	1.73	2.19	[−2.72, 6.18]
	Abdominal fat	−0.24	−0.002	0.001	[−0.01, 0.001]
	Income	−0.04	−0.15	0.60	[−1.36, 1.07]
	Global stress	−0.27	−2.46	1.57	[−5.64, 0.72]
F_2-isoprostanes (*n* =44)	Composite	0.32*	0.26*	0.12	[0.01, 0.51]
	Abdominal fat	0.22	0.00	0.00	[0.00, 0.00]
	Age	−0.20	−0.01	0.03	[−0.02, 0.004]
	Income	−0.03	−0.01	0.034	[−0.08, 0.06]
	Global stress	0.09	0.05	0.09	[−0.12, 0.23]
Weight stigma frequency					
Cortisol					
Morning serum (*n* = 44)	Frequency	0.36*	0.004*	0.002	[9E^{-5}, 0.01]
	Abdominal fat	−0.38*	−2E^{-4}*	1E^{-4}	[−4E^{-4}, −3E^{-5}]

Variable	Predictor	β	b	*SE*	95% CI
	Income	−0.05	−0.01	0.04	[−0.09, 0.07]
Daily total (AUC) (*n* = 37)	Frequency	0.32	0.004	0.002	[−0.001, 0.01]
	Abdominal fat	−0.21	0.00	0.00	[0.00, 0.00]
	Income	−0.15	−0.04	0.04	[−0.12, 0.05]
Awakening response (*n* = 41)	Frequency	0.38*	0.08*	0.04	[$1E^{-4}$, 0.17]
	Abdominal fat	−0.16	−0.002	0.002	[−0.01, 0.002]
	Income	0.25	1.15	0.78	[−0.44, 2.73]
Diurnal slope (*n* = 40)	Frequency	0.12	0.02	0.03	[−0.04, 0.08]
	Abdominal fat	−0.24	−0.002	0.001	[−0.01, 0.001]
	Income	−0.08	−0.27	0.62	[−1.52, 0.99]
F_2-isoprostanes (*n* =44)	Frequency	0.26	0.002	0.002	[−0.001, 0.01]
	Abdominal fat	0.23	0.00	0.00	[0.00, 0.00]
	Age	−0.07	−0.003	0.01	[−0.02, 0.01]
	Income	−0.09	−0.02	0.03	[−0.08, 0.05]
Weight stigma consciousness					
Cortisol					
Morning serum (*n* = 45)	Consciousness	0.33*	0.02*	0.01	[$5E^{-4}$, 0.03]
	Abdominal fat	−0.25	0.00	0.00	[0.00, 0.00]
	Global stress	−0.13	−0.09	0.11	[−0.30, −0.13]
Daily total (AUC) (*n* = 38)	Consciousness	0.32	0.02	0.01	[−0.002, 0.03]
	Abdominal fat	−0.07	0.00	0.00	[0.00, 0.00]
	Global stress	0.17	0.10	0.10	[−0.10, 0.30]
Awakening response (*n* = 42)	Consciousness	0.24	0.25	0.16	[−0.08, 0.057]
	Abdominal fat	−0.03	0.00	0.002	[−0.004, 0.003]
	Global stress	0.36*	4.83*	2.02	[0.73, 8.92]
Diurnal slope (*n* = 41)	Consciousness	0.10	0.07	0.12	[−0.18, 0.33]
	Abdominal fat	−0.20	−0.002	0.001	[−0.004, 0.001]
	Global stress	−0.27	−2.53	1.54	[−5.64, 0.59]
F_2-isoprostanes (*n* =45)	Consciousness	0.33*	0.02*	0.01	[0.002, 0.03]
	Abdominal fat	0.28*	$1E^{-4}$*	$7E^{-5}$	[$1E^{-6}$, $3E^{-4}$]

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Variable	Predictor	β	b	*SE*	95% CI
	Age	−0.25	−0.01	0.01	[−0.03, 0.001]
	Global stress	0.06	0.03	0.09	[−0.14, 0.21]

Note. Cortisol units are ln(mg/dl) for serum and ln(nmol/L) for saliva; F_2-isoprostane units are ln(ng/ml). Beta values are standardized units; b values are raw values. Unadjusted analyses are available in supplemental Table 2.

CI = confidence interval; AUC = area under the curve.

We provide values to more than three decimal places only when $p < .05$.

* $p < .05$ (two-tailed).