ASSOCIATIONS BETWEEN WEIGHT DISCRIMINATION AND METABOLIC HEALTH: ANALYSIS OF THE CORONARY ARTERY RISK DEVELOPMENT IN YOUNG ADULTS (CARDIA) STUDY

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Abstract

Background:

Concurrent with the recent rise in overweight and obesity, weight discrimination levels have also increased. Individuals who have experienced weight discrimination report higher levels of stress, depression, lack of diet control, and depletion of the self-control needed for weight management. What remains unclear, is the extent to which metabolic health may also be affected.

Method:

To address this, a secondary data analysis was performed on 1365 participants from year 25 of the Coronary Artery Disease in Young Adults (CARDIA) study who were living with overweight and obesity. Descriptive statistics and logistic regression analyses were performed on the presence of metabolic syndrome, diabetes, and abdominal obesity (and their associated measures) for their perception of the weight discrimination they experienced (none, low stress, or high stress).

Results:

In all cases, prevalence of the metabolic syndrome, diabetes, and abdominal obesity was higher among those reporting low and high stress weight discrimination compared to those with no history of weight discrimination. In the adjusted analyses, weight discrimination was associated with a 65% greater likelihood for having metabolic syndrome, 85% greater likelihood of diabetes, and between 2.5- and 3.9-times greater likelihood of abdominal obesity for low and high stress experiences, respectively.

Conclusion:

Exposure to weight discrimination may worsen metabolic health, as characterized by higher rates of metabolic syndrome, diabetes, and abdominal obesity. These associations may be amplified with higher levels of stress experienced from weight discrimination. Further longitudinal work is necessary to understand the temporal sequence, time lag, and any possible critical periods for weight bias internalization on metabolic health.

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1. Background

1.1 Obesity

According to the World Obesity Federation, obesity is defined as excessive weight in the form of fat that impairs health, is progressive in nature, and is a chronic disease that relapses[1–3]. Levels of obesity have been rising globally in all age and sociodemographic groups. Indeed, a third of people in the United States and a quarter of people in Canada are living with obesity [4]. In Canada, levels of obesity have been rising consistently from the 1980s, with a higher rate of growth for class II and III obesity [5]. Beyond the well-known association between obesity and chronic conditions (e.g. an increased risk of type 2 diabetes, hypertension, cancers, glucose intolerance, psychosocial stress, dyslipidemia, and cardiovascular diseases) [4–6], individuals with class II or III obesity have higher rates of premature death [5]. By 2025, it is estimated that the annual direct and indirect cost of managing obesity-related comorbidities worldwide will exceed $1.2 trillion (USD)[2].

1.2 Current challenges in obesity

At any given time, over 50% of individuals living with obesity may be trying to lose weight [7]. Common strategies for weight loss include dietary restriction, behavioural interventions (e.g. mindfulness, motivational interviewing, etc.), pharmacological, and surgical interventions [3,8]. Reviews of randomized controlled trials indicate that individuals who are overweight or individuals with obesity can lose approximately 10% of their initial weight using a set of comprehensive lifestyle modifications [9]. However, weight maintenance after successful weight loss is challenging, and regain is common [9–12].

Repeated episodes of weight loss and weight regain, denoted as weight cycling, are therefore an area of concern[10]. Weight cycling has been associated with an elevated risk of hypertension[13,14] and type 2 diabetes[15,16] however, there is no consensus as to how much weight cycling affects these associations independent of body weight. Different studies examining these associations use different definitions for weight cycling and often yield inconsistent results [17,18]. Furthermore, there are inconsistencies on whether weight cycling affects overall mortality risk [10,17].

Making matters worse, there is a disconnect between weight loss expectations, clinically healthy weight loss, and weight loss that can be feasibly achieved [19,20]. Prior to weight loss therapies, heavier individuals tend to set more unrealistic weight loss goals [21]. In an early study by Foster *et al.* [20], women living with

obesity listed that their "dream goal" was a 38% weight loss, that they would be "happy" with a 31% weight loss and find a 25% weight loss "acceptable", but would be "disappointed" with a 17% weight loss. After the program, despite an average weight loss of 16%, 47% of the women did not even reach the weight they had listed as a "disappointment" [20]. Because current pharmacological and behavioral treatments typically only lead to 10% weight loss - sufficient to improve the co-morbidities associated with obesity [12,22] - this degree of weight loss may be viewed as a disappointment, as their reported 'acceptable' range is 2 to 3 times more [21]. Despite considerable improvements in health, a 10% weight loss may still mean continued weight dissatisfaction, psychological harm, and social burden for a considerable proportion of individuals with obesity [12,22].

1.3 Weight bias, stigma, discrimination

With increasing prevalence of individuals with overweight and obesity there has been a concomitant increase in bias, stigmatization, and discrimination[23]. Weight bias refers to the negative attitudes and prejudices placed on individuals due to their weight[23]. These attitudes and prejudices are common throughout North America [12] and people living with overweight and obesity are often labeled as lazy, having poor willpower, have low self-esteem, not intelligent, and are subjected to blame for their extra weight [12]. These stereotypes promote weight stigma that arises in the form of social rejection, derogation, and devaluation [23–25]. These social stigmas, may in turn contribute to weight discrimination, which occurs when negative actions and unfair treatment is enacted towards individuals *due to* their weight [26]. While prejudices and stereotypes reflect negative attitudes, discrimination refers to a behaviour [26]. Weight bias, stigma, and discrimination are used interchangeably throughout the literature [27]. As a result, this thesis will focus on weight discrimination, but used studies focussing on weight bias and stigma to shape the theoretical approach.

Weight discrimination is pervasive in the United States and is the fourth most common form of discrimination after sex, race, and age discrimination. In one of the first comprehensive studies, Puhl *et al.* [26] examined weight discrimination across the United States and found that in all Body Mass Index (BMI) groups combined, approximately 5% of men and 10% of women had experienced it, with higher rates for relatively younger individuals. For women alone, weight or height discrimination is the third most common form of discrimination after gender and age, and women living with class I obesity have a three-fold greater risk than men for weight or height discrimination. When taken together, 40% of U.S. adults with class II obesity reported a history of weight discrimination that included being targeted for

their weight by family members[28], at school by educators and peers [25,29–32], in the workplace[33,34], and particularly, in healthcare environments[35–40]. Indeed, health care providers (physicians, medical students, nurses, and other professionals) reportedly exhibit negative attitudes towards their patients with (perceived) excess weight which may perpetuate as poorer quality of care [12,27,41,42]. Physicians also report less desire to help patients the heavier they are[41]. Compared to thinner patients, patients with obesity have reported receiving less time and less health education from healthcare providers, with their weight becoming the sole reason for their health problems when consulting healthcare services [12]. A sense that they are not being taken seriously can also result in these patients not wanting to discuss any weight related concerns [12]. This suggests that individuals with obesity are less likely to utilize healthcare and more likely to delay healthcare services or avoid it completely [12,42,43].

1.4 Health impacts

Experiences of weight discrimination can promote behaviours that lead to poor health outcomes. In both clinical and non-clinical samples, people with obesity and experiences of weight stigma have higher levels of stress[44], depression[25], body dissatisfaction[25,45], anxiety[42], and lower self-esteem[25,42,46]. Children that are bullied due to their weight also experience higher levels of stress, body dissatisfaction, and lower self-esteem even after adjusting for factors such as demographics and BMI[47].

Current research has shown that weight stigma may have an impact on behaviors surrounding physical activity and eating. Children that are targeted due to their weight are more likely to practice riskier forms of weight loss and binge eating than children with overweight who have not been targeted [12]. Furthermore, youths also cope with the stigma by eating more and refraining from physical activity[48]. Similarly, adults who face weight stigma are more likely to binge eat and may develop eating disorders[49,50]. Individuals experiencing weight stigma and who begin to internalize it, may avoid taking part in physical activity and exercise and lose the motivation to do so[51,52]. While indirect, these patterns appear to be independent of current BMI and body dissatisfaction[53]. Importantly, in experimental studies, women within the overweight range who were exposed to weight stigmatizing scenarios showed increases in caloric consumption suggesting that weight stigma also promotes overeating[49].

Some studies have also examined the associations between weight discrimination on physiological health. Among studies in cardiometabolic health, weight discrimination has been found to be an effect modifier (i.e. heighten the effect) of waist-to-hip ratio on glycosylated hemoglobin (HbA1c)[54] and is

associated with higher levels of C-reactive protein[55]. Among women with overweight, experimentally induced weight stigma showed increases in blood pressure [29]. Although the mechanism behind these physiological changes is not fully known, stress responses have been implicated. Prolonged weight discrimination may prompt chronic activation of the sympathetic nervous system, leading to further, adverse health outcomes[45]. Weight stigma has also been shown to cause elevated cortisol responses[56] and elevated stress responses[57]. Because cortisol is associated with greater abdominal adipose tissue[44], this pathway with weight discrimination may reinforce the positive feedback loop with obesity, diabetes, and cardiovascular disease [56]. Indeed, in a longitudinal study, Sutin and Terracciano[58] found that participants with weight discrimination were 2.5 times more likely to develop obesity, and three times more likely to remain within the obesity range at follow-up than those without discrimination[58]. Jackson *et al.* [59] found that participants with perceived weight discrimination had a higher likelihood of becoming, but not remaining within the obesity range [59]. Taken together, there is now agreement that weight discrimination can increase the co-morbidities associated with obesity, and further disease through the stresses brought on by discrimination[55,58,60]. What is less clear is the extent to which weight discrimination contributes to a disease state, inclusive of the role of stress (internalization) and different forms of weight discrimination that might occur at different times or settings, particularly in early life.

2. Rationale

Increasing levels of obesity are associated with higher levels of co-morbidities. Weight discrimination is a concurrent issue which, in a proportion of individuals, may contribute to an increased uptake of unhealthy weight loss behaviours [12,27,48–52]. Individuals reporting a history of weight discrimination may in turn partake in extreme forms of weight control, binge eating [58], and/or refraining from exercise which can contribute to difficulties in weight maintenance [51,52], more weight cycling [61–63], stress [44], and deteriorations in metabolic health [54,55]. What is currently unknown, is the extent to which poor metabolic health is exclusively due to body weight, or if there is an effect of weight discrimination.

3. Aims

To estimate the association between weight discrimination and metabolic dysfunction, type 2 diabetes, and abdominal obesity.

4. Manuscript

5. Abstract

Background:

Concurrent with the recent rise in overweight and obesity, weight discrimination levels have also increased. Individuals who have experienced weight discrimination report higher levels of stress, depression, lack of diet control, and depletion of the self-control needed for weight management. What remains unclear, is the extent to which metabolic health may also be affected.

Method:

To address this, a secondary data analysis was performed on 1365 participants with overweight and obesity from year 25 of the Coronary Artery Disease in Young Adults (CARDIA) study. Descriptive statistics assessed the prevalence of main outcomes of metabolic syndrome, diabetes, and abdominal obesity (and associated measures: e.g., anthropometrics, blood/lipid measures) for weight discrimination experienced (none, low stress, or high stress). Logistic regressions estimated the relationship between weight discrimination and main outcomes adjusting for age, sex, race, alcohol intake, physical activity, family income, and number of discrimination events.

Results:

In all cases, prevalence of the metabolic syndrome, diabetes, and abdominal obesity was higher among those reporting low and high stress weight discrimination compared to those with no history of weight discrimination. In the adjusted analyses, weight discrimination was associated with a 65% greater likelihood for having metabolic syndrome, 85% greater likelihood of diabetes, and between 2.5- and 3.9-times greater likelihood of abdominal obesity for low and high stress experiences, respectively.

Conclusion:

Exposure to weight discrimination may worsen metabolic health, as characterized by higher rates of metabolic syndrome, diabetes, and abdominal obesity. These associations may be amplified with higher

levels of stress experienced from weight discrimination. Further longitudinal work is necessary to understand the temporal sequence, time lag, and any possible critical periods for weight bias internalization on metabolic health.

6. Introduction

Overweight and obesity are known to impact on physical, functional, and psychological aspects of health[64], presenting as poorer quality of life, higher risk of disease, and chronic disease (i. e. different types of cancers, cardiovascular risks, type II diabetes mellitus, osteoarthritis, etc.) and experiences of pain and discomfort[64]. Current studies indicate that weight loss is difficult to maintain long term with most individuals experiencing weight regain and weight cycling which impair health and wellbeing[9–12]. There is also a disconnect between weight loss expectations and what is clinically feasible[19,20]. Taken together, this means that individuals with overweight or obesity are more likely to remain that way and remain susceptible to its psychological harm and social burden[12,22]. With recent increases in the prevalence of overweight and obesity there has been a concurrent rise in weight bias, stigmatization, and discrimination[23,26].

Weight biases are the negative attitudes and prejudices placed on individuals due to their weight[23]. These biases manifest themselves into weight stigma in the form of stereotypes (that individuals with obesity are lazy, have poor willpower, low self-esteem, are unintelligent, and are to blame for their extra weight [12]) which leads to social rejection, derogation, and devaluation [23–25]. Weight discrimination, on the other hand, is when these biases and stigma transmute into negative actions and unfair treatment enacted towards individuals [26].

Weight stigma and discrimination is pervasive, and can take the form of discrimination at home from family members[28], at school by educators and peers [25,29–32], in the workplace[33,34], and particularly within in healthcare environments[35–40]. Weight discrimination has been under-recognized until recently where its growing prevalence and impacts warrant further study[26,65]. The psychosocial stress from weight discrimination has served to promote several maladaptive self-care behaviours such as unhealthy eating[50], higher caloric consumption[49], binge eating[66], higher likelihood of developing eating disorders[49,50], and reducing physical activity[51,52], and motivation for physical activity[51,52]. These poor self-care behaviours contribute to deteriorations in metabolic health[54,55] where weight discrimination has been positively associated with C-reactive protein[55], cortisol (marker of stress)[67], glycosylated hemoglobin (HbA1c)[54], risk for diabetes[68], and obesity[68].

Currently, it is unknown whether these deteriorations in metabolic health are due to body weight, or if weight discrimination itself has an effect. The aim of this study is to therefore estimate the association between weight discrimination and metabolic syndrome, elevated blood glucose (i.e. oral glucose tolerance test, glycosylated hemoglobin, and type 2 diabetes risk), and abdominal obesity.

7. Methods

Data for this study was derived from the Coronary Artery Risk Development in Young Adults (CARDIA) and conducted under the auspices of an existing limited dataset access by the National Heart, Lung, and Blood Institute (NHLBI). Ethics approval was granted by York University (e2017-364).

7.1 CARDIA Study

The CARDIA study is a multicenter prospective cohort study that examined risk factors leading to the etiology and development of coronary heart disease in young adults. In 1985 and 1986, 5115 black and

white individuals between 18 to 30 years of age were recruited from four urban centres (Oakland, California; Minneapolis, Minnesota; Chicago, Illinois, and Birmingham, Alabama). The cohort was divided into subgroups based on sex, race/ethnicity, and education such that each subgroup contained a balanced number of individuals. Baseline data was collected in 1985-1986 then through follow-ups in spaced intervals of 2, 5, 7, 10, 15, 20, 25, and 30 years. Retention rates were 90% in year 2, 86% in year 5, 81% in year 7, 79% in year 10, 74% in year 15, 69% in year 20, 68% in year 25, and 66% in year 30. The study recorded information on traditional risk factors for heart disease such as anthropometrics, blood chemistry (i.e. %HbA1c, glucose, insulin, triglycerides, HDL cholesterol), blood pressure, lifestyle factors (i.e. physical activity, smoking status, education, and alcohol consumption), psychological factors and psychosocial factors. Even though the aim of the cohort was to examine the evolution of heart disease, the variety of risk factors and variables recorded and tracked has enabled notable health insights into other domains[69]. Further details of the CARDIA study have been described elsewhere[69].

7.2 Sample

Data from Year 25 of the CARDIA study (Y25) was used, which had 3473 participants. Data for the current analysis was limited to individuals within the overweight or obesity BMI categories (BMI: ≥25 $kg \cdot m^{-2}$)[70] leaving 1365 non-pregnant participants. Participants who did not respond to weight discrimination questionnaire were omitted and pair-wise deletion was used to generate the analytic sample (N~1363) for metabolic syndrome (MetS), type 2 diabetes mellitus (T2DM), and abdominal obesity analyses.

7.3 Exposures

The primary exposure variable for the current analysis was weight discrimination. Weight discrimination was recorded on Form 44[71] during year 25 (2010-11) of the CARDIA study for the first time using self-

administered questionnaires. Participants responded in a Yes/No format indicating if they experienced weight discrimination in seven different situations (at school, getting a job, getting housing, at work, at home, getting medical care, and on the street or public setting). Answering "Yes" to any of these situations prompted follow-up questions concerning how often these experiences of discrimination occurred (rarely, sometimes, or often) and how collectively stressful all of these experiences were (none, slightly, somewhat, moderate, or very). There were no differences seen in baseline BMI values between those who participated in the Year 25 CARDIA survey and those who did not[72].

7.4 Outcomes

Primary study outcomes were Y25 measures of weight (e.g. BMI and waist circumference (WC)), MetS, oral glucose tolerance test (OGTT), insulin levels, glycosylated hemoglobin (HbA1c), and Type 2 Diabetes Mellitus (T2DM).

7.4.1 Metabolic syndrome

MetS is a term given to the clustering of metabolic risk factors that elevates risk of cardiovascular disease, stroke, and diabetes[73,74]. Diagnosis of MetS was based on the presence of at least three out of five metabolic risk factors according to the revised National Cholesterol Education Program Adult Treatment panel III (NCEP ATP III) [73,74]:

1. Abdominal Obesity: waist circumference >102 cm in men or >88 cm in women.
2. Plasma triglycerides: ≥1.69 mmol/L or taking lipid medication.
3. Blood pressure: SBP>130 mmHg or DBP>85 mmHg or taking antihypertensive medications.
4. Fasting plasma glucose: ≥5.6 mmol/L or taking diabetes medications.
5. High Density Lipoprotein Cholesterol (HDL-C): <1.04 mmol/L in men and <1.29 mmol/L in women

7.4.2 Anthropometric measures

Waist circumference (WC) was measured using a Gulick II Plus anthropometric tape at the midpoint between the iliac crest and lowest lateral portion of the ribcage[75]. WC was measured twice to the nearest 0.5 cm at minimum waist girth and averaged[75]. Body weight was measured to the nearest 0.2 kg with participants in light clothing on balance beam scales[75]. Height was measured to the nearest 0.5 cm without shoes[75]. BMI was calculated as weight in kilograms divided by height squared in metres and used to group participants into established BMI categories [70].

7.4.3 Blood pressure

Blood pressure was measured using an automated blood pressure measurement monitor. Participants rested for five minutes before their seated blood pressure was measured three times form their right arm. The second and third systolic and diastolic blood pressures were averaged[76,77].

7.4.4 Biological measurements

Blood was collected by a phlebotomist from participants in a fasting state of at least eight hours. Venous blood samples were drawn and used for the measurement of glucose, insulin, and lipid levels. The serum and plasma was stored in cryovials and shipped to central laboratories for analyses[77,78]. Glucose was measured using a Roche Modular P hexokinase method[76]. Insulin levels were measured with a sandwich immunoassay[76]. Total cholesterol and triglyceride levels were determined enzymatically. Total HDL cholesterol was determined enzymatically with standard laboratory methods [78–80], whereas LDL cholesterol was calculated using the Friedwald Equation[81]. Details of the storage and analysis have been described elsewhere [77–80].

7.4.5 Oral Glucose Tolerance Test (OGTT)

Blood was collected by a phlebotomist from participants in a fasting state of at least eight hours. Participants then drank a 75-gram glucose solution (Glucola) as quickly as possible. Two hours after drinking the glucose drink 5mL of blood was drawn for OGTT glucose analyses. Details of the analyses have been described previously[76].

7.4.6 Diabetes

A history of diabetes mellitus was determined using self-report. At examinations, participants reported whether they have or had diabetes mellitus and if they are taking any medications for it [82]. The family history questionnaire asked participants if their natural parents had diabetes and at what age, and the number of full siblings with diabetes[83] [77]. Anyone reporting use of a T2DM medication, with fasting plasma glucose ≥7 mmol/L, HbA1c ≥ 6.5%, or OGTT was ≥ 11.1 mmol/L was considered to have T2DM[84].

7.5 Statistical Analysis

Sociodemographic, behavioural, and medical characteristics of the sample were compared in those with and without a history of weight discrimination using one-way analysis of variance (ANOVA) and chi-square analysis. To incorporate stress (i.e. internalization) from weight discrimination into the analyses, those with weight discrimination were dichotomized into a low stress (survey answers of none/slightly/somewhat)[71] and high stress (survey answers of moderate/very) [71].

The following analyses were performed to estimate the relationship between weight discrimination (none, low stress, high stress) with abdominal obesity, BMI-defined obesity, and cardiometabolic health measures (i.e. HbA1c, metabolic syndrome, and elevated blood glucose). Using continuous values and established clinical thresholds, the prevalence of abdominal obesity, BMI-defined obesity (30+ $kg \cdot m^{-2}$),

and metabolic health (MetS (overall and components), elevated blood glucose (≥5.6mM), elevated A1C (≥6.5%), insulin (≥90th percentile), and T2DM) were compared across weight discrimination groups using a one-way ANOVA and chi-square analysis, as appropriate. Tukey post-hoc tests were performed to identify group differences for any significant ANOVA main effects ($p<0.05$). Using the "no" weight discrimination group as the referent (OR=1.00), odds (95% CI) of metabolic dysfunction (MetS, T2DM, and abdominal obesity) were estimated through a series of logistic regressions: Model 1 examined the unadjusted raw relationships between weight discrimination and each outcome; Model 2 adjusted for any socio-demographic characteristics that were significant (i.e. age and sex), and; Model 3 adjusted for all other significant factors related to history of weight discrimination. This included total physical activity (EU), alcohol consumption (mL/day), family income, and the number of discrimination events (low (0-3), high (4-7). Only the total physical activity score was included in the model as it contained both moderate and heavy intensity scores, and to prevent violation of the collinearity assumption. After preliminary inspection, age- and sex-interactions by BMI category could not be investigated due to insufficient sample sizes (i.e. cell sizes of events ~$n<20$). All analyses were performed using SAS version 9.4 (SAS Institute, Cary, NC) with statistical significance established at $p \leq 0.05$.

7.5.1 Covariates

The covariates adjusted for were age, sex, race/ethnicity (black or white), alcohol consumption (mL/day), physical activity (expressed in Exercise Units, "EU"), number of discrimination events (low (0 to 3) high (4 to 7)), and family income. These covariates were chosen based on their associations with obesity and metabolic health.

The CARDIA Physical Activity History Questionnaire, an interview-based self report, was used to assess physical activity. Thirteen categories of physical activity, 8 vigorous and 5 moderate intensity, were scored whether it was performed for at least 1 hour of any 1 month in the past 12 months, the

consistency of the performance (how many months at that level), and the number of months an activity was performed frequently [85–87]. The sum of the scores are expressed as "Exercise Units" (EU) with separate scores for moderate intensity, vigorous intensity, and total activities [85–87]. As a reference, 300 EU is roughly equivalent to 150 minutes of moderate intensity activity per week or the amount of activity required to maintain health as per the American College of Sports Medicine recommendations [86,88]. The validity and reliability of this questionnaire is comparable to other physical activity questionnaires [89,90].

8. Results

8.1 Sociodemographic and behavioural

Sociodemographic and behavioural characteristics of the sample (year 25) are presented in **Table 1.** Overall, the mean age of the sample was 50.2 y and was comprised of 50.8% females, 52.8% white participants, approximately, 70% of whom had private or personal health insurance. Regardless of weight discrimination group, about 40% of participants had a high school level education, and 20% had a bachelor's degree, almost half of whom had had a combined family income of at least $75,000 (USD). Notably, over one-third of the high stress group reported a family income within the lowest range (<$5,000-$15,999) as compared to only 13% of the no discrimination, and 18.6% of the low stress groups. Whereas no differences in the proportion of former smokers were found across discrimination groups, alcohol consumption was significantly higher in the "none" versus "low" stress discrimination groups ($p<0.05$), with higher rates amongst males (versus females) at all levels (data not shown). Similarly, total physical activity scores were highest in the no weight discrimination group and lowest in the high stress group ($p<0.05$).

8.2 Clinical Characteristics

8.2.1 BMI, WC, and Obesity-Related Health Risk

After excluding participants with a BMI below the overweight range, average BMI of the sample was 32.3 $kg \cdot m^{-2}$ (**Table 2:** Clinical Outcomes). Overall, almost two-thirds of the sample were people with abdominal obesity, while BMI-defined obesity was present amongst 56.8%. Within obesity classes, the high stress weight discrimination group had a higher percentage of individuals with class II and III obesity, while the low stress group had a higher percentage of individuals with class I obesity. According to BMI cut-offs, 43% of the sample was overweight (>25 $kg \cdot m^{-2}$); however, nearly half of the people in the overweight group reported no weight discrimination. As expected, BMI levels were greater in relation to level of perceived stress of weight discrimination events ($p<.0001$, **Table 2:** Clinical Outcomes). Post hoc tests revealed that both low and high stress weight discrimination groups had a significantly higher BMI and waist circumference than those with no weight discrimination ($p<0.05$).

8.2.2 MetS and Diabetes

In general, few differences in blood and lipid measures were present between weight discrimination groups. Overall, 36.5% of the sample had MetS, the prevalence of MetS rose from 33.1% in no weight discrimination to 48.5% in high stress weight discrimination ($p=0.0008$). Although the prevalence of elevated HbA1c, fasting plasma glucose, 2-hour OGTT, and fasting insulin tended to have higher rates in the low and high stress weight discrimination groups as compared to the no discrimination group, only fasting insulin levels showed a significant difference between groups ($p=0.01$). Overall, 12.9% of the sample had T2DM, with higher proportions among those reporting greater discrimination severity ($p<0.01$). Finally, no differences in triglyceride, HDL-C, and blood pressure levels were observed across weight discrimination group ($P>0.05$).

8.2.3 Multivariable Findings

Odds of having metabolic syndrome, T2DM, or abdominal obesity in those with and without weight discrimination are described in **Table 3** and **Figure 1**, **Figure 2**, and **Figure 3**. Compared to participants in the no discrimination group (OR=1.00), participants who experienced low stress weight discrimination had 57% greater odds of having MetS (OR, 95% CI; 1.57, 1.17-2.10), and 91% greater odds for those with high stress weight discrimination (1.91, 1.16-3.14), effects that persisted after adjustment for age, sex, and race (2.02, 1.21-3.36). These effects remained in the low stress weight discrimination group, however, were attenuated after adjustment for sociodemographic and behavioral factors (1.65, 1.20-2.28).

As compared to no weight discrimination, those with low or high stress weight discrimination had almost two-fold greater odds of having T2DM (low stress: 1.83, 1.25-2.68; high stress: 1.94, 1.03-3.66), an effect that remained in the low, but not high-stress groups, after adjusting for covariates. Finally, when considering abdominal obesity as the outcome, even stronger effects were observed for both low and high-stress groups: in fully adjusted models, low and high stress weight discrimination was associated with 3.48 (2.32-5.22) and 4.86 times (2.05-11.52) greater odds of having abdominal obesity.

9. Discussion

This study examined the relationship between weight discrimination and metabolic health outcomes of MetS, T2DM and abdominal obesity in persons with overweight and obesity. Primary results suggest that weight discrimination is significantly associated with higher levels of MetS, T2DM, and abdominal obesity. Diabetes and other cardiometabolic diseases commonly observed in people with obesity are frequently related to stress, suggesting that the physiological response to discrimination can contribute

to their development[91,92]. The present study included stress (i.e. internalization) from weight discrimination revealing positive associations between stress and some metabolic health markers. Literature about stress caused by weight discrimination and weight-associated health problems is still limited.

9.1 Obesity

BMI levels were higher in those with weight discrimination consistent with prior studies showing a positive association between BMI and weight discrimination[26,93,94]. In a nationally representative study[58] of over 6,000 U.S. adults, weight discrimination alone was associated to a 2.5 times greater likelihood of developing obesity (2.54, 1.58–4.08). Among those who already had obesity, those experiencing weight discrimination were three times more likely to remain with obesity (3.2, 2.06-4.97). The present study complements and extends these findings in that a significantly greater proportion of individuals with abdominal obesity were observed in both the high and low stress weight discrimination groups. Incorporating abdominal obesity considers the distribution of body fat and it is an independent risk factor for cardiometabolic disorders such as systemic inflammation, insulin resistance, and dyslipidemia[95,96]. Furthermore, the present study also observed that higher stress amplifies the positive relationship between weight discrimination and abdominal obesity. A possible explanation for the association between weight discrimination and obesity is that on a behavioural level, individuals avoid physical activity[51,52] and indulge in overeating[49]. Indeed, binge eating is associated with weight stigmatizing experiences[50], weight based teasing in adolescents[97], and internalizing weight stigma[98]. Taken together, this demonstrates that weight discrimination is associated with higher levels of abdominal obesity, further demonstrating that individuals may be at a greater risk for other cardiometabolic disorders, which could be augmented from additional stress [58].

9.2 Diabetes

Overweight and obesity are strong predictors of T2DM[54,99], and while the majority of individuals with T2DM are also living with obesity, many individuals with obesity never develop T2DM[100]. This reinforces the multidimensional aspect of T2DM in that many other factors (e.g. stress, depression, diet, genes, etc.) may affect glycemic control[54]. The current study builds on existing literature showing that weight discrimination is related to a higher prevalence of T2DM. For example, a U.S. based study by Tomoko et al.[93] on over 21,000 people with obesity revealed that perceived weight discrimination was associated with a higher odds of diabetes (as well as high cholesterol, arteriosclerosis, and other heart conditions), independent of BMI, sociodemographics, and physical activity. While consistent, the association in the present study appeared stronger even after adjusting for significant covariates (OR:1.37 vs. OR:1.85)[93]. Previous work has also shown that self-reported weight discrimination is associated with higher levels of HbA1c, a measure of longer-term glycemic control, independent of BMI, total discrimination levels, and other sociodemographics[54,101]. Paradoxically, average HbA1c in the present study did not differ amongst those with and without weight discrimination. Weight discrimination amplifies the already existing positive association between waist-to-hip ratio and HbA1c[54].

9.3 Stress with MetS

The literature surrounding weight discrimination and MetS is sparse. One study examining weight bias internalization and MetS found that those with the highest (vs lowest) level of internalization had three times greater likelihood of MetS after adjusting for demographics and other factors. The majority of this sample was female (88%) and African-American (67%) for whom weight bias internalization is already known to be low[23,102]. The present study may not have examined weight bias internalization explicitly, but it did consider the internalization of weight discrimination through self-reported stress levels, results of which suggest that individuals with higher levels of reported stress had greater prevalence of MetS.

When considering individual components of MetS and weight discrimination, weight discrimination has been found to be associated with greater odds of high cholesterol and hypertension, in men, even after adjusting for BMI, sociodemographics, and physical activity[93]. Furthermore, a study experimentally inducing weight stigma demonstrated higher blood pressure levels across its participants, the majority of whom were white women[29]. Even with the overall consistency of these findings, results from the present study suggest that the stress (i.e. internalization) from weight discrimination may exacerbate increases in MetS components, providing a further explanation for variation in health risk associated with weight discrimination, and highlighting an actionable target.

9.4 Possible Mechanism

Weight stigmatization is a source of chronic stress which stimulates biochemical stress responses as seen in the literature by higher levels of blood pressure, C-reactive protein, oxidative stress, and cortisol which can have detrimental effects on metabolic health and diabetes [23,55,67,103]. Indeed, experimentally exposing individuals to weight stigma contributes to a rise in cortisol reactivity and blood pressure[23,29,104]. One possible mechanism is that stress can activate the hypothalamic pituitary adrenal (HPA) axis, initiating release of cortisol[105,106]. Enhanced cortisol reactivity in response to stress may also contribute to a preference for obesogenic (calorically dense and high fat) foods[107]. Activation of the HPA axis also promotes increased consumption of desirable foods through the release of endogenous opioids, which then contributes to a feed forward loop in which the cycle is repeated[108,109]. Taken together, higher levels of stress brought upon by weight discrimination may lead to emotional and mental distress and biochemical responses leading to increased calorie consumption and binge eating as a coping mechanism[23,49,58]. Further work is necessary to elucidate the physiological mechanism stress and weight discrimination.

9.5 Limitations

The CARDIA self-administered questionnaire concerning weight discrimination assessed lifelong experiences of weight discrimination and is subject to recall bias. How long ago an event of weight discrimination occurred, or if it happened on multiple occasions was not differentiated. Additionally, the sample was based out of four urban centers which may have attracted more educated and health-conscious participants. While the study did attempt to recruit a balanced number of black and white participants across their subgroups, it may not be reflective of the general United States population more broadly. In the current study, the analyses relied on health data from one time point (Y25) as it was the only survey that included weight discrimination data, which does not permit the exploration of longer-term outcomes associated with weight discrimination and possible weight changes over time. Furthermore, due to the cross-sectional nature of this study there is no certainty that the exposure of weight discrimination preceded the development of the outcomes.

9.6 Implications and Future Directions

Weight stigma is common[44] and thin ideal body size biases are introduced, and perpetuated, early in childhood[110], meaning that an individual can experience stigma even within the non-overweight/non-obesity range. This cross-sectional study focussed on people with overweight or obesity since they are more likely to experience weight stigma and discrimination than lower weight individuals, maximizing our chances of having sufficient statistical power for the proposed analysis[44]. Future studies should aim to longitudinally assess outcomes associated with weight discrimination, or how the relationship between a history of weight discrimination and metabolic health might change with weight change, since prior research suggests that perceptions of weight may be more important and impressionable than objective definitions[44].

While preliminary in nature, the current study contributes to the literature highlighting the importance of weight discrimination to metabolic health. It also contributes to a more basic understanding of the public health implications of weight discrimination, instead of a focus on persuading individuals to "lose weight" as treatment. Healthcare professionals may benefit from this information, as it may help in messaging to minimize stigmatization that could propagate health issues in patients who have experienced weight stigma or discrimination[111]. Furthermore, this research provides additional information on the relationship between obesity-related health risk and weight discrimination. Future work is necessary to explore longer-term health outcomes in individuals experiencing weight discrimination across the lifespan.

10. Tables and Figures

Table 1: Demographics

	Weight Discrimination				
	None (N=1068)	Low Stress (N=229)	High Stress (N=66)	Total (N=1363)	P-value
Age					**0.0064[1]**
Mean (SE)	50.4 (0.11)[a]	49.6 (0.24)[b]	50.8 (0.40)[a]	50.2 (0.10)	
Sex, n (%)					**<.0001[2]**
Male	581 (54.4%)	77 (33.6%)	12 (18.2%)	670 (49.2%)	
Female	487 (45.6%)	152 (66.4%)	54 (81.8%)	693 (50.8%)	
Race, n (%)					0.4628[2]
Black	501 (46.9%)	106 (46.3%)	36 (54.5%)	643 (47.2%)	
White	567 (53.1%)	123 (53.7%)	30 (45.5%)	720 (52.8%)	
Smoking Status, n (%)					0.8932[2]
Never	299 (28.3%)	71 (31.4%)	19 (29.2%)	389 (28.8%)	
Former	437 (41.3%)	89 (39.4%)	28 (43.1%)	554 (41.1%)	
Current	322 (30.4%)	66 (29.2%)	18 (27.7%)	406 (30.1%)	
Family History of Diabetes, n (%)					0.1230[2]
Yes	117 (22.9%)	20 (17.9%)	12 (34.3%)	149 (22.6%)	
Family Income, n (%)					**<.0001[2]**
<$5,000-$15,999	137 (13.0%)	42 (18.6%)	23 (35.4%)	202 (15.0%)	
$16,000-$34,999	140 (13.3%)	35 (15.5%)	7 (10.8%)	182 (13.5%)	
$35,000-$74,999	282 (26.8%)	69 (30.5%)	22 (33.8%)	373 (27.7%)	
$75,000+	495 (47.0%)	80 (35.4%)	13 (20.0%)	588 (43.7%)	
Source of Medical Care, n (%)					0.8556[2]
None	101 (9.5%)	19 (8.3%)	8 (12.1%)	128 (9.4%)	
Private or personal physician HMO*	745 (69.9%)	167 (72.9%)	42 (63.6%)	954 (70.1%)	
Other clinic (by appointment)	99 (9.3%)	21 (9.2%)	8 (12.1%)	128 (9.4%)	
Other	121 (11.4%)	22 (9.6%)	8 (12.1%)	151 (11.1%)	
Highest Degree Earned, n (%)					0.6576[2]
High school diploma or equivalency (GED)	435 (40.7%)	95 (41.5%)	27 (40.9%)	557 (40.9%)	
Associate degree (junior college)	148 (13.9%)	32 (14.0%)	8 (12.1%)	188 (13.8%)	
Bachelor's degree	248 (23.2%)	47 (20.5%)	14 (21.2%)	309 (22.7%)	
Master's degree	162 (15.2%)	40 (17.5%)	8 (12.1%)	210 (15.4%)	
No answer	75 (7.0%)	15 (6.6%)	9 (13.6%)	99 (7.3%)	
Alcohol Consumption (mL/Day)					**0.0029[1]**
Mean (SE)	16.1 (0.89)[a]	9.8 (1.13)[b]	10.4 (3.21)[ab]	14.8 (0.74)	

Physical Activity Total Intensity Score (Moderate & Heavy) (EU)					**0.0039[1]**
Mean (SE)	337.7 (8.23)[a]	303.3 (17.13)[ab]	236.9 (29.24)[b]	327.1 (7.23)	
Physical Activity Heavy Intensity Score (EU)					**0.0030[1]**
Mean (SE)	197.2 (6.35)[a]	170.3 (12.46)[ab]	118.8 (19.49)[b]	188.9 (5.50)	
Physical Activity Moderate Intensity Score (EU)					0.1917[1]
Mean (SE)	140.5 (3.31)	133.0 (7.10)	118.1 (14.00)	138.2 (2.94)	
Number of Discrimination Events, n (%)					**<.0001[2]**
Low (0 to 3)	1068 (100.0%)	187 (81.7%)	45 (68.2%)	1300 (95.4%)	
High (4 to 7)	0 (0.0%)	42 (18.3%)	21 (31.8%)	63 (4.6%)	

[1]ANOVA F-test p-value; [2]Chi-Square p-value;

[a,b,ab] Means in a row that are without a common letter differ significantly (P<0.05) as analyzed by Tukey Post Hoc test.

*HMO: Health Maintenance Organization;

EU: Exercise Units.

112,113

Table 2: Clinical Outcomes

	Weight Discrimination				
	None (N=1068)	Low Stress (N=229)	High Stress (N=66)	Total (N=1363)	P-value
% HbA1c					0.0554[1]
Mean (SE)	5.8 (0.03)	5.9 (0.07)	6.0 (0.16)	5.8 (0.03)	
Type 2 Diabetes, n (%)					**0.0021[2]**
Yes	120 (11.2%)	43 (18.8%)	13 (19.7%)	176 (12.9%)	
Self-Report Diabetes, n (%)					0.1649[2]
No	936 (87.6%)	188 (82.1%)	54 (81.8%)	1178 (86.4%)	
Yes	125 (11.7%)	39 (17.0%)	11 (16.7%)	175 (12.8%)	
Not Sure	7 (0.7%)	2 (0.9%)	1 (1.5%)	10 (0.7%)	
Medication for Diabetes, n (%)					0.0534[2]
No	975 (91.3%)	209 (91.3%)	58 (87.9%)	1242 (91.1%)	
Yes	92 (8.6%)	20 (8.7%)	7 (10.6%)	119 (8.7%)	
Not Sure	1 (0.1%)	0 (0.0%)	1 (1.5%)	2 (0.1%)	
Fasting Glucose (mmol/L)*					0.1197[1]
Mean (SE)	5.6 (0.04)	5.8 (0.11)	6.0 (0.29)	5.7 (0.04)	
2HR OGTT Glucose (mmol/L)					0.0710[1]
Mean (SE)	6.3 (0.08)	6.8 (0.23)	6.5 (0.42)	6.4 (0.07)	
Fasting Insulin (uU/ML)					**0.0110[1]**
Mean (SE)	12.5 (0.36)[b]	14.9 (0.63)[a]	13.7 (1.13)[ab]	13.0 (0.30)	
Triglycerides (mmol/L)*					0.7760[1]
Mean (SE)	3.4 (0.08)	3.5 (0.17)	3.3 (0.25)	3.4 (0.07)	
HDL Cholesterol (mmol/L)*					0.0610[1]
Mean (SE)	1.4 (0.01)	1.4 (0.03)	1.5 (0.05)	1.4 (0.01)	
Systolic Blood Pressure*					0.7097[1]
Mean (SE)	120.6 (0.47)	121.2 (1.16)	119.4 (2.03)	120.6 (0.43)	
Diastolic Blood Pressure*					0.1754[1]
Mean (SE)	75.8 (0.32)	77.2 (0.73)	76.4 (1.34)	76.1 (0.29)	
Waist Circumference (cm)*					**<.0001[1]**
Mean (SE)	98.7 (0.36)[b]	105.5 (0.92)[a]	106.0 (1.77)[a]	100.2 (0.34)	
Body Mass Index ($kg \cdot m^{-2}$)					**<.0001[1]**
Mean (SE)	31.2 (0.15)[b]	35.7 (0.44)[a]	37.3 (0.82)[a]	32.3 (0.16)	
Obesity BMI, n (%)					**<.0001[2]**
Yes	538 (50.4%)	180 (78.6%)	56 (84.8%)	774 (56.8%)	
Abdominal Obesity, n (%)					**<.0001[2]**
Yes	591 (55.4%)	187 (81.7%)	59 (89.4%)	837 (61.5%)	
Body Mass Index ($kg \cdot m^{-2}$)					**<.0001[2]**
Overweight	530 (49.6%)	49 (21.4%)	10 (15.2%)	589 (43.2%)	

Obesity I	330 (30.9%)	77 (33.6%)	16 (24.2%)	423 (31.0%)	
Obesity II	130 (12.2%)	38 (16.6%)	17 (25.8%)	185 (13.6%)	
Obesity III	78 (7.3%)	65 (28.4%)	23 (34.8%)	166 (12.2%)	
Metabolic Syndrome, n (%)					**0.0008**[2]
Yes	353 (33.1%)	100 (43.7%)	32 (48.5%)	485 (35.6%)	
Metabolic Syndrome Components, n (%)					**0.0002**[2]
0	187 (17.5%)	18 (7.9%)	4 (6.1%)	209 (15.3%)	
1	265 (24.8%)	43 (18.8%)	17 (25.8%)	325 (23.8%)	
2	263 (24.6%)	68 (29.7%)	13 (19.7%)	344 (25.2%)	
3	213 (19.9%)	52 (22.7%)	18 (27.3%)	283 (20.8%)	
4	94 (8.8%)	35 (15.3%)	10 (15.2%)	139 (10.2%)	
5	46 (4.3%)	13 (5.7%)	4 (6.1%)	63 (4.6%)	

[1]ANOVA F-test p-value; [2]Chi-Square p-value;

[a,b,ab] Means in a row that are without a common letter differ significantly (P<0.05) as analyzed by Tukey Post-Hoc test;

*Represent the main components of metabolic syndrome [112,113]

Table 3: Odds ratios of metabolic syndrome, type 2 diabetes, and abdominal obesity according to weight discrimination level

	Model 1		Model 2		Model 3	
	Events/ Total	Odds Ratio (95% CI)	Events/ Total	Odds Ratio (95% CI)	Events/ Total	Odds Ratio (95% CI)
Metabolic Syndrome	485/1363		485/1363		478/1343	
Weight Discrimination						
None	353/1068	Reference	353/1068	Reference	349/1054	Reference
Low Stress	100/229	**1.57 (1.17-2.10)**	100/229	**1.71 (1.27-2.30)**	98/224	**1.65 (1.20-2.28)**
High Stress	32/66	**1.91 (1.16-3.14)**	32/66	**2.02 (1.21-3.36)**	31/65	1.69 (0.97-2.94)
Type 2 Diabetes	176/1363		176/1363		173/1343	
Weight Discrimination						
None	120/1068	Reference	120/1068	Reference	118/1054	Reference
Low Stress	43/229	**1.83 (1.25-2.68)**	43/229	**1.96 (1.32-2.90)**	43/224	**1.85 (1.21-2.83)**
High Stress	13/66	**1.94 (1.03-3.66)**	13/66	1.87 (0.97-3.61)	12/65	1.42 (0.69-2.93)
Abdominal Obesity	837/1363		837/1363		821/1343	
Weight Discrimination						
None	591/1068	Reference	591/1068	Reference	580/1054	Reference
Low Stress	187/229	**3.59 (2.52-5.13)**	187/229	**3.25 (2.25-4.69)**	183/224	**3.48 (2.32-5.22)**
High Stress	59/66	**6.80 (3.08-5.03)**	59/66	**4.85 (2.16-10.85)**	58/65	**4.86 (2.05-11.52)**

Model 1: Unadjusted.

Model 2: Adjusted for Age and Sex and Race.

Model 3: Adjusted for Age, Sex, Race, Number of Discrimination Events, Family Income, Alcohol Consumption, and Total Physical activity score.

Odds ratios with covariates are shown in Odds ratios of metabolic syndrome, type 2 diabetes, and abdominal obesity according to weight discrimination level with covariatesSupplementary Table 1.

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Figure 1: Odds of metabolic syndrome according to weight discrimination level

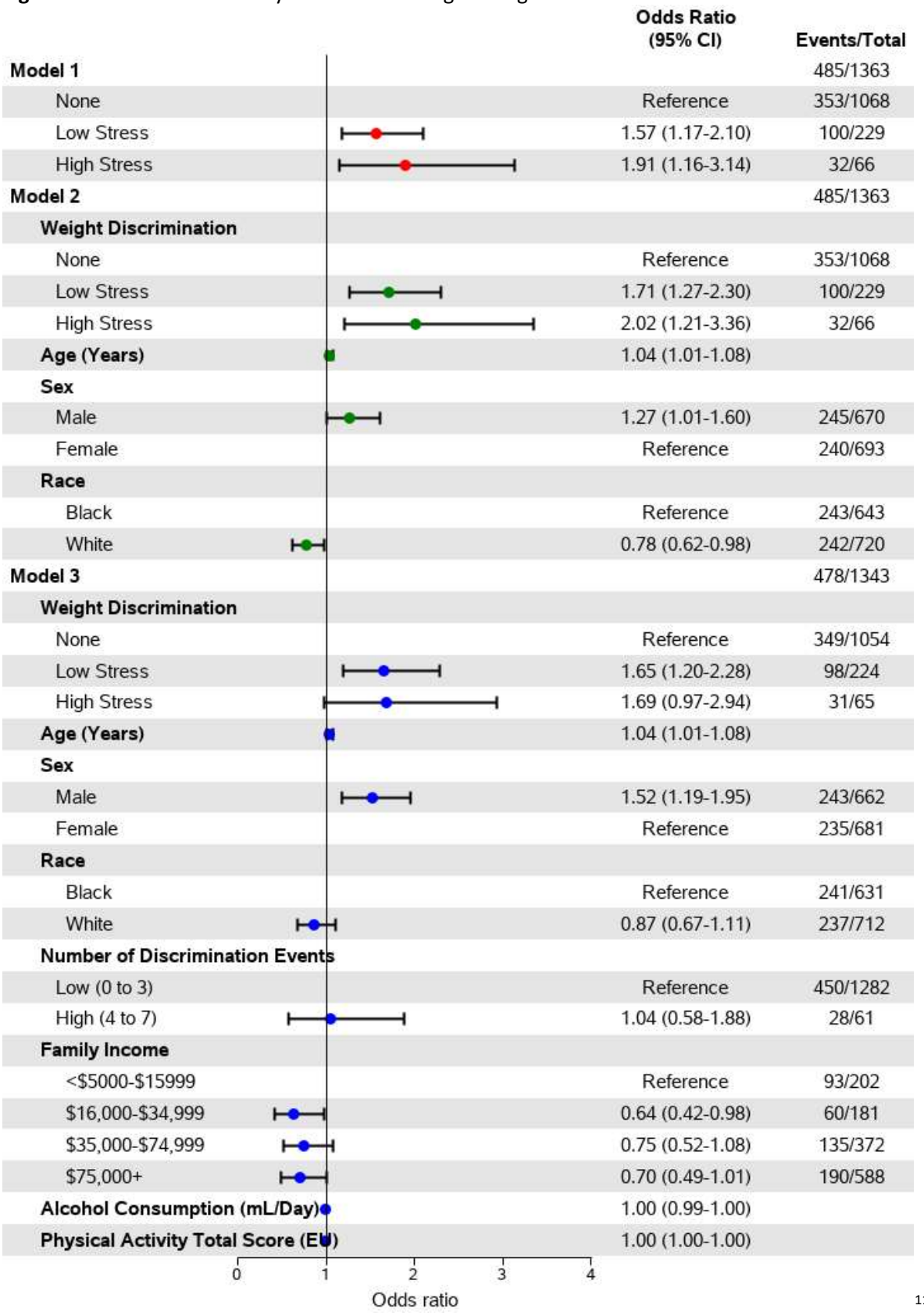

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Figure 2: Odds of type 2 diabetes according to weight discrimination level

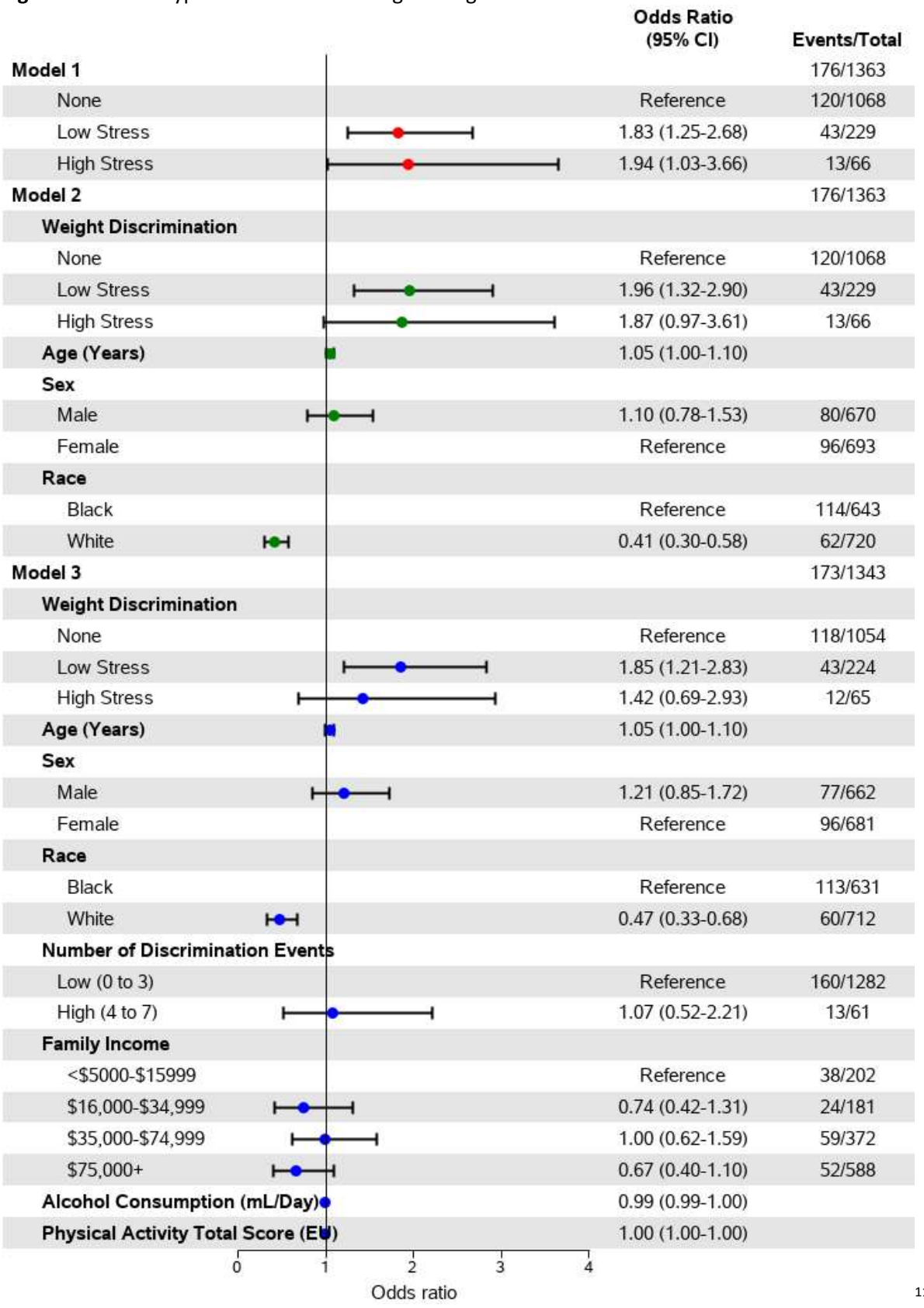

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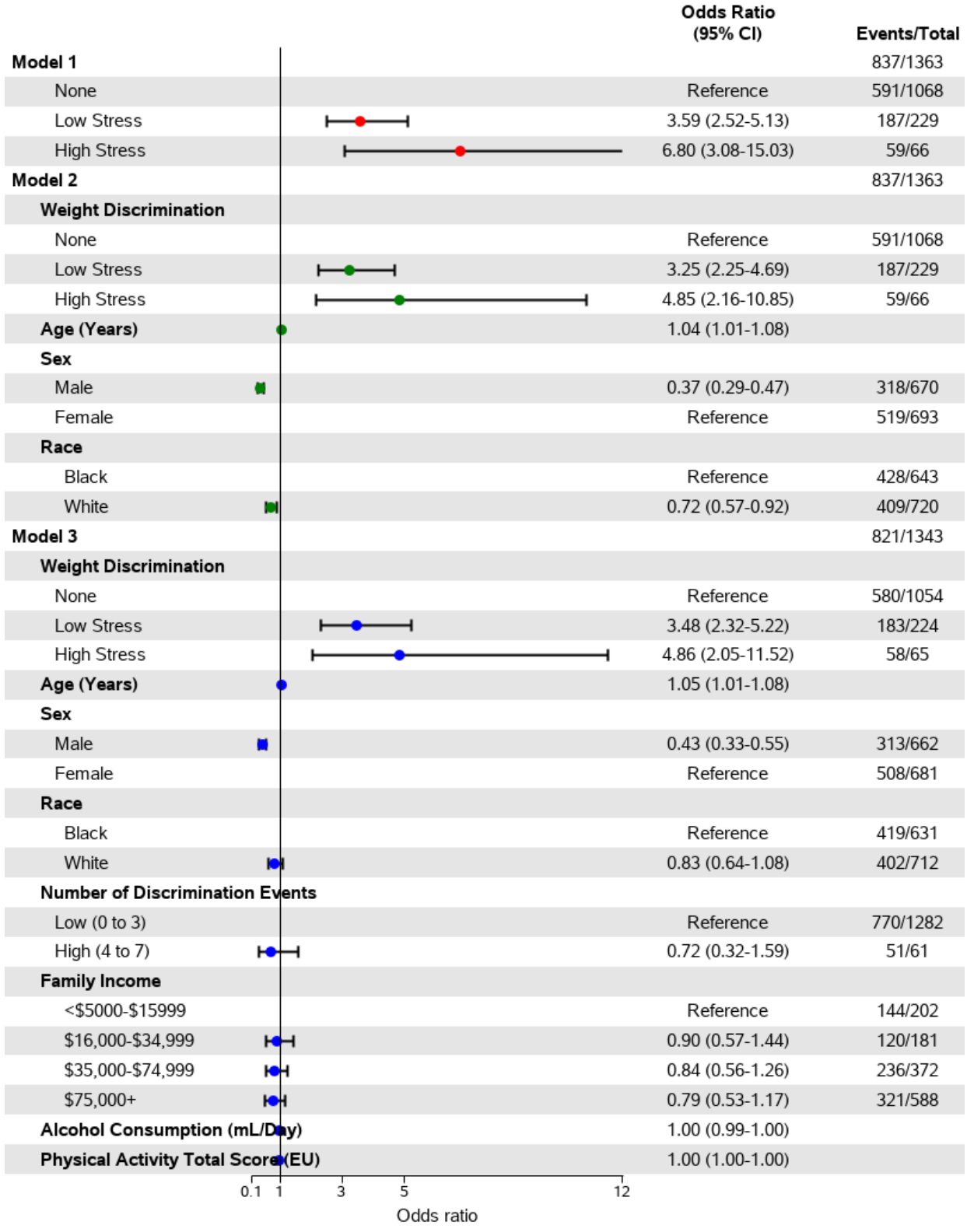
Figure 3: Odds of abdominal obesity according to weight discrimination level



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11. Extended Discussion

11.1 Operationalization of the exposure

As with any secondary analysis, there were a few study and survey constraints that limited the scope of the analyses. One of the persistent drawbacks of a cross-sectional study design is the issue of reverse causality. The CARDIA study did not specifically ask when weight discrimination occurred making it difficult to determine whether exposure to weight discrimination preceded the development of MetS, T2DM, and abdominal obesity. One method to overcome this limitation was to narrow the analysis to individuals who only reported home or school weight discrimination which would have occurred early in life and likely preceded development of MetS, abdominal obesity, and T2DM. However, this analysis was abandoned because the number of individuals who reported solely of home and school weight discrimination was low, resulting in insufficient statistical power. The current study examined weight discrimination across all events and not the individual sources of weight discrimination. Future studies with larger sample sizes are required to the latter.

At year 25 the CARDIA study began assessing weight discrimination through seven sources of weight discrimination: at school, getting a job, getting housing, at work, at home, getting medical care, and public setting[71]. Only those who responded "Yes" to any of these sources had to report the frequency (rarely, sometimes, or often)[71]. While examining the survey data nearly all the individuals who responded "No" to any of the seven sources of weight discrimination then reported its frequency as "Rarely". This artificially increased the amount of people who reported "rare" frequency as opposed to "sometimes", or "often" [71]. Since each source of discrimination has its own frequency, separating all participants into "rare", "sometimes", and "often" across the seven weight discrimination sources would result in multiple group combinations (approximately 21 groups) that would prevent any analysis due to

limited sample size[71]. Attempts to incorporate the frequency of a single source of weight discrimination into the analysis would have restricted this study to focus on individuals reporting only one source of seven. This would be necessary to avoid the influence of other discrimination sources on the outcomes. As a result, the current sample does not allow for reliable analyses using only one source.

11.2 Stress-Internalization

Weight discrimination or stigma can cause individuals with obesity to internalize the negative stereotypes and attitudes on themselves (i.e. weight bias internalization)[23]. Assessing weight bias internalization may be a better way to show the psychological distress of weight stigma than the event alone[23,115]. This internalization is shown to be higher in those with more weight stigma events[116,117]. The present study adds to the research associating cardiometabolic health and internalization of weight bias by dichotomizing the weight discrimination group into a high and low stress (a proxy measure for internalization). In doing so, the present study was able to compliment existing literature, suggesting, that higher levels of stress (i.e. internalization) may lead to higher levels of cardiometabolic risk[23].

Much of the literature discuss stress as a common theme due to weight discrimination; however, there are very few studies to date that actually measure stress. In contrast, the present study focussed on the stress specifically from weight discrimination and its association on the measured outcomes. In agreement with the present study, others have adjusted for indices of total life event stress and found that perceived weight discrimination was significantly associated with diabetes, arteriosclerosis, and some heart conditions[93]. This same study found that people with perceived weight discrimination report more stressful general life events than those without weight discrimination[93]. This suggests that repeated stressful experiences may lead to difficulties in coping with stress and heighten the sensitivity to any stressor which could translate to worse health outcomes[93]. The cross-sectional nature of this and the present study do not allow for differentiation of whether chronic stress from weight discriminations

increases sensitivity to stress in general, or chronic stress in general increases sensitivity to stress from weight discrimination[93]. Nonetheless, stressors do appear to mediate the observed relationships between weight discrimination and health outcomes. Among the possible mechanisms is that internalization of weight stigma can promote unhealthy eating that offset more positive weight loss efforts[98]. Future longitudinal studies are required to study this relationship further[93,118,119].

11.3 Obesity and Central Obesity

The relationship between generalized obesity and cardiometabolic risk is well established; however, the distribution of body fat is important to consider as well[120,121]. Obesity defined by BMI levels may underestimate "at-risk" individuals with abdominal obesity (centrally obesity) yet still within the normal weight BMI range[120]. Abdominal obesity is an independent risk factor for metabolic disorders and in comparison with BMI defined obesity is a better predictor for cardiovascular risk[95,120]. Furthermore, abdominal obesity is one of components used to diagnose MetS and is associated with T2DM and hypertension independent of BMI defined obesity[120]. Future studies may wish to examine abdominal obesity separate from BMI defined obesity as its risks increase with more abdominal fat.

11.4 Sex

This study shows that of those who are more stressed, women are more likely to report weight discrimination than men **(Table 1)**. This finding supports the notion and is consistent with prior research that shows women are more susceptible than men to weight stigma[26,57], potentially experiencing nearly twice as much weight discrimination as men (~10% vs ~5%)[26]. Women with class I obesity (BMI 30-35) report three times more weight discrimination than men[26]. However, the present study was not sufficiently powered to stratify by sex across obesity. The literature examining sex differences among weight discrimination and chronic disease is growing, but inconsistent. One study examining weight discrimination and all-cause mortality found no sex differences[91], whereas another study found women

with weight discrimination to have higher odds of diabetes, arteriosclerosis, and cardiovascular conditions while men had higher odds of arthritis, minor heart conditions, and hypertenstion[93]. Furthermore, HPA axis activation in response to stress can differ by sex, leading to differential development of diseases [93,122].

11.5 Race/Ethnicity

Racial differences also remain with perceived weight discrimination[23,72]. The CARDIA study maintained a balanced number of black and white individuals; however, this study showed a trend towards more white individuals experiencing low stress weight discrimination and more black individuals experiencing high stress weight discrimination($p<0.05$). Evidence of race-by-sex interactions are also apparent. Of note, a prior analysis of the CARDIA study[72] revealed overweight and obese white women reported more weight discrimination than men (30% vs 12% respectively), whereas 25% of African American women, and 15% of African American men reported weight discrimination. This research supports the present study's findings that more women experience weight discrimination than men. Furthermore, this study suggests that racial differences may play a role in attitudes towards perceptions of weight discrimination that could serve as a protective or detrimental factor on clinical outcomes[72,123].

11.6 Final Thoughts

The present study adds to the growing body of research on weight discrimination and health, suggesting that weight discrimination goes beyond mental health implications and can manifest itself in elevations in cardiometabolic risk. This study compliments existing work showing that internalization of weight discrimination seen in the form of stress can further amplify these risk factors. Given that weight loss is difficult to maintain long term, persons with overweight or obesity are more likely to remain so and will more than likely continue to experience discrimination and stress. Chronic exposure to this stress may make it difficult to cope with other life events, which further accentuate pre-existing cardiometabolic

risk. Currently, clinicians and health care have limited strategies to cope with rising levels of metabolic syndrome, diabetes, and weight gain. The present study helps bring forth the notion that assessing weight discrimination in parallel with current strategies may help identify a subset of patients who would benefit from additional psychological supports.

Supplementary Tables and Figures

Supplementary Table 1: Odds ratios of metabolic syndrome, type 2 diabetes, and abdominal obesity according to weight discrimination level with covariates

	Model 1		Model 2		Model 3	
	Events/ Total	Odds Ratio (95% CI)	Events/ Total	Odds Ratio (95% CI)	Events/ Total	Odds Ratio (95% CI)
Metabolic Syndrome	485/1363		485/1363		478/1343	
Weight Discrimination*						
None	353/1068	Reference	353/1068	Reference	349/1054	Reference
Low Stress	100/229	**1.57 (1.17-2.10)**	100/229	**1.71 (1.27-2.30)**	98/224	**1.65 (1.20-2.28)**
High Stress	32/66	**1.91 (1.16-3.14)**	32/66	**2.02 (1.21-3.36)**	31/65	1.69 (0.97-2.94)
Age at Year 25 Exam				**1.04 (1.01-1.08)**		**1.04 (1.01-1.08)**
Sex						
Male			245/670	**1.27 (1.01-1.60)**	243/662	**1.52 (1.19-1.95)**
Female			240/693	Reference	235/681	Reference
Race						
Black			243/643	Reference	241/631	Reference
White			242/720	0.78 (0.62-0.98)	237/712	0.87 (0.67-1.11)
Number of Discrimination Events						
Low (0 to 3)					450/1282	Reference
High (4 to 7)					28/61	1.04 (0.58-1.88)
Family Income						
<$5000-$15999					93/202	Reference
$16,000-$34,999					60/181	0.64 (0.42-0.98)
$35,000-$74,999					135/372	0.75 (0.52-1.08)
$75,000+					190/588	0.70 (0.49-1.01)
Alcohol Consumption (mL/Day)						1.00 (0.99-1.00)
Physical Activity Total Score**						1.00 (1.00-1.00)
Type 2 Diabetes	176/1363		176/1363		173/1343	
Weight Discrimination*						
None	120/1068	Reference	120/1068	Reference	118/1054	Reference
Low Stress	43/229	**1.83 (1.25-2.68)**	43/229	**1.96 (1.32-2.90)**	43/224	**1.85 (1.21-2.83)**
High Stress	13/66	**1.94 (1.03-3.66)**	13/66	1.87 (0.97-3.61)	12/65	1.42 (0.69-2.93)
Age at Year 25 Exam				1.03 (0.99-1.08)		**1.05 (1.00-1.10)**

Sex						
Male			80/670	1.10 (0.78-1.53)	77/662	1.21 (0.85-1.72)
Female			96/693	Reference	96/681	Reference
Race						
Black			114/643	Reference	113/631	Reference
White			62/720	0.41 (0.30-0.58)	60/712	0.47 (0.33-0.68)
Number of Discrimination Events						
Low (0 to 3)					160/1282	Reference
High (4 to 7)					13/61	1.07 (0.52-2.21)
Family Income						
<$5000-$15999					38/202	Reference
$16,000-$34,999					24/181	0.74 (0.42-1.31)
$35,000-$74,999					59/372	1.00 (0.62-1.59)
$75,000+					52/588	0.67 (0.40-1.10)
Alcohol Consumption (mL/Day)						0.99 (0.98-1.00)
Physical Activity Total Score**						1.00 (1.00-1.00)
Abdominal Obesity*	837/1363		837/1363		821/1343	
Weight Discrimination*						
None	591/1068	Reference	591/1068	Reference	580/1054	Reference
Low Stress	187/229	**3.59 (2.52-5.13)**	187/229	**3.25 (2.25-4.69)**	183/224	**3.48 (2.32-5.22)**
High Stress	59/66	**6.80 (3.08-5.03)**	59/66	**4.85 (2.16-10.85)**	58/65	**4.86 (2.05-11.52)**
Age at Year 25 Exam				**1.04 (1.01-1.08)**		**1.05 (1.01-1.08)**
Sex						
Male			318/670	0.37 (0.29-0.47)	313/662	0.43 (0.33-0.55)
Female			519/693	Reference	508/681	Reference
Race						
Black			428/643	Reference	419/631	Reference
White			409/720	0.72 (0.57-0.92)	402/712	0.83 (0.64-1.08)
Number of Discrimination Events						
Low (0 to 3)					770/1282	Reference
High (4 to 7)					51/61	0.72 (0.32-1.59)
Family Income						

<$5000-$15999			144/202	Reference
$16,000-$34,999			120/181	0.90 (0.57-1.44)
$35,000-$74,999			236/372	0.84 (0.56-1.26)
$75,000+			321/588	0.79 (0.53-1.17)
Alcohol Consumption (mL/Day)				1.00 (0.99-1.00)
Physical Activity Total Score**				1.00 (1.00-1.00)

***Main Effect**

****Odd ratios for Physical activity scores only appear significant due to rounding**

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