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The Role of Psychological Distress, Eating Styles, Dietary Intake, and Gender in Cardiometabolic Risk

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THE ROLE OF PSYCHOLOGICAL DISTRESS, EATING STYLES, DIETARY INTAKE, AND GENDER IN CARDIOMETABOLIC RISK

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Approximately one-third of U.S. adults are at increased risk for life-threatening diseases such as atherosclerosis and type 2 diabetes mellitus. Such individuals are considered healthy without any diagnosed cardiometabolic conditions but may have a constellation of cardiometabolic complications that include obesity, glucose intolerance, hyperinsulinemia, dyslipidemia, hypertension, insulin resistance, and hypertriglyceridemia. When most of these preclinical conditions comorbidly occur, the condition has been referred to as metabolic syndrome (MetS). MetS is considered to reflect one or more early pathophysiological processes in cardiometabolic disease; however, the extent to which these complications and their underlying pathophysiology interact with behavioral factors such as stress, diet, and physical activity have not been clearly established. For example, diet consisting of high total caloric intake and high fat composition is posited to contribute to obesity and other cardiometabolic risk factors, but research is inconsistent regarding the effect of psychological distress (i.e., anxiety, stress, depression, anger) on dietary intake and whether dietary intake mediates a relationship between distress and preclinical cardiometabolic disease risk.

One factor that has been suggested to play a role in the distress – dietary intake relationship is eating style. Research on eating styles has identified four main types that may be related to distress and dietary intake: restrained, disinhibited, emotional, and

external eating. Restrained eaters consciously restrict food intake to control body weight and body shape. Disinhibited eating refers to overeating that occurs following failure of restraint. Emotional eaters consume foods to reduce and alleviate negative emotions, such as anxiety. External eating occurs in response to immediate food-related external stimuli, regardless of internal physiological cues of hunger. Current evidence suggests each of these eating styles moderates the relationship between distress and dietary intake. There is also some research to suggest a relationship between eating styles and weight gain, body mass index (BMI), and development of obesity. However, no study has examined the interrelationships among psychological distress, eating style, and central obesity, and whether these relationships differ according to gender. Moreover, the extent to which distress and eating style may be associated with cardiometabolic risk beyond obesity is unknown. Thus, the main aim of the present study was to test a model of mediation and moderation to evaluate how psychological distress, eating styles, dietary intake, and gender are associated with measures of cardiometabolic risk in healthy individuals (Figure 1). Four hundred sixty-four participants contributed data from two different studies: Obesity, Metabolic Syndrome, and Meal-Related Glycemia (SUGAR) and Markers Assessing Risk for Cardiovascular Health (MARCH). All participants were aged 18-55 years, had no major systemic disease, were not using medications having a cardiovascular, carbohydrate, endocrine, or psychiatric effect, and had no history of substance or alcohol abuse or dependence.

The study employed a structural equation modeling (SEM) approach to assess the following aims: 1) to develop composite, latent factors to reflect psychological distress, eating style, and dietary intake using confirmatory factor analysis (CFA) and to develop a

hybrid model of cardiometabolic risk; and 2) to simultaneously test the interrelationships among factors in a comprehensive model so that the strength of direct and indirect effects can be evaluated while statistically controlling for the other factors and covariates in the model. Latent factor models of psychological distress and eating style fit the data and were statistically acceptable, and a hybrid model of cardiometabolic risk fit the data and its CFA components were acceptable. A latent factor model of dietary intake would have likely fit the data and been statistically acceptable given the high intercorrelations among dietary variables, but no such factor was created because dietary variables failed to confirm the hypothesized associations with other model components (e.g., waist girth, eating styles); thus, these measures were excluded from further SEM analyses.

Final model results showed that psychological distress was positively related to restrained, emotional, and external eating styles, but only restrained eating was directly associated with greater waist girth. Distress was not directly related to cardiometabolic risk, but an indirect effect was found in which higher levels of distress led to greater waist girth via higher levels of restrained eating. Waist girth, in turn, served as a significant mediator between restrained eating and worse insulin sensitivity, higher blood pressure, diminished glucose tolerance, and greater dyslipidemia. These effects were significant when controlling for age, gender, education, and physical activity, and when analyzed in a comprehensive SEM model simultaneously including distress, eating style, and cardiometabolic risk variables. Of note, results suggest the possibility for a reversed effect such that waist girth leads to restrained eating. Findings also suggest that emotional eating may lead to distress. In contrast, the relationship between distress and the other two eating styles, restrained and external eating, appeared unidirectional such

that distress leads to restrained and external eating but not the reverse. Future studies using longitudinal data are needed to better understand these relationships in regards to causality.

Data from the MARCH subsample was excluded from the above final modeling analyses because eating style data were only available for the SUGAR subsample. Thus, the role of gender in how distress, eating styles, and cardiometabolic risk are interrelated could not be examined due to the small number of women in the SUGAR study (n = 38). It remains unknown whether the significant effect of distress on each of the eating styles found in the current study was driven primarily by men, women, or both equally. Similarly, the sample size would not permit the evaluation of whether gender moderated the effect of restrained eating on central obesity. Given that women in the current study reported more restrained, emotional, and external eating than men, future studies with larger samples should follow-up by assessing for potential moderating effects of gender.

The present findings suggest that decreasing restrained eating style may lead to less central fat deposition and hence reduced cardiometabolic risk. Such "non-diet" interventions show potential for improved cardiometabolic health, but more research is needed. Particularly needed are studies examining prevention and intervention outcomes based on type of restrained eating – flexible versus rigid – to better understand how these different subtypes operate and how they can be altered effectively to improve health.

Dedications

To all my family and friends who have given me support, love, and laughter during my graduate school training.

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Chapter I:

Introduction

The prevalence rate of obesity is increasing worldwide but particularly in the United States where it has doubled over the past two decades (Flegal, Carroll, Kuczmarski, & Johnson, 1998; Mokdad et al., 2001; Ogden et al., 2006). The estimated prevalence of obesity in the United States is 32.2%, with an additional 34.1% of Americans falling into the overweight category (Ogden et al., 2006). In other words, Americans are more likely to be overweight or obese than to be normal weight. Obesity has a well-established relationship with the development of several diseases including type 2 diabetes mellitus, cardiovascular disease, stroke, and cancer (National Task Force on the Prevention and Treatment of Obesity, 2000). If an individual fulfills criteria for metabolic syndrome (MetS), a constellation of cardiometabolic conditions including central obesity, glucose intolerance, hyperinsulinemia, dyslipidemia, hypertension , insulin resistance, and hypertriglyceridemia, the likelihood for developing cardiovascular disease and diabetes is further increased (Deen, 2004; Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, 2001). However, the extent to which these cardiometabolic disease risk conditions interact together and with behavioral factors such as stress, diet, and physical activity have not been clearly established. For example, diet consisting of high total caloric intake and high fat composition is posited to contribute to obesity and other cardiometabolic risk factors, but research is inconsistent regarding the effect of psychological distress (i.e., anxiety, stress, depression, anger) on dietary intake and whether dietary intake mediates a relationship between distress and preclinical cardiometabolic disease risk (e.g., Epel, Lapidus, McEwen, & Brownell, 2001; Liu et al.,

2007; Stone & Brownell, 1994; World Health Organization, 2000). One factor that has been suggested to play a role in the distress – dietary intake relationship is eating style, including restrained, disinhibited, emotional, and external eating (e.g., Conner, Fitter, & Fletcher, 1999; Greeno & Wing, 1994; O'Connor, Jones, Conner, McMillan, & Ferguson, 2008; Oliver, Wardle, & Gibson, 2000; Schotte, Cools, & McNally, 1990; Wardle, Steptoe, Oliver, & Lipsey, 2000). A study examining interrelationships among psychological distress, eating style, dietary intake, gender, and cardiometabolic risk factors may indicate that psychological interventions targeting distress and eating style may be beneficial for disease prevention and management.

Background on obesity and MetS is first presented. Next, factors that contribute to the development of obesity and other cardiometabolic risk factors are briefly reviewed. Dietary intake, one factor that contributes to cardiometabolic risk, is then examined in more detail with a focus on the interrelationships among dietary intake, psychological distress, eating styles, gender, obesity, and MetS.

Obesity and Metabolic Syndrome

Obesity

As already discussed, obesity has a well-established relationship with the development of several diseases including type 2 diabetes mellitus, cardiovascular disease, stroke, and cancer (National Task Force on the Prevention and Treatment of Obesity, 2000). Excess fat surrounding the abdominal visceral organs versus subcutaneous fat places an individual at particular risk for adverse health consequences (National Task Force on the Prevention and Treatment of Obesity, 2000; World Health

Organization, 2000). Development of obesity is multifactorial; origins of obesity are reviewed in the section "Factors Contributing to Cardiometabolic Risk."

The current overweight and obesity classifications are defined by body mass index (BMI), a simple measure calculated by weight and height (Deurenberg & Yap, 1999; World Health Organization, 2000). The BMI ranges for overweight and obesity, respectively, are 25.0-29.9 kg/m^2 and $\geq$30 kg/m^2 (World Health Organization, 2000). BMI is a limited measure of obesity because it does not distinguish between different types of body mass (i.e., adiposity vs. fat-free body mass) nor different types of body fat distributions (Deurenberg & Yap, 1999; Kushner & Blatner, 2005). In contrast, waist circumference measurement provides a simple and practical method of identifying individuals at increased risk for obesity-related illness due to abdominal fat distribution (World Health Organization, 2000). Other measures of central visceral fat, such as those derived using computerized tomography (CT), provide even more precise measurements but require additional resources (Deurenberg & Yap, 1999).

Metabolic Syndrome

When abdominal obesity co-occurs with other individual cardiometabolic risk factors, the likelihood for developing cardiovascular disease and diabetes is further increased (Deen, 2004). Specifically, persons may be classified with MetS, which refers to a constellation of conditions that usually includes central obesity; in addition, other comorbid conditions may be present such as glucose intolerance, insulin resistance, dyslipidemia, elevated triglycerides, hypertension, and prothrombotic and proinflammatory states (Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, 2001). The MetS classification systems, described next,

were derived and have evolved to provide clinicians with simple measures by which to identify persons that may be at risk for disease endpoints by virtue of the impact of the risk-factor clustering (Grundy, 2008).

There are several proposed definitions for MetS, but the most widely used is that defined by the National Cholesterol Education Program's Adult Treatment Panel III (NCEP ATP III; Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, 2001). The NCEP ATP III definition requires the presence of three or more of the following: 1) abdominal obesity defined as a waist circumference of >102 cm in men or >88 cm in women; 2) a fasting triglyceride level of ≥150 mg/dL; 3) a high-density lipoprotein cholesterol (HDL-C) level of <40 mg/dL in men or <50 mg/dL in women; 4) blood pressure of ≥130/85 mm Hg; 5) a fasting glucose level of ≥110 mg/dL. The American Heart Association and the National Heart, Lung, and Blood Institute proposed a modified NCEP ATP III definition that uses a fasting glucose cut-off of 100 mg/dL as criteria for MetS (Grundy et al., 2005). Since the 2001 NCEP ATP III definition, the International Diabetes Federation (IDF) Task Force Consensus Group has released a new set of MetS criteria (Alberti, Zimmet, & Shaw, 2006). The IDF definition requires individuals to meet a central obesity criteria that differs according to ethnicity, as well as to meet two additional MetS criteria that use the same cut-offs as the modified NCEP ATP III criteria.

The prevalence of MetS has been analyzed in a sample of 6,436 men and women from the National Health and Nutrition Examination Survey (NHANES) III (1988-1994) and in 1,677 participants from NHANES 1999-2000 (Ford, Giles, & Mokdad, 2004). Using the original NCEP ATP III definition, the age-adjusted MetS prevalence for

NHANES III and NHANES 1999-2000 were 24.1% and 27.0%, respectively. Using the modified NCEP ATP III definition, the age-adjusted MetS prevalence for NHANES III and NHANES 1999-2000 were 29.2% and 32.3%, respectively.

Although the pathogenesis of MetS and each of its components is complex and not fully elucidated, two features appear to stand out as possible causative factors: insulin resistance and central obesity (Alberti et al., 2006). Potential pathophysiological mechanisms have been proposed whereby insulin resistance or compensatory hyperinsulinemia could influence lipidemia and cardiovascular function (Alberti et al., 2006; Mather, Anderson, & Verma, 2001; Reaven & Laws, 1994). Some research also suggests insulin resistance leads to obesity; the compensatory hyperinsulinemia associated with insulin resistance may result in an increase of fat stores because while insulin's effect on glucose metabolism is diminished, the anti-lipolytic effect is still preserved (Kahn & Flier, 2000). Not only does insulin resistance lead to obesity, but research demonstrates that obesity leads to insulin resistance and other MetS risk factors (Alberti et al., 2006). Central adiposity is a source of factors such as free fatty acids, leptin, tumor necrosis factor-α (TNF-α), adiponectin, plasminogen activator inhibitor-1 (PAI-1), lipoprotein lipase, and resistin (Alberti et al., 2006; Wyne, 2005). Dysregulation of these factors can lead to dyslipidemia, elevated triglycerides, a prothrombotic state, insulin resistance and hyperinsulinemia via lipotoxicity in the muscle and liver, and hypertension and initiation of atherosclerotic disease via endothelial activation and damage (Wyne, 2005).

Summary

There are life-threatening health risks, such as atherosclerosis and type 2 diabetes mellitus, associated with obesity, and risk for disease development further increases when persons fulfill criteria for MetS (Deen, 2004; National Task Force on the Prevention and Treatment of Obesity, 2000). Given that approximately one-third of the adults in the U.S. are at an increased risk for disease development because they fulfill MetS criteria and/or are obese, it is crucial to investigate factors that contribute to cardiometabolic risk to identify possible prevention and treatment strategies.

Factors Contributing to Cardiometabolic Risk

Obesity results when energy intake exceeds energy expenditure (Hill & Melanson, 1999). Specifically, in the presence of excess calories in conjunction with an absence of a concomitant increase in energy expenditure, the body will convert and store calories as triglycerides in adipose tissue and, over time, this can lead to obesity (Wilborn et al., 2005; Zimmermann et al., 2004). The determinants of energy intake and expenditure are multifactorial and include genetic and physiological components, as well as modifiable lifestyle factors such as physical activity and diet (de Ferranti & Mozaffarian, 2008; Wilborn et al., 2005). The following sections review the role of genetics, physical inactivity, and diet in obesity and other cardiometabolic risk factors.

Genetics

An individual's genetic makeup does not necessarily cause obesity, but it can lower the threshold for its development (McArdle, Katch, & Katch, 2001). Defects in certain deoxyribonucleic acid (DNA) sequences [e.g., genes for leptin (LEP), leptin receptors (LEPR), proopiomelanocortin (POMC), plasma cell membrane glycoprotein-1 (PC-1),

and melanocortin 4 receptors (MC4R)] appear to be associated with early onset morbid obesity and hyperphagia, and well as hyperinsulinemia and hyperglycemia (Bray, 2008). Twin and family studies estimate the heritability of body mass, percent body fat, and fat patterning to range from 37% to 70% (Bouchard et al., 1996; Carey, Nguyen, Campbell, Chisholm, & Kelly, 1996; Comuzzie et al., 1995; Lyon & Hirschhorn, 2005; Moll, Burns, & Lauer, 1991; Rice, Borecki, Bouchard, & Rao, 1993; Rice et al., 1997). Genetics appear to also play a role in MetS, with the primary genes and pathways related to MetS being the aromatase gene, LEPR gene, POMC, serotoninergic system, and the hypothalamo-pituitary-adrenal (HPA) axis genes corticotrophin-releasing hormone (CRH) and the glucocorticoid receptor (GR) gene (Wilborn et al., 2005).

Although individual variation in obesity and MetS have a genetic component, the rapid rise in obesity and MetS in recent years, in concert with the fact that the human genotype cannot be changed substantially over the span of just a few decades, make it improbable that a population genetic change is responsible for these increases in obesity and MetS (Hill & Melanson, 1999; McArdle et al., 2001; Speakman, 2006). Instead, obesity and MetS likely result from gene-environment interactions (Bray, 2008; Speakman, 2006). Genetic variations may interact with physical inactivity, diet, or both to influence body weight and adiposity (Bray, 2008; Webber, 2003).

Physical Inactivity

Energy expenditure is determined by resting metabolic rate, the thermic effect of food, and physical activity (Hill & Melanson, 1999; Wilborn et al., 2005). Of these three determinants of energy expenditure, studies suggest level of physical activity is the primary contributor to the development of obesity (Castañeda et al., 2005; DiPietro,

1995; Hill & Melanson, 1999). Indeed, studies consistently demonstrate that sedentary lifestyle is a contributory factor in weight gain (McArdle et al., 2001; Rissanen, Heliövaara, Knekt, Reunanen, & Aromaa, 1991). In contrast, regular exercise can alter body composition favorably by enhancing fat mobilization and catabolism (McArdle et al., 2001). Fat mobilization and catabolism involve breaking down triglycerides stored in adipose tissue into glycerol and fatty acids so that it can be released into circulation and used as an energy source by active muscle (McArdle et al., 2001; Zimmermann et al., 2004). In addition to accelerating body fat loss, regular exercise can also retard lean tissue loss and increase fat-free body mass (McArdle et al., 2001). Conserving or increasing fat-free body mass helps to lower adiposity indirectly by maintaining a higher level of resting metabolism, average daily metabolic rate, and possibly fat oxidation during rest (McArdle et al., 2001).

Besides central obesity, physical activity prevents and helps in the treatment of other cardiometabolic risk factors, including improving glucose tolerance, reducing insulin resistance, decreasing plasma triglyceridemia, increasing HDL-C concentrations, and decreasing blood pressure (Esposito, Ciotola, Maiorino, & Giugliano, 2008; Ivy, 1997; Ross et al., 2000). Of note, some of these improvements occurred independent of weight loss (e.g., Ross et al., 2000). Improvements in any of these cardiometabolic factors reduce the overall risk of developing MetS (Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, 2001). Indeed, a study with Finnish men demonstrated that, compared with sedentary men, the risk of developing MetS was 50% lower in those who engaged in moderate or vigorous leisure time physical activity for more than 3 hours per week (Laaksonen et al., 2002).

Diet

When caloric intake exceeds energy expenditure, the result is increased body fat and weight gain (McArdle et al., 2001). Excess calories are converted into triglycerides and stored in adipose tissue (Wilborn et al., 2005). However, not all macronutrients contribute to obesity equally (de Ferranti & Mozaffarian, 2008; Wilborn et al., 2005). Dietary fat appears to be the key macronutrient that undermines the body's weight regulatory system; compared with carbohydrates and protein, fat is more energy-dense, has a lower ability to bring eating to an end and to suppress hunger, has a lower ability to stimulate its own oxidation on intake, and has a higher storage capacity in the body (World Health Organization, 2000). Moreover, fats are often considered to have greater flavor or palatability, which could lead to increased fat consumption (Willett, 2002; World Health Organization, 2000). However, whether dietary fat plays an important role in the rising prevalence of obesity is not clear and remains a topic of debate (Bray & Popkin, 1998; Willett, 2002). Besides fat, increased intake of refined carbohydrates may contribute to the development of obesity (Qi & Cho, 2008). Fruits and vegetables, in contrast, are low-energy-dense foods high in water and fiber and may help sustain satiety while concurrently reducing energy intake (Wilborn et al., 2005). Although some studies found fruit and vegetable intake to be inversely related to BMI and body fatness, findings are inconsistent across studies (Qi & Cho, 2008; Wilborn et al., 2005). Inconsistencies of the dietary intake – obesity relationship across studies appear to at least partially result from varying control of confounding by other factors, such as sociodemographic and lifestyle variables (e.g., physical activity, food availability) (Qi & Cho, 2008; Willett, 2002).

Dietary intake appears to also play a role in other cardiometabolic risk factors. Observational studies found that healthy diets (i.e., diets rich in fruits and vegetables and high in monounsaturated fats) were negatively associated with fasting plasma glucose, triglycerides, and blood pressure, positively correlated with HDL-C, and were associated with a lower likelihood of having MetS (Kokkinos, Panagiotakos, & Polychronopoulos, 2005; Panagiotakos et al., 2004; Williams et al., 2000). In addition, findings from the Framingham Offspring Study suggest consuming cereal fiber and whole grains is inversely associated with insulin resistance and is related with a 38% reduction of MetS prevalence (McKeown et al., 2004). Further, an interventional study with 180 MetS patients demonstrated that, compared with a prudent diet consisting of <30% fat, 50-60% carbohydrate, and 15-20% protein, a Mediterranean-style diet of increased consumption of whole grains, vegetables, fruits, nuts, and olive oil was associated with decreased insulin resistance and a greater reduction of MetS prevalence by approximately 50% two years later (Esposito et al., 2004). Research also suggests diets rich in fruits, vegetables, whole grains, nuts, and omega-3 fatty acids are associated with better endothelial function and reduced levels of inflammatory markers, including TNF-α, C-reactive protein (CRP), and interleukin-6 (IL-6) (de Ferranti & Mozaffarian, 2008; Esposito et al., 2004; Giugliano, Ceriello, & Esposito, 2006).

Factors Contributing to Dietary Intake

Although it is presently unclear how and to what extent certain components of diet and their interactions with other factors (e.g., genes, physical activity, food availability) impact obesity and other cardiometabolic risk factors, dietary intake clearly plays a role as it is the sole source of energy intake of the energy balance equation (McArdle et al.,

2001; Qi & Cho, 2008; Willett, 2002). Thus, a closer examination of the factors that influence food choice and quantity consumed are needed to obtain a more comprehensive understanding of dietary intake. The role of genetics on dietary intake has already been briefly discussed. The following sections will expand upon this and review other factors influencing dietary intake, including physiology, food environment, psychological distress, eating styles, and gender, with an emphasis on the latter three components.

Physiology

Several hormones and corresponding genetic components have been found to have an effect on energy intake (Wilborn et al., 2005). Leptin and ghrelin are hormones involved in appetite regulation; leptin is an adipocyte-derived hormone that suppresses appetite, and ghrelin is predominantly a stomach-derived peptide that stimulates appetite (Cummings & Foster, 2003; Wilborn et al., 2005). Ghrelin, in addition to increasing appetite, promotes weight gain by decreasing metabolic rate, sympathetic nervous system activity, fat catabolism, and body temperature (Cummings & Foster, 2003). However, obesity is associated with reduced plasma ghrelin concentrations and higher levels of circulating leptin (Tschöp et al., 2001; Wilborn et al., 2005). One theory that has been proposed to explain elevated leptin levels in obesity is that genetic mutations in the leptin signaling cascade (e.g., POMC, PC-1, MC4R) result in leptin resistance (Weinsier, Hunter, Heini, Goran, & Sell, 1998; Wilborn et al., 2005). The effect of ghrelin on weight is likely mediated through central antagonism of leptin and other anorectic cytokines, suggesting low ghrelin in obesity may result via high leptin levels (Shintani et al., 2001). The leptin resistance theory, or any other theory, has yet to be confirmed as a physiological mechanism of obesity; this is likely due to the complexity

underlying the relationships among ghrelin, leptin, and other factors involved in appetite regulation such as neuropeptide Y, norepinephrine, and IL-6 (Webber, 2003; Wilborn et al., 2005). In addition, physiological contributors to dietary intake that work via appetite regulation may be overridden by external factors, such as those discussed in the following section (Weinsier et al., 1998).

Food Environment

There has been a growing interest in the role of the environment in influencing individuals' eating behavior (Giskes et al., 2007). Environmental factors that can promote food intake include enticing food advertisements, social eating behaviors, large food diversity, high palatability of food, widespread availability of high-fat and energy-dense foods, and eating outside the home (Webber, 2003; Weinsier et al., 1998). A review of 217 fast-food and sit-down restaurants found that a minority of both types of restaurants offered any main dish that could be considered healthy by current dietary guidelines (Saelens, Glanz, Sallis, & Frank, 2007). In addition, school and worksite food environments can have an adverse impact on dietary intake because multiple meals and snacks can be consumed daily in these environments, and the types of foods available are often high-calorie, low-nutrition foods (Sallis & Glanz, 2009; Story, Kaphingst, & French, 2006). Interventions targeting the food environment in schools and worksites, which require more widespread organizational efforts, are promising and seem to positively affect food intake (Sallis & Glanz, 2009)

Although systematic research is lacking regarding whether energy intake is related to the cost and availability of food, analyses of major trends in food supply by the United States Department of Agriculture (USDA) Economic Research Service suggest a greater

availability of low-cost, energy-dense foods, such as refined grains, added sugars, and fats, which is conducive to increased energy intake (Hill & Melanson, 1999; Putnam, Allshouse, & Kantor, 2002). The availability of low-cost, energy-dense foods may especially impact those in poverty (Basiotis, Kramer-LeBlanc, & Kennedy, 1998; Drewnowski, 2004). Portion size in the United States has increased and may also contribute to overeating; the portion of food served at fast-food restaurants and other restaurants and the size of candy bars and soft drinks have increased over the past few decades (Hill & Melanson, 1999).

Psychological Distress

It is widely accepted that eating behavior can be influenced by changes in emotional states such as anxiety, anger, joy, depression, and sadness (Canetti, Bachar, & Berry, 2002). The impact of psychological distress on dietary intake has been examined with animal studies and both cross-sectional and longitudinal human studies.

Animal studies. A review of the impact of stressors of varying severity on food intake in rats found that severe stress (e.g., immobilization, chronic noise) leads to decreased food intake, whereas milder forms of stress (e.g., tail pinch, handling) have no effect or lead to increased food intake (Torres & Nowson, 2007). Research demonstrates that subsequent weight gain is dependent on not just absolute food intake level but also the nutrient composition of food (World Health Organization, 2000). Results from studies using the rat model to examine the effect of stress on intake of specific foods, such as those high in sucrose, are mixed; some studies reported an increase, some found a decrease, and others noted no change in sweet fluid or sweet food intake in stressed rats (e.g., Baker, Kentner, Konkle, Santa-Maria Barbagallo, Bielajew, 2006; Matthews,

Forbes, & Reid, 1995; Silveira et al., 2000; Torres & Nowson, 2007). The nature and duration of the stressors, as well as the food used (e.g., rat chow vs. cookies) may be responsible for these discrepancies.

Besides capturing stress, chronic exposure to mild, unpredictable stress is at the core of one of the animal models of depression because it can cause all the behavioral symptoms of major depressive disorder, including anhedonia, decreased sexual behavior, decreased locomotor activity, and changes in sleep (Herzog et al., 2009; Willner, 1997; Willner et al., 1998). Female rats exhibiting depressive-like behaviors that were induced from chronic social instability showed reduced food intake and reduced preference for sucrose (Herzog et al., 2009). Similar results were found for male rats, such that those with social defeat-induced depression-like behavioral and biological symptoms exhibited decreased sweet water and food consumption compared with rats that were never attacked or defeated (Becker et al., 2008).

Findings from animal studies must be interpreted with caution because the relationship among stress, depressive-like behaviors, and dietary intake is impacted by research methodological choices such as type of stress and measures of food and dietary intake. Moreover, one must be cautious when using animal studies to draw conclusions about human psychology, especially regarding constructs that involve cognition such as depression (American Psychiatric Association, 2000).

Human studies. A review of the few studies on the effects of laboratory-induced stress on food intake indicates that consumption is not significantly impacted following acute stress (Torres & Nowson, 2007). For example, compared with a control group, men and women who were required to prepare a 4-minute speech consumed similar

amounts of food overall, as well as similar amounts of specific food categories, such as high- and low-fat sweet foods (Oliver et al, 2000). In another study with 59 women, consumption of a range of foods, including high- and low-fat sweet foods, did not differ following the non-stress condition and the stress condition that consisted of 45 minutes of visuospatial puzzles, serial subtractions, and speech preparation and delivery (Epel et al., 2001). Although these laboratory studies included manipulation checks of the stress conditions via measures of blood pressure or mood changes, acute stress captured in the laboratory is conceptually distinct from chronic stress experienced on a daily basis, the latter of which is more likely to negatively impact dietary intake (Cohen, Kessler, & Gordon, 1997; Torres & Nowson, 2007). Thus, perceived stress measured by self-report questionnaires may more accurately reflect the type of stress relevant for food choice and eating behaviors. Indeed, in a survey of 212 undergraduate students, the majority perceived their eating patterns to be influenced by the type of stress experienced in daily life (Oliver & Wardle, 1999). However, the direction of this effect was not uniform, with 42% reporting eating more and 38% eating less. Regardless of the direction of food intake change, the majority of the undergraduates reported that when under stress, they are more likely to eat sweets and chocolate, cake and biscuits, and savory snacks. These findings suggest that stress is perceived in changing food choices towards more energy-dense, snack-type foods with high palatability.

Of the few additional human studies on stress and food composition, results confirm the findings of Oliver and Wardle (1999) that chronic life stress is related to high-fat diets and a greater preference for sweet foods (Liu et al., 2007; Ng & Jeffery, 2003; O'Connor et al., 2008; Torres & Nowson, 2007). A large cross-sectional study of 12,110 adults

found that higher stress scores as measured by the 4-item Perceived Stress Scale (PSS) were associated with self-reports of more frequent consumption of high-fat foods (Ng & Jeffery, 2003). In addition, some research suggests that not only stress but also depressive symptoms are associated with poorer dietary intake. In a cross-sectional study with 2,579 college students living in one of seven Chinese cities, self-reports of perceived stress, depression, and food consumption frequency were obtained and analyzed (Liu et al., 2007). Perceived stress was measured using 3 of 10 items of the PSS, depression was measured using 3 of 20 items of the Center for Epidemiological Studies Depression Scale (CES-D), and food consumption frequency was measured using a food frequency questionnaire focusing on foods consumed during the previous month. Foods high in carbohydrates and/or energy were grouped into five categories: fresh fruits, sweets, ready-to-eat foods (e.g., instant noodles), snack food items, and fast-food. Possible covariates (e.g., gender, city, smoking, alcohol use) were entered into stepwise logistic regression models. In adjusted models, perceived stress was inversely correlated with fresh fruits and positively correlated with ready-to-eat foods and snack food items. Depression was inversely correlated with fresh fruits and positively correlated with ready-to-eat foods and fast-food. Thus, these findings suggest perceived stress and depression are associated with poorer dietary intake.

Stone and Brownell (1994) conducted a longitudinal study to examine stress and eating in the natural environment. Daily records of stress were completed by 158 participants over 84 days, resulting in 16,188 person-days of observation. Because the study was designed to examine stress and coping, the only measure of food intake available was the response to a question on whether eating patterns changed during each

of the 84 days. Similar to the findings previously presented (Oliver & Wardle, 1999), Stone and Brownell found that compared with those reporting low levels of stress, those reporting high levels of stress engaged in both hyperphagia and hypophagia. However, participants reported eating less-than-usual significantly more frequently than eating more-than-usual. Within-subjects analyses indicated stress as a significant predictor of less-than-usual eating but not of more-than-usual eating. Taken together, the investigators concluded that the predominant response to stress was eating less.

Another prospective, naturalistic study on stress and eating was conducted in 422 adults by O'Connor and colleagues (2008). In this study, participants used daily diaries to provide information about food intake and stressors/hassles encountered over 4 weeks. Those who experienced at least one hassle reported consuming significantly more between-meal snacks and less main meals and vegetables on the day of the hassle(s). Results also suggest that type of stress or hassle impacts eating behavior. Findings demonstrated that increased snacking was associated with work-related hassles (e.g., meeting a deadline), whereas decreased snacking was associated with physical stressors (e.g., feeling ill, threat of attack by dog). Decreased eating related to physical stressors was also observed in a retrospective survey of U.S. Marines (Popper, Smits, Meiselman, & Hirsch, 1989). During the first day of combat, 68% of the Marines reported eating less than usual due to lack of time and feeling nervous, tense, and scared.

Researched less frequently, anger is another component of psychological distress that may impact eating (Canetti et al., 2002). For example, Macht (1999) found that in 107 women and 103 men asked to report how eating behavior could change during various emotions, impulsive and sensory eating were rated as occurring more frequently

during anger compared with fear, sadness, and joy. Moreover, a review of psychological characteristics and cardiometabolic risk concluded that anger is associated with MetS, elevated central obesity, and insulin resistance (Goldbacher & Matthews, 2007). Thus, although anger is not often considered along with depression and anxiety, anger should be taken into account as it may impact dietary intake, obesity, and other cardiometabolic risk factors.

Summary of psychological distress and dietary intake. In a review conducted by Torres and Nowson (2007), the authors concluded that chronic stress can lead to increased food intake, which can in turn result in weight gain, abdominal obesity, and increased risk for diseases such as coronary heart disease and type 2 diabetes. Thus, research on distress and dietary intake is pertinent as it may guide the development of effective disease prevention strategies. Unfortunately, the impact of distress on eating is not clear. Some investigators found that distress was associated with increased consumption of food, especially for energy-dense foods (Liu et al., 2007; Ng & Jeffery, 2003; O'Connor et al., 2008; Oliver & Wardle, 1999), others failed to find any differences in food intake between high- and low-stress conditions or groups (Epel et al., 2001; Oliver et al., 2000), and others reported that distress is related to decreased food intake (Stone & Brownell, 1994; O'Connor et al., 2008; Oliver & Wardle, 1999; Popper et al., 1989). Factors that may play a role in determining if and how distress is related to food intake include distress severity and type, eating styles, and gender (Greeno & Wing, 1994; Torres & Nowson, 2007). The potential moderating effects of eating styles and gender on the distress – dietary intake relationship has been an area of particular research interest recently, and the existing literature is reviewed next.

Eating Style

Maladaptive eating styles that may moderate the relationship between distress and dietary intake include restrained, disinhibited, emotional, and external eating (Conner et al., 1999; Greeno & Wing, 1994; Herman & Polivy, 1980; Oliver et al., 2000; Ouwens, van Strien, & van der Staak, 2003). Of note, only stress, anxiety, and depression have been examined in this context; no research has been conducted on the relationship between anger and eating styles.

The eating styles described next are not conceptually independent from one other (Heatherton & Baumeister, 1991; Herman & Polivy, 1980; Macht, 2008). Rather, the various eating styles overlap, and these shared characteristics were of particular research interest in the current study. However, to be consistent with how eating styles has traditionally been described and for clearer understanding, eating styles are presented below in separate sections with their overlapping characteristics noted throughout the sections.

Restrained and Disinhibited Eating

Eating is affected by the balance between the desire for food and the effort to resist that desire (Canetti et al., 2002). The cognitive effort to resist that desire is referred to as restraint (Herman & Polivy, 1980). **Restrained eaters are individuals who report that they consciously restrict food intake in order to control body weight and body shape, and that controlling their eating requires considerable effort** (Greeno & Wing, 1994; Herman & Mack, 1975; Herman & Polivy, 1980). As described by Ouwens and colleagues (2003), restrained eaters include successful and unsuccessful dieters. Successful dieters are characterized by high restraint and low tendency toward

overeating, whereas unsuccessful dieters are characterized by high restraint and high tendency towards overeating (Ouwens et al., 2003). Of note, different questionnaires of restrained eating can capture slightly different constructs; the Restraint Scale (RS) measures unsuccessful dieting, whereas the restraint subscales of the Three Factor Eating Questionnaire (TFEQ), Dutch Eating Behavior Questionnaire (DEBQ), and Eating Inventory (EI) measure both successful and unsuccessful dieting (de Lauzon et al., 2004; Ganley, 1988; Herman & Mack, 1975; van Strien, Frijters, Bergers, & Defares, 1986). The restraint theory as it pertains to obesity, however, specifically refers to unsuccessful dieting in which attempts to regulate food intake lead to episodic overeating (Herman & Polivy, 1980). **Disinhibited eating refers to overeating that occurs following failure of restraint** (de Lauzon et al., 2004; Ganley, 1988). Thus, **restraint and disinhibition are closely associated; self-control of restrained eaters may be temporarily released by disruptive events or "disinhibitors"** (Herman & Polivy, 1980). Therefore, unsuccessful dieting is represented when using a disinhibition scale in combination with the TFEQ, DEBQ, or EI (Ouwens et al., 2003). In contrast, disinhibition is embedded in the Restraint Scale and so no additional scale is needed to measure unsuccessful dieting (Herman & Polivy, 1980; Ouwens et al., 2003). Factors that appear to lead to disinhibited eating in restrained eaters include alcohol, forced preload of food, specific cognitions about food, and psychological distress (e.g., anxiety and depression) (Herman & Polivy, 1980).

Restrained and disinhibited eating, psychological distress, and dietary intake. The relationships among restraint, psychological distress, and dietary intake have been examined in laboratory studies in which stressful conditions are presented to produce

distress in participants. In these studies, restrained and unrestrained eaters are often defined as such by a median split of Restraint Scale scores; thus, the terms "restrained" and "unrestrained" eaters used in this section refer to this definition unless otherwise specified. Using this median split categorization, restrained eaters ate more when exposed to an ego-threat (i.e., failing at an easy task or anticipating having to give a speech in front of an evaluative audience) compared with when exposed to a control condition, whereas unrestrained eaters did not differ in amount consumed between the ego-threat and control conditions (Heatherton, Herman, & Polivy, 1991). Restraint as measured by the DEBQ has also been shown to be associated with greater intake after an ego-threatening Stroop color-naming task than after a control condition (Wallis & Hetherington, 2004). Physical threat via anticipation of a "fairly painful electric shock" seems to have a different effect; compared with a control condition, unrestrained individuals have been found to eat significantly less, whereas restrained eaters consumed non-significantly more (Heatherton et al., 1991; Herman & Polivy, 1975). Although group comparisons were not conducted in these studies, others have assessed group differences. For example, Schotte and colleagues (1990) found that when exposed to a film with emotionally neutral content, unrestrained and restrained eaters did not differ in amount of popcorn consumed. In contrast, when participants watched segments of a frightening film, restrained eaters ate more than unrestrained eaters.

Although the effectiveness of the various laboratory-induced stressors on self-rated changes in anxiety and mood were confirmed with manipulation checks, acute stress captured in the laboratory does not necessarily generalize to chronic stress or psychological distress experienced in the outside world on a daily basis (Cohen et al.,

1997). To address this issue, Weinstein and colleagues (1997) examined self-reported food intake in response to stress experienced in daily life in 49 men and 52 women, as well as factors that may affect the relationship between eating and stress (e.g., restraint). Findings included a significant correlation such that those who reported eating more in response to general or chronic stress had higher scores on the Restraint Scale. Moreover, for women, the disinhibition subscale of the EI correctly classified 84.6% of the women into one of two food intake groups: eating more during general stress versus not eating more. A later study examined not only amount of food consumed but also type of foods eaten (Wardle et al., 2000). In this study, data were collected at four time points over a 6-month period from 58 women and 32 men working at a department store in London. Compared with the low-work-stress time point, the high-work-stress time point was characterized by significantly more hours of work (an average of 15 hours more per week), more interference between work and home, and higher levels of perceived stress as measured by the PSS. Workers were classified into high- and low-restraint groups based on median split of the dietary restraint scale of the DEBQ, and food intake was assessed by a 24-hour recall. The high-restraint group had higher energy intake, higher fat and saturated fat intake, and greater percentage of energy derived from saturated fat in the high- than low-work-stress session, but the low-restraint group did not demonstrate food intake changes as a function of high- and low-work stress sessions. Moreover, among those in the high-restraint group, a greater difference in PSS score between the high- and low-work-stress sessions was associated with a greater energy difference. In other words, restrained eaters did not just eat more overall, they specifically ate more sweet and fatty foods in the high-work-stress time, and food intake was greater among

those who had a larger increase in perceived stress between the low- and high-workload sessions.

Restrained and disinhibited eating, obesity, and other cardiometabolic risk factors. As Wardle and colleagues (2000) discussed, adverse dietary changes associated with prolonged or frequent distress can increase the likelihood of weight gain and increased cardiovascular risk in restrained eaters. Indeed, in a sample of 12 clinically depressed patients grouped into restrained and unrestrained eaters, the restrained eaters reported a weight gain since the onset of their depression whereas the unrestrained eaters reported a weight loss (Polivy & Herman, 1976). Several other studies have examined the relationship between body weight and size and restraint and disinhibited eating (Chaput et al., 2009; Dykes, Brunner, Martikainen, & Wardle, 2004; Hainer et al., 2006; Hays & Roberts, 2008; Lluch, Herbeth, Méjean, & Siest, 2000; van Strien, Herman, & Verheijden, 2009). In a cross-sectional analysis of 1,470 women aged 45-68 years, disinhibition was strongly and positively associated with several measures of body weight and size, including BMI, weight adjusted for height, waist circumference, hip circumference, and waist-hip ratio (Dykes et al., 2004). In the same study, restraint, as measured by the TFEQ, was not directly associated with body weight and size measures, but restraint was related to body size through its interaction with disinhibition; results suggested that higher levels of restraint may help to limit weight gain in the presence of high disinhibition. In another cross-sectional study, data from 1,320 members of 387 families ranging in age from 11 to 65 years revealed that the DEBQ restraint subscale score was positively correlated with BMI and weight of both adults and children (Lluch et al., 2000). A similar positive association between the DEBQ restraint subscale

score and BMI was found in a sample of 1,342 men and women in the Netherlands (van Strien et al., 2009).

Findings of longitudinal studies include a significant association between disinhibition and weight gain over 20 years in 535 women aged 55-65 years, and disinhibition and restraint associated with weight gain and development of obesity over 6 years in 283 men and women (Chaput et al., 2009; Hays & Roberts, 2008). Regarding other cardiometabolic risk factors, Hainer and colleagues (2006) cross-sectionally assessed relationships among restraint and disinhibition (measured by the EI), body adiposity, and prevalence of selected diseases, including hypertension and diabetes, in 3,053 Czech men and women. Restraint was negatively and disinhibition was positively related to BMI and waist circumference. Hypertension, hyperlipidemia, and cardiovascular disease were positively associated with restraint and disinhibition, even after adjusting for BMI and age. In addition, a significant positive association between restraint and diabetes was found.

Summary of restrained and disinhibited eating. Restrained eaters can be successful or unsuccessful in their efforts to restrict food intake (Ouwens et al., 2003). Current evidence suggests restraint with periods of disinhibition, which can be conceptualized as unsuccessful dieting, moderates the relationship between dietary intake and laboratory-induced stressors, as well as intake and stress experienced outside the laboratory (e.g., Schotte et al., 1990; Wardle et al., 2000). In both cross-sectional and longitudinal studies, findings indicate restrained and disinhibited eating are associated with body weight and size (e.g., Chaput et al., 2009; Dykes et al., 2004). Only one study has examined restraint and disinhibition in relation to cardiometabolic risk factors other than

obesity; findings of this study provide initial evidence for an eating styles – MetS relationship (Hainer et al., 2006).

Emotional Eating

Emotional eating theory postulates that negative emotions, such as anxiety, induce eating and in turn reduce and alleviate the negative emotion (Macht, 2008). The psychosomatic theory of obesity proposed by Kaplan and Kaplan (1957) describes two consequences of overeating: 1) an immediate, rewarding anxiety-reducing effect; and 2) a negative, long-term effect of increased adipose tissue. In emotional eating, the immediate reduction of negative emotion is a stronger reinforcer for eating than the negative long-term effects are a deterrent for eating, resulting in a learned association between eating and improved emotionality that leads to compulsive eating in response to negative emotions (Kaplan & Kaplan, 1957). The mechanisms underlying improved emotion following eating are not clear, but potential mechanisms being investigated include reduced activity of the HPA axis, increased serotonin in the brain, and eating as a distraction from distress (Macht, 2008).

As discussed in the previous section of restrained and disinhibited eating, psychological distress or negative emotion can lead to disinhibited eating in restrained eaters (Herman & Polivy, 1980). Emotional eating refers to eating during periods of distress or negative emotion, so in this manner, **emotional eating can overlap with restraint and disinhibition** (Macht, 2008). Indeed, a significant relationship between emotional and restrained eating has been reported (e.g., van Strien et al., 1986). In addition, a factor analysis of the EI subscales (i.e., dietary restraint, perceived hunger,

and disinhibition) revealed that the disinhibition factor split into two new factors, weight lability and emotional eating (Ganley, 1988).

Emotional eating, psychological distress, and dietary intake. Relationships among eating styles, stress, and self-reported changes in food intake in response to stress were examined in 89 undergraduate women (Wallis & Hetherington, 2009). In this study, participants completed the DEBQ and a perceived stress and eating questionnaire. The latter questionnaire is a self-report measure requiring the women to indicate any changes in eating in response to stress in general and to a recent, specific stressful experience, as well as the amount of three snack foods (i.e., crisps, chocolate, and biscuits) eaten around the time of the specific stressful experience. Based on a median split of DEBQ emotional eating scores, participants classified as emotional eaters versus non-emotional eaters reported they were more likely to overeat under general and specific stress conditions. Moreover, compared with non-emotional eaters, emotional eaters tended to report eating more of the snack foods under stress. No differences in stress-related eating were found between restrained and unrestrained eaters, possibly because restraint as measured by the DEBQ captures both successful and unsuccessful restraint. These findings are limited in that they rely on self-reported, perceived stress-induced food intake changes rather than a more precise measurement. Thus, a second component of the study was conducted to assess eating styles and food intake following a laboratory stress. In this part, 26 undergraduate women were exposed to two conditions, a Stroop task with ego-threatening words (e.g., worthless) and with neutral words (e.g., wavering). After each task, the women were left alone and allowed to eat ad libitum two types of snacks, a high-fat sweet choice (i.e., chocolate) and a low-fat sweet choice (i.e., dried fruit). A

significant interaction between condition and restraint group emerged such that the unrestrained women consumed slightly more after the ego-threat than after the neutral task, whereas the high restraint women ate less after the ego-threat than after the neutral task. These interaction effects appear to be driven by the low-fat, dried fruit intake more than the high-fat, chocolate intake. Hence, these findings demonstrated that the restrained eaters exhibited successful suppression of intake after ego-threat, with dried fruit possibly serving as a diet reminder. No significant interaction effects between condition and emotional eating status were found for snack intake. Thus, the main findings of the two study components differ. The survey provided evidence for a link between emotional eating and poor dietary intake following stress, whereas the results of the experimental study suggested a stronger association among restraint, acute stress, and inhibited food intake. These differences likely resulted from the varied experimental methods, limited sample characteristics (i.e., small sample size, restricted age range, inclusion of undergraduate women only), and assessment of conceptually different types of stress (i.e., acute laboratory-induced stress versus general or specific stressors experienced outside the laboratory).

At least two other studies have investigated the associations among emotional eating, stress, and eating using experimental methods in which acute stressors are presented in the laboratory setting with measurement of subsequent food intake (Oliver et al., 2000; Wallis & Hetherington, 2004). A study by Wallis and Hetherington (2004) was conducted prior to the one described above (Wallis & Hetherington, 2009). In the 2004 study, chocolate intake was assessed in 38 females following a neutral, ego-threatening, and incongruent Stroop color-naming task; the incongruent word/color condition was

included to assess intake in response to a cognitively demanding stressor. Perceived stress ratings revealed that both the ego-threat and incongruent conditions were significantly more stressful than the neutral, control condition. The women completed the DEBQ, and based on median split of restraint and emotional eating scores, four groups were created – high restraint/high emotional (HR/HE), high restraint/low emotional (HR/LE), low restraint/high emotional (LR/HE), and low restraint/low emotional (LR/LE). The LR/LE group ate similar amounts of chocolate across conditions, whereas the HR/LE group consumed significantly more in the ego-threat and incongruent conditions than in the control condition; group comparisons revealed that the HR/LE group ate more than the LR/LE group in the ego-threat condition. The HR/HE and LR/HE groups also consumed significantly more after the ego-threat than after the control task. These findings demonstrate high restraint associated with greater chocolate intake after both ego-threatening and cognitively demanding stressors than after a control task, and emotional eating associated with greater intake in only the ego-threat compared with the control condition. As previously described in their 2009 study, Wallis and Hetherington reported high restraint associated with diminished intake following stress and no relationships among emotional eating, stress, and food intake; the authors suggest differences between the 2004 and 2009 findings may be explained by varied selection of food versus only one food choice after stress.

Another research group examined whether a laboratory-induced, acute stressor altered food choice in 68 men and women, as well as the effects of emotional eating and restraint on the acute stress – dietary intake relationship (Oliver et al., 2000). The stressed group prepared a 4-minute speech, expecting it to be filmed and assessed after a midday meal,

whereas the control group listened to a passage of neutral text before eating the meal. The meal was a buffet lunch consisting of bland, salty, and sweet foods, with low- and high-fat choices within each of these main food categories, resulting in a total of six categories. No differences in overall intake or intake of the six food categories were found between the stressed and control groups. Emotional and restrained eating were measured by the DEBQ. There were no significant differences in consumption between restrained and unrestrained eaters, nor any significant interaction effects between group and restraint level. In contrast, stressed emotional eaters ate more sweet high-fat foods and a more energy-dense meal than stressed non-emotional eaters; in the unstressed control group, food intake did not differ between emotional and non-emotional eaters.

To examine the associations between eating and mood in a naturalistic setting, Lowe and Fisher (1983) used daily diaries to obtain measures of food intake and mood for 12 consecutive days in 47 undergraduate women. Mood was assessed by a checklist of words referring to positive moods (e.g., "calm or relaxed," "content," "excited") and negative moods (e.g., "angry or frustrated," "worried," "depressed or blue") with an intensity rating for each word. Emotional eating was not measured by an eating styles questionnaire. Instead, emotional eating was operationalized based on number of calories consumed following positive or negative moods. A significant positive correlation was found between intensity of negative moods and meals and snacks eaten. Despite study limitations, which include the lack of a validated eating styles questionnaire and limited sample characteristics, this study provides evidence that increased consumption is related to negative moods in a naturalistic setting.

Another study conducted in a naturalistic setting examined data collected from 192 men and 229 women (mean age = 40 years) who completed daily diaries over 4 weeks (O'Connor et al., 2008). Daily diaries required participants to provide free responses to questions regarding food eaten between meals, fruit and vegetables eaten, perceived change, if any, of amount of main meals eaten, and stressors/hassles experienced with corresponding intensity ratings. Daily hassles were positively associated with between-meal snacks and negatively related to vegetable consumption and perceived main meal intake. Eating styles, assessed by the DEBQ and TFEQ, were tested as potential moderators for the hassles – food intake relationship. Significant moderation effects were found; individuals high on restraint, emotional eating, and disinhibition showed a stronger positive association between daily hassles and snacking, and when eating styles were analyzed simultaneously, only emotional eating remained a significant moderator. These findings suggest that eating styles significantly moderate the daily hassles – snacking relationship, and that emotional eating, in particular, serves as the preeminent moderator.

Emotional eating and obesity. A factor analysis of an 80-item questionnaire on food, nutrition, and eating themes identified seven factors, including emotional eating (Scherwitz & Kesten, 2005). This emotional eating factor included items that measured eating prompted by feeling sad, anxious, depressed, and frustrated, and prompted by craving food. Cross-sectional analyses based on 5,256 participants' data indicated that this emotional eating factor was positively associated with self-reported frequency of overeating and likelihood of being obese (Scherwitz & Kesten, 2005). Using the DEBQ to measure emotional eating in 1,320 adults and children of 387 families from France,

emotional eating was positively correlated with BMI in women, but emotional eating was not associated with BMI in men, boys, or girls (Lluch et al., 2000). However, a positive correlation between the DEBQ emotional eating subscale score and BMI was found in both men and women in a sample of 1,342 individuals from the Netherlands (van Strien et al., 2009).

Only one study has examined emotional eating and weight longitudinally (Hays & Roberts, 2008). In this study, data from 535 women aged 55-65 years were collected over 20 years. Eating behavior was obtained by the EI, which contains a disinhibition scale that was further divided by Hays and Roberts (2008) into habitual disinhibition, situational disinhibition, and emotional disinhibition subscales, the latter referring to "overeating in response to emotional states such as depression." The emotional disinhibition subscale was a significant correlate of later weight gain and BMI. Although this study lacked a more validated measure of emotional eating and was limited by the sample characteristics (i.e., inclusion of only older women), the findings provide evidence that emotional eating may impact later weight gain and development of obesity.

Summary of emotional eating. Emotional eating refers to consuming foods in order to reduce and alleviate negative emotions, such as anxiety (Macht, 2008). Some laboratory studies suggest emotional eating moderates the relationship between acute stress and food intake, whereas others do not find this moderating effect (Oliver et al., 2000; Wallis & Hetherington, 2004; Wallis & Hetherington, 2009). The number of studies on emotional eating and stress experienced outside the laboratory is limited, but there is evidence for a moderating effect of emotional eating on the daily hassles – snacking relationship (O'Connor et al., 2008). There is also little research on emotional

eating and obesity, but the available studies suggest a cross-sectional and longitudinal relationship between emotional eating and obesity and/or BMI (e.g., Hays & Roberts, 2008; Scherwitz & Kesten, 2005). Besides obesity, no cardiometabolic risk factors have been assessed in relation to emotional eating.

External Eating

Externality theory posits **external eating occurs in response to immediate food-related external stimuli, regardless of internal physiological cues of hunger** (Rodin, 1980). In contrast, internal eaters are more responsive to internal cues of hunger and satiety (Schachter, Goldman, & Gordon, 1968). External food cues include sight, smell, taste, and number of food cues (Rodin, 1980; van Strien et al., 1986).

External eating is related to restrained and disinhibited eating in that external food cues can serve as disinhibitors to restraint (Herman & Polivy, 1980). **External eating is also associated with emotional eating**; stress can result in both emotional and external eating (Heatherton & Baumeister, 1991; Macht, 2008). With regards to stress and external eating, stress may reduce internal cues of hunger and increase external food cues (Heatherton & Baumeister, 1991). Consequently, it has been suggested that stress might lead to increased eating in external eaters (Conner et al., 1999).

External eating, psychological distress, and dietary intake. Less research has been conducted on external eating than the other eating styles in terms of psychological distress and dietary intake. However, some studies have been conducted and suggest that external eating moderates the relationship between stress and eating (Conner et al., 1999; O'Connor et al., 2008). Seven days of daily diary entries regarding daily hassles and number of between-meal snacks consumed were obtained from 60 undergraduate

students (Conner et al., 1999). Restrained, emotional, and external eating were assessed by the DEBQ. The relationship between number of hassles and number of snacks was only moderated by external eating; restrained and emotional eating were not significant moderators. The moderating effects of external eating remained significant in a model containing restrained and emotional eating, average severity of hassles, and gender. At low levels of external eating, the hassles – snacking relationship was not significant. At high levels of external eating, the relationship between number of hassles and number of snacks was significant and positive, such that more hassles were associated with more snacking. The authors hypothesized that their failure to find significant moderating effects for the other eating styles may be attributed to limited sample size, the measure of eating behavior used (i.e., number of snacks consumed), and/or some other study characteristic, such as the use of self-report diaries rather than more objective measures. In a later study conducted by the same research group, hassles, eating styles, and food intake was assessed for 4 weeks in a larger and more diverse sample – 193 men and 229 women, ranging in age from 18-65 years (O'Connor et al., 2008). Restrained, emotional, and external eating were again assessed by the DEBQ, and disinhibition was measured by the TFEQ. Significant moderation effects were found for all eating styles; those high on external eating, restraint, emotional eating, and disinhibition showed a stronger positive association between daily hassles and snacking. However, when eating styles were analyzed simultaneously, external eating was no longer significant and only emotional eating remained a significant moderator. These results suggest that although external eating may moderate the relationship between stress and eating, emotional eating

may be more influential than external eating when examined in a larger, more diverse sample over a longer period of time.

Although this line of research suggests external eating may moderate the daily stress – snacking relationship, it is unknown whether this moderating effect would generalize to the relationship between chronic psychological distress and dietary intake beyond snacking.

External eating and obesity. In one of the first studies to prospectively examine externality on later weight gain, Rodin and Slochower (1976) analyzed data from girls aged 9-15 years who attended an 8-week summer camp. "External responsiveness" was quantified as a composite of: 1) amount of food eaten under conditions of high cue salience; 2) immediate recall of objects and words briefly presented on a slide; and 3) difference in affective scores given to positive versus neutral stimuli. The latter two measures assume high external responsiveness is associated with better recall of salient stimuli and greater arousal to affect-inducing cues. Note, however, that only the first measure of the composite (i.e., amount of food eaten under salient food cues) refers specifically to eating whereas the other two measures do not. In normal weight girls (n = 92), a significant positive correlation between external responsiveness and weight change over 8 weeks was found. In a later review article by the same research group, additional data from this study were discussed; of the 12 overweight girls who gained weight, 86% reached their highest weights at week 8 (Rodin, 1980). In contrast, of the normal weight girls who gained weight, 70% reached their highest weights before week 8 and then began to lose weight. As discussed by Rodin (1980), this pattern suggests that high external responders of normal weight may experience short-term weight gain but

long-term regulation of normal weight, whereas overweight external responders may have become overweight after long-term regulatory mechanisms that have failed to inhibit the weight gain.

Since the Rodin and Slochower (1976) study, research on obesity and external eating measured by validated instruments in an adult sample has been conducted (Lluch et al., 2000; van Strien et al., 2009). Cross-sectional analyses of eating styles and body morphology in 760 adult men and women revealed no significant association between the DEBQ external eating subscale score and BMI (Lluch et al., 2000). This finding was replicated in a sample of 1,342 men and women in the Netherlands in which no significant relationship between external eating and BMI was found (van Strien et al., 2009). Other factors, such as dietary restraint, emotional eating, and gender, appear more influential than external eating on weight gain and obesity in these studies (Lluch et al., 2000; van Strien et al., 2009).

A prospective twin cohort study on eating styles and obesity also suggest external eating is not a major factor in obesity (Keski-Rahkonen et al., 2007). Data were obtained from monozygotic and dizygotic twins (2,333 women and 2,060 men) from the FinnTwin16 study, who were 16 at baseline and ranged from ages 22-27 at the later assessment time-point. Eating styles, which included restrictive/overeating, snacking, health-conscious, emotional, and external eating, were assessed by a brief questionnaire developed by the researchers. Individuals were considered external eaters if they responded "usually" or "often" to the item "My eating is triggered by seeing food, food advertisements, etc." Using this categorization of external eating and controlling for participants' baseline BMI, obese women and overweight men were more likely to be

external eaters than normal weight women and men. In multivariate analyses including all eating styles, gender, and baseline BMI, only restricting/overeating and health-conscious eating styles were associated with later BMI. Logistic regression analyses of 39 female and 45 male monozygotic twin pairs discordant for overweight status indicated the risk of restrictive/overeating was greater in the obese or overweight twin than the non-overweight twin. Thus, although external eating appears to play a role in later weight gain and development of overweight or obesity, these study findings suggest restrictive/overeating, which is similar to the restraint and disinhibition eating style constructs, are more influential than external eating. However, a major study limitation is the use of a brief eating styles questionnaire lacking reliability and validity data.

Summary of external eating. External eating occurs in response to immediate food-related external stimuli, regardless of internal physiological cues of hunger (Rodin, 1980). Some research suggests external eating moderates the daily stress – snacking relationship, but it is unknown whether this moderating effect would generalize to the relationship between chronic psychological distress and food intake beyond snacking (Conner et al., 1999; O'Connor et al., 2008). Research on external eating and body weight are lacking, and the few studies available have conflicting results that are at least partially due to differences in methods to measure external eating and varying sample characteristics (Keski-Rahkonen et al., 2007; Lluch et al., 2000; Rodin & Slochower, 1976; van Strien et al., 2009). In general, the research suggests that it is possible external eating is related to obesity, but the impact of the other eating styles on obesity appears stronger (Keski-Rahkonen et al., 2007; Lluch et al., 2000; van Strien et al., 2009). No

study on external eating and cardiometabolic risk factors besides obesity has been reported.

Gender

Studies have found that the associations among distress, eating styles, dietary intake, and obesity sometimes differ by gender. There is an abundance of research in this area, and the following is intended to provide a brief overview by highlighting a subset of the available studies on gender differences.

Gender, stress, and dietary intake. Grunberg and Straub (1992) found a significant interaction between gender and acute, laboratory-induced stress. In this study, participants were shown either a film about industrial accidents (stress condition) or a pleasant travelogue (control condition). Men in the stress versus the control condition had significantly decreased food consumption, whereas women in the stressed versus the control condition had increased food consumption. Outside the laboratory, gender has also been found to moderate the relationship between stress and food intake; the association between daily hassles and snacking has been found to be stronger in women than in men (O'Connor et al., 2008).

Gender and eating styles. Compared with men, women have reported higher DEBQ restraint and emotional eating scores, but external eating scores have been similar between men and women (van Strien et al., 2009). Self-reported food intake in response to stress experienced in daily life was significantly and positively associated with EI disinhibition scores for women but not for men (Weinstein et al., 1997). Further, the DEBQ emotional eating score was positively correlated with BMI in women, but emotional eating was not associated with BMI in men (Lluch et al., 2000).

In sum, the interrelationships among distress, eating styles, dietary intake, and obesity cannot be assumed uniform across men and women. Specifically, the impact of distress and eating styles on food intake and obesity appears to be stronger in women compared with men (e.g., Lluch et al., 2000; O'Connor et al., 2008).

Move Towards a More General "Maladaptive Eating Style" Construct?

Restrained, disinhibited, emotional, and external eating have each been defined by emphasizing the unique characteristics of that particular eating style, but there are clearly conceptual similarities and overlap among all these eating styles (Heatherton & Baumeister, 1991; Herman & Polivy, 1980; Macht, 2008). Restraint and disinhibition are closely associated; by definition, disinhibition requires prior restraint/inhibition (Herman & Polivy, 1980). Emotional and external eating are related to restraint and disinhibition in that negative emotion and external food cues can lead to disinhibited eating in restrained eaters (Herman & Polivy, 1980). External and emotional eating also overlap with one another in that stress can result in both types of eating (Heatherton & Baumeister, 1991; Macht, 2008). Despite these similarities among separate eating styles, no study has examined the validity of a more general maladaptive eating style construct. Such a construct that characterizes the shared variance or overlap among restrained, disinhibited, emotional, and external eating may be a stronger predictor of adverse health consequences. Although there is no research on a maladaptive eating style construct, there are studies on a construct of adaptive eating style, called "intuitive eating," which is described next.

Intuitive eating. Tribole and Resch (1995) developed a reference volume to help chronic dieters "rediscover the pleasures of eating and rebuild body image." In this book,

the authors identify 10 core principles of intuitive eating: 1) reject the diet mentality; 2) honor your hunger; 3) make peace with food; 4) challenge the food police; 5) feel your fullness; 6) discover the satisfaction factor; 7) cope with your emotions without using food; 8) respect your body; 9) exercise – feel the difference; and 10) honor your health – gentle nutrition (Tribole & Resch, 1995). Upon examination of each principle, nearly all target at least one of the maladaptive eating styles reviewed, including restrained (principles 1-4, 6, and 8), disinhibited (principles 2, 3, and 7), emotional (principle 7), and external eating (principle 5) (Herman & Mack, 1975; Herman & Polivy, 1980; Macht, 2008; Rodin, 1980).

In a continued effort to emphasis adaptive versus maladaptive eating style, the Intuitive Eating Scale (IES) was developed and evaluated using data from 1,260 college women (Tylka, 2006). Based on review of the literature, Tylka (2006) identified three key aspects to intuitive eating and created 28 items to reflect these central characteristics for the initial IES item pool. After factor analyses, 7 items were deleted, resulting in a total of 21 items in the final IES. The three key features of intuitive eating reflected by the IES are: 1) unconditional permission to eat when hungry and what food is desired at the moment (9 items); 2) eating for physical rather than emotional reasons (6 items); and 3) reliance on internal hunger and satiety cues to determine when and how much to eat (6 items). Structural equation modeling (SEM) analyses confirmed these three intuitive eating features, and that these three latent factors were moderately related to one another and loaded on a second-order latent factor of intuitive eating. Of note, the three first-order factors are roughly the opposites of restrained, emotional, and external eating. Thus, it is reasonable to suggest a similar model of eating style would be confirmed using

scale and subscale scores obtained from measures such as the Restraint Scale, DEBQ, and TFEQ. Expanding beyond the use of only IES items by using multiple questionnaires would provide further confirmation and confidence of three eating styles that are distinct but related components of a broader eating style construct.

The first- and second-order latent factors of intuitive eating have not been examined in subsequent studies, but research has been conducted using the total score of IES (Avalos & Tylka, 2006; Tylka, 2006). For example, IES scores were negatively related to body dissatisfaction, pressure for thinness, and body mass (Tylka, 2006). More research is needed to determine whether and how eating styles is related to cardiometabolic risk.

Rationale and Specific Aims

Approximately one-third of U.S. adults are at increased risk for life-threatening diseases such as atherosclerosis and type 2 diabetes mellitus because they fulfill MetS criteria and/or are obese (Deen, 2004; Ford et al., 2004; National Task Force on the Prevention and Treatment of Obesity, 2000; Ogden et al., 2006). Therefore, it is crucial to investigate factors that contribute to cardiometabolic risk to identify possible prevention and treatment strategies. Poor dietary intake (e.g., high total caloric intake, high fat composition) is posited to contribute to obesity and other cardiometabolic risk factors, but research is inconsistent regarding the effect of psychological distress (i.e., anxiety, stress, depression, anger) on dietary intake and whether dietary intake mediates a relationship between distress and MetS (e.g., Epel et al., 2001; Liu et al., 2007; Stone & Brownell, 1994; World Health Organization, 2000).

One factor that has been suggested to play a role in the distress – dietary intake relationship is eating style (Greeno & Wing, 1994). Research on eating styles has identified four main types that may be related to distress and dietary intake: restrained, disinhibited, emotional, and external eating. **Restrained** eaters consciously restrict food intake to control body weight and body shape (Herman & Mack, 1975; Herman & Polivy, 1980). Restrained eaters can be successful or unsuccessful in their efforts to restrict food intake (Ouwens et al., 2003). The restraint theory as it pertains to obesity specifically refers to unsuccessful dieting in which attempts to regulate food intake lead to episodic overeating (Herman & Polivy, 1980). **Disinhibited** eating refers to overeating that occurs following failure of restraint (de Lauzon et al., 2004; Ganley, 1988). Consequently, restraint and disinhibition are closely associated; self-control of restrained eaters may be temporarily released by disrupting events or "disinhibitors" (Herman & Polivy, 1980). **Emotional** eating refers to consuming foods to reduce and alleviate negative emotions, such as anxiety (Macht, 2008). **External** eating occurs in response to immediate food-related external stimuli, regardless of internal physiological cues of hunger (Rodin, 1980). Emotional and external eating are related to restraint and disinhibition in that negative emotion and external food cues can lead to disinhibited eating in restrained eaters (Herman & Polivy, 1980). External and emotional eating also overlap with one another in that stress can result in both types of eating (Heatherton & Baumeister, 1991; Macht, 2008). Of note, despite the similarities and overlap among the separate eating styles, no study to date has used multiple eating style measures to examine whether a validated composite latent factor of a more general eating style

construct can be created by the separate eating styles using confirmatory factor analysis (CFA).

As reviewed in the previous sections, current evidence suggests each of the eating styles moderates the relationship between distress and dietary intake (e.g., Conner et al., 1999; O'Connor et al., 2008; Oliver et al., 2000; Schotte et al., 1990; Wardle et al., 2000). The impact of distress and eating styles on dietary intake and obesity appears to be stronger in women compared with men (e.g., Lluch et al., 2000; O'Connor et al., 2008). There is also some research to suggest a relationship between eating styles and weight gain, BMI, and development of obesity (e.g., Chaput et al., 2009; Dykes et al., 2004; Hays & Roberts, 2008; Keski-Rahkonen et al., 2007; Scherwitz & Kesten, 2005). However, no study has comprehensively examined the interrelationships among psychological distress, eating style, dietary intake, and obesity, and whether these relationships differ according to gender. Moreover, the extent to which distress and eating style may be associated with cardiometabolic risk is unknown. A study examining these interrelationships may guide how psychological interventions targeting distress and eating style should be conducted to maximize benefit for disease prevention and management. Thus, **the main aim of the present study was to test a model of mediation and moderation to evaluate how psychological distress, eating styles, dietary intake, and gender are associated with cardiometabolic risk in healthy individuals** (Figure 1).

Structural equation models were used to test specific direct and indirect relationships among psychological distress, eating style, dietary intake, and cardiometabolic risk. The **specific aims** of this project were to use SEM to:

1. Operationalize and validate theory-driven constructs of psychological distress, eating style, dietary intake, and cardiometabolic risk using CFA.
2. Test the hypothesis that dietary intake is associated with cardiometabolic risk.
3. Examine whether distress and eating style each have a direct effect on dietary intake.
4. Assess whether a relationship between distress and dietary intake is moderated by eating style (i.e., test for an interaction effect between eating style and distress on dietary intake).
5. Test a combined, comprehensive model of moderation and mediation to examine whether dietary intake mediates the predicted relationship between distress and cardiometabolic risk, accounting for the predicted moderation effects of eating style on the association between distress and dietary intake (Figure 1).

Potential covariates included age, ethnicity, income, education, smoking status, and self-reported physical activity level. Gender differences were proposed to be examined by running separate models for men and women and comparing models for differences, as well as by conducting multiple group SEM analyses (Muthén, 1989).

Anticipated Results

Based on previous research findings, it was hypothesized that poor dietary intake (e.g., high total caloric intake, high fat composition) would be significantly associated with measures of cardiometabolic risk. Psychological distress (i.e., anxiety, stress,

depression, anger) and eating style were each expected to have direct effects on dietary intake; distress and maladaptive eating style (i.e., high levels of restrained, disinhibited, emotional, and external eating) were hypothesized to be positively associated with poor dietary intake. In a model containing distress, eating style, and dietary intake, it was anticipated that there would be a relationship between distress and dietary intake that would be moderated by eating style, such that individuals with high levels of maladaptive eating style would exhibit a significantly stronger positive association between distress and poor dietary intake compared with those low on the maladaptive eating style factor. The association between distress and dietary intake was also expected to differ according to gender; the distress – poor dietary intake relationship was hypothesized to be stronger in women than in men. In addition, the distress – dietary intake association may differ by an interaction of eating style and gender. For example, distress may be associated with poor dietary intake in women with maladaptive eating style but not in men with maladaptive eating style or in women with adaptive eating style. Lastly, the combined model of mediation and moderation was anticipated to be significant such that poor dietary intake would mediate the relationship between distress and cardiometabolic risk measures, and that maladaptive eating style and gender would moderate the distress – dietary intake relationship as described above.

Chapter II:

Methods

Participants

Four hundred sixty-four participants contributed data from two different studies: Obesity, Metabolic Syndrome, and Meal-Related Glycemia (SUGAR) and Markers Assessing Risk for Cardiovascular Health (MARCH). For both studies, participants were primarily recruited from Miami-Dade and Broward counties of Florida via newspaper advertisement and chain-referral. The objective of the SUGAR study was to investigate meal-related glycemia, vascular functioning, and postprandial metabolic mediators (e.g., insulinemia) in individuals with and without MetS. Thus, persons with high risk for coronary heart disease (CHD) and persons with low CHD risk were recruited. Specifically, the high CHD risk participants consisted of individuals with BMI >25 kg/m^2 and ≥3 features of MetS according to NCEP ATP III and IDF criteria, whereas the low CHD risk group had BMI ≤25 kg/m^2 and <3 MetS features. The high and low risk groups were matched according to age, gender, and ethnicity composition. In addition, all SUGAR participants: 1) were aged 18-55 years; 2) had no nicotine use in the past year, no history of substance or alcohol dependency, no current substance or alcohol dependency or abuse, and negative urine toxicology screen; 3) within 3 months of study entry, were taking no prescribed medication having a cardiovascular, carbohydrate, endocrine, or psychiatric effect; 4) within 3 weeks of study entry, were not taking statin medication for a diagnosed cholesterol condition; 5) had no history of cardiovascular, metabolic (other than MetS), or endocrine disorder; 6) presented no electrocardiogram (ECG) arrhythmias associated with tachycardia, bradycardia [<50 beats per minute

(bpm)], or heart block; 7) for women, were not pregnant and had no intention to become pregnant; 8) were willing to participate in 3 visits that included extensive personal and medical history assessments, toxicity check, blood drawing, echocardiographic assessment, and a 2-day, 3-night stay for 48-hour continuous glucose monitoring; and 9) provided informed consent.

The methods of the MARCH study have been described previously (Goldstein et al., 2001; Klaus et al., 2009). In brief, the MARCH study was designed to assess preclinical cardiovascular disease risk in relation to MetS indices in healthy men and women. Healthy status was based on physical examination by physician, medical history, fasting blood chemistry analysis, and 12-lead ECG. MARCH participants: 1) were aged 18-55 years; 2) had no hypertensive history or diastolic blood pressure (DBP) ≥90 mmHg; 3) had no cardiac arrhythmia; 4) had no history of cardiorespiratory, carbohydrate, or metabolic disorders, or other major systemic disorders (e.g., myocardial infarction, stroke, syncope, chest pain, diabetes mellitus, seizure disorder, asthma, chronic bronchitis, renal disease); 5) within 3 months of study entry, no regular use of medications that have cardiovascular, carbohydrate, neuroendocrine, or psychiatric effects; 6) had no history of substance or alcohol abuse, and negative urine toxicology screen; 7) for women, were not pregnant and were not using birth control pills; 8) were willing to participate in 3 visits that included extensive personal and medical history assessments, toxicity check, blood drawing, echocardiographic assessment, reactivity assessments, pharmacological challenges, and glucose monitoring; and 9) provided informed consent.

Procedures

The protocol of both studies consisted of a screening session followed by two assessment visits.

Screening Session: SUGAR and MARCH

Screening session procedures for both the SUGAR and MARCH studies included obtaining informed consent, family medical history, and all current and pre-existing personal medical history through self-report and a comprehensive physical examination. All participants underwent casual sphygmomanometric blood pressure, 12-lead ECG, height, weight, and waist and hip girth assessments, and BMI and waist girth-to-hip girth ratio (WHR) were calculated. Urine was tested with a pregnancy and toxicology screen for alcohol, barbiturates, benzodiazepines, cannabinoids, lysergic acid diethylamide (LSD), phencyclidine (PCP), tetrahydrocannabinol (THC), morphine, and amphetamines. Current and past substance and alcohol use were assessed by self-report questionnaires, and in the SUGAR study, substance and alcohol abuse and dependence were further evaluated by the structured clinical diagnostic interview (SCID-1 v2.0; Spitzer, Williams, Gibbons, & First, 1996). Blood samples for comprehensive metabolic panels were collected from all participants; these assays included a complete blood count (CBC) with iron, total iron binding capacity (TIBC), ferritin and chemistries assessing liver function, creatinine and blood urea nitrogen (BUN), erythrocyte sedimentation rate, creatine phosphokinase, thyroid stimulating hormone, serum folate, vitamin B12, rheumatoid factor, and antinuclear antibody testing. SUGAR but not MARCH participants underwent a 60-minute graded exercise stress test to screen for the presence of subclinical CHD risk (Gibbons et al., 1997). In the MARCH study, all study eligible participants were included and screened

for presence of subclinical CHD risk; the study evaluated stressor reactivity in the context of CHD risk.

Assessment Sessions: MARCH

The two MARCH assessment sessions were administered in random order and were held within a 3-week time span. Both sessions included a 24-hour dietary intake recall interview concerning the previous day's food consumption, and one of the two visits included completion of a packet of psychosocial questionnaires.

During **one session**, participants underwent an oral glucose tolerance test (OGTT) followed by a protocol examining cardiovascular responses evoked by pharmacological agents and mental stressors; the latter tests are not described herein. The **other session** included a fasting blood draw followed by a euglycemic hyperinsulinemia clamp. Following the washout procedure from the clamp, a light lunch meal was provided and then after approximately 60 minutes, an echocardiographic evaluation of cardiac structure and function was performed. Procedures and results of the echocardiography examination are not reported herein.

Assessment Sessions: SUGAR

Both SUGAR assessment visits included urine toxicology and pregnancy screens and 24-hour dietary intake recall interviews. The **first assessment session** also included: a fasting blood draw, euglycemic hyperinsulinemia clamp, psychosocial questionnaires, Structured Interview for Sleep Disorders (Schramm et al., 1993), baseline echocardiography examination, carotid intimal-media thickness (IMT) and brachial artery endothelial-dependent and -independent vasodilation assessments, and height, weight, and waist and hip girth measurements. The **second assessment session** was scheduled

within 3 weeks after the first assessment visit and consisted of three in-patient assessment days. Participants arrived the morning of day 1 and were discharged the morning after day 3 assessments, totaling a 3-day, 3-night stay in the research laboratory. The first day of the in-patient stay assessed glycemic and insulinemic response to a standard OGTT; participants also had a CT scan to derive abdominal adiposity and completed a battery of psychosocial questionnaires administered by an interview process. Throughout the two following assessment days, participants were provided meals of low glucose content on one day and high glucose content on the other day. The order in which participants received low and high glucose-content days were counterbalanced across participants. The two assessment days included blood draws, serial brachial artery reactive hyperemia tests, and continuous glucose monitoring.

Of the assessments completed in the SUGAR study, those of interest for the current investigation included the 24-hour dietary intake recall interviews, psychosocial questionnaires, fasting blood draws, euglycemic hyperinsulinemia clamp, OGTT, and height, weight, and waist and hip girth measurements.

Blood Assays

Numerous measures were obtained from blood assays. The current study analyses were limited to the assays and measures described below.

Serum **cholesterol** and **triglycerides** were measured enzymatically by autoanalyzer (Cobas-Mira Plus, Roche Diagnostics, Branchburg, NJ) using procedures previously described (Allain, Poon, Chan, Richmond, & Fu, 1974; McGowan, Artiss, Strandbergh, & Zak, 1983). HDL-C was measured after precipitation of apoB-containing lipoproteins with dextran sulfate (Warnick, Benderson, & Albers, 1982), and low-density

lipoprotein cholesterol (LDL-C) was calculated by the Friedewald method (Friedewald, Levy, & Fredrickson, 1972). Respectively, intra- and inter-assay coefficients of variation (CVs) for cholesterol were <2.5% and <3.5%, and for triglycerides were <3.9% and <1.1%.

Serum **glucose** was measured by an enzymatic glucose oxidase method using a YSI 2300 Stat Plus glucose analyzer (Yellow Springs Instruments, Yellow Springs, OH). Serum **insulin** was determined by RIA insulin-specific kit (Linco Research, St. Charles, MO). The assay sensitivity limit was 2 μU/ml and characterized by ED50 of 26 ± 1 μU/ml. Intra-assay CV was 4-8% and inter-assay CV was 6-11%.

Euglycemic Hyperinsulinemia Clamp

Participants underwent a euglycemic hyperinsulinemia clamp procedure to derive **insulin sensitivity**. Pharmacologic hyperinsulinemia was achieved by infusion of insulin, wherein the dosage was based on body surface area (BSA). The insulin infusate was prepared in 250 ml 0.9% NaCl to which 10 ml of the participant's blood was added (2 ml/50ml infusate) to prevent the absorption of the regular recombinant human insulin (Humulin-R, Eli Lilly, Indianapolis, IN) to glass or plastic surfaces. A 10-minute priming infusion was followed by a constant infusion at 40 $\mu U/m^2/min$ for approximately 150 minutes, using a calibrated IMed Gemini PC-2TX infusion pump (Alaris Medical Systems, San Diego, CA). Glucose infusion was begun 4 minutes after the initiation of insulin infusion and was empirically set at 2.0 mg/kg•min and then increased at 10 minutes to 2.5-3 mg/kg•min. Blood glucose was clamped to maintain euglycemia within 5% of the fasting value by feedback controlled infusion of 20% dextrose. To monitor blood glucose concentration and adjust the dextrose infusion, whole blood

glucose was measured every 5 minutes by an enzymatic glucose oxidase method using an YSI 2300 STAT Plus glucose analyzer (Yellow Springs Instruments, Yellow Spring, OH). During this steady-state phase, the rate of glucose infusion is equal to the rate of total body glucose uptake and therefore was a measure of tissue insulin sensitivity, labeled "M." Specifically, M was defined as the mean exogenous glucose disposal rate in mg/kg•min by calculating the steady-state glucose infusion rate over consecutive 20-minute periods and applying a space correction factor (DeFronzo, Tobin, & Andres, 1979).

Oral Glucose Tolerance Test

Estimates of post-prandial **glucose and insulin metabolic function** were determined using the OGTT standard procedures as per the National Diabetes Data Group (1979). Oral glucose (75 gm, which is equivalent to 300 calories) was administered as a solution in 25-35 gm/dL of flavored water, consumed within 5 minutes. Baseline, pre-prandial blood samples were obtained at -15 and 0 minutes before glucose was consumed. Blood samples were then obtained at 30, 60, 90, and 120 minutes in the MARCH study, and at 15, 30, 60, 90, 120, 150, and 180 minutes in the SUGAR study. Area under the curve (AUC) for glucose and insulin responses were computed (Allison, Paultre, Maggio, Mezzitis, & Pi-Sunyer, 1995).

Anthropometric Measurements

Body mass index. Measured height and weight were used to calculate BMI. BMI was calculated as weight divided by height squared (kg/m^2) (Kushner & Blatner, 2005).

Waist girth-to-hip girth ratio. The circumference of the waist at the level of the umbilicus and the circumference of the hips at the level of the greater trochanters were

measured twice per visit, resulting in a total of six waist and hip measurements. The mean of these six measurements were used to calculate WHR.

Dietary Intake

Dietary nutrients were assessed in the MARCH study with the Nutritionist V nutrition-analysis software (First DataBank, Inc., San Francisco, CA) and in the SUGAR study with the Food Processor SQL nutrition software (ESHA Research, Salem, OR). Using a validated protocol, a trained assessor during a structured interview obtained detailed information of meals and snacks eaten in the last 24 hours. Twenty-four-hour dietary recalls have been shown to be correlated with actual dietary intake, but intake tends to be underestimated using this method (Greger & Etnyre, 1978). The food consumption information was entered into the nutrition software programs to yield an analysis indexing daily total caloric intake, and breakdown of macronutrients composition (i.e., carbohydrate, fat, and protein) in addition to a nutrient analysis. The average of the recall assessments were used in analyses. Unadjusted, raw values for total caloric and macronutrient intake, and intake of macronutrients as a percentage of total caloric intake were analyzed. In addition, because what is considered a nutritionally adequate diet can differ according to individual differences such as gender and age, adjusted dietary intake variables were calculated based on reference intake values defined by the Food and Nutrition Board (FNB) of the National Academy of Sciences (Institute of Medicine, 2005).

The FNB has developed a set of recommendations for average daily nutrient intake, the latest revision published in a series of reports from 1997 to 2005 and collectively referred to as Dietary Reference Intakes (DRIs) (Institute of Medicine, 2005). Scientific

data was used to develop the DRIs, and reference values were defined by either specific criteria for nutrient adequacy or by a specific endpoint of adverse effect. DRIs comprise a set of various types of reference values, including Estimated Energy Requirements (EERs), Recommended Dietary Allowances (RDAs), and Acceptable Macronutrient Distribution Ranges (AMDRs), each of which were used in the current project and described next (Institute of Medicine, 2005). Assessing dietary intake using the methods and reference values described below has been conducted previously and reported by others (e.g., Clark & Fox, 2009; Nthangeni et al., 2002; Sharma et al., 2008).

EER is defined as "the average dietary energy intake that is predicted to maintain energy balance in a healthy adult of a defined age, gender, weight, height, and level of physical activity consistent with good health" (Institute of Medicine, 2005). Energy intakes above the EER would be expected to result in weight gain. The Institute of Medicine (2005) warns that their EER prediction equations are not intended for overweight or obese individuals; instead, total energy expenditure (TEE) values should be used to estimate energy requirements in a population consisting of overweight, obese, and normal weight individuals. Like the EER, TEE accounts for individual difference variables that affect daily energy expenditure, including age, gender, weight, height, and level of physical activity. For the present study, TEE was estimated for participants using the methods outlined by Gerrior and colleagues (2006), which are based on the Institute of Medicine (2005) report. Average daily total caloric intake computed from the two 24-hour dietary recalls was then divided by the TEE, thus yielding a percentage and used in analyses.

The TEE equation uses reported daily physical activity, but the Paffenbarger Physical Activity Index (PPAI), used in the present studies, was created with the intention to assess weekly, not daily, physical activity (refer to "Covariates" for a detailed description of this index). The methods for estimating physical activity differ between the PPAI and the TEE equation (e.g., estimated basal energy expenditure is used in the TEE equation but not the PPAI). Thus, simply dividing the PPAI score by seven would not be an appropriate method to obtain a score of daily physical activity to use in the TEE equation. Instead, specific items of the PPAI were used to derive an estimate of daily physical activity that would be more consistent with the TEE method. For each physical activity participants reported engaging in during the past week, the number of times they participated in the activity in the past week and the amount of time spent in that activity per session were provided. When estimating daily physical activity, the assumption was made that the participant engaged in all activities listed for the week in one day versus more than one day. For example, if a participant reported engaging in three activities in the past week, all three activities were used to estimate daily physical activity. This method results in an accurate estimation if the participant did indeed engage in all three activities in one day, but if the participant did not engage in all three activities in one day, the daily physical activity measure would be overestimated. Note that the detailed physical activity data used to estimate daily activity was collected for both MARCH and SUGAR studies, but only data from the SUGAR subsample was available for analyses. In short, TEE values were only estimated for SUGAR participants, and the values may be overestimated. Because the purpose of TEE estimates is to account for variables (e.g., age, gender) when evaluating dietary intake, a second method was used to

accomplish this same goal. For this second method, a measure of total calories was statistically adjusted for all the variables from which TEE is derived (i.e., age, gender, weight, height, and physical activity). The PPAI score (i.e., total kcals exerted per week) was used in place of the daily physical activity measure used in the TEE calculation. This second method of adjusting dietary intake allows for the use of data from both MARCH and SUGAR studies. Further, it relies on fewer assumptions than the first method and is less susceptible to overestimation.

RDA is defined as "the average daily dietary nutrient intake level sufficient to meet the nutrient requirement of nearly all (97 to 98 percent) healthy individuals," based on an individual's age and gender (Institute of Medicine, 2005). The RDA for carbohydrate intake is based on the average amount of glucose used by the brain, and it is set at 130 g per day for both adult men and women, regardless of age. The RDA for protein intake is based on meta-analysis of nitrogen balance studies, and for both men and women is set at 0.8 g per kg of body weight. Average dietary intakes of carbohydrate and protein computed from the 24-hour recalls were divided by their respective RDA value, and these percentages were used in analyses. Note that RDAs reflect the recommended minimum nutrient intake levels but fail to capture recommended maximum nutrient intake levels.

An RDA is not set for fat because there is insufficient scientific data for RDA determination (Institute of Medicine, 2005). However, an AMDR has been estimated for fat. An AMDR is expressed as a range of percentages of total caloric intake and is defined as "a range of intakes for a particular energy source that is associated with reduced risk of chronic diseases while providing adequate intakes of essential nutrients." The AMDR for total fat is set at 20-35% of total caloric intake. AMDRs are also set for

carbohydrate and protein and are 45-65% and 10-35%, respectively. Participants' total fat, carbohydrate, and protein intake, expressed as a percentage of total caloric intake, were compared with the corresponding AMDR to determine whether intakes were within, less than, or greater than the AMDR.

Psychosocial Questionnaires

Participants completed a packet of psychosocial questionnaires that included, among other scales, instruments to assess eating styles and psychological distress.

Eating Style

Eating style was assessed in the SUGAR participants by three self-report questionnaires, described in detail below.

Dutch Eating Behavior Questionnaire. The initial item pool for the DEBQ consisted of 100 items, derived from three existing questionnaires: the Eating Patterns Questionnaire (EPQ), the Fragenbogen für Latente Adipositas [the Latent Obesity Questionnaire] (FLA), and the Eating Behavior Inventory (EBI) (O'Neil et al., 1979; Pudel, Metzdorff, & Oetting, 1975; van Strien et al., 1986; Wollersheim, 1970). Specifically, development of the DEBQ involved: selecting items from the EPQ, FLA, and EBI about restrained, emotional, and external eating for the DEBQ; administering the preliminary questionnaire to normal weight and overweight subjects; factor analyzing the data; and then revising, deleting, or adding items to achieve stability (van Strien et al., 1986).

The final version of the DEBQ has 33 items from which three scales were derived: restrained eating (10 items; e.g., "Do you try to eat less at mealtimes than you would like to eat?"), emotional eating (13 items; e.g., "Do you have a desire to eat when you are

irritated?"), and external eating (10 items; e.g., "If food smells and looks good, do you eat more than usual?") (Appendix A; van Strien et al., 1986). Response options are: "never" [1], "seldom" [2], "sometimes" [3], "often" [4], and "very often" [5]. Scores for each scale were calculated by dividing the sum of the item responses by the total number of items endorsed. A high scale score reflects a high degree of the eating behavior in question. Each of the scales has been shown to display good factorial validity, high convergent and discriminant validity, and good internal consistency with Cronbach's alpha coefficients ranging from .80 to .98 (e.g., van Strien, 1996; van Strien et al., 1986; van Strien, Herman, Engels, Larsen, & van Leeuwe, 2007; van Strien et al., 2009; van Strien, van de Laar, et al., 2007). Of note, results of factor analyses suggest the DEBQ actually contains four factors: restrained eating, external eating, eating in response to diffuse emotions, and eating in response to clearly labeled emotions (van Strien et al., 1986). Consequently, the developers of the DEBQ assert that three scales for emotional eating can be obtained from the DEBQ: a two-dimensional scale of general emotional eating (13 items), a homogeneous scale of eating in response to diffuse emotions (4 items; e.g., "Do you have a desire to eat when you have nothing to do?"), and a homogeneous scale of eating in response to clearly labeled emotions (9 items; e.g., "Do you have a desire to eat when you are depressed or discouraged?") (van Strien et al., 1986).

Three-Factor Eating Questionnaire – Revised 18-item version. The TFEQ-R18 (Appendix B) was shortened and revised from the original 51-item TFEQ (Karlsson, Persson, Sjöström, & Sullivan, 2000; Stunkard & Messick, 1985). The initial item pool for the original TFEQ consisted of 67 items and were derived from three sources: the

Restraint Scale, the FLA, and 17 newly written items based on clinical experience (Herman & Polivy, 1980; Pudel et al., 1975; Stunkard & Messick, 1985). Development of the TFEQ then involved: administering the 67-item preliminary questionnaire to normal weight and obese individuals recruited to capture the full spectrum of restrained eating; factor analyzing the data; revising the questionnaire based on the resulting factor structure; and then repeating this three-step process (i.e., administering, factor analyzing, and revising items) with the revised questionnaire (Stunkard & Messick, 1985). The final questionnaire includes 51 items and contains three factors: dietary restraint, disinhibition, and hunger.

The TFEQ-R18, used in the current study, was derived from the 51-item TFEQ (Karlsson et al., 2000; Stunkard & Messick, 1985). The TFEQ-R18 was developed based on data of 4,377 middle-aged, obese individuals using a step-by-step procedure, including multi-trait/multi-item and factor analysis, to boost internal consistency and discriminant capacity among the scales (Karlsson et al., 2000). The final 18 items measure three factors: cognitive restraint (6 items; e.g., "I deliberately take small helpings as a means of controlling my weight"), uncontrolled eating (9 items; e.g., "Do you go on eating binges though you are not hungry?"), and emotional eating (3 items; e.g., "When I feel blue, I overeat"). Of note, the original TFEQ contains a disinhibition scale and hunger scale, whereas the TFEQ-R18 groups these scales into the single factor referred to as uncontrolled eating, which reflects extreme appetite as well as external eating cues (Karlsson et al., 2000). Response options are on a 4-point scale, and scale scores were obtained by summing items. Raw scale scores were then transformed to a 0-100 scale [((raw score – lowest possible raw score)/possible raw score range) x 100] (de Lauzon

et al., 2004). Higher scores on the respective scales indicate greater restrained, uncontrolled, or emotional eating. The TFEQ-R18 has been found to be a psychometrically valid instrument with satisfactory results from tests of the internal structure and Cronbach's alpha coefficients above the .70 standard (Karlsson et al., 2000). Multi-trait/multi-item scaling analyses further revealed that the TFEQ-R18 has stronger convergent and discriminant validity compared with the original 51-item TFEQ (Karlsson et al., 2000; Stunkard & Messick, 1985).

Restraint Scale. Herman and Mack (1975) created an eating habits questionnaire containing 38 items related to eating and dieting habits and weight history to examine whether restrained and unrestrained eaters differed in amount of ice cream consumed after a preload of 0, 1, or 2 milkshakes. From this questionnaire, 10 items were selected by the investigators based on face validity before its administration to create an index of restraint. Because the restraint index items were selected only on the basis of face validity, analyses were conducted in respect to reliability; only 5 items demonstrated internal consistency and were thus used to calculate a restraint index score for the study analyses (Herman & Mack, 1975). Based on this initial questionnaire, the investigators have developed and refined the restraint index into what is currently referred to as the Restraint Scale (RS) (Herman & Polivy, 1980).

The revised RS consists of 10 items and assesses unsuccessful restrained eating by capturing dieting efforts and weight fluctuations (Appendix C; Herman & Polivy, 1980). As such, the RS differs from the DEBQ and TFEQ restrained eating scales because the latter instruments measure only the degree of food restriction, which can include both successful and unsuccessful restriction (Scagliusi et al., 2005). RS items related to

dieting behaviors have Likert scale response options. Questions about weight fluctuation are based on specific categories of weight loss or gain. Of note, it has been suggested that the RS may erroneously classify obese individuals as restrained eaters because they may obtain higher scores due to greater fluctuations in body weight without genuine restraint (van Strien et al., 1986).

RS score was calculated by summing the scores for all items, with a 0-35 range of scores such that higher scores reflect more dietary restraint. General practice with this scale is to classify those participants with scores that are 15 or higher as restrained eaters and those with lower scores as unrestrained eaters (Roth, Herman, Polivy, & Pliner, 2001). Study results have established reliability and validity of the RS, with satisfactory test-retest reliability and construct, criterion, and concurrent validity (e.g., Gorman & Allison, 1995; Ruderman, 1986).

Psychological Distress

Self-report questionnaires on psychological distress included the Beck Depression Inventory, the Perceived Stress Scale, the Profile of Mood States, the Cook-Medley Hostility Scale, Spielberger's State-Trait Anxiety Inventory, and Spielberger's State-Trait Anger Expression Inventory.

Beck Depression Inventory. The BDI is a 21-item survey that quantifies the severity of cognitive, affective, behavioral, and somatic symptoms of depression during the past week (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). Response format requires endorsement of one of four statements, rank-ordered by severity of content. Item scores range from 0 for a neutral statement (e.g., "I do not feel sad") to 3 for the most severe statement (e.g., "I am so sad or unhappy that I can't stand it"). Higher BDI scores

indicate more depressive symptomatology. The BDI has adequate psychometric properties, including internal consistency above .84, test-retest reliability above .59, high content validity, and validity in differentiating between depressed and non-depressed individuals (Beck et al., 1961; Richter, Werner, Heerlein, Kraus, & Sauer, 1998).

Perceived Stress Scale. The PSS contains 14 items that assess the degree to which an individual appraises one's life as stressful during the last month (Cohen, Kamarck, & Mermelstein, 1983). Sample items include: "How often have you been upset because of something that happened unexpectedly?" and "How often have you felt nervous and 'stressed'?" Response options are on a 0-4 scale: "never" [0], "almost never" [1], "sometimes" [2], "fairly often" [3], and "very often" [4]. Scores range from 0 to 56 with higher scores indicating higher perceived stress. The PSS demonstrates test-retest reliability, adequate internal validity, and concurrent and predictive ability (Cohen et al., 1983).

Profile of Mood States. The POMS is a 65-item, adjective rating scale that captures six mood/affective states: Tension-Anxiety (e.g., "tense," "panicky"), Depression-Dejection (e.g., "worthless," "blue"), Anger-Hostility (e.g., "furious," "grouchy"), Vigor-Activity (e.g., "energetic," "cheerful"), Fatigue-Inertia (e.g., "worn out," "exhausted"), and Confusion-Bewilderment (e.g., "forgetful," "unable to concentrate") (McNair, Lorr, & Droppleman, 1971). Items are answered according to how the individual has been feeling during the past week using a 0-4 scale: "not at all" [0], "a little" [1], "moderately" [2], "quite a bit" [3], and "extremely" [4]. Scores can be derived for each of the mood states as well as for an overall Total Mood Disturbance rating. The six

scales have been shown to have internal consistencies above .90, test-retest reliabilities of .61-.69, and adequate predictive and construct validity (McNair et al., 1971).

Cook-Medley Hostility Scale. Derived from the Minnesota Multiphasic Personality Inventory to identify teachers who had difficulty getting along with their students, the Cook-Medley Hostility Scale (Ho) is a 50-item, true-false scale that assesses cynical attitudes, misanthropic beliefs, and aggressive responding style (Cook & Medley, 1954). Items include: "I would certainly enjoy beating a crook at his own game," "When someone does me a wrong I feel I should pay him back if I can, just for the principle of the thing," and "I have often met people who were supposed to be expert who were no better than I." True and false responses are converted to a numerical score of 0 or 1 and then summed. Thus, Ho scale scores range from 0 to 50, with a higher score depicting an individual characterized by dislike and distrust of others. Since the development of the Ho, subsequent researchers have defined subscales of the original scale, including paranoid alienation and cynicism based on factor analysis, and cynicism, hostile attributions, hostile affect, aggressive responding, social avoidance, and other based on face validity (Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989; Costa, Zonderman, McCrae, & Williams, 1986). The Ho has established reliability, convergent validity, and discriminant validity (Barefoot et al., 1989).

Spielberger's State-Trait Anxiety Inventory. State anxiety refers to a transitory emotional state that varies in intensity and fluctuates over time whereas trait anxiety is a disposition to experience anxiety as a personality trait with more enduring levels of anxiety (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983). The STAI consists of two 20-item measures, the STAI State and the STAI Trait (Spielberger et al., 1983). The

STAI State assesses how individuals feel "right now, at this given moment" (e.g., "I feel at ease," "I feel upset") using a 4-point intensity scale ranging from "not at all" [1] to "very much so" [4]. The STAI Trait targets how respondents "generally feel" (e.g., "I am a steady person," "I lack self-confidence") using a 4-point frequency scale from "almost never" [1] to "almost always" [4]. Some items are reverse-coded so that higher total scores indicate more anxiety. Scores for both the STAI State and STAI Trait range from 20 to 80. Both measures have demonstrated adequate internal consistency and convergent and discriminant validity with other measures of state and trait anxiety (Barnes, Harp, & Jung, 2002; Spielberger et al., 1983). As would be expected given the nature of the constructs, the STAI Trait has stronger test-retest reliability compared with the STAI State (average r = .88 vs. .70) (Barnes et al., 2002).

Spielberger's State-Trait Anger Expression Inventory. The STAXI was developed to measure components of anger in the context of both normal and abnormal personality and consists of 44 items that are divided into three anger scales: State Anger (10 items), Trait Anger (10 items), and Anger Expression (24 items) (Spielberger, 1988). Participants in the current project completed the Trait Anger and Anger Expression scales only. The STAXI Trait Anger scale measures the disposition to experience anger and can be used to calculate a total Trait Anger score as well as two subscale scores. These two subscales assess: 1) a general propensity to experience anger when provoked by others (Anger Reaction; 4 items; e.g., "It makes me furious when I am criticized in front of others"); and 2) a disposition to experience anger when not provoked by others (Anger Temperament; 4 items; e.g., "I have a fiery temper"). The STAXI Anger Expression scale contains three subscales to assess how often anger is: 1) expressed toward other

persons or environmental objects (Anger-Out; 8 items; e.g., "I argue with others"); 2) experienced but suppressed (Anger-In; 8 items; e.g., "I tend to harbor grudges I don't tell anyone about"); or 3) controlled (Anger-Control; 8 items; e.g., "I am patient with others"). For the Trait Anger and Anger Expression scales, participants are asked "how you generally feel" or "how often you generally react or behave," and response options range from "almost never" [1] to "almost always" [4]. Scores for Anger Trait range from 10 to 40, and subscales of the Anger Expression range from 8 to 32; higher scores indicate higher levels of that facet of anger. The STAXI has demonstrated good internal consistency for all subscales (Cronbach's alpha coefficients = .73-.91) and good validity among a range of clinical and non-clinical groups (Spielberger, 1988; Spielberger, 1996). For example, college students who scored at or above the 75th percentile on the STAXI Trait Anger scale reported significantly more frequent consequences of angry behavior than did students who scored in the lower 25th percentile, demonstrating concurrent and discriminant validity (Deffenbacher et al., 1996).

Covariates

Potential covariates included age, ethnicity, income, education, smoking status, and physical activity level; these measures were obtained by self-report. Regarding physical activity assessment, the Paffenbarger Physical Activity Index (PPAI), a self-report questionnaire, was administered to participants (Paffenbarger, Wing, & Hyde, 1978). Based on coding catalogs of physical activity and energy expenditure described by Ainsworth et al. (1993; 2000), kilocalories (kcals) exerted per week were determined by summing the following: 1) number of flights of stairs climbed multiplied by 2; 2) number of city blocks walked multiplied by 8; and 3) total minutes engaged in other

physical activities during the past week multiplied by either 3.5, 5.25, or 7, depending on level of exertion required by the activities.

Statistical Analyses

This project used SEM to test a model of mediation and moderation to evaluate how psychological distress, eating style, dietary intake, and gender are associated with cardiometabolic risk in healthy individuals using a cross-sectional design. Data preparation and screening procedures required for valid SEM analyses were conducted with the Statistical Package for the Social Sciences (SPSS version 17.0 for Windows) and included thorough inspection of descriptive statistics, confirmation of distributional assumptions (e.g., normality), investigation and elimination of outliers, transformation or exclusion of non-normally distributed variables, assurance of reliable measurement, linear relationships between variables, and determination of missing data patterns (Kline, 1998; Ullman, 2001). Missing data were handled in preliminary correlation analyses using pairwise deletion. Procedures to handle missing data in main analyses are discussed below in Aim 1. SEM analyses were conducted using M*plus* version 5.21 software. Required for both measurement and structural model analyses, acceptable model goodness were indicated by: 1) a non-significant χ^2 test; 2) comparative fit index (CFI) > .95; 3) root mean square error of approximation (RMSEA) < .06; and 4) standardized root mean squared residual (SRMR) < .08 (Hu & Bentler, 1999; MacCallum & Austin, 2000; Ullman, 2001). Potential covariates for analyses included age, ethnicity, income, education, smoking status, and physical activity level. The specific proposed SEM analyses for each aim are described below. [Note that actual SEM analyses diverged from these planned analyses due to unanticipated results. The

modifications in analytic approach are described where appropriate in the Results section.]

Aim 1

To operationalize and validate theory-driven constructs of psychological distress, eating style, dietary intake, and cardiometabolic risk, CFA were used to assess measurement models in which measured variables (i.e., indicators) were tested for the strength and statistical significance of their correlation with the latent (i.e., unmeasured) construct on which they are specified to load. Acceptable loadings of indicators onto a latent factor was defined as standard loading estimates (λ) > .40 that were statistically significant (i.e., $p < .05$). For each CFA, model fit and modification indices were inspected, and measurement models were modified in order to optimize fit if modifications were theoretically defensible.

Indicators of **psychological distress** that were examined were scale and subscale scores of the BDI, PSS, POMS, Ho, STAI, and STAXI. Indicators of **eating style** that were examined were scale and subscale scores of the DEBQ, TFEQ-R18, and RS. Three first-ordered latent factors relating to eating style (i.e., restrained, emotional, and external eating) were analyzed with multiple indicators each. In addition, a model would be tested to examine whether each of these eating styles loaded significantly onto a second-order latent factor of maladaptive eating style. Only SUGAR participants, which represented approximately 27% of the total sample size, completed eating style questionnaires. Consequently, analyses involving eating style measures were conducted using the SUGAR data only. For all other missing data, full information maximum likelihood (FIML) estimation was used to mathematically compute parameter estimates using all

available data, thereby reducing bias in parameters. The use of FIML is supported by statistical theory as well as a number of recent empirical studies (see Enders, 2006 for a review).

Measures of **dietary intake** considered for factor indicators included total caloric intake as well as food composition (i.e., breakdown of fat, carbohydrate, protein, etc.). The **cardiometabolic risk** construct was tested as a hybrid model versus a pure measurement model. Hybrid SEM analyses refer to models that include both measurement and structural components, the latter referring to pathways between variables. Conducting hybrid SEM analyses for cardiometabolic risk allows for testing and replicating specific relationships among cardiometabolic risk factors that our laboratory previously reported (Klaus et al., 2009). As depicted in Figure 2, three observed variables (i.e., waist girth, insulin sensitivity, and glucose tolerance) and two latent variables (i.e., blood pressure and lipids) were included in the proposed cardiometabolic risk model with specified interrelationships, based on findings from Klaus and colleagues.

Aim 2

To test the hypothesis that the dietary intake factor is associated cardiometabolic risk, hybrid SEM analyses were conducted. A direct effect of dietary intake on cardiometabolic risk was tested for significance by examining the ratio of the parameter estimate (regression coefficient) to its standard error.

Aim 3

To examine whether the distress factor and eating style factor each have a direct effect on the dietary intake factor (i.e., distress → dietary intake; eating style → dietary

intake), the ratio of the parameter estimate (regression coefficient) to its standard error were examined for each.

Aim 4

To assess whether a relationship between the psychological distress factor and dietary intake factor is moderated by the eating style factor, hybrid SEM analyses were conducted to test for an interaction effect between eating style and distress on dietary intake.

Aim 5

The final aim was to examine whether dietary intake mediates the predicted relationship between psychological distress and cardiometabolic risk, while accounting for the predicted moderation effects of eating style on the association between distress and dietary intake. To analyze these relationships, elements were added to the hybrid model specified under Aim 4. Specifically, cardiometabolic risk and a path between dietary intake and cardiometabolic risk were added, allowing for mediation analyses. Indirect effects were computed to test mediational mechanisms linking psychological distress, dietary intake, and cardiometabolic risk, taking into account a potential interaction between distress and eating style. A direct effect of eating style on dietary intake, specified under Aim 3, was also tested and included in this model. In addition, indirect effects of eating style on cardiometabolic risk, mediated by dietary intake, were tested for significance. Gender differences would be indicated by discordant parameter estimates from multiple group SEM analyses, or by discordant results from analyses conducted separately for men and women.

Chapter III:

Results

Sample Characteristics

Demographic characteristics of the MARCH subsample (n = 339), SUGAR subsample (n = 125), and combined total sample (n = 464) are presented in Table 1. Results from t-tests and χ^2 analyses revealed that several characteristics differed between study subsamples: compared with the SUGAR subsample, the MARCH subsample consisted of a smaller proportion of Hispanic participants and a higher proportion of White participants, Black participants, and women; MARCH participants were younger and had a higher total family income; and the MARCH subsample consisted of a small proportion of participants who had never smoked and a higher proportion of current smokers. No differences in education and physical activity were found between the MARCH and SUGAR participants. As a combined total sample, mean participant age was 36 years, 56.5% were men, 27.8% were current or former smokers, and 56.7%, 27.2%, 11.6%, and 4.5% identified themselves as Hispanic, Black, White, and other race/ethnicity, respectively. On average, participants had approximately 13 years of education and a total annual family income of $17,600. Mean physical activity score was 1,674 kcal/wk, indicative of a sedentary lifestyle (Paffenbarger, Hyde, Wing, & Hsieh, 1986).

Data Screening

All variables and covariates were assessed for fulfillment of statistical analyses assumptions. For several measures, box-and-whisker plots revealed outliers. However, because these values were plausible, represented a very small proportion of all available

data used in modeling analyses, and influential observation analyses (evaluated by Cook's *D*, dffits, and standardized, studentized, and studentized deleted residuals) indicated these values did not have greater influence relative to all other data, they were retained in the dataset.

Examination of distribution diagnostics revealed significant departures of normality for triglycerides and fasting insulin; these variables were positively skewed. To improve approximation of normality, log-transformations were applied. These transformations resulted in all skewness values less than ±3 and all kurtosis values less than ±10, demonstrating acceptable approximations of normality for all variables, consistent with requirements for SEM. Transformed variables were used in subsequent analyses, but nontransformed means and standard errors for triglycerides and fasting insulin are reported for ease of interpretation.

Descriptive Statistics

Cardiometabolic Risk

Table 2 includes means and standard errors for cardiometabolic risk variables for the two subsamples and combined sample, adjusted for the demographic variables found to differ between subsamples (i.e., age, gender, ethnicity, and smoking status; 15.3% of income data were missing and consequently not included as a covariate in analyses so as to retain cardiometabolic risk data from all participants). Subsample comparisons were conducted by analyses of covariance (ANCOVAs) and multinomial logistic regression. Compared with MARCH participants, SUGAR participants displayed: higher BMI, BSA, weight, waist girth, and WHR; a higher proportion of obese participants; elevated levels of triglycerides, LDL-C, and total cholesterol; higher total cholesterol-to-HDL-C ratio

(TC:HDL-C); lower insulin sensitivity; and higher DBP and SBP. No subsample differences were found for HDL-C, fasting insulin or glucose, or AUC for insulin or glucose during the OGTT ($AUC_{Insulin}$, $AUC_{Glucose}$).

Metabolic Syndrome Criteria

The percentage of participants fulfilling MetS criteria is detailed in Table 3. Subsample comparisons were analyzed by binary logistic and ordinal regressions, and demographic covariates (i.e., age, gender, ethnicity, and smoking status) were included in analyses. Of the combined total sample, 15.8% and 20.8% fulfilled MetS according to the NCEP ATP III and modified NCEP ATP III criteria, respectively. A higher percentage of SUGAR versus MARCH participants fulfilled MetS criteria. Regarding individual MetS criteria, a higher proportion of the SUGAR subsample met the elevated waist girth and elevated BP criteria. No subsample differences were found for percentage of participants meeting the elevated triglycerides, low HDL-C, and elevated fasting glucose criteria. As a combined total sample, 26.5%, 31.7%, 21.1%, 13.8%, 5.2%, and 1.7% fulfilled 0, 1, 2, 3, 4, or 5 of the modified NCEP ATP III MetS criteria, respectively.

Dietary Intake

As can be seen in Table 4, SUGAR participants reported eating more total calories, carbohydrates, protein, total fat, and saturated fat than MARCH participants. However, the proportion of protein and total fat in total caloric intake (i.e., % calories from protein and fat) did not differ between subsamples, and proportion of calories from carbohydrates was higher in the MARCH subsample ($p = .052$). Monounsaturated and polyunsaturated fats did not differ between subsamples.

Regarding the dietary intake variables derived using dietary reference values, compared with MARCH participants: SUGAR participants reported higher carbohydrate and protein intakes relative to their respective RDAs (i.e., total carbohydrate intake/carbohydrate RDA and total protein intake/protein RDA); the SUGAR subsample had a higher proportion of participants who reported carbohydrate intake below the carbohydrate AMDR; and the SUGAR subsample had a lower proportion of participants within the carbohydrate AMDR. No subsample differences were found for proportion of participants with reported carbohydrate, protein, or fat intakes above their respective AMDRs. Data show that reported total caloric intake was lower than predicted TEE. Predicted TEE was likely overestimated as described previously in the Methods section, especially given that the study sample has a larger than normal proportion of persons who are overweight and obese.

Although total calories relative to TEE could not be calculated for the total sample, a measure of total calories statistically adjusted for all the variables TEE is derived from (i.e., age, gender, weight, height, and physical activity) was examined. ANCOVA results indicate that after adjusting for age, gender, weight, height, and physical activity, the subsample differences remained significant such that SUGAR participants reported eating more total calories than MARCH participants.

Eating Style and Psychological Distress

Descriptive statistics for eating style variables for SUGAR participants are presented in Table 5. On average, participants were not restrained eaters as classified by RS scores (RS cut-off for restrained eating = 15; Roth et al., 2001). Unlike the RS, the DEBQ and TFEQ-R18 subscales do not have recommended cut-off scores. However, as can be seen

in Table 5, participants on average reported TFEQ-R18 scores in the lower quarter of the subscale ranges. In addition, mean DEBQ scores were in the lower half of the scale range.

Means and standard deviations for psychological distress measures for MARCH, SUGAR, and the total sample are presented in Table 6. Results from *t*-tests revealed that MARCH versus SUGAR participants exhibited higher levels of psychological distress as indicated by significantly higher scores on all questionnaires. ANCOVA results indicate that after controlling for age, gender, ethnicity, and smoking status, these subsample differences in distress remained significant, with the exception that the BDI somatic subscale became a trend ($p = .087$) and STAI state anxiety was no longer significantly different between subsamples ($p = .11$) (Table 7). BDI subsample and combined sample means suggest that on average, study participants were not depressed (standard BDI cut-off for depression = 10; Beck et al., 1961).

Missing Data

As mentioned previously in the Methods section, eating style data were provided by SUGAR participants only. DEBQ and RS measures were complete with no missing data, and TFEQ measures had 3.2% of data missing. All demographic variables used in analyses as covariates (i.e., age, gender, ethnicity, and smoking status) were complete with no data missing, whereas physical activity had 8.4% of data missing. All other study measures (i.e., cardiometabolic, dietary intake, and psychological distress variables) had 0% to 3.0% of data missing, and those study measures used for SEM analyses had 0% to 2.2% data missing.

Confirmatory Factor Analyses

In the present investigation, latent factor models of psychological distress and eating style fit the data and were statistically acceptable. A hybrid model of cardiometabolic risk fit the data and its CFA components were also acceptable. In contrast, analyses of dietary intake measures failed to confirm the hypothesized associations with other model components; thus, these measures were excluded from further SEM analyses. Detailed descriptions of psychological distress, eating style, cardiometabolic risk, and dietary intake CFAs are presented below.

Psychological Distress

A correlation matrix for the distress measures is presented in Table 8. The majority of bivariate correlations between distress measures were significant. Measures of anxiety, stress, depression, and anger did not consistently correlate more strongly within than between their theoretical constructs; for example, a measure of depressive symptomatology (BDI total score) was more strongly correlated with a measure of anxiety (STAI Trait) than with another measure of depression (POMS Depression-Dejection). Furthermore, the CFAs that modeled distinct distress constructs (e.g., depression, anxiety) did not fit the data; model results and modification indices suggested strong correlations and overlap among the theoretically distinct distress components. Thus, measures of various distress constructs (i.e., depression, perceived stress, anxiety, and anger) were used to model one, more general latent factor. Henceforth, this factor will be referred to as "distress".

CFA results for the final distress factor model are illustrated in Figure 3. The distress latent variable was comprised of 4 indicators: BDI (total score), PSS, POMS Tension-

Anxiety, and POMS Anger-Hostility. These 4 distress measures were chosen as candidate indicators based on results reported in the literature, previous research conducted in our laboratory, their correlations among other model components (Table 9), and their correlations among each other (e.g., Goldbacher & Matthews, 2007; Ng & Jeffery, 2003; Wardle et al., 2000). Bivariate correlations between .40 and .80 are desirable in attempting to formulate latent variables (Kline, 1998; Ullman, 2001); in this study, bivariate correlations among BDI, PSS, POMS Tension-Anxiety, and POMS Depression-Dejection ranged from .39 to .72 (Table 8). Based on model modification indices and theoretical reasoning, a significant correlation (r = .29) between the error variances of POMS Tension-Anxiety and POMS Anger-Hostility was specified in the distress factor model to significantly improve model fit [$\Delta\chi^2$ (1) = 23.7, p < .001]. CFA results indicate that the final distress factor model fit the data [χ^2 (1) = 1.0, p = .32; CFI = 1.000; RMSEA < .001; SRMR = .006)], and all factor loadings (λ) were >.40 and significant (λ's = .59-.73, p's < .001). The total variance (R^2) in BDI, PSS, POMS Tension-Anxiety, and POMS Anger-Hostility explained by the distress model were 40.1%, 47.2%, 53.0%, and 34.5%, respectively.

Eating Style

Correlations among eating style variables are included in Table 9. Correlations among DEBQ restrained eating, TFEQ-R18 cognitive restraint, and RS ranged from .44 to .49, the correlation between DEBQ emotional eating and TFEQ-R18 emotional eating was .52, and the correlation between DEBQ external eating and TFEQ-R18 uncontrolled eating was .40. These correlations were all significant and within the desirable range for creating latent variables (Kline, 1998; Ullman, 2001). For the most

part, correlations were stronger for measures within than between theoretical eating styles. For example, a measure of restrained eating (DEBQ restrained eating) was more strongly correlated with other measures of restrained eating (TFEQ-R18 cognitive restraint and RS; *r*'s = .44-.49) than with measures of emotional or external eating (*r*'s = .09-.31). There were two exceptions to this pattern. First, a measure of external eating (TFEQ-R18 uncontrolled eating) was more strongly correlated with a measure of emotional eating (TFEQ-R18 emotional eating; *r* = .58) than with the other measure of external eating (DEBQ external eating; *r* = .40). Second, DEBQ external eating was more strongly correlated with DEBQ emotional eating (*r* = .42) than with the other measure of external eating (TFEQ-R18 uncontrolled eating; *r* = .40). These exceptions likely result from similar response patterns within questionnaire type (e.g., DEBQ vs. TFEQ-R18). Of note, most correlations between measures of different eating styles (e.g., a measure of restrained eating and a measure of emotional eating) were moderately to highly significant. This finding suggests the presence of an underlying, unified construct reflecting maladaptive eating style.

A CFA was first conducted to examine three separate eating style constructs: restrained eating, emotional eating, and external eating. As depicted in Figure 4, indicators of **restrained eating** were DEBQ restrained eating, TFEQ-R18 cognitive restraint, and RS; indicators of **emotional eating** were DEBQ emotional eating and TFEQ-R18 emotional eating; and indicators of **external eating** were DEBQ external eating and TFEQ-R18 uncontrolled eating. To obtain acceptable model fit, four significant correlations between indicator error variances were specified in the model based on modification indices and theoretical reasoning (Figure 4). These modifications

resulted in a significant improvement in model fit [$\Delta\chi^2$ (4) = 50.6, $p < .0001$], and the model fit indices for the eating style factor model presented in Figure 4 indicate excellent fit of the data [χ^2 (7) = 7.6, $p = .37$; CFI = .997; RMSEA = .027; SRMR = .035)]. All λ's were >.40 and significant (λ's = .57-.93, p's < .001). Correlations among the latent variables were: restrained eating with emotional eating, $r = .47$, $p < .001$; restrained eating with external eating, $r = .37$, $p < .05$; and emotional eating with external eating, $r = .59$, $p < .001$. The total variance in DEBQ restrained eating, TFEQ-R18 cognitive restraint, RS, DEBQ emotional eating, TFEQ-R18 emotional eating, DEBQ external eating, and TFEQ-R18 uncontrolled eating explained by the eating style model illustrated in Figure 4 were 44.0%, 35.9%, 56.4%, 87.1%, 32.6%, 60.4%, and 33.5%, respectively.

Building on the model from Figure 4, a second CFA of eating style was conducted to test whether the restrained, emotional, and external eating latent variables would load onto a second-order maladaptive eating style factor. As shown in Figure 5, CFA results confirmed that each of the first-order latent variables loaded significantly onto a second-order maladaptive eating style factor (λ's = .55-.87, p's < .05). Model fit indices were identical to those from the model in Figure 4. Other equivalent results between the models with and without the second-order maladaptive eating style factor were: λ's for the measured variables loading onto the first-order eating style variables; correlations between measured variable error variances; and the total variance in the measured variables explained by the eating style model. The total variance in the restrained, emotional, and external eating latent variables explained by the model with the second-order eating style factor were 29.7%, 75.5%, and 45.4%, respectively.

Cardiometabolic Risk

A correlation matrix for the cardiometabolic risk measures is presented in Table 10. Modeling for cardiometabolic risk was guided by previous results from our laboratory using data from the MARCH project (Klaus et al., 2009). The proposed cardiometabolic risk model depicted in Figure 2 was modified from the original Klaus et al. model; some variables (e.g., cardiac mass and inflammation measures) were not used in the present investigation and were consequently omitted from the current model.

As illustrated in Figure 6, the cardiometabolic risk model includes three observed, measured variables (i.e., waist girth, insulin sensitivity, and glucose tolerance), two latent variables (blood pressure and lipids), and hypothesized relationships among variables. Note that the sign of $AUC_{Glucose}$ values were reversed so that a positive value would reflect greater glucose tolerance. The model was adjusted for age, gender, ethnicity, smoking status, education, and physical activity. This model had a χ^2 with 27 degrees of freedom of 53.6, $p = .002$. Although the χ^2 is significant, the χ^2 fit index is very sensitive to sample size (Kline, 1998). Specifically, as sample size increases, the χ^2 statistic magnifies even small discrepancies. Less sample-sensitive indices for the cardiometabolic risk model suggest good model fit (CFI = .981; RMSEA = .046; SRMR = .017).

Blood pressure indicators include DBP and SBP, and lipids indicators include triglycerides and TC:HDL-C; all λ's were >.40 and significant (λ's = .79-.89, p's < .001). All proposed direct effects were significant (p's < .01), except for the direct effect of glucose tolerance on blood pressure (β = -.07, n.s.). Some indirect effects were also significant. Waist girth had a significant total effect (i.e., direct + indirect effects) on

lipids (β = .40, $p < .001$), comprised of: a significant direct effect (waist → lipids; β = .21, $p < .001$), a significant indirect effect via insulin sensitivity (waist → insulin sensitivity → lipids; β = .15, $p < .001$), and a significant indirect effect via insulin sensitivity and glucose tolerance (waist → insulin sensitivity → glucose tolerance → lipids; β = .04, $p < .01$). Insulin sensitivity had a significant total effect on lipids (β = -.32, $p < .001$), comprised of: a significant direct effect (insulin sensitivity → lipids; β = -.25, $p < .001$), and a significant indirect effect via glucose tolerance (insulin sensitivity → glucose tolerance → lipids; β = -.07, $p < .01$). The effect of waist girth on blood pressure was fully accounted for by a direct effect (waist → blood pressure; β = .30, $p < .001$); an indirect effect via insulin sensitivity and glucose tolerance was not significant (waist → insulin sensitivity → glucose tolerance → blood pressure; β = .02, n.s.).

Dietary Intake

Nearly all intercorrelations among dietary intake variables were significant (Table 11). Dietary intake variables were also significantly related to cardiometabolic risk variables, including measures of central obesity, insulin sensitivity, lipidemia, blood pressure, and glucose tolerance (Table 11); however, most *r*'s among dietary intake and cardiometabolic risk variables were .23 or less, indicating a small effect size (Cohen, 1992). To examine whether relationships between dietary intake and cardiometabolic risk remained significant after adequate control for confounding variables, partial correlations were conducted using age, gender, ethnicity, smoking status, education, and physical activity as covariates. Waist girth was used as the cardiometabolic risk variable in partial correlation analyses because waist girth was hypothesized to be the primary

entry point from which diet is related to other cardiometabolic risk variables. Results from these analyses indicated that higher waist girth was significantly associated with lower %calories from carbohydrates, lower total protein/RDA, higher total fat, higher %calories from total fat, and higher saturated fat (*r*'s = -.12, -.30, .09, .12, and .10, respectively; *p*'s < .05); all other dietary intake variables were not significantly related to waist girth.[1]

Because one of the main aims of the current project was to examine dietary intake as it relates to eating style, the above analyses were repeated with the SUGAR subsample only, as eating style was not assessed for MARCH participants. Similar to the results of the combined sample, most intercorrelations among dietary intake variables were significant for SUGAR participants (Table 12). In contrast to results of the combined sample, the SUGAR subsample exhibited fewer significant correlations between dietary intake and cardiometabolic variables (Table 12). Closer examination of these correlations suggests that some are no longer significant due to diminished power to detect small effects, indicated by a similar magnitude of *r*'s between the combined sample and SUGAR subsample. For other correlations that were no longer significant for the SUGAR subsample, it appears actual differences between the SUGAR and MARCH subsamples correlations exists, indicated by different magnitudes of *r*'s. In the SUGAR subsample, waist girth was only significantly correlated with monounsaturated fat. Results from partial correlation analyses controlling for age, gender, ethnicity, smoking status, education, and physical activity indicated that waist girth was not significantly

[1] Of note, RDA for protein is calculated as a constant multiplied by weight. Thus, the significant correlation between total protein/RDA and waist girth is spurious because it is confounded by the use of weight in the RDA calculation.

associated with any dietary intake variable. Thus, anticipated links between dietary intake and waist girth were not found in the current study for the SUGAR participants.

In the SUGAR subsample, correlational results between dietary intake and measures of distress and eating style were also mostly non-significant (Table 12). Poor dietary intake was expected to be related to higher levels of distress and maladaptive eating style. In the correlation matrix containing the 13 dietary intake variables and 4 distress indicators, only one dietary intake-distress correlation was significant; one significant correlation in the context of 51 non-significant correlations is not adequate evidence for a relationship between dietary intake and distress for the SUGAR participants. Moreover, this one significant relationship was the inverse of what was expected; higher caloric intake was associated with lower scores of perceived stress (r = -.20; p < .05). Regarding eating styles, the most consistent findings were significant correlations between measures of restrained eating and dietary intake such that poor dietary intake (e.g., higher total caloric intake, higher carbohydrate intake) was associated with lower scores of restrained eating. Low scores of restrained eating reflect adaptive (vs. maladaptive) eating style, so the direction of these correlations were opposite of what was hypothesized. Of the 7 eating style indicators and 13 dietary intake variables, only significant correlations between DEBQ external eating and measures of total caloric intake (r's = .15 - .20, p's < .05) were consistent with hypotheses. Partial correlation results controlling for age, gender, ethnicity, smoking status, education, and physical activity indicated that all significant correlations between dietary intake variables and distress and eating style indicators presented in Table 12 and described above remained significant after adjustment for covariates.

Relationships were also assessed between dietary intake and the latent factors (vs. observed variables) of distress and eating style. In these SEM hybrid analyses, the distress factor and all three first-ordered eating styles factors were entered as predictors for one of the dietary intake variables, adjusting for gender, age, education, and physical activity. Inclusion of ethnicity and smoking status prevented accurate parameter estimation, so these two covariates were omitted from modeling analyses.[2] Results from these SEM analyses with latent factors were similar to the results from correlational analyses with observed variables. Specifically, restrained eating had a direct, negative effect on total calories ($\beta = -.31$, $p < .05$), total calories/TEE ($\beta = -.44$, $p < .01$), carbohydrates ($\beta = -.31$, $p < .05$), carbohydrates/RDA ($\beta = -.31$, $p < .05$), protein/RDA ($\beta = -.34$, $p < .05$), and saturated fat ($\beta = -.32$, $p < .05$), and external eating had a direct, positive effect on total calories ($\beta = .27$, $p < .05$) and total calories/TEE ($\beta = .28$, $p < .05$).

Moderation analyses using SEM were also conducted to examine whether an interaction between distress and eating style had an effect on any dietary intake variable. Analyses were conducted with every possible combination of variables; that is, an interaction between each eating style factor (i.e., restrained, emotional, and external eating) and the distress factor was tested as a predictor for each dietary intake variable (see Table 4 for a listing of these 16 dietary intake variables), resulting in 48 analyses. No significant moderation effects were found in these analyses.

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[2] Without running a simulation study of the data, it cannot be known with certainty the cause of parameter estimation biases (Muthén & Muthén, 2002). Based on rules of thumb, however, it appears the combination of a small-to-moderate sample size with a complex model may explain estimation biases (Kline, 1998; Muthén & Muthén, 2002). Smoking and ethnicity, in particular, may result in parameter estimation biases because they are categorical variables with two to four categories (Muthén & Muthén, 2002). Categorical variables with two to four categories are not typically normally distributed, and differences in variable distribution affects the sample size needed for accurate parameter estimation (Kline, 1998; Muthén & Muthén, 2002).

In sum, dietary intake descriptive and correlation analyses failed to confirm the hypothesized associations with other model components. Poor dietary intake (e.g., high total caloric intake, high carbohydrate intake, high fat composition) was expected to be related to greater waist girth and higher levels of distress and maladaptive eating style. In the combined sample, this hypothesis was somewhat confirmed such that higher waist girth was significantly associated with higher total fat, higher %calories from total fat, and higher saturated fat. However, in the SUGAR subsample, waist girth was not significantly related to any dietary intake measures of total calories, carbohydrates, proteins, or fats. Moreover, correlations between dietary intake and distress and eating style were mostly non-significant. In addition, no moderation effects (i.e., an interaction between distress and eating style) significantly impacted any dietary intake variable. One consistent finding was the relationship between poor dietary intake and low restrained eating, which was contrary to the study's hypothesized findings. Dietary intake was included to be examined as a predictor of cardiometabolic risk and to be tested as a mediator linking distress and eating style to cardiometabolic risk. However, given that dietary intake was not related to waist girth in the SUGAR study cohort, dietary intake was thus excluded from further modeling analyses. Consequently, study aims 2-5 (see Methods section) were modified such that dietary intake was omitted from analyses.

Path Analyses

Path analyses were conducted using data from SUGAR participants only because eating style data was missing for MARCH participants. As such, before investigating path relationships, analyses were conducted to assess whether measurement models of distress and cardiometabolic risk, assessed with combined sample data described in the

previous section, were similar between SUGAR and MARCH subsamples. Figures 7 and 8 illustrate the distress model for the MARCH and SUGAR subsamples, respectively. Both models fit the data, and all factor loadings remained significant. Similarly, Figures 9 and 10 depict the cardiometabolic risk model for the MARCH and SUGAR subsamples. Examination of these models reveals differences in cardiometabolic risk between the subsamples. Specifically, (1) a path from insulin sensitivity to lipids is significant for MARCH but for not SUGAR participants; (2) a path from glucose tolerance to blood pressure is significant for MARCH but not for SUGAR participants; (3) a path from glucose tolerance to lipids is significant for MARCH but is a trend ($p = .06$) for SUGAR participants; and (4) all other paths are similar between subsamples. Figure 11 illustrates a trimmed, more parsimonious cardiometabolic risk model for the SUGAR subsample with non-significant pathways omitted [$\Delta\chi^2 (2) = 1.2$, $p = .55$]; note the effect of glucose tolerance on lipids was retained in the model and went from being a trend to statistically significant. For the SUGAR cardiometabolic risk model, ethnicity and smoking status were omitted because their inclusion prevented accurate parameter estimation as previously described. Omission versus inclusion of these covariates may account for the differences found between the MARCH and SUGAR cardiometabolic risk models, especially given that ethnicity and smoking status had significant effects on some model components. However, in a cardiometabolic risk model for the MARCH subsample that controlled for age, gender, education, and physical activity but not for ethnicity or smoking status, the direct effect of insulin sensitivity on lipids and the direct effect of glucose tolerance on blood pressure remained significant (respectively: $\beta = -.28$, $p < .001$; $\beta = -.15$, $p < .05$). These results suggest that the differences between the

MARCH and SUGAR cardiometabolic risk models do not appear to be caused by omission or inclusion of ethnicity or smoking status. Instead, differences are likely due to actual subsample differences in the cardiometabolic risk interrelationships.

Although not explicitly stated throughout, the following analyses described below were conducted using data from SUGAR participants only. All analyses included age, gender, education, and physical activity as covariates by specifying direct effects of each covariate on each latent factor and/or observed variable included in the model of interest. Unless otherwise noted, analyses involving cardiometabolic risk were conducted by specifying the trimmed cardiometabolic risk model for SUGAR (Figure 11).

Effect of Distress on Cardiometabolic Risk

As illustrated in Figure 12, the model of distress and cardiometabolic risk fit the data [χ^2 (59) = 73.2, p = .10; CFI = .977; RMSEA = .044; SRMR = .051)]. Distress did not have a significant effect on waist girth (β = -.06, n.s.).

Effect of Eating Style on Cardiometabolic Risk

The second-order maladaptive eating style factor had a significant effect on waist girth (β = .42, p < .05), but this model did not fit the data according to the χ^2 test and CFI criteria [χ^2 (98) = 131.6, p = .01; CFI = .947; RMSEA = .052; SRMR = .061)]. Consequently, modifications were made that resulted in good model fit according to all fit indices [χ^2 (88) = 107.6, p = .08; CFI = .969; RMSEA = .042; SRMR = .055)]; this fit was significantly improved from the previous model [$\Delta\chi^2$ (10) = 23.9, p < .01]. There were three modifications: examining eating style by its first-order factors versus the second-order factor, adding a correlation, and fixing an error variance to 0. Specifically, modification index suggestions included adding a path from restrained eating to waist

girth. Thus, the model was modified such that, instead of the second-order maladaptive eating style factor, a path from the 3 first-order eating style factors to waist girth was specified to assess the unique contribution of each on cardiometabolic risk (Figure 13). This modeling change incorporated the suggested modification index wherein a path from restrained eating to waist girth was added; the model was further modified to test for the effects of emotional and external eating on waist girth. A significant correlation between the error variances of DEBQ restrained eating and TFEQ-R18 cognitive restraint ($r = .22$, $p < .01$) was specified and was also guided by modification index suggestions. In addition, the error variance of RS was fixed to 0 to eliminate the resulting negative error variance. The correlation between the error variances of DEBQ restrained eating and TFEQ-R18 cognitive restraint was theoretically justified because these two restraint subscales can reflect both successful and unsuccessful restraint, whereas RS captures only unsuccessful restraint (Ouwens et al., 2003). Whether parameter estimates were significant or not did not differ as a function of whether the correlation between the error variances of DEBQ restrained eating and TFEQ-R18 cognitive restraint was specified. In other words, adding the correlation simply helped to improve model fit without disrupting other model components and interrelationships.

As shown in Figure 13, restrained eating had a direct effect on waist girth ($\beta = .40$, $p < .001$), whereas emotional and external eating did not (respectively: $\beta = .02$, n.s.; $\beta = .03$, n.s.). Paths from waist girth, in turn, led to the other cardiometabolic risk variables as previously described. To assess whether restrained eating had a direct effect on any other cardiometabolic risk factors, a model was specified in which a path was specified from restrained eating to waist girth, insulin sensitivity, glucose tolerance,

blood pressure, and lipids (Figure 14). This model fit the data well [χ^2 (82) = 100.7, p = .08; CFI = .971; RMSEA = .043; SRMR = .047)] and revealed that restrained eating did not have significant direct effects on insulin sensitivity (β = .02, n.s.), glucose tolerance (β = -.07, n.s.), blood pressure (β = -.09, n.s.), or lipids (β = .10, n.s.). Instead, the model indicated that restrained eating had a unique, direct effect on waist girth (β = .42, p < .001). Although restrained eating did not have a direct effect on cardiometabolic risk factors besides waist girth, the restrained eating factor had indirect effects on all other cardiometabolic risk factors. Specifically, restrained eating had a significant indirect effect on: insulin sensitivity via waist girth (restrained eating → waist → insulin sensitivity; β = -.24, p < .001); glucose tolerance via waist girth and insulin sensitivity (restrained eating → waist → insulin sensitivity → glucose tolerance; β = -.12, p < .01); blood pressure via waist girth (restrained eating → waist → blood pressure; β = .14, p < .05); and lipids (β = .20, p < .01), comprised of a significant indirect effect via waist girth (restrained eating → waist → lipids; β = .15, p < .01) and a significant indirect effect via waist girth, insulin sensitivity, and glucose tolerance (restrained eating → waist → insulin sensitivity → glucose tolerance → lipids; β = .03, p < .05). In sum, waist girth served as a significant mediator between restrained eating and worse insulin sensitivity, diminished glucose tolerance, higher blood pressure, and greater dyslipidemia. Given that restrained eating had a unique effect on waist girth, it should be noted that maladaptive eating style was not examined further as a second-order factor in the remaining analyses.

Effect of Distress on Cardiometabolic Risk, Moderated by Eating Style

To evaluate whether eating style moderated a relationship between distress and waist girth, an interaction term was created and tested for significance. Specifically, the distress factor, the restrained eating factor, and an interaction between distress and restrained eating were specified as predictors for waist girth, which in turn was specified to lead to the other cardiometabolic risk variables as illustrated in Figure 11. As before, age, gender, education, and physical activity were included as covariates, and emotional and external eating were included and specified to correlate with distress and restrained eating. However, given the SEM analytical demands on model parameter estimation when creating an interaction term from two latent factors, this model was modified by omitting emotional and external eating, glucose tolerance, blood pressure, and lipids. Waist girth and insulin sensitivity were chosen to remain in the model because these cardiometabolic factors were hypothesized to serve as entry points from distress and eating styles to the other cardiometabolic risk components. To assess whether results change when glucose tolerance, blood pressure, and lipids were excluded, the models that evaluated the effects of eating style and distress on cardiometabolic risk (see the previous two sections) were repeated with only waist girth and insulin sensitivity to represent cardiometabolic risk. These results demonstrated that the role of eating style and distress on waist girth did not change if the larger cardiometabolic risk model was trimmed to only include waist girth and insulin sensitivity. Specifically, in the trimmed model of eating style that fit the data [χ^2 (34) = 39.9, p = .23; CFI = .982; RMSEA = .037; SRMR = .044)], restrained eating had a direct effect on waist girth (β = .40, p < .001) whereas emotional and external eating did not (respectively: β = .02, n.s.; β = .03, n.s.),

and waist girth had a direct effect on insulin sensitivity (β = -.57, $p < .001$). These results are similar to those of the more comprehensive model depicted in Figure 13. The trimmed model of distress fit the data [χ^2 (20) = 20.0, $p = .46$; CFI = 1.000; RMSEA < .001; SRMR = .047)] and was similar to the model shown in Figure 12 in that distress did not have an effect on waist girth (β = -.06, n.s.), and waist girth had a direct effect on insulin sensitivity (β = -.57, $p < .001$).

Thus, the final moderation model included paths to waist girth from the distress factor, the restrained eating factor, and an interaction between distress and restrained eating, and a path to insulin sensitivity from waist girth, adjusted for age, gender, education, and physical activity. Results revealed a significant direct effect for restrained eating on waist girth ($B = 1.2$, $p < .01$) but no significant effects for distress or the interaction between distress and restrained eating on waist girth (respectively: $B = -.26$, n.s.; $B = -.07$, n.s.). In other words, the effect of distress on waist girth was not moderated by restrained eating.

Effect of Distress on Cardiometabolic Risk, Mediated by Eating Style

To assess whether eating style mediated a relationship between distress and waist girth, a model was tested in which the following were specified: a path from distress to waist girth and restrained, emotional, and external eating, a path from restrained eating to waist girth, and a path from waist girth to insulin sensitivity (Figure 15). Paths from emotional and external eating to waist girth were omitted because emotional and external eating failed to demonstrate significant effects on waist girth in previous analyses, and also because including these paths significantly worsened model fit [$\Delta\chi^2$ (1) = 11.7, $p < .001$; χ^2 (82) = 105.7, $p = .04$; CFI = .960; RMSEA = .048; SRMR = .058)].

Therefore, the model depicted in Figure 15 was tested. This model fit the data well [χ^2 (81) = 93.9, p = .15; CFI = .978; RMSEA = .036; SRMR = .060)]. Distress had a direct effect on restrained eating (β = .45, p < .01), emotional eating (β = .60, p < .001), and external eating (β = .54, p < .01). Restrained eating had a direct effect on waist girth (β = .49, p < .001). The direct effect of distress on waist girth was not significant (β = -.14, n.s.), but a significant indirect positive effect of distress on waist girth was mediated by restrained eating (β = .22, p < .01). The positive indirect effect and negative (albeit non-significant) direct effect of distress on waist girth canceled each other out; hence the result is a non-significant total effect (β = .07, n.s.). In other words, restrained eating mediated a positive effect of distress on waist girth, but the overall total effect of distress on waist girth was not significant due to a negative but non-significant direct effect of distress on waist girth.

Final Model

Compared with Figure 15, Figure 16 illustrates the final model in which the non-significant path from distress to waist girth has been omitted, depicting a more parsimonious model with good fit of the data according to all model fit indices [$\Delta\chi^2$ (1) = 1.4, n.s.; χ^2 (82) = 95.4, p = .15; CFI = .977; RMSEA = .036; SRMR = .062)]. Table 13 includes unstandardized and standardized parameter estimates for factor loadings and direct effects. Table 14 lists the proportion of the variance (R^2) in each observed and latent variable explained by the final model.

The impact of covariates on model variables is shown in Table 15. There was a trend for an effect of physical activity on waist girth such that less kcals exerted per week was associated with greater waist girth (β = -.16, p = .06). Age and education did not have

significant effects on any model variables. The effect of gender on model components is discussed in the section "Gender Differences."

Test of reversal hypothesis: the effect of waist girth on restrained eating. The final model depicts higher levels of restrained eating resulting in greater waist girth. To explore the directionality of this association statistically, the final model illustrated in Figure 16 was repeated with one modification: a path was reversed such that the effect from waist girth to restrained eating was specified. This model fit the data [χ^2 (79) = 91.7, p = .16; CFI = .979; RMSEA = .036; SRMR = .058)]. The effect of waist girth on restrained eating was significant (β = .39, p < .001). All other model results were similar to those of the model in Figure 16. Model fit did not differ between the model specifying restrained eating causing waist girth and the model specifying waist girth causing restrained eating [$\Delta\chi^2$ (3) = 3.7, n.s.]. Together, these results suggest the possibility that central obesity results in restrained eating cannot be ruled out.

Test of reversal hypothesis: the effect of eating styles on distress. The final model depicts higher levels of distress resulting in higher levels of restrained, emotional, and external eating. To explore the directionality of these associations statistically, the final model illustrated in Figure 16 was repeated with five modifications: three paths were reversed such that the effects from restrained, emotional, and external eating to distress were specified, and two additional correlations were specified (restrained eating with external eating, and restrained eating with emotional eating). As shown in Figure 17, this model fit the data [χ^2 (81) = 99.9, p = .08; CFI = .968; RMSEA = .043; SRMR = .062)]. Emotional eating had a direct effect on distress (β = .41, p < .05). However, the paths from restrained eating and external eating to distress were not significant (respectively:

β = .08, n.s.; β = .21, n.s). All other model results were similar to those of the model in Figure 16. These findings suggest that emotional eating may lead to distress, or that a bidirectional between emotional eating and distress exists. In contrast, the relationship between distress and the other two eating styles, restrained and external eating, appeared unidirectional such that distress leads to restrained and external eating.

Gender Differences

Of the covariates, gender was the most influential. Compared with men, women had higher scores of restrained and emotional eating and lower waist girth, and there was a trend for higher scores of external eating ($p = .07$) and worse insulin sensitivity ($p = .0994$) in women than men. Psychological distress did not differ by gender.

Further evidence for gender differences in cardiometabolic risk comes from the model containing data from both MARCH and SUGAR participants (Figure 6). Similar to results from the final model of the SUGAR subsample (Figure 16), as would be expected women of the combined, total sample displayed lower waist girth (β = -.29, $p < .001$); however, women also demonstrated worse insulin sensitivity (β = -.13, $p < .01$) than men. Compared with men, women also had lower blood pressure (β = -.23, $p < .001$), lower lipids (β = -.23, $p < .001$), and higher glucose tolerance (β = .21, $p < .001$). To examine whether interrelationships among cardiometabolic risk factors differ by gender, cardiometabolic risk was modeled for women and men separately, controlling for age, education, and physical activity. As can be seen by comparing Figure 18 with Figure 19, a direct effect of glucose tolerance on lipids was significant for men (β = -.24, $p < .01$) but not women (β = -.07, n.s.). For all other parameter estimates, achieving or not achieving statistical significance did not differ by gender. Although

both women and men had a significant effect of insulin sensitivity on lipids, the magnitude of this effect seems stronger in women than men (women: $\beta = -.32$, $B = -.21$, $SE = .054$, $p < .001$; men: $\beta = -.17$, $B = -.12$, $SE = .056$, $p < .05$). Similarly, the significant effect of waist girth on blood pressure appears stronger in women than men (women: $\beta = .44$, $p < .001$; men: $\beta = .23$, $p < .001$), but examination of unstandardized parameter estimates and standard errors suggests this difference is partly due to greater parameter variance in men than women (women: $B = .24$, $SE = .044$; men: $B = .19$, $SE = .052$).

Models using data from only the SUGAR subsample could not be statistically evaluated when divided by gender. The sample size of women in the SUGAR study was 38; sample sizes under 100 are considered small in regards to conducting SEM analyses (Kline, 1998). When complex SEM analyses, such as those illustrated in the final model, are conducted using data from a small sample, parameter estimations may not be trustworthy (Kline, 1998; Muthén & Muthén, 2002). Thus, whether interrelationships among cardiometabolic risk, distress, and eating style differ according to gender could not be accurately assessed.

Summary of Results

This project had five proposed aims. Some aims were modified due to unanticipated results. Below are the originally proposed aims and their corresponding results, followed by a brief summary of results obtained from modified analyses. [Note that the main reason for modified analyses was that dietary intake was not related to waist girth in the SUGAR subsample and was thus excluded from modeling analyses.]

Aim 1: Operationalize and validate theory-driven constructs of psychological distress, eating style, dietary intake, and cardiometabolic risk.

Latent factor models of psychological distress and eating style fit the data and were statistically acceptable, and a hybrid model of cardiometabolic risk fit the data and its CFA components were acceptable. A latent factor model of dietary intake would have likely fit the data and been statistically acceptable given the high intercorrelations among dietary variables, but no such factor was created because dietary variables failed to confirm the hypothesized associations with other model components (e.g., waist girth, eating style) in the SUGAR subsample; thus, these measures were excluded from further SEM analyses.

Aim 2: Test the hypothesis that dietary intake is associated with cardiometabolic risk.

In the combined sample, this hypothesis was somewhat confirmed such that higher waist girth was significantly associated with higher total fat, higher %calories from total fat, and higher saturated fat. However, in the SUGAR subsample, waist girth was not significantly related to any dietary intake measures of total calories, carbohydrates, proteins, or fats.

Aim 3: Examine whether distress and eating style each have a direct effect on dietary intake.

Correlations between dietary intake and distress and eating style were mostly non-significant. SEM analyses further confirmed a relationship between distress and dietary intake and a relationship between eating style and dietary intake were generally lacking. One rather consistent finding, however, supported a relationship between poor dietary

intake and low restrained eating; the direction of this relationship was contrary to the study hypothesis.

Aim 4: Assess whether a relationship between distress and dietary intake is moderated by eating style.

No significant moderation effects were found.

Aim 5: Test a combined, comprehensive model of moderation and mediation to examine whether dietary intake mediates the predicted relationship between distress and cardiometabolic risk, accounting for the predicted moderation effects of eating style on the association between distress and dietary intake.

Given that results for aims 2-4 did not support the smaller components of the larger model discussed in aim 5, a comprehensive model was not analyzed. Results did not support the model specified in aim 5.

Aims 2-5 were proposed to also examine gender differences. However, due to the small number of women in the SUGAR study (n = 38), results from SEM analyses of gender as a potential moderator would not be trustworthy. Group mean gender differences could be assessed and results showed that, compared with men, women reported more restrained, emotional, and external eating, lower waist girth, lower blood pressure, lower lipids, higher glucose tolerance, and worse insulin sensitivity. Distress did not differ by gender. Gender differences in the interrelationships among the cardiometabolic risk factors were examined using both SUGAR and MARCH data, and results revealed that a direct effect of glucose tolerance on lipids was significant for men but not women.

Additional Results

Maladaptive eating style was directly related to increased cardiometabolic risk. Specifically, restrained eating was associated with greater waist girth. Waist girth, in turn, served as a significant mediator between greater restrained eating and poorer insulin sensitivity, higher blood pressure, diminished glucose tolerance, and greater dyslipidemia. Emotional and external eating were not significantly related to waist girth. Of note, results suggest the possibility for a reversed effect such that waist girth leads to restrained eating. Findings also suggest that emotional eating may lead to distress. In contrast, the relationship between distress and the other two eating styles, restrained and external eating, appeared unidirectional such that distress leads to restrained and external eating but not the reverse.

Distress did not have a direct effect on waist girth, nor was a distress – waist girth relationship moderated by eating style. However, when a mediation model was assessed, results demonstrated that: (1) higher levels of distress were related to higher levels of restrained, emotional, and external eating, and (2) distress had an indirect, positive effect on waist girth via restrained eating, but the total effect of distress on waist girth was not significant. This finding appears to have occurred because the positive, indirect effect was canceled out by a negative (albeit non-significant), direct effect of distress on waist girth.

In short, higher levels of distress were related to higher levels of all three maladaptive eating styles, but only higher levels of restrained eating led to greater cardiometabolic

risk via waist girth. Distress was not directly related to cardiometabolic risk, but an indirect effect was found in which higher levels of distress led to greater waist girth via higher levels of restrained eating.

Chapter IV:

Discussion

In the present study, maladaptive eating style was related to measures of increased cardiometabolic risk. Specifically, restrained eating was associated with greater waist girth, a finding that is consistent with previous results demonstrating an association between restrained eating and body weight and size (Chaput et al., 2009; Dykes et al., 2004; Hays & Roberts, 2008; Lluch et al., 2000; van Strien et al., 2009). A novel finding from the current project is that waist girth served as a significant mediator between restrained eating and worse insulin sensitivity, higher blood pressure, diminished glucose tolerance, and greater dyslipidemia. Thus, this study is the first to provide evidence that eating style is associated with health risk beyond central obesity to the broader context of cardiometabolic risk as indexed by other factors. In conjunction with previous results demonstrating a link between restrained eating and disease states such as diabetes and cardiovascular disease, current study findings suggest that restrained eating may lead not only to greater cardiometabolic risk but also to later cardiometabolic disease development (Hainer et al., 2006).

Emotional and external eating were not significantly related to the central obesity measure, which is consistent with some findings (e.g., Hays & Roberts, 2008; Keski-Rahkonen et al., 2007; Lluch et al., 2000) but not others (e.g., Hays & Roberts, 2008; Keski-Rahkonen et al., 2007; Lluch et al., 2000; Scherwitz & Kesten, 2005; van Strien et al., 2009). Latent factors of restrained, emotional, and external eating were significantly correlated with each other, and each loaded significantly onto a second order latent factor of maladaptive eating style. Despite this overlap, only restrained eating was

related to cardiometabolic risk measures. A prospective twin study found similar results (Keski-Rahkonen et al., 2007). Restrained eating may be the only eating style directly related to central obesity because restrained eating is less conditional than emotional or external eating. That is, whether an individual engages in emotional or external eating is conditional upon that individual's eating occurring in response to negative emotions or external food cues. In contrast, an individual with a restrained eating style can become disinhibited and overeat not only by negative emotions and external foods cues, but also by their pervasive cognitive focus on food and their conscious efforts to restrict food intake. Therefore, consistent with the observed findings, the restrained eating style may be more influential on actual eating and consequent weight gain than the emotional and external eating style.

Restrained Eating and Central Obesity: Direction of Effects?

Just as it is possible that restrained eating can lead to obesity, it is also entirely possible that obesity can cause the development of a restrained eating style in an attempt to diet and lose weight. Indeed, current study results suggest the possibility for a reversed effect such that waist girth leads to restrained eating. Given the current study was cross-sectional in design, directionality of the observed relationship between restrained eating and waist girth cannot be determined definitively. It could be that restrained eating leads to central obesity, as proposed, but it could also be that central obesity leads to restrained eating. Yet a third possibility is that a reciprocal relationship exists. The initial work on restrained eating was based on the premise that restrained eating is the cause, not the result, of overeating and weight gain (Herman & Polivy, 1980). Few prospective studies are available, but there is some support for this direction

of effects. For example, a positive association between initial restrained eating and later changes in weight, BMI, and waist girth has been observed in women (Drapeau et al., 2003; Provencher, Polivy, et al., 2009). Regarding the hypothesis that obesity causes restrained eating, review of the items from the restrained eating measures provides some suggestion as to how obesity or weight gain can subsequently lead to higher scores of restrained eating. In these surveys (Appendices A-C), items include "if you have put on weight, do you eat less than you usually do?" (DEBQ), "I deliberately take small helpings as a means of controlling my weight" (TFEQ), and "how many pounds over your desired weight were you at your maximum weight?" (RS). Beyond an informal review of restrained eating items, there are also longitudinal results, albeit limited, to suggest obesity may lead to restrained eating. For example, in a study of 466 adults and 271 adolescents assessed at baseline and 2-year follow-up, participants completed the TFEQ, and several measures of adiposity were obtained (e.g., percentage body fat, waist girth, BMI; de Lauzon-Guillain et al., 2006). Baseline adiposity was associated with a larger increase in restrained eating, but baseline restrained eating did not predict change in adiposity. In short, some prospective studies have found that restrained eating causes obesity whereas others have found that obesity causes restrained eating. Conflicting results may be due to failure to account for factors that may moderate the restrained eating – adiposity relationship, such as gender, obesity status, and dieting history (e.g., de Lauzon-Guillain et al., 2006; Drapeau et al., 2003; Keski-Rahkonen et al., 2007; Konttinen, Haukkala, Sarlio-Lähteenkorva, Silventoinen, & Jousilahti, 2009; Provencher, Drapeau, Tremblay, Després, & Lemieux, 2003; Provencher, Polivy, et al., 2009). Thorough investigation of the role of these candidate moderating variables and other

relevant factors are needed to help explain the processes underlying the observed link between restrained eating and waist girth.

Psychological Distress and Eating Style

Whereas investigators have previously conceptualized eating style primarily as an individual difference variable that impacts a person's eating behavior under distress (e.g., Polivy & Herman, 1999; Wardle et al., 2000), current findings do not support a moderating role for eating style. Instead, psychological distress had a direct effect on each of the eating styles: higher levels of distress (i.e., depression, stress, anxiety, and anger) were related to higher levels of restrained, emotional, and external eating. Failure to find a moderating effect may be due to conceptually different types of distress measured in the current versus past studies. Psychological distress in the current study was assessed by self-report questionnaires that were more likely to reflect chronic stress or psychological distress, whereas distress in previous studies was typically treated as an independent variable that was manipulated to measure food intake after acute stress (e.g., Heatherton et al., 1991; Oliver et al., 2000; Wallis & Hetherington, 2004). Of the research literature on distress and eating style, only a small minority focuses on chronic distress; results from such studies are generally consistent with the present findings in that distress was found to be related to maladaptive eating styles (e.g., Johnson & Wardle, 2005). Both sets of findings are possible. That is, acute stress may differentially impact eating behavior of individuals with varying maladaptive eating style, and chronic distress may be directly related with maladaptive eating style in a more linear fashion. Future studies assessing both acute and chronic distress are needed to test the hypothesis that maladaptive eating style is directly related to chronic distress, but that eating style also

moderates the relationship between acute stress and dietary intake and consequent cardiometabolic risk.

Although the path between distress and eating styles in the present study was specified from distress to maladaptive eating styles, findings of the reversal hypothesis suggest that emotional eating may lead to distress. In contrast, the relationship between distress and the other two eating styles, restrained and external eating, appeared unidirectional such that distress led to restrained and external eating. However, given the cross-sectional study design of the current project, caution must be used when interpreting these results. Longitudinal studies are needed to provide a better understanding of the direction of effects between distress and maladaptive eating.

Few longitudinal studies on eating style and distress exist. Prospective studies have shown that restricted food intake, whether resulting from an acute laboratory manipulation, dieting pattern, or restrained eating style, causes worsened mood, heightened irritability, and depression (Keys, Brozek, Henschel, Mickelsen, & Taylor, 1950; Laessle, Platte, Schweiger, & Pirke, 1996; McFarlane, Polivy, & McCabe, 1999; Polivy, 1996; Stice, Hayward, Cameron, Killen, & Taylor, 2000). No study, however, has examined longitudinally whether psychological distress causes subsequent changes in measures of restrained eating style.

Although direction of causality between distress and maladaptive eating style is unclear due to the scarcity of prospective studies, theories have been formulated to explain the relationship between distress and eating style. For example, the psychosomatic theory of obesity posits emotional eating results from the learned association, over time, between eating and improved emotionality (Kaplan & Kaplan,

1957). Logically, those with long-term distress have more opportunities to engage in emotional eating. Thus, distress may cause emotional eating in this manner. The current findings are consistent with this theory; of the three eating styles, distress had the strongest impact on emotional eating. Assuming distress causes emotional eating style and that this possibility was the case for the current study sample, it would appear that those high in emotional eating have not been successful with reducing negative emotions, indicated by their higher, not lower, levels of distress. Of course, it could be that poor dietary intake alleviates negative emotions temporarily but the underlying, chronic distress remains. Less clear is how distress might cause restrained or external eating. One possibility is that distress primarily leads to increases in emotional eating, resulting in the development of a more general maladaptive eating style that includes restrained and external eating.

Cardiometabolic Risk Model

In the present study, the MARCH subsample model of cardiometabolic risk replicated the Klaus et al. (2009) model. These results were expected, as the Klaus et al. model was analyzed using data from the MARCH study. The current project also replicated the previous model with independent data obtained from the SUGAR study, with the exception that no direct effects were found for insulin sensitivity on lipids or glucose tolerance on blood pressure. These differences may be attributed to the differences between the MARCH and SUGAR subsamples. For example, SUGAR participants exhibited greater cardiometabolic risk than MARCH participants as indicated by several measures. The effects of insulin sensitivity on lipids and glucose tolerance on blood pressure may become less pronounced in individuals with higher versus lower levels of

cardiometabolic risk. Research is needed to explore these potential moderating effects of risk severity on the relationships among risk factors.

Gender Differences

Due to the small number of women in the SUGAR study in conjunction with complex SEM analyses, gender could not be statistically examined as a potential moderator in interrelationships among cardiometabolic risk, distress, and eating style. However, group mean gender differences could be assessed. Distress did not differ by gender. Women reported more restrained, emotional, and external eating than men. Whereas the gender differences in restrained and emotional eating were significant, the difference in external eating was a trend (p = .07). van Strien and colleagues (2009) found similar results, wherein women reported higher DEBQ restraint and emotional eating scores than men, but external eating scores were similar between men and women.

Several gender differences were found for cardiometabolic risk. Compared with premenopausal women, men displayed greater cardiometabolic risk as indicated by greater waist girth, higher blood pressure, and greater dyslipidemia; these findings are consistent with the research literature (Regitz-Zagrosek, Lehmkuhl, & Mahmoodzadeh, 2007). In contrast, women displayed greater cardiometabolic risk as indicated by worse insulin sensitivity than men, which is also consistent with previous studies (e.g., Wilkin & Murphy, 2006). However, one finding of gender differences conflicts with prior research results. Impaired glucose tolerance has been reported to be more common among women than men, but women from the current study sample had higher glucose tolerance than men than women (Huang, Shimel, Lee, Delancey, & Strother, 2007; Regitz-Zagrosek et al., 2007; Unwin, Shaw, Zimmet, & Alberti, 2002). Moreover,

results revealed that a direct effect of diminished glucose tolerance on greater dyslipidemia was significant for men but not women. It is unclear why women in the current study sample failed to demonstrate the expected results (i.e., more diminished glucose tolerance than men and a direct effect of glucose tolerance on lipids). It is possible the present findings are sample-specific and may limit the external validity of the glucose tolerance results for women.

Psychological Distress and Central Obesity

In a review conducted by Torres and Nowson (2007), the authors concluded chronic stress can lead to weight gain. In the current study, psychological distress was not directly related to central adiposity indexed by waist circumference. However, as others have noted, the effect of distress on weight gain may be moderated by gender, eating styles, and distress severity and type (Greeno & Wing, 1994; Torres & Nowson, 2007). The current study attempted to investigate the moderating effects of gender and eating styles. Eating styles did not serve as moderators, but restrained eating did mediate the relationship between distress and waist girth.

Type of distress may also moderate the relationship between distress and obesity, but assessing the unique contribution of different types of distress was not an aim of the current study. Instead, distress was measured as the shared variance, or overlap, of self-report scores of depression, anxiety, stress, and anger. Whether different types of distress (e.g., depression vs. anger) differentially impacted waist girth was not examined, so potential effects of one type of distress on central obesity may have become masked by combining measures to create a more general latent factor of distress. Future studies investigating the differential effects of various types of distress on central obesity would

provide a more comprehensive understanding of the potential impact of distress on cardiometabolic health.

In short, the current study found that distress was only indirectly related to central obesity via restrained eating. However, distress and central obesity may be associated through other mechanisms. An in-depth examination of distress and central obesity with potential moderating factors, such as gender and distress type, may indicate that distress is more likely to lead to central obesity for a particular subset of individuals.

Dietary Intake

Poor dietary intake (e.g., high total caloric intake, high fat composition) was expected to be related to greater waist girth and higher levels of distress and maladaptive eating style. In the combined sample, this hypothesis was somewhat confirmed such that greater waist girth was significantly associated with higher total fat, higher %calories from total fat, and higher saturated fat. However, in the SUGAR subsample, waist girth was not significantly related to any dietary intake measures of total calories, carbohydrates, proteins, or fats. Poor dietary intake may have been related to greater waist girth in the MARCH but not the SUGAR subsample because the MARCH study consisted of a larger sample and consequently had more power to detect significant effects. However, inspection of correlations indicate that the magnitudes of *r*'s between waist girth and total fat, %calories from total fat, and saturated fat are not similar but instead differ between the two studies. Thus, it is unlikely that with merely more power, dietary intake would have been significantly associated with waist girth in the SUGAR subsample. Failure to detect a significant relationship between dietary intake and central obesity in the SUGAR participants may instead be due to differences between SUGAR

and MARCH participants. For example, compared to the MARCH subsample, the SUGAR subsample displayed greater cardiometabolic risk and reported poorer dietary intake. These subsample mean differences may impact (i.e., strengthen/weaken) relationships among dietary intake and cardiometabolic risk variables via moderating effects.

Correlations between dietary intake and distress and eating style were mostly non-significant. One rather consistent finding, however, supported a relationship between poor dietary intake and low restrained eating, which is in contrast to the study's hypothesized findings. High, rather than low, levels of restrained eating was expected to be associated with poor dietary intake via a restraint-disinhibition pattern of eating. One way to interpret the current findings is that, at the snapshot time of the study, participants with high versus low levels of restrained eating were successfully restricting food intake and were subsequently less likely to report poor dietary intake at the time of assessment. In contrast, the significant positive association between restrained eating and waist girth may reflect the long-term toll restrained eating style has had on the body through a chronic pattern of both successful periods of restraint and unsuccessful periods of disinhibition.

Restraint and Disinhibition

Restrained eating, as measured in the current study, represented unsuccessful dieting efforts. Restrained eating was conceptualized in this way to be consistent with the restraint theory. The restraint theory refers to unsuccessful dieting in which attempts to regulate food intake lead to episodic overeating (Herman & Polivy, 1980). In other words, restrained eating is thought to cause obesity when it is characterized by a pattern

of restraint and disinhibition (de Lauzon et al., 2004; Ganley, 1988). The restraint theory posits a casual path from restrained eating to overeating, and although previous research findings demonstrate that dieting does indeed precede overeating for some individuals, overeating was found to precede dieting for others (e.g., Brewerton, Dansky, Kilpatrick, & O'Neil, 2000; Grilo & Masheb, 2000). Future research examining how and for whom the restraint-disinhibition cycle emerges may help to identify ways to prevent such an eating pattern from developing.

Whereas some restrained eaters overeat, as described by the restraint theory, other restrained eaters do successfully restrict food intake and do not overeat. Future studies should examine what factors determine success or failure in restrained eaters. Recent findings demonstrate that self-regulatory processes moderate the effect of food cues on restrained eaters such that food cues activate the dieting goal in successful restrained eaters and inhibit the dieting goal in unsuccessful restrained eaters (Papies, Stroebe, & Aarts, 2008). Another recent study found that restrained eaters only overate when they were also impulsive, which was determined by their ability to inhibit pre-potent responses on a stop-signal task (Jansen et al., 2009). These results confirm the restraint theory of obesity in that restrained eating in combination with disinhibition is related to poor dietary intake (Jansen et al., 2009; Papies et al., 2008). More research is needed to determine why some restrained eaters have poor inhibitory control and what types of prevention and treatment strategies may help to increase inhibitory control.

Clinical Applications

In contrast to traditional dieting programs, findings from the current study suggest interventions that decrease, not increase, restrained eating style would lead to weight loss

(McTigue et al., 2003). Some interventions have indeed been designed to abandon dieting efforts and restrictive eating habits. These interventions are referred to as non-diet, un-diet, and natural eating, and include specific programs such as Health At Every Size (HAES), Diet Free Forever (DFF), and Intuitive Eating (Bacon, Stern, van Loan, & Keim, 2005; Ciliska, 1998; Lowe, Foster, Kerzhnerman, Swain, & Wadden, 2001; Provencher, Bégin, et al., 2009; Steinhardt, Bezner, & Adams, 1999; Tribole & Resch, 1995). Interventions vary slightly in how sessions are conducted and what specific topics are covered, but all are psychoeducational group interventions that include specific goals to reduce restrictive dieting behaviors and replace them with internally-regulated eating. Despite efforts, research shows that interventions which successfully reduce restrained eating do not result in weight loss (Bacon et al., 2002; Bacon et al., 2005; Ciliska, 1998; Lowe et al., 2001; Steinhardt et al., 1999). In contrast, interventions that increase restrained eating have been found to result in weight loss (Bacon et al., 2002; Lowe et al., 2001; Teixeira et al., 2009). However, follow-up assessments of these studies were conducted within a year or less, and weight gain that negates initial weight loss has been shown to manifest at a later follow-up (e.g., Bacon et al., 2005). In short, research thus far suggests that neither traditional nor non-diet interventions are overwhelmingly successful with long-term weight loss. Of note, a measurement of weight does not capture weight distribution. Redistribution of weight, such as changes in central adiposity, was not assessed in the studies reviewed above; such assessments but may have revealed positive non-diet intervention effects on cardiometabolic health.

"Non-diet" interventions (e.g., Health At Every Size) may fail at achieving weight loss, but research shows that such programs lead to better cardiometabolic health in obese

women as indicated by lowered triglycerides, LDL-C, total cholesterol, and SBP; comparison groups that increased restrained eating did not exhibit these cardiometabolic improvements (Bacon et al., 2002; Bacon et al., 2005). Moreover, non-diets were associated with improved psychological well-being, such as increased self-esteem, decreased depressive symptoms, and decreased body dissatisfaction; these improvements were less consistently observed with traditional diets (Bacon et al., 2002; Ciliska, 1998; Steinhardt et al., 1999). Compared with traditional diets, non-diets also had better attendance and significantly lower attrition rates (e.g., Bacon et al., 2002). Taken together, the evidence suggests non-diet interventions may have better treatment outcome results than traditional diets.

As mentioned previously, research evidence does not support weight loss following non-diet programs. Rather than conclude non-diet interventions are hopelessly ineffective at weight loss, closer examination of program components may provide insight into underlying intervention processes. For example, one aspect of non-diets that should be investigated further is rigid versus flexible restrained eating. Flexible restrained eating is a more graduated and lenient approach to eating and weight control, whereas rigid restrained eating is a more dichotomous, all-or-nothing, and excessively disciplined eating pattern (Westenhoefer, 1991). These subtypes of restrained eating are related to disinhibition differently; flexible restrained eating is associated with low disinhibition, whereas rigid restrained eating is associated with high disinhibition (Westenhoefer, 1991). Thus, flexible restrained eating and successful dieting appear to be related constructs. Indeed, Teixeira and colleagues (2009) found that increases in flexible restrained eating predicted weight loss 2 years later in overweight and obese

women; rigid restrained eating did not predict later weight change. Further, studies indicate weight loss and weight maintenance is accompanied by decreased disinhibition but not decreased restrained eating per se (Provencher, Bégin, et al., 2009; Teixeira et al., 2009). These findings suggest that high disinhibition, which reflects failure at restrained eating and is related specifically to rigid restrained eating, should be the primary target for intervention. Altering restrained eating may only lead to weight loss in so much that disinhibition is decreased. Therefore, future clinical obesity research should distinguish between flexible and rigid restrained eating, with attempts to increase flexible restrained eating, decrease rigid restrained eating, and decrease disinhibition.

As presented throughout this section already, more research is needed to increase effectiveness and efficacy of obesity intervention programs. More studies are needed with men; the majority of non-diet research has been conducted with women only (e.g., Bacon et al., 2005; Ciliska, 1998; Lowe et al., 2001). As current studies focus largely on interventions for people who are already overweight or obese, more research is needed on prevention efforts. In addition, given the significant relationship found between psychological distress and eating style in previous studies and in the current project, intervention and prevention programs should examine how improvements in distress may impact changes in eating style and consequent weight change. Lastly, cardiometabolic measures such as insulin sensitivity, cholesterol, and blood pressure should be obtained as outcome variables along with weight when possible.

Eating Disorders

The focus of the current study was the interrelationships among distress, eating style, and obesity, but distress and eating style can also lead to weight problems at the other end

of the spectrum, such as anorexia nervosa and bulimia nervosa (e.g., Hsu, 1996; Lilly, 2003; Morris & Twaddle, 2007; Zandian, Ioakimidis, Bergh, & Södersten, 2007). Specifically, dieting has been shown to be a major risk factor for the development of anorexia nervosa and bulimia nervosa (Hsu, 1996). In addition, anxiety and depressive symptoms are associated with these eating disorders (Lilly, 2003; Morris & Twaddle, 2007; Zandian et al., 2007). Although all participants in the current project were not using any medications having a psychiatric effect, participants were not specifically screened for eating disorders. Future studies of obesity and eating style should include an eating disorders screen to assess potential differences in the underlying processes among these subpopulations.

Strengths and Limitations

One study limitation was that eating style data was not collected from MARCH participants, resulting in a lower sample size for the analyses of primary interest. Reduced sample size was expected to present limitations such as inability to run more complex SEM analyses and inability to include several important covariates. However, the sample size of the SUGAR study alone was sufficient to run several complex SEM models adjusted for age, gender, education, and physical activity. Inclusion of physical activity data is a particular strength of the current project because the majority of research fails to account for energy expenditure.

In the present study, only restrained eating was found to have an effect on waist girth. Potential effects of emotional and external eating on central adiposity might have been masked by suppressed ranges. Indeed, previous studies have reported means that are higher than those of the current project for emotional and external eating (Konttinen

et al., 2009; O'Connor et al., 2008; Oliver et al., 2000; Tucker & Bates; 2009; van Strien et al., 2009). In contrast, prior studies have reported means for restrained eating that are similar to those of the present study (Lluch et al., 2000; O'Connor et al., 2008; Oliver et al., 2000; Wardle et al., 2000), although some have reported higher means (Konttinen et al., 2009; van Strien et al., 2009). Likely reasons behind these differences in eating style measure means include differences in sample characteristics, such as health status. That is, the current sample was healthy in that participants did not have any medical or psychiatric disorders. If other studies included individuals with medical or psychiatric conditions, their sample means for maladaptive eating styles may be higher for this reason. Unfortunately, detailed medical and psychiatric information was not collected or reported in prior studies, making it impossible to determine whether differences in sample characteristics is the source of eating style differences between these studies and the present study. In addition to differences in sample characteristics, differences in methodology may have impacted eating style scores. In the current project, the eating style questionnaires were included within a larger packet of many psychosocial questionnaires. In contrast, other studies may have given participants only the eating styles questionnaires to complete. Thus, participants may have responded to the eating style questionnaires differently across studies based on how the eating style questionnaires were presented.

Data was collected cross-sectionally. Thus, the direct, causal effects portrayed in the SEM models may in fact be in the opposite direction. For example, it could be that restrained eating led to central obesity, but it is also entirely possible that central obesity caused the development of a restrained eating style in an attempt to diet and lose weight.

Moreover, a reciprocal relationship may exist. Unfortunately, the current study design did not allow for distinction among these mechanisms. This study does, however, provide evidence of a significant relationship between restrained eating and measures of cardiometabolic risk that requires further exploration by longitudinal and experimental studies.

Dietary intake was excluded from the final analyses because preliminary analyses failed to confirm the hypothesized associations between dietary intake and other model components. Intake tends to be underestimated by 24-hour dietary recalls, and this may explain why the expected relationships were not found to be significant (Greger & Etnyre, 1978). Another explanation is that the dietary recalls were not representative of participants' typical dietary intake. Previous studies have examined dietary intake with prospective daily diaries, and use of such diaries in the future may help to obtain more accurate measures of intake (e.g., O'Connor et al., 2008). In addition, investigators should consider assessing history of dietary intake to account for the variability in duration of current and past eating patterns. For example, 30 years of poor dietary intake are likely to be more influential on cardiometabolic risk than only 2 years of poor dietary intake. Other methods of dietary intake assessment, such as estimated and weighted food records, food frequency questionnaires, and electronic diaries, may be useful in future research as well (Medlin & Skinner, 1988). Despite the dietary intake limitations, this study is one of the few to measure natural eating rather than laboratory-based eating. As more research is conducted, including multiple measures of dietary intake should be considered.

Regarding strengths, this study is one of the minority that includes outcome variables beyond measures of central obesity. Numerous cardiometabolic risk measures were collected and examined, including measures of insulin sensitivity, glucose tolerance, lipidemia, and blood pressure. Rather than funneling cardiometabolic risk data into a dichotomous variable reflecting whether or not MetS criteria was met, SEM analyses were used to examine the effects the risk factors had on one another. SEM analyses provided additional study strengths, such as the creation and use of latent variables. This project is the first to use restrained, emotional, and external eating latent factors that were created from multiple eating style questionnaires. Moreover, a second-order factor of maladaptive eating style created by each of the three separate eating styles has never before been reported.

Inclusion of all three eating styles in one model provided a way to examine the effects of unique versus shared variance of the eating styles. In other words, the current study assessed whether what was unique to a particular eating style or what was shared among all the eating styles was more strongly related to model components. For example, in a comprehensive SEM model including all three eating styles and multiple cardiometabolic risk measures, a significant, direct association was found between restrained eating and waist girth. This relationship with waist girth was unique to restrained eating; no such relationship was found for emotional or external eating. Likewise, the direct relationship with restrained eating was unique to waist girth; no such relationship was found for the other cardiometabolic risk factors (i.e., insulin sensitivity, glucose tolerance, dyslipidemia, blood pressure). In short, the comprehensive SEM analyses conducted in the present study allowed for more detailed and confident conclusions.

Summary and Conclusions

Psychological distress was positively related to restrained, emotional, and external eating styles, but only restrained eating was directly associated with greater waist girth. Distress was not directly related waist girth, but an indirect effect was found in which higher levels of distress led to greater waist girth via higher levels of restrained eating. Waist girth, in turn, served as a significant mediator between restrained eating and worse insulin sensitivity, higher blood pressure, diminished glucose tolerance, and greater dyslipidemia. These findings confirm the Klaus et al. (2009) model of cardiometabolic risk whereby central obesity served as an entry point to additional cardiometabolic risk factors. Model effects were significant when controlling for several important covariates, including age, gender, education, and physical activity, and when simultaneously analyzed in a comprehensive SEM model including distress, eating style, and cardiometabolic risk variables.

Unfortunately, the role of gender in how distress, eating styles, and cardiometabolic risk are interrelated could not be examined due to the small number of women in the SUGAR study in conjunction with complex SEM analyses. That is, it remains unknown whether the significant effect of distress on each of the eating styles found in the current study was driven primarily by men, women, or both. Similarly, small sample size prevented the evaluation of whether gender moderated the effect of restrained eating on central obesity. Given that women reported more restrained, emotional, and external eating than men, future studies with larger samples should follow-up by assessing for potential moderating effects of gender on the interrelationships among distress, eating styles, dietary intake, and cardiometabolic risk.

The clinical implication of the present findings is that decreasing restrained eating style may lead to less central fat deposition and hence reduced cardiometabolic risk. Such "non-diet" interventions show potential for improved cardiometabolic health, but more research is needed. Particularly needed are studies examining prevention and intervention outcomes based on type of restrained eating – flexible versus rigid – to better understand how these different restrained eating subtypes operate and how they can be altered effectively to improve health.

The cross-sectional results obtained in the current study need to be assessed with longitudinal designs to better address issues of causality. Moreover, a longitudinal study that incorporates an experimental component could further contribute knowledge about the effects of chronic versus acute distress on dietary intake, and how eating style may function differently in these separate processes.

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Table 1

Demographic Characteristics

Variable	MARCH (n = 339)	SUGAR (n = 125)	Total (n = 464)
	M (SD)	*M (SD)*	*M (SD)*
Age (yrs)*	35.45 (9.09)	38.95 (8.51)	36.39 (9.07)
Gender*			
% Men	51.6%	69.6%	56.5%
% Women	48.4%	30.4%	43.5%
Ethnicity*			
% White	14.4%	4.0%	11.6%
% Hispanic	49.0%	77.6%	56.7%
% Black	31.6%	15.2%	27.2%
% Other	5.0%	3.2%	4.5%
Total family income ($K)*	18.70 (14.59)[a]	15.11 (16.52)[b]	17.61 (15.27)[c]
Education (yrs)	13.45 (3.17)	13.30 (2.68)	13.41 (3.04)
Physical activity (kcal/wk)	1653.37 (1433.87)[d]	1731.93 (1456.41)[e]	1674.44 (1438.65)[f]
Smoking status*			
% Current smoker	18.9%	2.4%	14.4%
% Former smoker	12.1%	16.8%	13.4%
% Never smoked	69.0%	80.8%	72.2%

[a]n = 274. [b]n = 119. [c]n = 393. [d]n = 311. [e]n = 114. [f]n = 425.
*p < .05, for MARCH and SUGAR subsample comparison.

Table 2

Cardiometabolic Risk Variables, Adjusted for Age, Gender, Ethnicity, and Smoking Status

Variable	MARCH (n = 339)		SUGAR (n = 125)		Total (n = 464)	
	adj. *M (SE)*		adj. *M (SE)*		adj. *M (SE)*	
BMI (kg/m^2)*	27.18	(0.25)	29.83	(0.43)	28.51	(0.24)
Weight classification*						
% Normal weight or less	31.3%		16.8%		27.4%	
% Overweight	44.2%		38.4%		42.7%	
% Obese	24.5%		44.8%		29.9%	
BSA (m^2)*	1.88	(0.01)	1.94	(0.02)	1.91	(0.01)
Weight (kg)*	78.23	(0.82)	84.47	(1.40)	81.35	(0.78)
Height (m)†	1.69	(0.004)	1.68	(0.006)	1.69	(0.003)
Waist girth (cm)*	88.87	(0.66)	97.35	(1.13)	93.11	(0.63)
WHR*	0.86	(0.003)	0.91	(0.005)	0.89	(0.003)
Insulin sensitivity (mg/kg•min)*	5.78	(0.17)	4.84	(0.29)	5.31	(0.16)
Fasting insulin (μU/mL)	12.16	(1.03)	12.02	(1.06)	12.11	(1.03)
Fasting glucose (mg/dL)	88.42	(0.49)	88.40	(0.84)	88.41	(0.47)
$AUC_{Insulin}$	9866.05	(326.61)	8992.18	(560.72)	9429.12	(312.95)
$AUC_{Glucose}$	15067.79	(163.93)	15435.44	(281.43)	15251.61	(157.07)
Triglycerides (mg/dL)*	92.90	(1.03)[a]	106.66	(1.05)	99.54	(1.03)[b]
LDL-C (mg/dL)*	104.31	(1.64)[a]	115.15	(2.80)	109.73	(1.56)[b]
HDL-C (mg/dL)	45.95	(0.65)[a]	44.78	(1.10)	45.37	(0.62)[b]
Total cholesterol (mg/dL)*	171.36	(1.80)[a]	184.09	(3.08)	177.72	(1.72)[b]
TC:HDL-C*	3.95	(0.07)[a]	4.42	(0.12)	4.19	(0.07)[b]
DBP (mmHg)*	75.26	(0.49)[c]	82.45	(0.84)	78.85	(0.47)[d]
SBP (mmHg)*	114.89	(0.63)[c]	118.50	(1.07)	116.70	(0.60)[d]

[a]n = 335. [b]n = 460. [c]n = 336. [d]n = 461.

*$p < .05$, †$p < .10$, for MARCH and SUGAR subsample comparison controlling for age, gender, ethnicity, and smoking status.

For abbreviations, refer to Glossary of Abbreviations and Symbols.

Table 3

Percentage of Participants Fulfilling Metabolic Syndrome Criteria

MetS criteria	MARCH	SUGAR	Total
Waist girth*	26.8%	54.4%	34.3%
Triglycerides	18.2%	28.8%	21.1%
HDL-C	48.1%	48.0%	48.0%
Blood pressure*	18.2%	39.2%	23.9%
Fasting glucose (≥110 mg/dL)	3.2%	5.6%	3.9%
Fasting glucose (≥100 mg/dL)	16.2%	23.2%	18.1%
MetS			
NCEP ATP III criteria*	11.9%	26.4%	15.8%
Modified NCEP ATP III criteria*	16.0%	33.6%	20.8%
Number of MetS criteria[a] fulfilled*			
0	29.5%	18.4%	26.5%
1	35.7%	20.8%	31.7%
2	18.9%	27.2%	21.1%
3	11.2%	20.8%	13.8%
4	4.1%	8.0%	5.2%
5	0.6%	4.8%	1.7%

[a] Modified NCEP ATP III criteria.
*$p < .05$, for MARCH and SUGAR subsample comparison controlling for age, gender, ethnicity, and smoking status.
For abbreviations, refer to Glossary of Abbreviations and Symbols.

Table 4

Descriptive Statistics for Average Daily Dietary Intake

Variable	MARCH (n = 339)	SUGAR (n = 125)	Total (n = 464)
	M (SD)	*M (SD)*	*M (SD)*
Calories			
Total calories (kcal)*	1825.91 (831.94)	2103.99 (932.72)	1900.83 (868.05)
Total calories (kcal)*[a]	1872.33 (46.17)[b]	2114.09 (78.90)[c]	1993.21 (44.39)[d]
Predicted TEE (kcal)[e]	-	2687.06 (625.85)	-
Total calories/TEE[e]	-	0.80 (0.34)	-
Carbohydrates			
Total carbohydrates (g)*	240.56 (119.58)	269.62 (130.33)	248.39 (123.10)
% calories from carbs†	53.38 (10.47)	51.23 (10.79)	52.80 (10.59)
Total carbohydrates/RDA*	1.85 (0.92)	2.07 (1.00)	1.91 (0.95)
AMDR = 45-65%*			
% less than AMDR	17.7%	30.4%	21.1%
% within AMDR	69.9%	56.8%	66.4%
% more than AMDR	12.4%	12.8%	12.5%
Protein			
Total protein (g)*	74.08 (36.90)	89.90 (40.14)	78.34 (38.41)
% calories from protein	16.71 (5.07)	17.45 (4.67)	16.91 (4.97)
Total protein/RDA*	1.21 (0.62)	1.37 (0.64)	1.25 (0.63)
AMDR = 10-35%			
% less than AMDR	4.4%	2.4%	3.9%
% within AMDR	94.7%	96.8%	95.2%
% more than AMDR	0.9%	0.8%	0.9%

Variable	MARCH (n = 339)	SUGAR (n = 125)	Total (n = 464)
	M (SD)	*M (SD)*	*M (SD)*
Fat			
Total fat			
Total fat (g)*	63.86 (36.30)	75.55 (46.66)	67.01 (39.65)
% calories from total fat	30.39 (8.47)	31.32 (9.87)	30.64 (8.87)
AMDR = 20-35%			
% less than AMDR	10.9%	13.6%	11.7%
% within AMDR	58.7%	48.8%	56.0%
% more than AMDR	30.4%	37.6%	32.3%
Saturated fat (g)*	20.69 (12.98)	24.98 (18.22)	21.84 (14.68)
Monounsaturated fat (g)	18.71 (12.54)	18.28 (10.35)	18.59 (11.98)
Polyunsaturated fat (g)	10.51 (7.71)	10.09 (8.21)	10.39 (7.84)

[a] adjusted for age, gender, weight, height, and Paffenbarger Physical Activity Index score; adj. *M (SE)* reported.
[b] n = 311. [c] n = 114. [d] n = 425.
[e] predicted TEE was not calculated for MARCH participants due to lack of available data (i.e., detailed physical activity data).
*p < .05, †p < .10, for MARCH and SUGAR subsample comparison.
For abbreviations, refer to Glossary of Abbreviations and Symbols.

Table 5

Descriptive Statistics for Eating Style Variables for SUGAR Participants (n = 125)

Variable	min	max	*M*	*SD*
DEBQ				
Restrained	1.00	4.70	2.37	1.01
Emotional	1.00	4.38	1.60	0.84
Diffuse emotions	1.00	4.75	1.80	0.95
Clearly labeled emotions	1.00	4.56	1.51	0.83
External	1.00	5.00	2.60	0.85
TFEQ-R18[a]				
Cognitive Restraint	0.00	88.89	25.04	22.99
Emotional	0.00	77.78	14.05	20.87
Uncontrolled	0.00	66.67	19.38	17.53
RS	1.00	19.00	8.35	4.76

[a]*n* = 121.
For abbreviations, refer to Glossary of Abbreviations and Symbols.

Table 6

Descriptive Statistics for Psychological Distress Variables

Variable	MARCH			SUGAR			Total		
	n	*M*	(*SD*)	*n*	*M*	(*SD*)	*n*	*M*	(*SD*)
Beck Depression Inventory									
Cognitive*	335	3.06	(4.09)	125	0.90	(2.40)	460	2.47	(3.82)
Somatic*	336	1.87	(2.42)	125	0.99	(1.76)	461	1.63	(2.29)
Total*	335	4.91	(5.79)	125	1.89	(3.80)	460	4.09	(5.49)
Perceived Stress Scale*	334	20.95	(7.12)	125	18.71	(7.75)	459	20.34	(7.36)
Profile of Mood States									
Tension-Anxiety*	338	6.65	(5.36)	120	4.07	(5.19)	458	5.97	(5.43)
Depression-Dejection*	334	5.55	(6.71)	119	3.07	(6.06)	453	4.90	(6.63)
Anger-Hostility*	335	7.59	(5.73)	119	3.56	(7.51)	454	6.53	(6.48)
Vigor-Activity	336	17.94	(6.47)	119	17.40	(7.28)	455	17.80	(6.69)
Fatigue-Inertia*	334	3.87	(4.68)	120	2.00	(3.98)	454	3.38	(4.58)
Confusion-Bewilderment*	336	4.84	(3.66)	119	3.13	(3.12)	455	4.39	(3.61)
Total Mood Disturbance*	331	10.62	(25.42)	119	-1.52	(24.64)	450	7.41	(25.75)
Cook-Medley Hostility Scale[a]									
Cynicism*	333	6.82	(3.57)	120	4.67	(3.53)	453	6.25	(3.68)
Hostile Attributions*	332	4.34	(2.83)	120	2.43	(2.48)	452	3.83	(2.86)
Hostile Affect*	335	1.88	(1.33)	120	1.03	1.13	455	1.65	(1.33)

Variable	MARCH			SUGAR			Total		
	n	*M (SD)*		*n*	*M (SD)*		*n*	*M (SD)*	
Aggressive Responding*	331	4.12	(1.88)	120	2.81	(1.53)	451	3.77	(1.88)
Social Avoidance*	336	1.56	(1.12)	120	0.93	(0.90)	456	1.40	(1.10)
Other*	332	2.55	(1.71)	120	1.89	(1.16)	452	2.38	(1.61)
Cynicism*	332	11.99	(6.06)	120	7.43	(5.80)	452	10.78	(6.32)
Paranoid Alienation*	334	4.21	(3.08)	120	2.09	(2.33)	454	3.65	(3.05)
Total Hostility Score*	332	21.19	(9.81)	120	13.76	(8.33)	452	19.22	(9.99)
STAI State Anxiety*	334	34.22	(8.32)	120	32.52	(7.26)	454	33.77	(8.08)
STAI Trait Anxiety*	334	34.32	(9.28)	121	30.69	(7.36)	455	33.36	(8.95)
STAXI Trait Anger									
Anger Reaction*	338	7.87	(2.65)	121	6.01	(2.57)	459	7.38	(2.75)
Anger Temperament*	338	6.02	(2.32)	121	4.95	(1.79)	459	5.74	(2.24)
Total Trait Anger*	338	16.70	(4.84)	121	13.47	(4.49)	459	15.85	(4.95)
STAXI Anger Expression									
Anger-In*	337	14.51	(3.98)	121	12.35	(3.47)	458	13.94	(3.96)
Anger-Out*	338	14.42	(3.55)	121	12.70	(3.15)	459	13.96	(3.53)
Anger-Control	338	23.26	(4.93)	121	22.97	(5.21)	459	23.18	(5.00)

[a] the first 6 subscales are defined by Barefoot et al. (1989), and the next 2 subscales are defined by Costa et al. (1986).
*$p < .05$, for MARCH and SUGAR subsample comparison.
For abbreviations, refer to Glossary of Abbreviations and Symbols.

Table 7

Psychological Distress Variables, Adjusted for Age, Gender, Ethnicity, and Smoking Status

Variable	MARCH			SUGAR			Total		
	n	adj. *M (SE)*		*n*	adj. *M (SE)*		*n*	adj. *M (SE)*	
Beck Depression Inventory									
Cognitive*	335	2.94	(0.20)	125	1.21	(0.35)	460	2.08	(0.20)
Somatic†	336	1.75	(0.12)	125	1.31	(0.21)	461	1.53	(0.12)
Total*	335	4.68	(0.29)	125	2.51	(0.50)	460	3.59	(0.28)
Perceived Stress Scale*	334	20.89	(0.41)	125	18.88	(0.69)	459	19.88	(0.39)
Profile of Mood States									
Tension-Anxiety*	338	6.65	(0.29)	120	4.09	(0.51)	458	5.37	(0.29)
Depression-Dejection*	334	5.38	(0.36)	119	3.55	(0.63)	453	4.46	(0.35)
Anger-Hostility*	335	7.51	(0.35)	119	3.77	(0.60)	454	5.64	(0.34)
Vigor-Activity	336	17.92	(0.37)	119	17.45	(0.65)	455	17.69	(0.36)
Fatigue-Inertia*	334	3.68	(0.24)	120	2.54	(0.42)	454	3.11	(0.24)
Confusion-Bewilderment*	336	4.74	(0.19)	119	3.40	(0.34)	455	4.07	(0.19)
Total Mood Disturbance*	331	10.12	(1.40)	119	-0.16	(2.43)	450	4.98	(1.35)
Cook-Medley Hostility Scale[a]									
Cynicism*	333	6.79	(0.19)	120	4.76	(0.33)	453	5.78	(0.19)
Hostile Attributions*	332	4.32	(0.15)	120	2.48	(0.26)	452	3.40	(0.14)
Hostile Affect*	335	1.86	(0.07)	120	1.07	(0.12)	455	1.47	(0.07)

Variable	MARCH			SUGAR			Total		
	n	*M (SD)*		*n*	*M (SD)*		*n*	*M (SD)*	
Aggressive Responding*	331	4.16	(0.10)	120	2.71	(0.17)	451	3.43	(0.10)
Social Avoidance*	336	1.56	(0.06)	120	0.95	(0.10)	456	1.25	(0.06)
Other*	332	2.58	(0.09)	120	1.83	(0.15)	452	2.20	(0.08)
Cynicism*	332	11.93	(0.32)	120	7.62	(0.56)	452	9.77	(0.31)
Paranoid Alienation*	334	4.27	(0.16)	120	1.93	(0.28)	454	3.10	(0.15)
Total Hostility Score*	332	21.18	(0.51)	120	13.79	(0.89)	452	17.48	(0.50)
STAI State Anxiety	334	34.16	(0.45)	120	32.68	(0.77)	454	33.42	(0.43)
STAI Trait Anxiety*	334	34.15	(0.49)	121	31.16	(0.84)	455	32.65	(0.47)
STAXI Trait Anger									
Anger Reaction*	338	7.88	(0.15)	121	5.98	(0.25)	459	6.93	(0.14)
Anger Temperament*	338	6.03	(0.12)	121	4.91	(0.21)	459	5.47	(0.12)
Total Trait Anger*	338	16.70	(0.26)	121	13.46	(0.46)	459	15.08	(0.26)
STAXI Anger Expression									
Anger-In*	337	14.53	(0.21)	121	12.31	(0.37)	458	13.42	(0.21)
Anger-Out*	338	14.33	(0.19)	121	12.93	(0.33)	459	13.63	(0.18)
Anger-Control	338	23.22	(0.28)	121	23.08	(0.48)	459	23.15	(0.27)

[a] the first 6 subscales are defined by Barefoot et al. (1989), and the next 2 subscales are defined by Costa et al. (1986).
*$p < .05$, †$p < .10$, for MARCH and SUGAR subsample comparison, controlling for age, gender, ethnicity, and smoking status.
For abbreviations, refer to Glossary of Abbreviations and Symbols.

Table 8

Correlation Matrix for Psychological Distress Variables

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. BDI tot	1													
2. BDI cog	.94*	1												
3. BDI som	.83*	.59*	1											
4. PSS	.44*	.40*	.38*	1										
5. POMS total	.55*	.51*	.46*	.57*	1									
6. POMS anx	.46*	.43*	.38*	.51*	.87*	1								
7. POMS dep	.50*	.48*	.40*	.47*	.89*	.75*	1							
8. POMS ang	.39*	.38*	.30*	.39*	.83*	.72*	.79*	1						
9. POMS vig	-.29*	-.26*	-.25*	-.38*	-.43*	-.21*	-.16*	-.04	1					
10. POMS fat	.47*	.42*	.44*	.42*	.82*	.71*	.72*	.66*	-.18*	1				
11. POMS conf	.50*	.45*	.45*	.51*	.86*	.75*	.77*	.64*	-.30*	.70*	1			
12. Ho total	.37*	.36*	.29*	.41*	.39*	.36*	.38*	.40*	-.03	.31*	.37*	1		
13. Ho cyn[a]	.33*	.32*	.26*	.35*	.31*	.27*	.33*	.32*	.01	.26*	.29*	.89*	1	
14. Ho att[a]	.33*	.32*	.28*	.37*	.38*	.33*	.38*	.41*	-.03	.30*	.37*	.88*	.70*	1

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14
15. Ho aff[a]	.35*	.36*	.26*	.39*	.42*	.39*	.34*	.40*	-.15*	.35*	.37*	.69*	.53*	.61*
16. Ho agg res[a]	.21*	.23*	.12*	.25*	.24*	.25*	.22*	.26*	-.01	.17*	.23*	.76*	.59*	.58*
17. Ho soc avo[a]	.27*	.25*	.22*	.31*	.32*	.33*	.29*	.32*	-.03	.28*	.29*	.66*	.49*	.55*
18. Ho other[a]	.23*	.21*	.20*	.29*	.19*	.19*	.19*	.20*	<-.01	.12*	.22*	.69*	.50*	.53*
19. Ho cyn[b]	.33*	.33*	.26*	.37*	.36*	.32*	.37*	.39*	.01	.31*	.34*	.96*	.93*	.85*
20. Ho par alien[b]	.39*	.39*	.29*	.42*	.40*	.39*	.35*	.38*	-.14*	.29*	.38*	.84*	.64*	.74*
21. STAI state	.46*	.44*	.38*	.47*	.57*	.53*	.43*	.42*	-.40*	.37*	.52*	.29*	.18*	.30*
22. STAI trait	.64*	.60*	.54*	.62*	.66*	.56*	.56*	.46*	-.42*	.49*	.60*	.42*	.34*	.39*
23. STAXI trait	.26*	.26*	.19*	.36*	.55*	.48*	.45*	.56*	-.21*	.39*	.46*	.42*	.31*	.42*
24. STAXI ang reac	.22*	.23*	.15*	.27*	.45*	.42*	.38*	.45*	-.09*	.38*	.39*	.35*	.25*	.36*
25. STAXI ang temp	.22*	.21*	.19*	.33*	.47*	.38*	.38*	.48*	-.25*	.29*	.39*	.33*	.24*	.32*
26. STAXI ang-in	.33*	.33*	.25*	.41*	.43*	.44*	.39*	.42*	-.09*	.32*	.40*	.49*	.38*	.45*
27. STAXI ang-out	.16*	.16*	.12*	.24*	.34*	.32*	.27*	.40*	-.09*	.23*	.29*	.36*	.26*	.34*
28. STAXI ang-con	-.22*	-.21*	-.19*	-.34*	-.31*	-.20*	-.19*	-.23*	.43*	-.12*	-.25*	-.14*	-.11*	-.12*

Variables	15	16	17	18	19	20	21	22	23	24	25	26	27	28
15. Ho aff[a]	1													
16. Ho agg res[a]	.43*	1												
17. Ho soc avo[a]	.44*	.45*	1											
18. Ho other[a]	.36*	.51*	.45*	1										
19. Ho cyn[b]	.65*	.69*	.58*	.56*	1									
20. Ho par alien[b]	.68*	.66*	.63*	.63*	.69*	1								
21. STAI state	.30*	.23*	.20*	.17*	.23*	.36*	1							
22. STAI trait	.38*	.28*	.29*	.27*	.37*	.46*	.58*	1						
23. STAXI trait	.46*	.35*	.26*	.20*	.38*	.45*	.38*	.45*	1					
24. STAXI ang reac	.39*	.29*	.21*	.15*	.32*	.34*	.31*	.37*	.86*	1				
25. STAXI ang temp	.36*	.29*	.22*	.18*	.28*	.40*	.33*	.39*	.84*	.48*	1			
26. STAXI ang-in	.45*	.37*	.34*	.33*	.43*	.51*	.29*	.45*	.50*	.45*	.39*	1		
27. STAXI ang-out	.32*	.32*	.23*	.22*	.32*	.38*	.25*	.24*	.56*	.41*	.51*	.45*	1	
28. STAXI ang-con	-.25*	-.07†	-.06	-.09*	-.10*	-.25*	-.31*	-.29*	-.25*	-.09*	-.31*	-.06	-.18*	1

[a] subscales defined by Barefoot et al. (1989). [b] subscales defined by Costa et al. (1986).
*$p < .05$. †$p < .10$.
For abbreviations, refer to Table 6 or 7.

Table 9

Correlation Matrix for All Variables in SEM Path Analyses

Variables	1	2	3	4	5	6	7	8	9	10	11
Psychological distress											
1. BDI tot	1										
2. PSS	.44*	1									
3. POMS anx	.46*	.51*	1								
4. POMS ang	.39*	.39*	.72*	1							
Restrained eating											
5. DEBQ res[a]	.17*	.16*	.06	.12†	1						
6. TFEQ res[a]	.15†	.08	.15*	.20*	.44*	1					
7. RS[a]	.36*	.07	.16*	.21*	.49*	.44*	1				
Emotional eating											
8. DEBQ emo[a]	.31*	.29*	.32*	.26*	.28*	.19*	.37*	1			
9. TFEQ emo[a]	.20*	.21*	.23*	.24*	.23*	.36*	.21*	.52*	1		
External eating											
10. DEBQ ext[a]	.19*	.13†	.15†	.10	.31*	.10	.31*	.42*	.21*	1	
11. TFEQ uncon[a]	.20*	.14†	.31*	.24*	.09	.34*	.16*	.31*	.58*	.40*	1

Variables	1	2	3	4	5	6	7	8	9	10	11
Central obesity											
12. Waist girth	-.02	.03	-.07†	-.08*	.01	.11	.36*	.17*	.08	.11	.03
Insulin sensitivity											
13. M	<-.01	-.02	.06†	.11*	-.08	-.09	-.19*	.09	<.01	-.07	-.07
Lipidemia											
14. TG	-.09*	-.06	-.06	-.11*	.10	.08	.19*	.06	.03	.15*	.11
15. TC:HDL-C	-.10*	-.03	-.06	-.14*	.11	.12†	.15†	.04	.15*	.14†	.10
Blood pressure											
16. DBP	-.09*	-.11*	-.13*	-.14*	-.16*	-.05	.06	.02	<-.01	-.12†	-.09
17. SBP	-.09*	-.05	-.08*	-.10*	-.25*	-.10	-.04	-.01	-.05	-.10	-.14†
Glucose tolerance											
18. $AUC_{Glucose}$[b]	.03	-.04	.05	.07†	-.16*	-.11	-.13†	-.04	-.11	-.06	-.16*

Variables	12	13	14	15	16	17	18
Central obesity							
12. Waist girth	1						
Insulin sensitivity							
13. M	-.55*	1					
Lipidemia							
14. TG	.39*	-.40*	1				
15. TC:HDL-C	.41*	-.35*	.71*	1			
Blood pressure							
16. DBP	.39*	-.24*	.25*	.25*	1		
17. SBP	.36*	-.19*	.22*	.20*	.69*	1	
Glucose tolerance							
18. $AUC_{Glucose}$ [b]	-.37*	.46*	-.41*	-.31*	-.27*	-.20*	1

[a] data for SUGAR participants only.
[b] sign of $AUC_{Glucose}$ values reversed so that a positive value reflects greater glucose tolerance.
*$p < .05$. †$p < .10$.
For abbreviations, refer to Glossary of Abbreviations and Symbols.

Table 10

Correlation Matrix for Cardiometabolic Risk Variables

Variables	1	2	3	4	5	6	7	8	9
1. BMI	1								
2. BSA	.64*	1							
3. Weight	.84*	.95*	1						
4. Height	.03	.78*	.56*	1					
5. Waist girth	.87*	.79*	.89*	.32*	1				
6. WHR	.47*	.54*	.55*	.32*	.76*	1			
7. Insulin sensitivity	-.55*	-.36*	-.47*	-.01	-.55*	-.39*	1		
8. Fasting insulin	.41*	.32*	.38*	.08†	.40*	.27*	-.47*	1	
9. Fasting glucose	.30*	.29*	.31*	.14*	.37*	.38*	-.26*	.31*	1

Variables	1	2	3	4	5	6	7	8	9
10. $AUC_{Insulin}$	.39*	.28*	.35*	.03	.39*	.28*	-.54*	.64*	.17*
11. $AUC_{Glucose}$[a]	-.27*	-.26*	-.28*	-.12*	-.37*	-.46*	.46*	-.25*	-.58*
12. Triglycerides	.29*	.26*	.29*	.10*	.39*	.49*	-.40*	.24*	.37*
13. LDL-C	.21*	.16*	.19*	.02	.26*	.31*	-.17*	.12*	.17*
14. HDL-C	-.24*	-.22*	-.23*	-.09*	-.30*	-.37*	.31*	-.25*	-.26*
15. Total cholesterol	.19*	.14*	.17*	.01	.26*	.31*	-.18*	.10*	.20*
16. TC:HDL-C	.31*	.27*	.30*	.09*	.41*	.50*	-.35*	.27*	.33*
17. DBP	.30*	.36*	.36*	.23*	.39*	.41*	-.24*	.11*	.24*
18. SBP	.25*	.41*	.38*	.33*	.36*	.35*	-.19*	.13*	.22*

Variables	10	11	12	13	14	15	16	17	18
10. $AUC_{Insulin}$	1								
11. $AUC_{Glucose}$ [a]	-.36*	1							
12. Triglycerides	.19*	-.41*	1						
13. LDL-C	.09*	-.13*	.31*	1					
14. HDL-C	-.17*	.23*	-.48*	-.15*	1				
15. Total cholesterol	.08*	-.18*	.46*	.92*	.05	1			
16. TC:HDL-C	.21*	-.31*	.71*	.67*	-.71*	.60*	1		
17. DBP	.14*	-.27*	.25*	.21*	-.08*	.24*	.25*	1	
18. SBP	.18*	-.20*	.22*	.13*	-.10*	.16*	.20*	.69*	1

[a] sign of $AUC_{Glucose}$ values reversed so that a positive value reflects greater glucose tolerance.
*$p < .05$. †$p < .10$.
For abbreviations, refer to Glossary of Abbreviations and Symbols.

Table 11

Correlation Matrix for Dietary Intake and Cardiometabolic Risk Variables for Total Sample (n = 464)

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Total cals	1												
2. Total cals/TEE[a]	.88*	1											
3. Total carbs	.90*	.79*	1										
4. %cals from carbs	-.08†	-.01	.31*	1									
5. Total carbs/RDA	.90*	.79*	1.00*	.31*	1								
6. Total protein	.78*	.67*	.58*	-.32*	.58*	1							
7. %cals from protein	-.19*	-.23*	-.35*	-.49*	-.35*	.38*	1						
8. Total protein/RDA	.66*	.63*	.49*	-.27*	.49*	.91*	.39*	1					
9. Total fat	.86*	.71*	.60*	-.43*	.60*	.67*	-.16*	.54*	1				
10. %cals from fat	.18*	.12†	-.15*	-.84*	-.15*	.15*	<.01	.09*	.59*	1			
11. Saturated fat	.79*	.65*	.54*	-.40*	.54*	.62*	-.14*	.51*	.92*	.54*	1		
12. Monounsaturated fat	.72*	.53*	.51*	-.36*	.51*	.58*	-.09*	.48*	.80*	.48*	.69*	1	
13. Polyunsaturated fat	.52*	.30*	.32*	-.35*	.32*	.42*	-.11*	.37*	.67*	.49*	.47*	.67*	1

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13
Central obesity													
14. Waist girth	.14*	-.06	.09*	-.15*	.09*	.10*	<.01	-.24*	.16*	.14*	.15*	.11*	.04
Insulin sensitivity													
15. M	.04	.07	.10*	.16*	.10*	.05	-.04	.25*	-.06	-.16*	-.05	-.08†	-.06
Lipidemia													
16. TG	.07†	-.01	.09*	.02	.09*	.09*	.08*	-.05	.01	-.07†	.02	.01	-.01
17. TC:HDL-C	.05	.05	.07†	<-.01	.07†	.09*	.09*	-.05	-.01	-.05	<-.01	-.05	-.04
Blood pressure													
18. DBP	.15*	.13†	-.13*	-.05	.13*	.14*	<.01	-.03	.11*	.02	.13*	.06	-.04
19. SBP	.23*	.17*	.21*	-.06†	.21*	.21*	.01	.05	.18*	.04	.17*	.12*	.04
Glucose tolerance													
20. $AUC_{Glucose}$ [b]	.03	.02	.05	.09*	.05*	.03	-.05	.15*	.02	-.04	.01	.03	.06†

[a] data for SUGAR participants only.
[b] sign of $AUC_{Glucose}$ values reversed so that a positive value reflects greater glucose tolerance.
*$p < .05$. †$p < .10$.
For abbreviations, refer to Glossary of Abbreviations and Symbols.

Table 12

Correlation Matrix for Dietary Intake and Cardiometabolic Risk, Psychological Distress, and Eating Style Variables for the SUGAR Subsample (n = 125)

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Total cals	1												
2. Total cals/TEE	.88*	1											
3. Total carbs	.87*	.79*	1										
4. %cals from carbs	-.07	-.01	.37*	1									
5. Total carbs/RDA	.87*	.79*	1.00*	.37*	1								
6. Total protein	.79*	.67*	.57*	-.28*	.57*	1							
7. %cals from protein	-.23*	-.23*	-.36*	-.41*	-.36*	.34*	1						
8. Total protein/RDA	.63*	.63*	.44*	-.26*	.44*	.89*	.41*	1					
9. Total fat	.83*	.71*	.49*	-.50*	.49*	.65*	-.20*	.50*	1				
10. %cals from fat	.18*	.12†	-.23*	-.90*	-.23*	.14†	-.03	.09	.64*	1			
11. Saturated fat	.76*	.65*	.43*	-.45*	.43*	.60*	-.17*	.50*	.92*	.57*	1		
12. Monounsaturated fat	.60*	.53*	.35*	-.41*	.35*	.54*	-.10	.38*	.68*	.49*	.61*	1	
13. Polyunsaturated fat	.37*	.30*	.15†	-.40*	.15†	.28*	-.15*	.18*	.55*	.51*	.32*	.52*	1

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13
Central obesity													
14. Waist girth	.12†	-.06	.12†	.01	.12†	.05	-.08	-.34*	.10	.03	.05	.17*	.10
Insulin sensitivity													
15. M	.04	.07	.09	.14†	.09	.07	-.01	.29*	-.04	-.15*	.01	-.11	-.11
Lipidemia													
16. TG	.01	-.01	.08	.14†	.08	-.03	-.02	-.20*	-.05	-.15†	-.08	-.06	-.04
17. TC:HDL-C	.09	.05	.14†	.07	.14†	.01	-.07	-.17*	.03	-.04	<-.01	-.07	.05
Blood pressure													
18. DBP	.18*	.13†	.23*	.05	.23*	.14†	-.01	-.01	.06	-.05	.06	.09	-.02
19. SBP	.26*	.17*	.29*	.03	.29*	.21*	-.03	.04	.14†	-.02	.14†	.12†	.08
Glucose tolerance													
20. $AUC_{Glucose}$ [a]	-.05	.02	-.06	.07	-.06	-.04	-.08	.09	-.02	-.04	<-.01	.01	.09

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13
Psychological distress													
21. BDI tot	.05	.03	.08	.06	.08	.04	-.05	-.02	.01	-.04	.02	.06	-.01
22. PSS	-.11	-.20*	-.07	.09	-.07	-.13†	-.05	-.11	-.11	-.08	-.09	-.04	-.06
23. POMS anx	.02	-.03	.02	.02	.02	.03	-.03	.02	.02	-.01	.02	.06	.12
24. POMS ang	.01	-.03	<-.01	-.03	<-.01	.02	.01	.03	.03	.03	.02	.02	.10
Restrained eating													
25. DEBQ res	-.17*	-.19*	-.19*	-.11	-.19*	-.05	.16*	-.06	-.12†	.04	-.19*	-.07	-.05
26. TFEQ res	-.14†	-.20*	-.16*	-.15†	-.16*	-.05	.16*	-.11	-.07	.09	-.11	-.10	-.04
27. RS	-.13†	-.21*	-.09	.04	-.09	-.08	.05	-.22*	-.13†	-.07	-.12†	-.10	-.07
Emotional eating													
28. DEBQ emo	.05	.06	.08	.08	.08	.06	<.01	.03	-.02	-.09	.01	<-.01	-.09
29. TFEQ emo	-.01	.02	.03	.02	.03	-.02	.05	-.01	-.04	-.05	.01	-.08	-.09
External eating													
30. DEBQ ext	.15*	.20*	.14†	.08	.14†	.13†	-.08	.08	.11	-.05	.12†	.13†	<-.01
31. TFEQ uncon	-.07	-.03	-.01	.10	-.01	-.06	.04	-.06	-.10	.13†	-.09	-.08	<.01

[a] sign of $AUC_{Glucose}$ values reversed so that a positive value reflects greater glucose tolerance.
*$p < .05$. †$p < .10$.
For abbreviations, refer to Glossary of Abbreviations and Symbols.

Table 13

Final Model Factor Loadings and Direct Effects

Parameter	Unstandardized estimate	*SE*	Standardized estimate	*p*
Factor Loadings				
Distress → BDI	1.000	.000	.589	< .001
Distress → PSS	.828	.240	.478	< .001
Distress → POMS anx	.715	.161	.617	< .01
Distress → POMS ang	.859	.221	.513	< .001
Restrained → DEBQ res	1.000	.000	.471	< .001
Restrained → TFEQ res	1.068	.213	.440	< .001
Restrained → RS	2.010	.334	1.000[a]	< .001
Emotional → DEBQ emo	1.000	.000	.976	< .001
Emotional → TFEQ emo	.686	.153	.537	< .001
External → DEBQ ext	1.000	.000	.727	< .001
External → TFEQ uncon	.840	.211	.583	< .001
Direct Effects				
Distress → Restrained	.477	.159	.451	< .01
Distress → Emotional	1.135	.298	.622	< .001
Distress → External	.759	.244	.554	< .01
Restrained → Waist	1.133	.288	.421	< .001
Waist → Insulin sensitivity	-.295	.035	-.588	< .001

[a] value resulting from fixing error variance of RS from a negative value to 0.
For abbreviations, refer to Glossary of Abbreviations and Symbols.

Table 14

Proportion of Variance of Latent and Observed Variables Explained by Final Model

Variable	R^2	%
Latent Variables		
Distress	.037	3.7%
Restrained	.245	24.5%
Emotional	.385	38.5%
External	.335	33.5%
Observed Variables		
BDI	.347	34.7%
PSS	.228	22.8%
POMS anx	.381	38.1%
POMS ang	.263	26.3%
DEBQ res	.222	22.2%
TFEQ res	.194	19.4%
RS	1.000[a]	100.0%[a]
DEBQ emo	.953	95.3%
TFEQ emo	.288	28.8%
DEBQ ext	.529	52.9%
TFEQ uncon	.339	33.9%
Waist	.224	22.4%
Insulin sensitivity	.392	39.2%

[a] value resulting from fixing error variance of RS from a negative value to 0.
For abbreviations, refer to Glossary of Abbreviations and Symbols.

Table 15

Effects of Covariates on Model Variables

Parameter	Unstandardized estimate	*SE*	Standardized estimate	*p*
Age → Distress	-.058	.064	-.111	n.s.
Age → Restrained	-.041	.050	-.073	n.s.
Age → Emotional	.079	.088	.082	n.s.
Age → External	-.017	.081	-.023	n.s.
Age → Waist	.122	.124	.082	n.s.
Age → Insulin sensitivity	-.089	.055	-.118	n.s.
Gender → Distress[a]	-.103	.077	-.160	n.s.
Gender → Restrained[a]	.138	.065	.202	< .05
Gender → Emotional[a]	.223	.111	.190	< .05
Gender → External[a]	.197	.108	.223	.067
Gender → Waist[a]	-.425	.150	-.231	< .01
Gender → Insulin sensitivity[a]	-.111	.067	-.120	.099
Education → Distress	.035	.066	.063	n.s.
Education → Restrained	.058	.052	.098	n.s.
Education → Emotional	.001	.091	.001	n.s.
Education → External	-.025	.083	-.033	n.s.
Education → Waist	-.136	.129	-.086	n.s.
Education → Insulin sensitivity	-.049	.057	-.062	n.s.
Physical activity → Distress	-.002	.061	-.004	n.s.
Physical activity → Restrained	.004	.047	.009	n.s.
Physical activity → Emotional	-.044	.082	-.052	n.s.
Physical activity → External	-.065	.074	-.102	n.s.
Physical activity → Waist	-.210	.113	-.160	.063
Physical activity → Insulin sensitivity	.066	.051	.101	n.s.

[a] gender was dummy coded such that 1 = female and 0 = male; thus, positive (vs. negative) estimates reflect an effect of being female (vs. male) on the dependent variable.
For abbreviations, refer to Glossary of Abbreviations and Symbols.

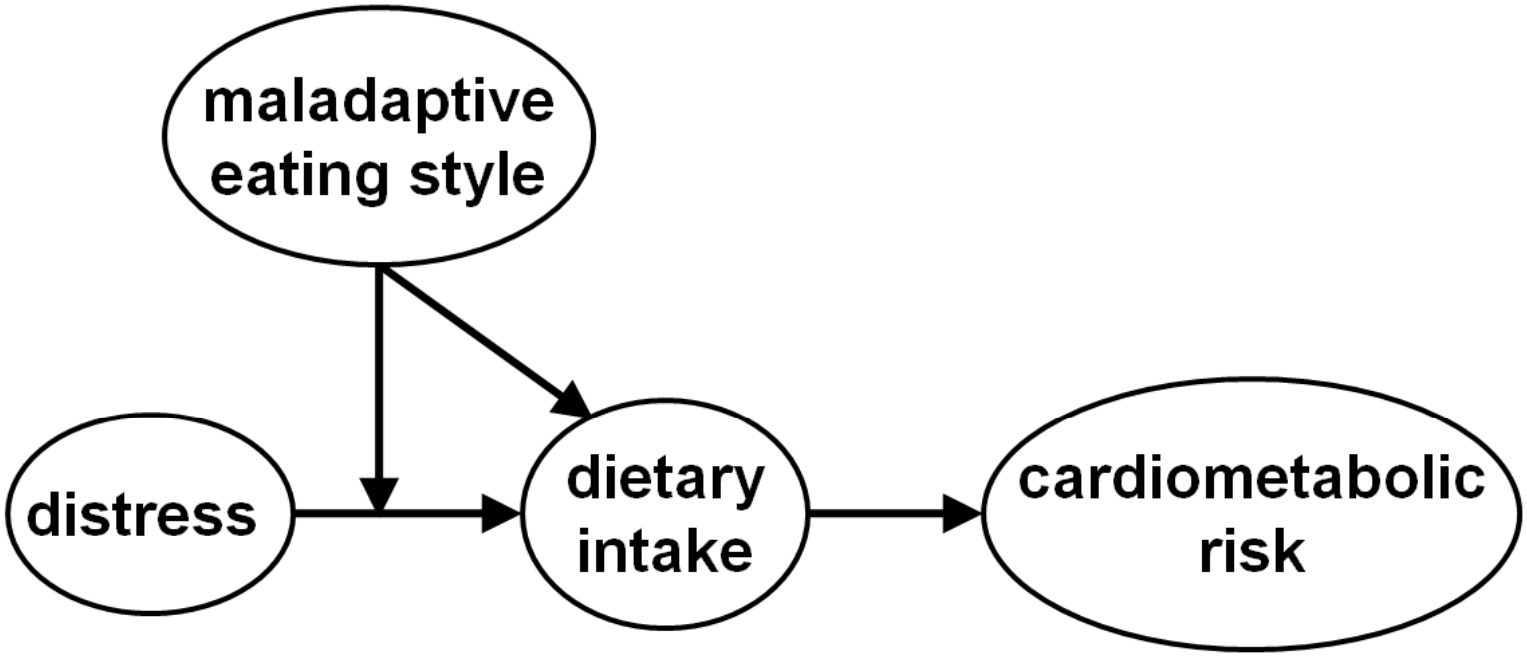

Figure 1. Proposed final model.

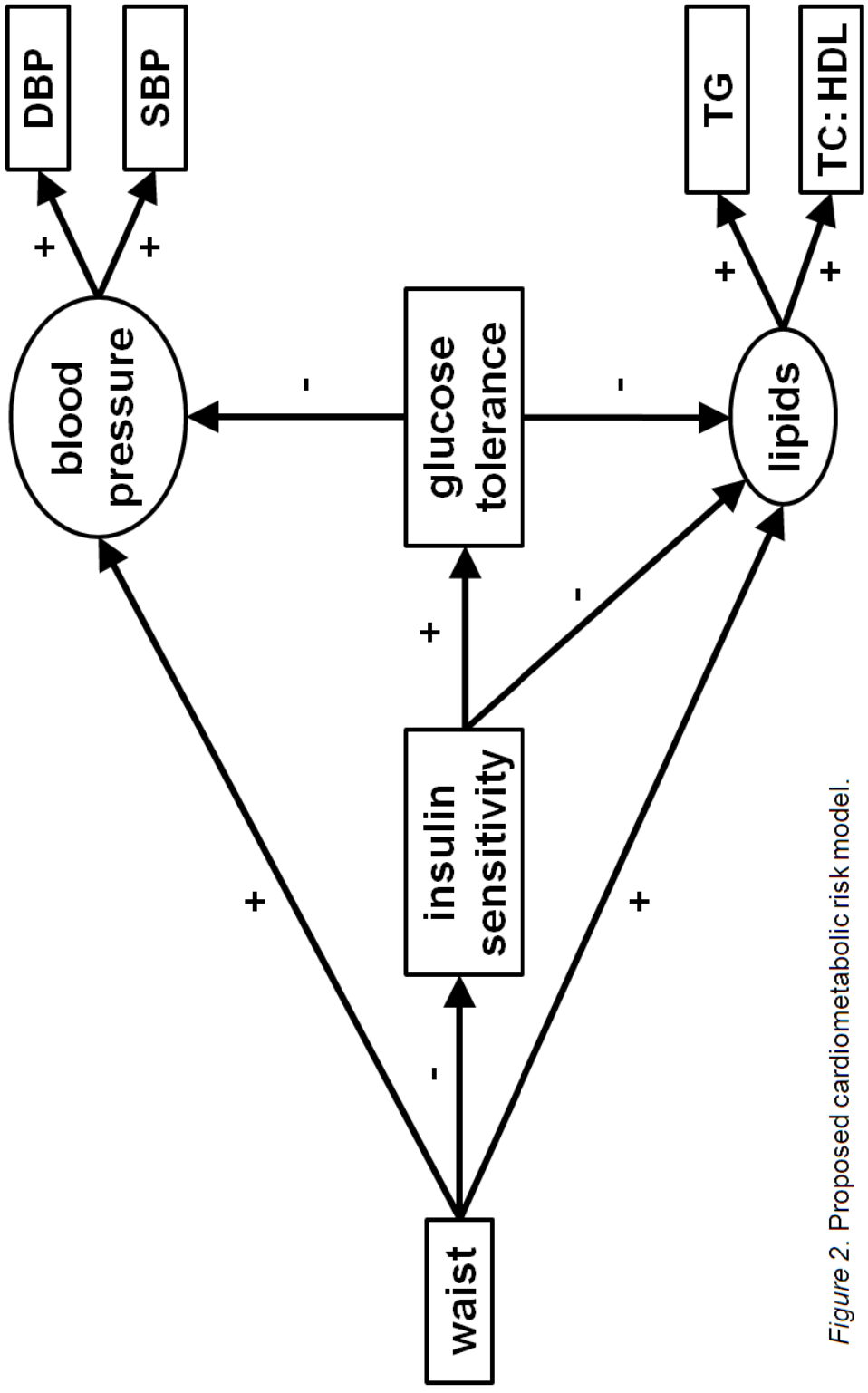

Figure 2. Proposed cardiometabolic risk model.

Waist = waist girth. Insulin sensitivity = M. Glucose tolerance = area under the curve for glucose during the oral glucose tolerance test (sign reversed). TG = triglycerides. TC:HDL = total cholesterol-to-HDL-C ratio. DBP = diastolic blood pressure. SBP = systolic blood pressure.

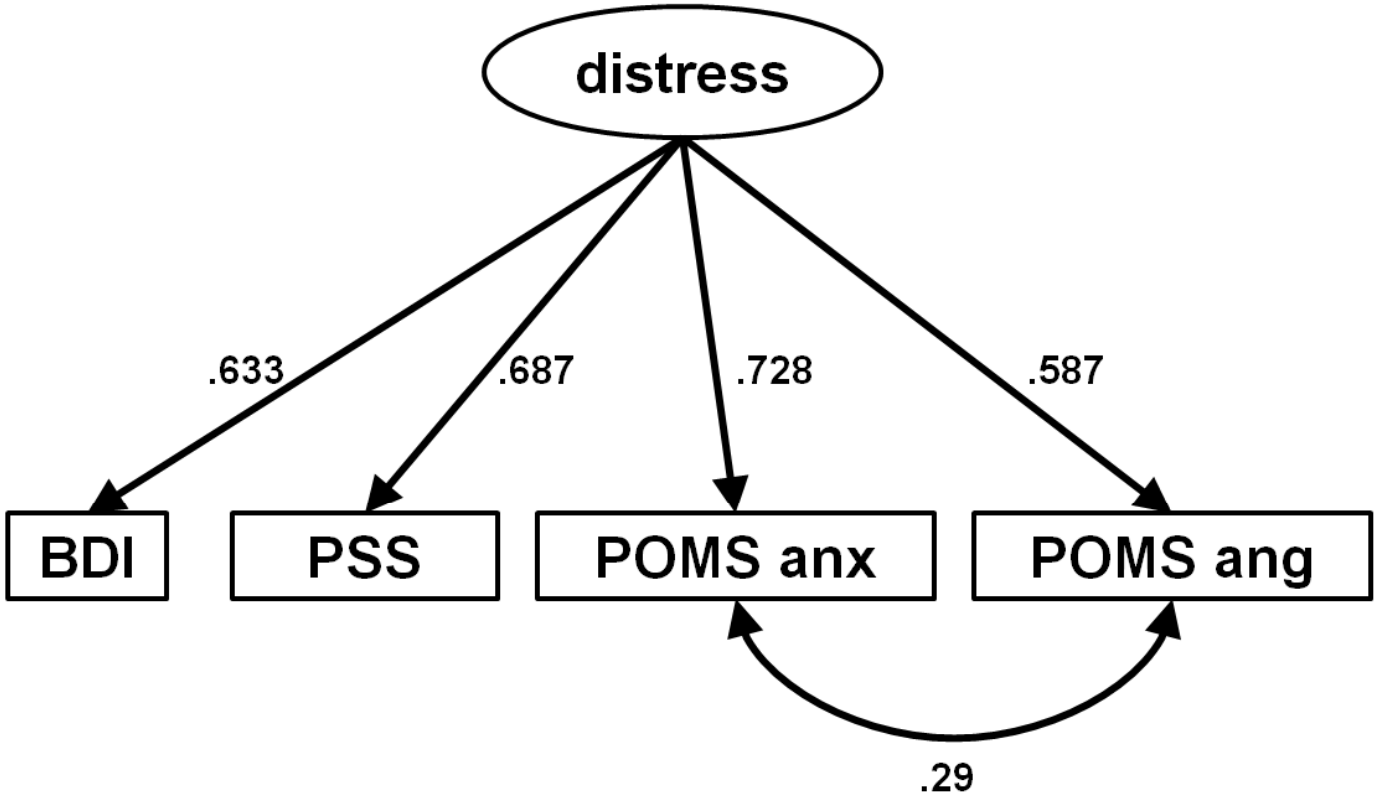

Figure 3. Distress factor model (n = 464). Standardized parameter estimates reported.

Model fit indices: χ^2 (1) = 0.976, p = .32; CFI = 1.000; RMSEA < .001; SRMR = .006.
BDI = Beck Depression Inventory. PSS = Perceived Stress Scale.
POMS anx = Profile of Mood States (POMS) Tension-Anxiety subscale.
POMS ang = POMS Anger-Hostility subscale.

——— p < .05
– – – p = n.s.

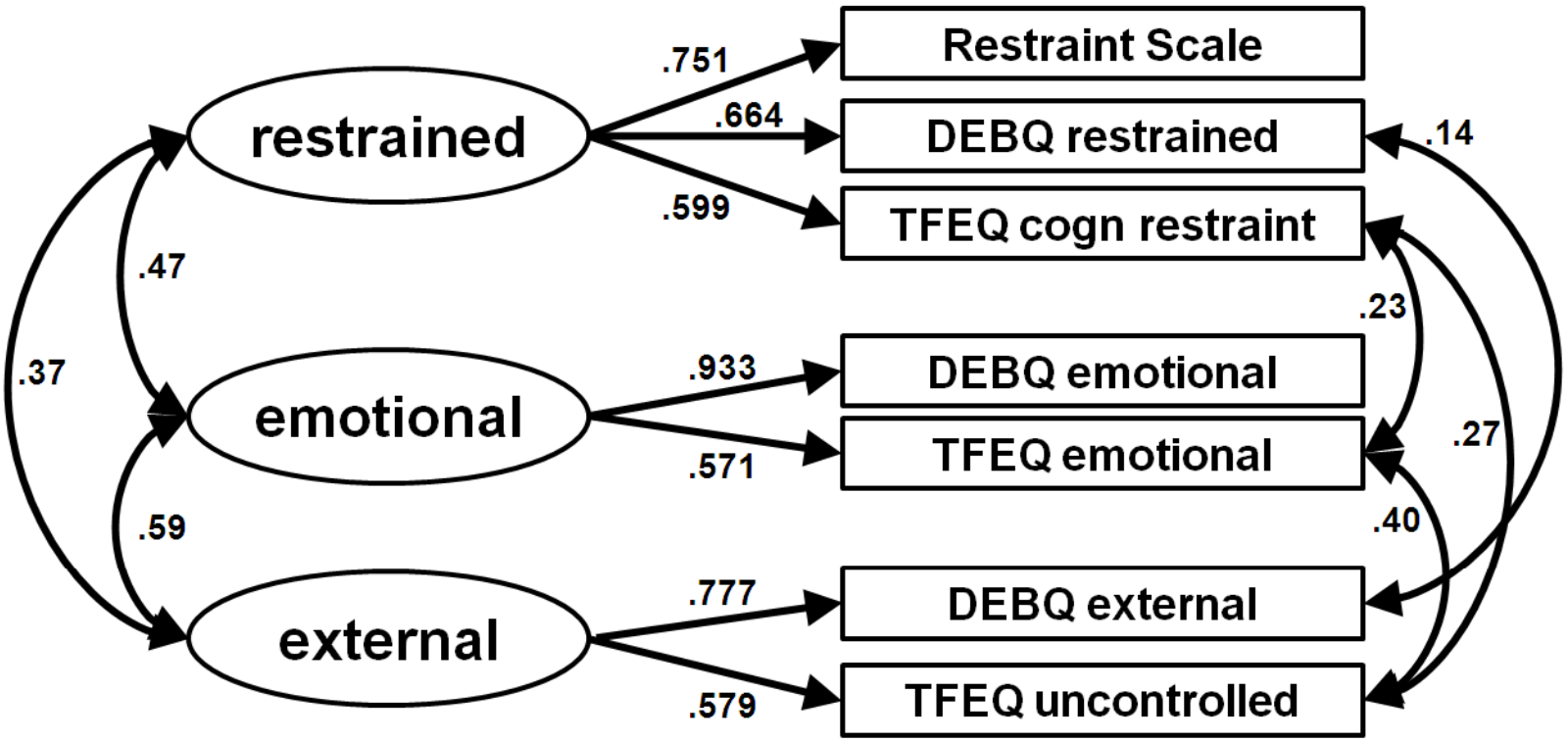

Figure 4. Eating style factor model (n = 125). Standardized parameter estimates reported.

Model fit indices: χ^2 (7) = 7.629, p = .37; CFI = .997; RMSEA = .027; SRMR = .035.
DEBQ = Dutch Eating Behavior Questionnaire. TFEQ = Three-Factor Eating Questionnaire – Revised 18-item version.

——— $p < .05$
– – – p = n.s.

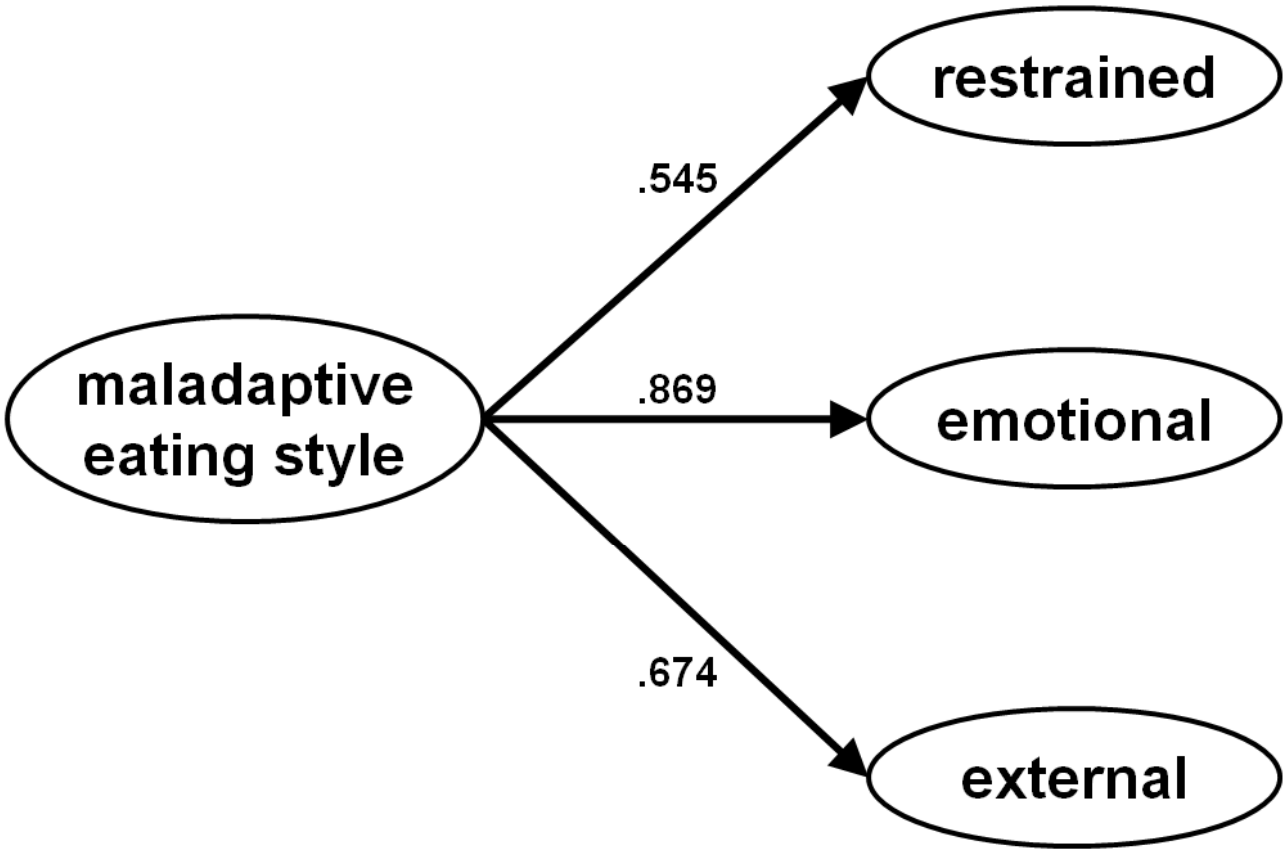

Figure 5. Eating style factor model with second-order eating style factor included (*n* = 125). Standardized factor loadings reported. Refer to Figure 4 for additional parameter estimates of the first-order eating style factors.

Model fit indices: χ^2 (7) = 7.629, *p* = .37; CFI = .997; RMSEA = .027; SRMR = .035.

p < .05
p = n.s.

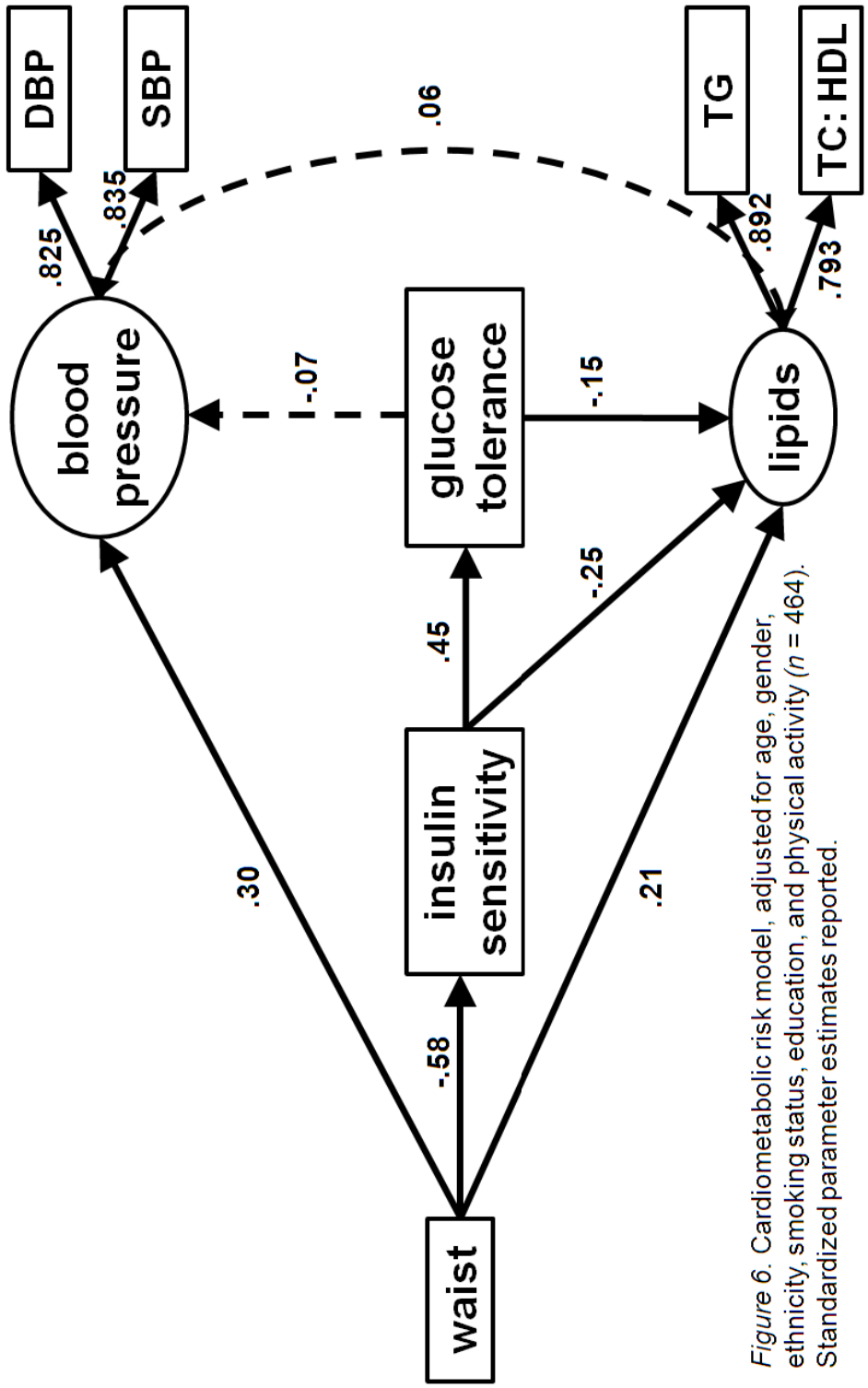

Figure 6. Cardiometabolic risk model, adjusted for age, gender, ethnicity, smoking status, education, and physical activity (n = 464). Standardized parameter estimates reported.

Model fit indices: χ^2 (27) = 53.638, p = .002; CFI = .981; RMSEA = .046; SRMR = .017. Waist = waist girth. Insulin sensitivity = M. Glucose tolerance = area under the curve for glucose during the oral glucose tolerance test (sign reversed). TG = triglycerides. TC:HDL = total cholesterol-to-HDL-C ratio. DBP = diastolic blood pressure. SBP = systolic blood pressure.

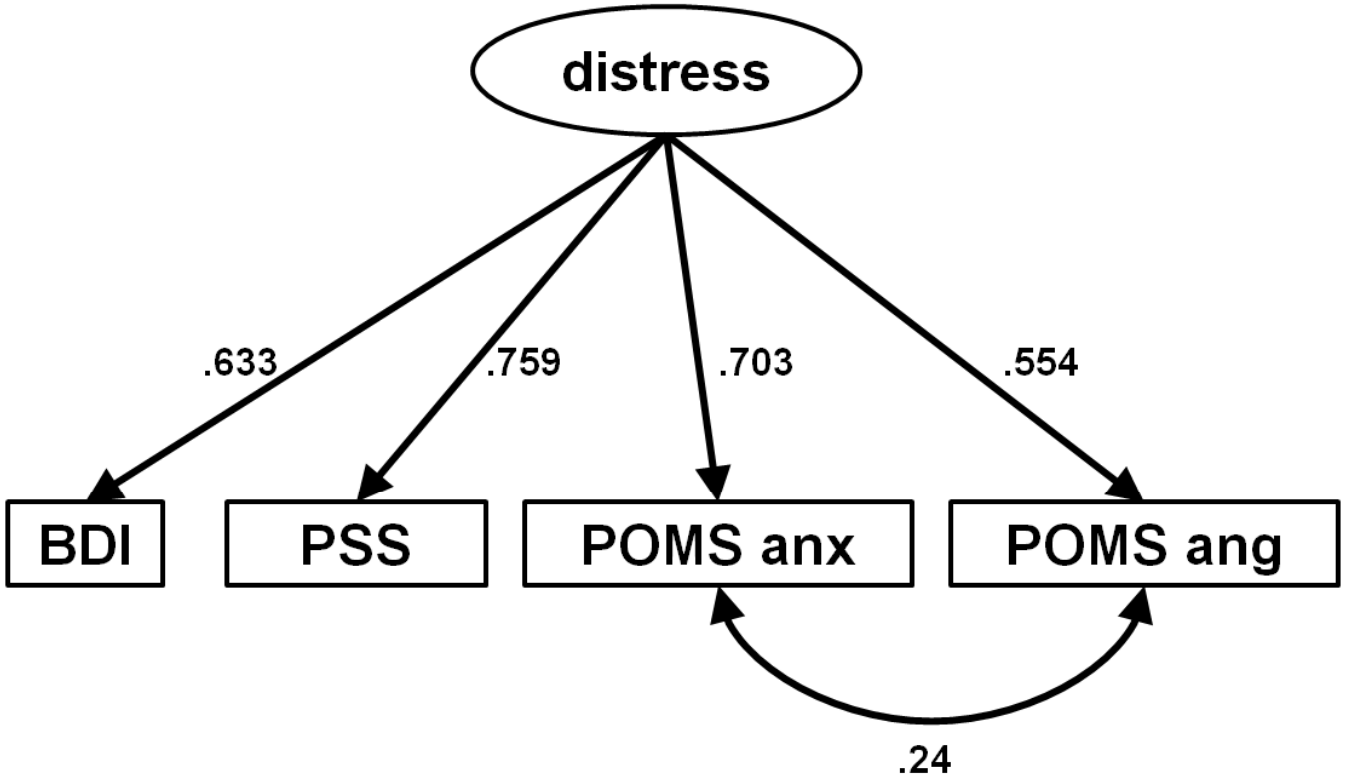

Figure 7. Distress factor model for the MARCH subsample (*n* = 339). Standardized parameter estimates reported.

Model fit indices: χ^2 (1) = 0.705, *p* = .40; CFI = 1.000; RMSEA < .001; SRMR = .006.
BDI = Beck Depression Inventory. PSS = Perceived Stress Scale.
POMS anx = Profile of Mood States (POMS) Tension-Anxiety subscale.
POMS ang = POMS Anger-Hostility subscale.

p < .05
p = n.s.

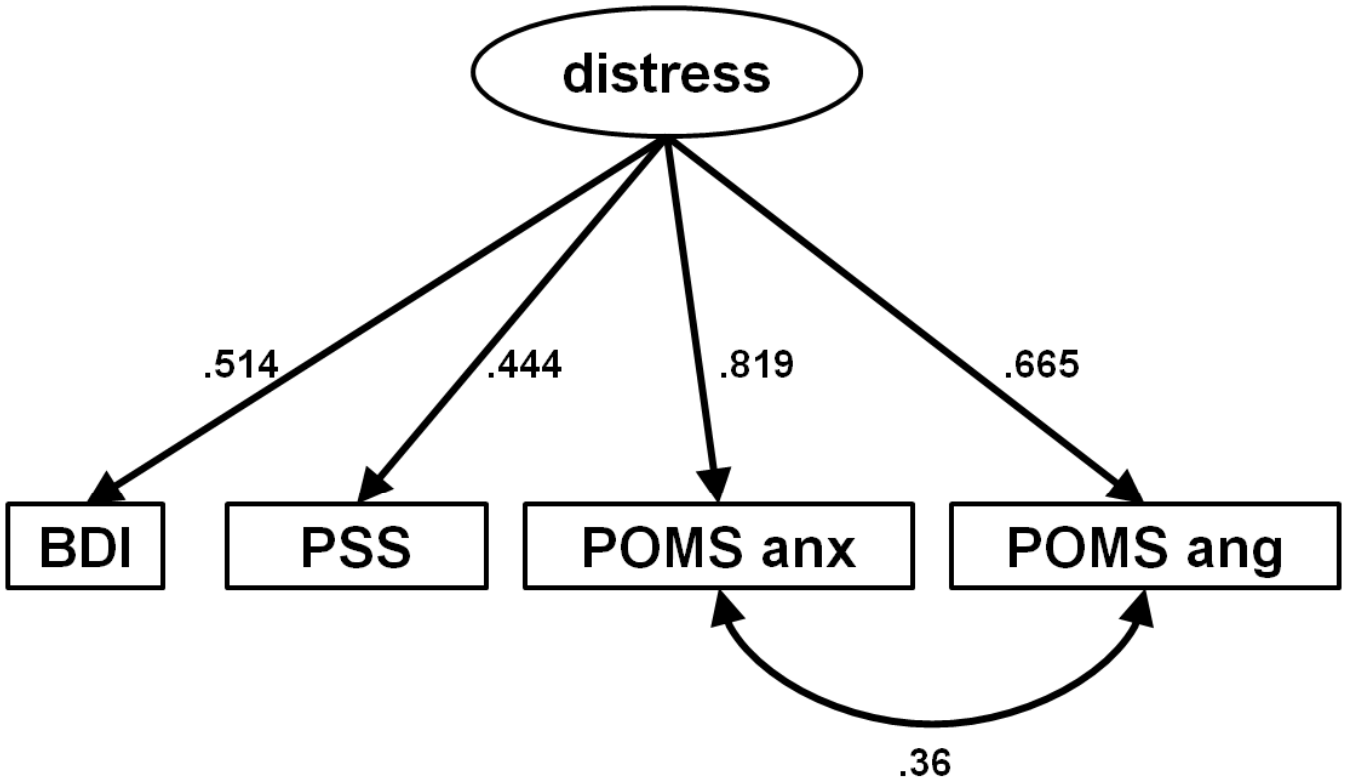

Figure 8. Distress factor model for the SUGAR subsample (n = 125). Standardized parameter estimates reported.

Model fit indices: χ^2 (1) = 0.023, p = .88; CFI = 1.000; RMSEA < .001; SRMR = .002.
BDI = Beck Depression Inventory. PSS = Perceived Stress Scale.
POMS anx = Profile of Mood States (POMS) Tension-Anxiety subscale.
POMS ang = POMS Anger-Hostility subscale.

p < .05
p = n.s.

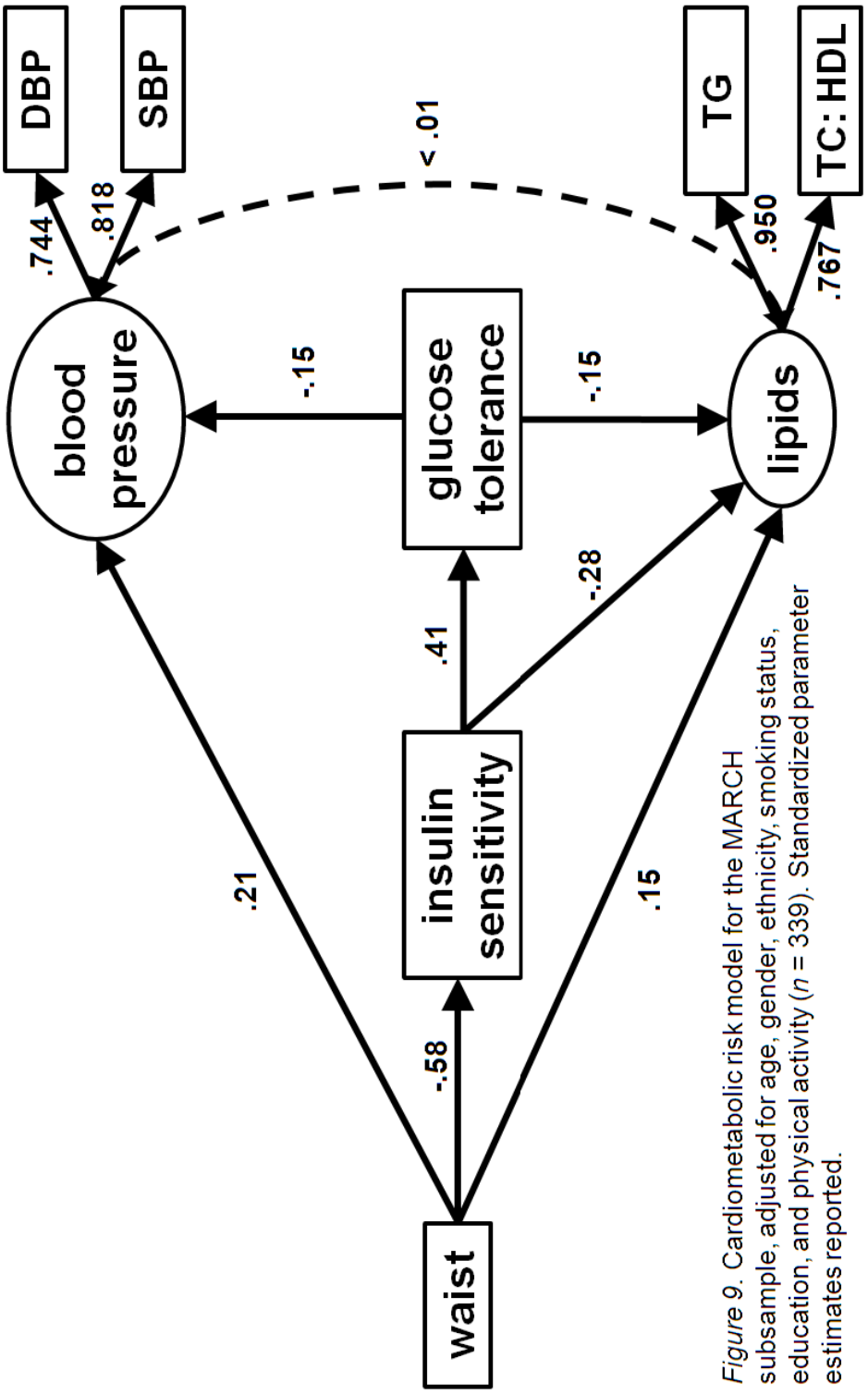

Figure 9. Cardiometabolic risk model for the MARCH subsample, adjusted for age, gender, ethnicity, smoking status, education, and physical activity (n = 339). Standardized parameter estimates reported.

Model fit indices: χ^2 (27) = 33.728, p = .17; CFI = .993; RMSEA = .027; SRMR = .016. Waist = waist girth. Insulin sensitivity = M. Glucose tolerance = area under the curve for glucose during the oral glucose tolerance test (sign reversed). TG = triglycerides. TC:HDL = total cholesterol-to-HDL-C ratio. DBP = diastolic blood pressure. SBP = systolic blood pressure.

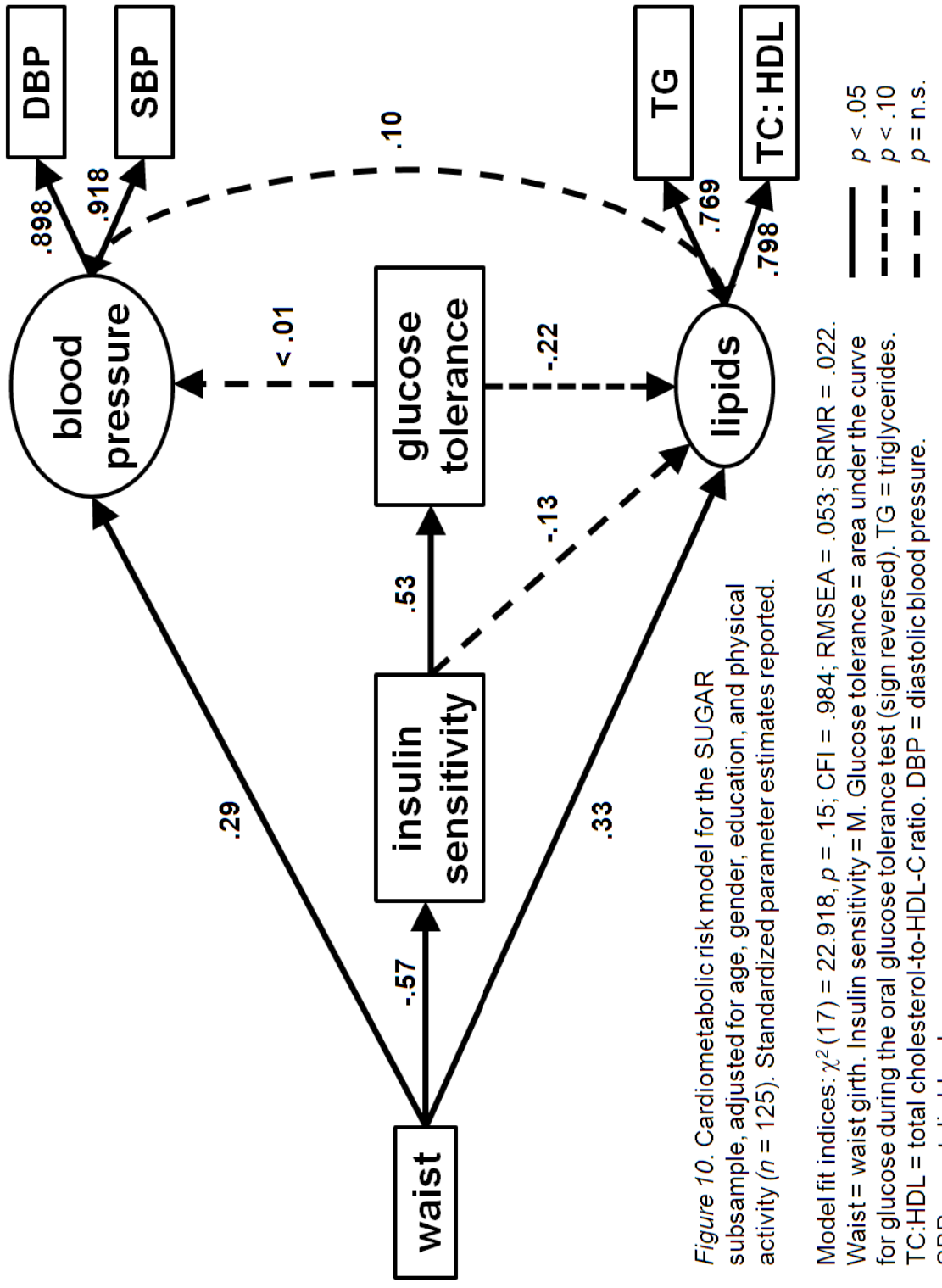

Figure 10. Cardiometabolic risk model for the SUGAR subsample, adjusted for age, gender, education, and physical activity (n = 125). Standardized parameter estimates reported.

Model fit indices: $\chi^2(17) = 22.918$, $p = .15$; CFI = .984; RMSEA = .053; SRMR = .022. Waist = waist girth. Insulin sensitivity = M. Glucose tolerance = area under the curve for glucose during the oral glucose tolerance test (sign reversed). TG = triglycerides. TC:HDL = total cholesterol-to-HDL-C ratio. DBP = diastolic blood pressure. SBP = systolic blood pressure.

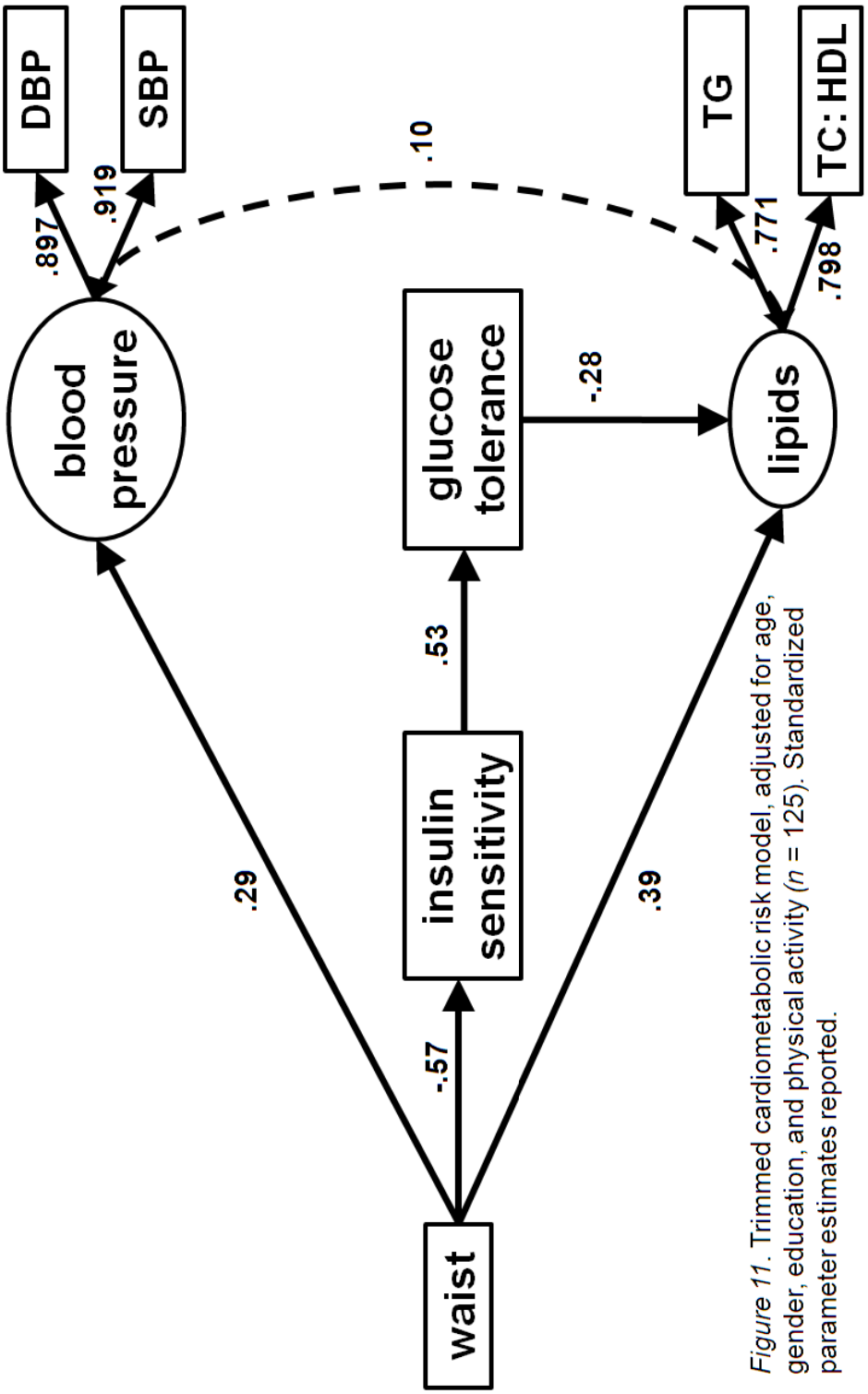

Figure 11. Trimmed cardiometabolic risk model, adjusted for age, gender, education, and physical activity (n = 125). Standardized parameter estimates reported.

Model fit indices: χ^2 (19) = 24.115, p = .19; CFI = .986; RMSEA = .046; SRMR = .024.
Waist = waist girth. Insulin sensitivity = M. Glucose tolerance = area under the curve for glucose during the oral glucose tolerance test (sign reversed). TG = triglycerides. TC:HDL = total cholesterol-to-HDL-C ratio. DBP = diastolic blood pressure. SBP = systolic blood pressure.

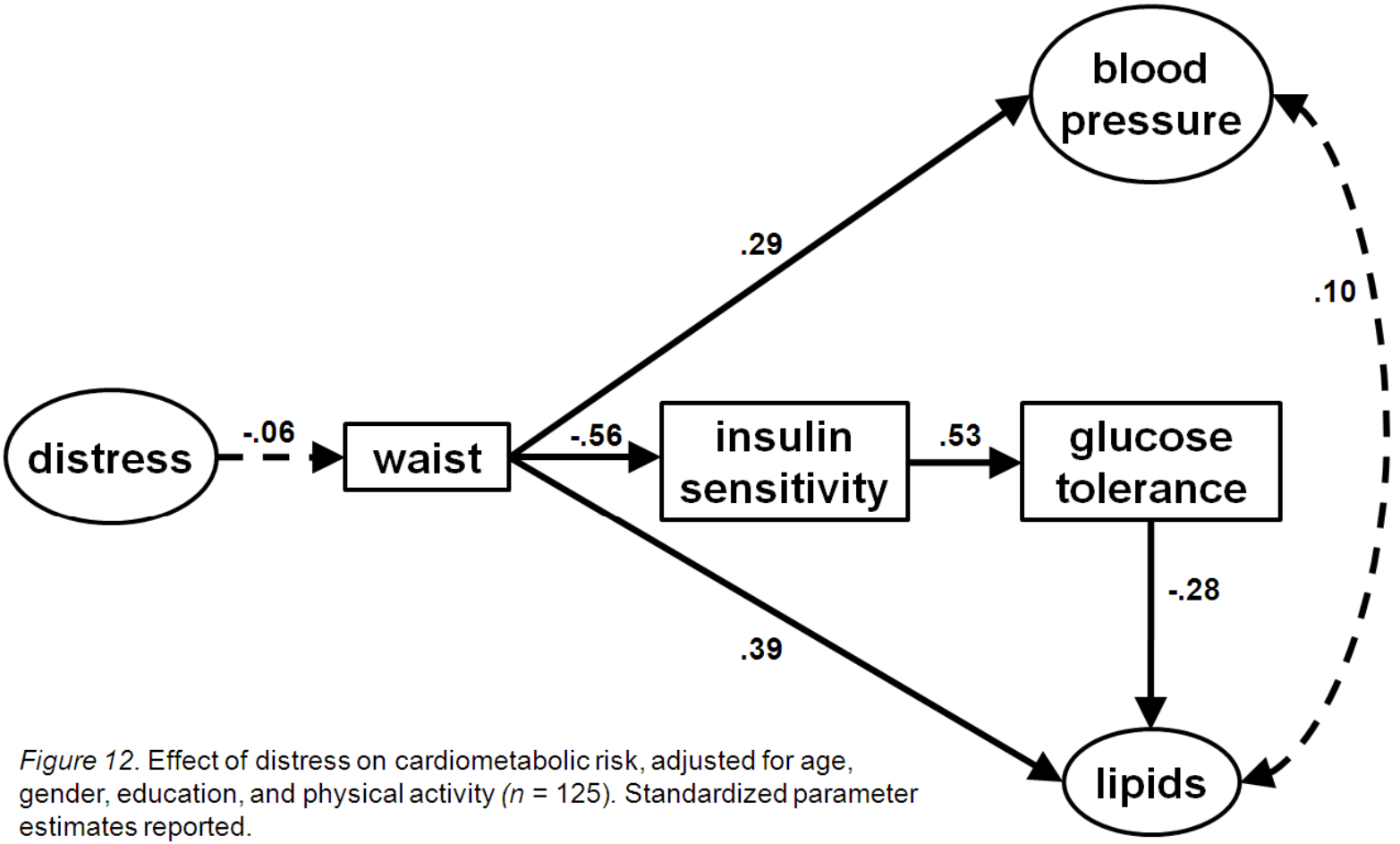

Figure 12. Effect of distress on cardiometabolic risk, adjusted for age, gender, education, and physical activity *(n* = 125). Standardized parameter estimates reported.

Model fit indices: χ^2 (59) = 73.160, *p* = .10; CFI = .977; RMSEA = .044; SRMR = .051. Waist = waist girth. Insulin sensitivity = M. Glucose tolerance = area under the curve for glucose during the oral glucose tolerance test (sign reversed). Refer to Figure 11 for details about the blood pressure and lipids factors.

p < .05
p = n.s

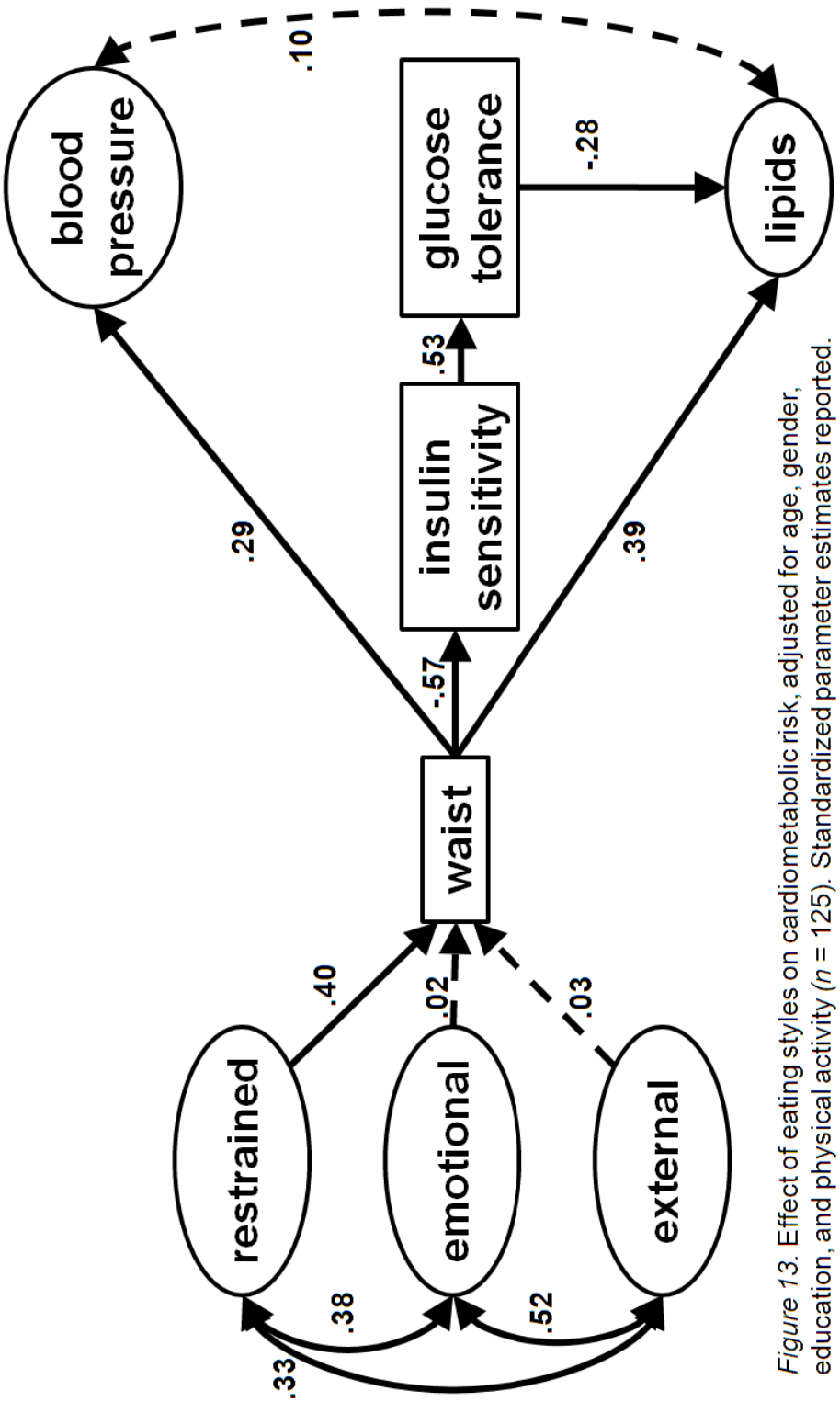

Figure 13. Effect of eating styles on cardiometabolic risk, adjusted for age, gender, education, and physical activity (n = 125). Standardized parameter estimates reported.

Model fit indices: χ^2 (88) = 107.658, p = .08; CFI = .969; RMSEA = .042; SRMR = .055. Waist = waist girth. Insulin sensitivity = M. Glucose tolerance = area under the curve for glucose during the oral glucose tolerance test (sign reversed). Refer to Figures 4 and 11 for details about the restrained, emotional, external, blood pressure, and lipids factors.

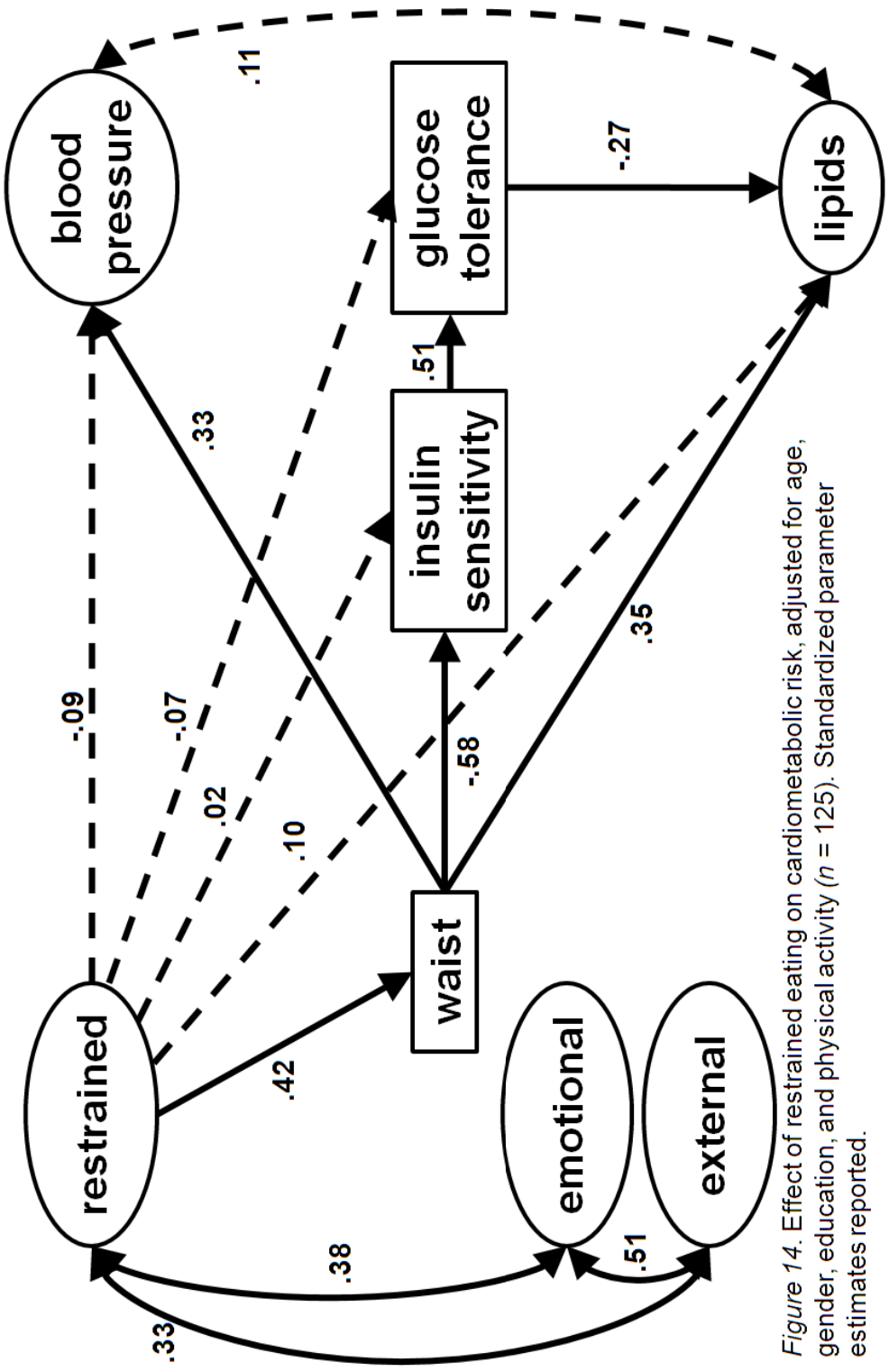

Figure 14. Effect of restrained eating on cardiometabolic risk, adjusted for age, gender, education, and physical activity (n = 125). Standardized parameter estimates reported.

Model fit indices: χ^2 (82) = 100.653, p = .08; CFI = .971; RMSEA = .043; SRMR = .047. Waist = waist girth. Insulin sensitivity = M. Glucose tolerance = area under the curve for glucose during the oral glucose tolerance test (sign reversed). Refer to Figures 4 and 11 for details about the restrained, emotional, external, blood pressure, and lipids factors.

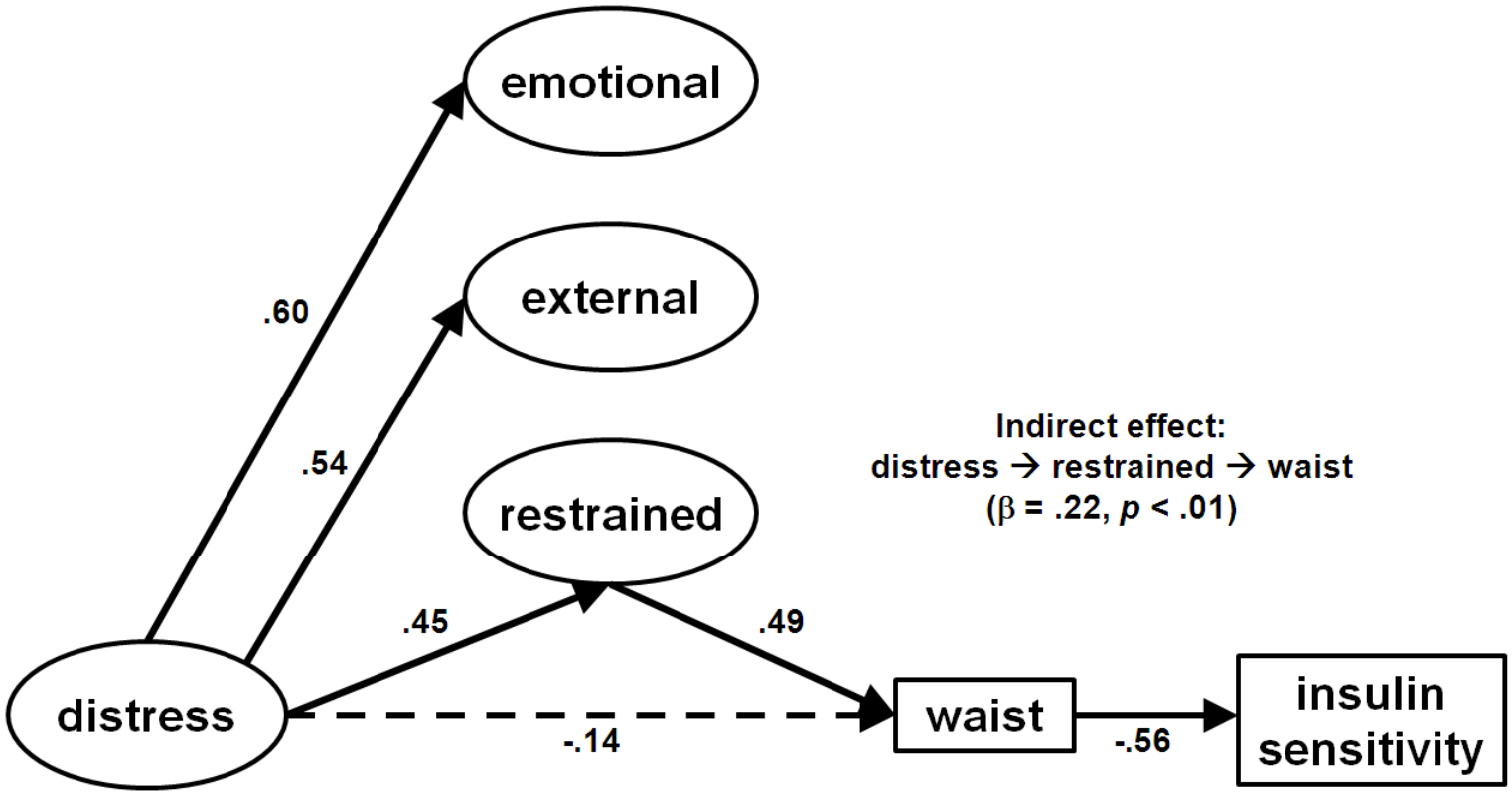

Figure 15. Effects of distress and eating styles on cardiometabolic risk, adjusted for age, gender, education, and physical activity *(n* = 125). Standardized parameter estimates reported.

Model fit indices: χ^2 (81) = 93.949, *p* = .15; CFI = .978; RMSEA = .036; SRMR = .060. Waist = waist girth. Insulin sensitivity = M. Refer to Figures 3 and 4 for details about the distress, emotional, external, and restrained factors.

p < .05
p = n.s

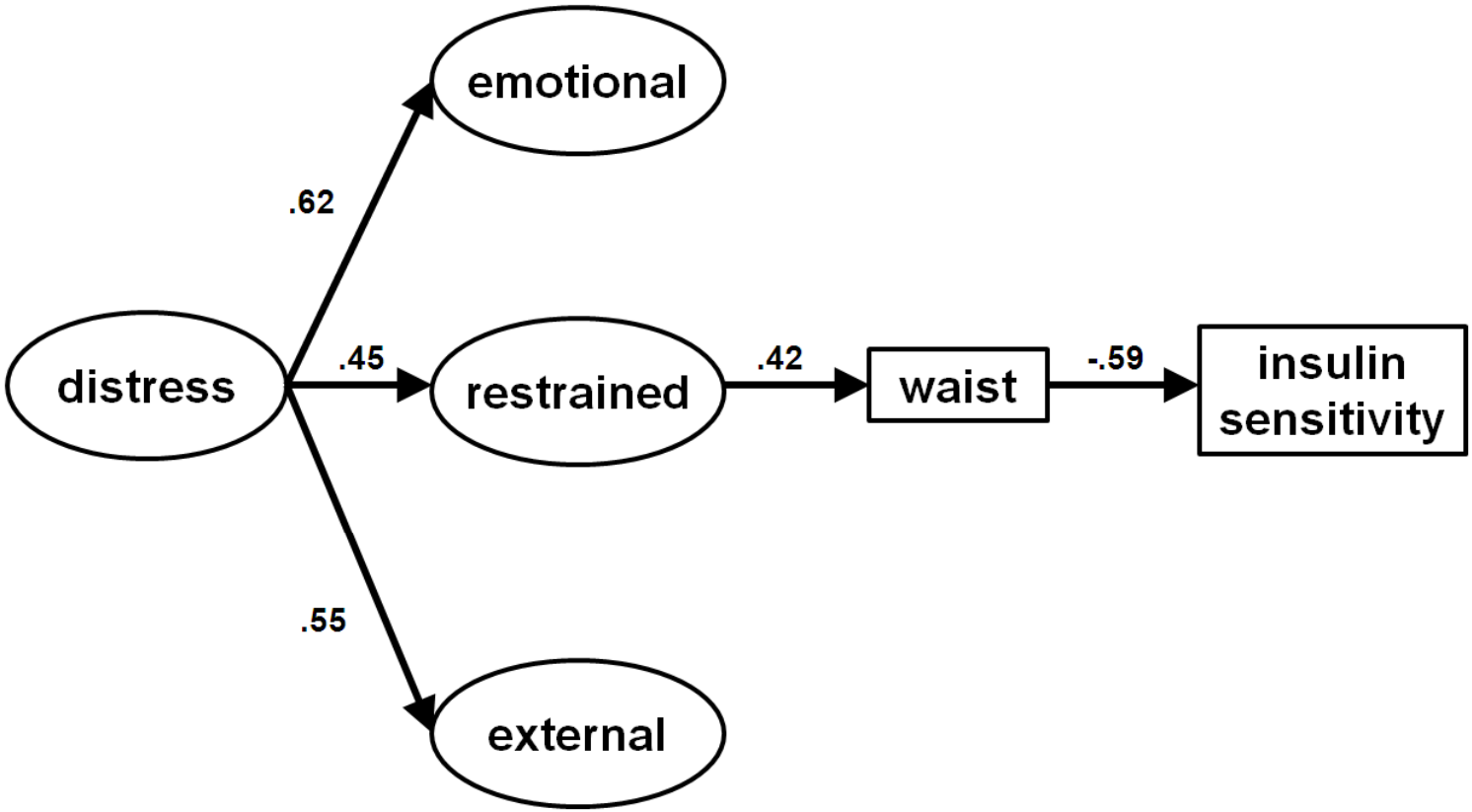

Figure 16. Effects of distress and eating styles on cardiometabolic risk, adjusted for age, gender, education, and physical activity *(n* = 125). Standardized parameter estimates reported.

Model fit indices: χ^2 (82) = 95.397, *p* = .15; CFI = .977; RMSEA = .036; SRMR = .062. Waist = waist girth. Insulin sensitivity = M. Refer to Figures 3 and 4 for details about the distress, emotional, restrained, and external factors.

p < .05
p = n.s

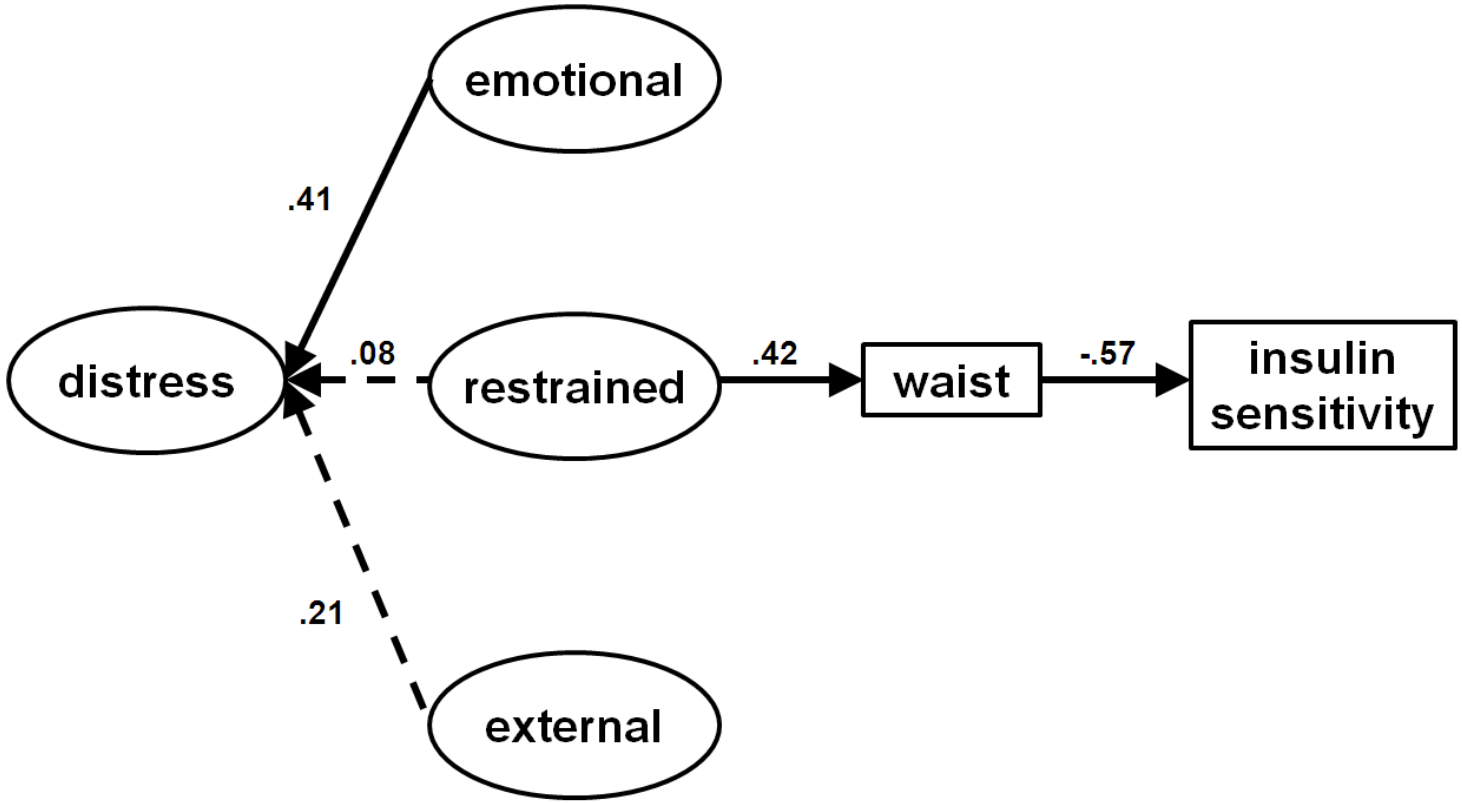

Figure 17. Reversal hypothesis: effect of eating styles on distress and cardiometabolic risk, adjusted for age, gender, education, and physical activity *(n* = 125). Standardized parameter estimates reported.

Model fit indices: χ^2 (81) = 99.903, *p* = .08; CFI = .968; RMSEA = .043; SRMR = .062. Waist = waist girth. Insulin sensitivity = M. Refer to Figures 3 and 4 for details about the distress, emotional, restrained, and external factors.

p < .05
p = n.s

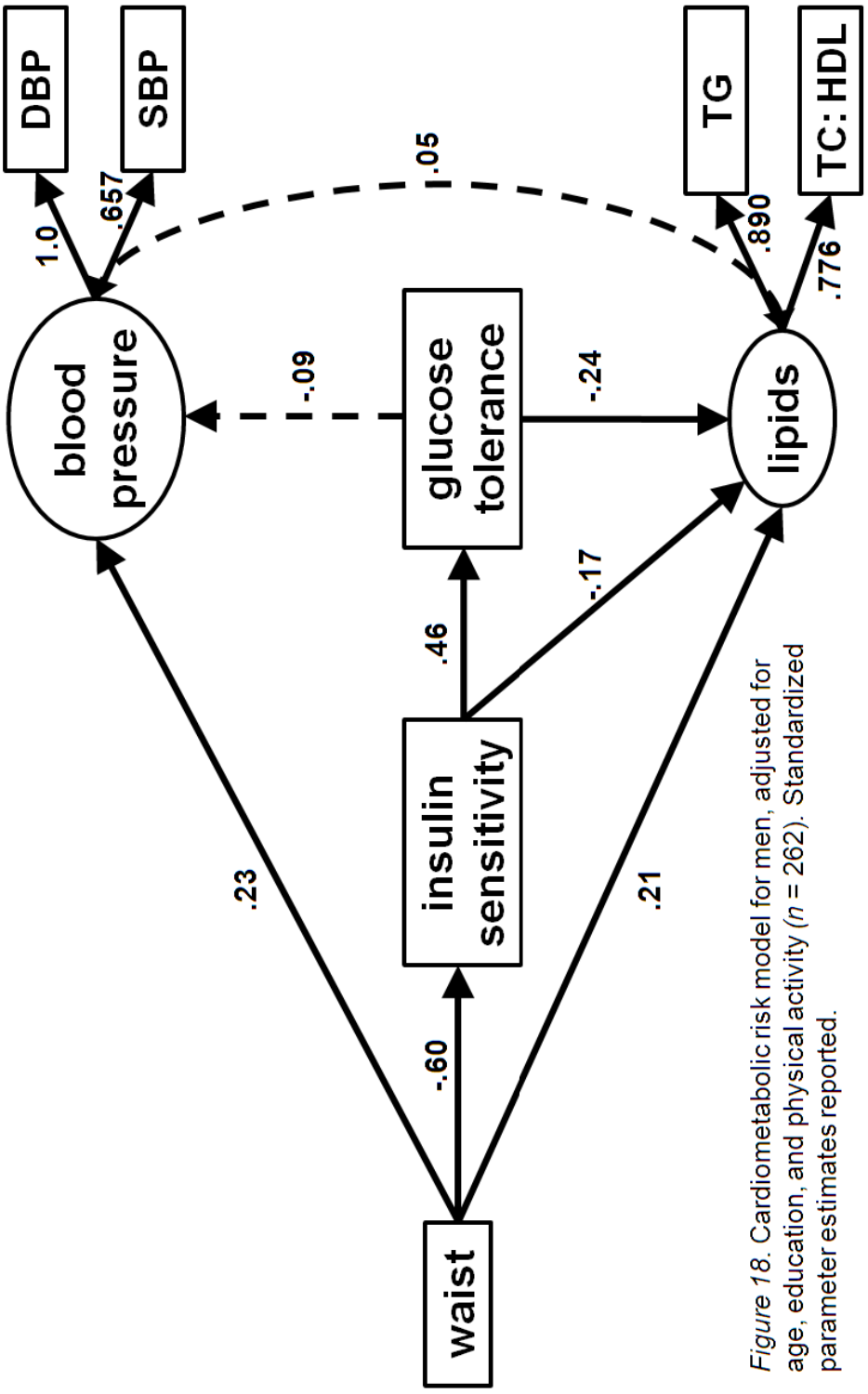

Figure 18. Cardiometabolic risk model for men, adjusted for age, education, and physical activity (n = 262). Standardized parameter estimates reported.

Model fit indices: χ^2 (16) = 20.831, p = .19; CFI = .993; RMSEA = .034; SRMR = .020. Waist = waist girth. Insulin sensitivity = M. Glucose tolerance = area under the curve for glucose during the oral glucose tolerance test (sign reversed). TG = triglycerides. TC:HDL = total cholesterol-to-HDL-C ratio. DBP = diastolic blood pressure. SBP = systolic blood pressure.

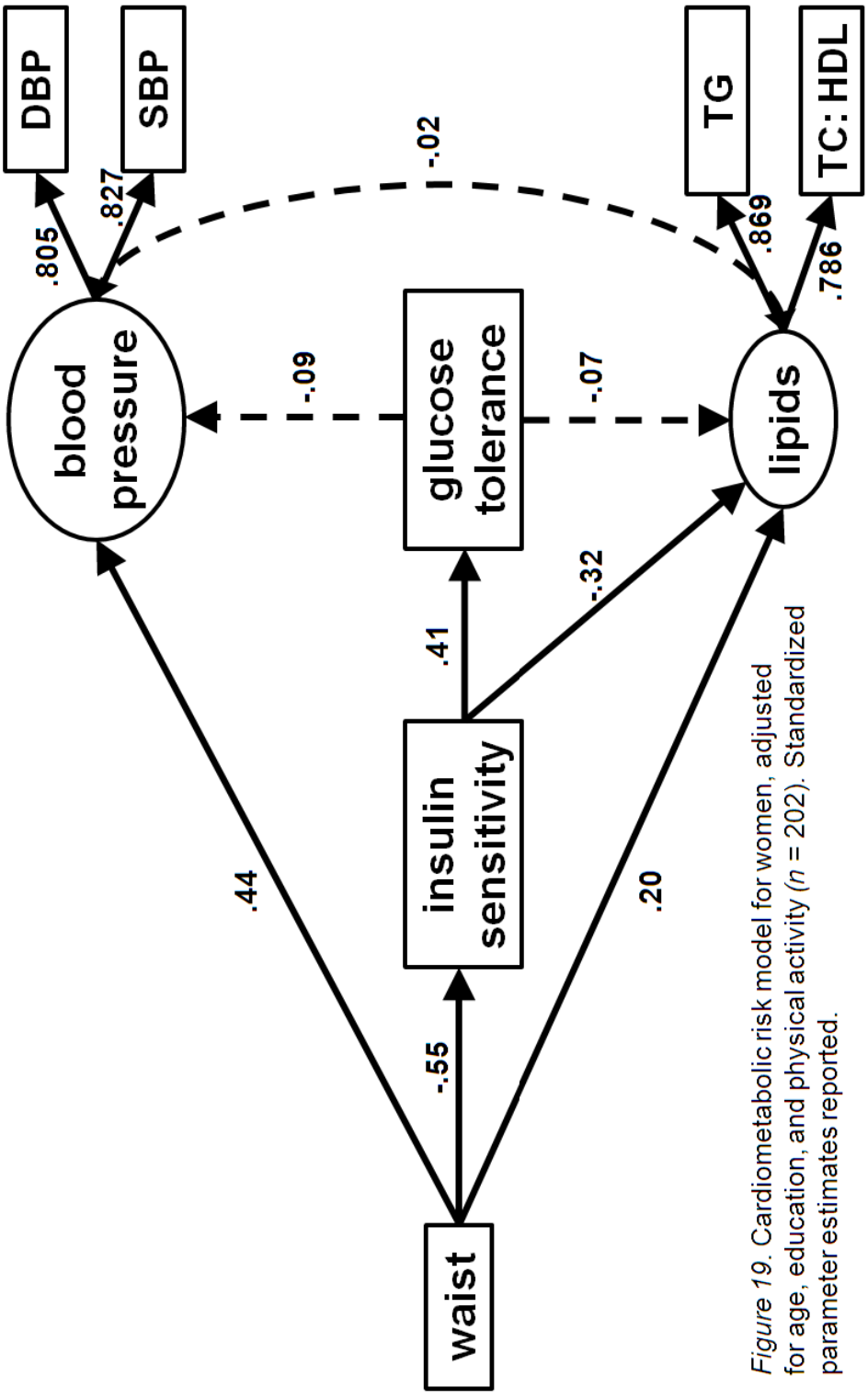

Figure 19. Cardiometabolic risk model for women, adjusted for age, education, and physical activity (n = 202). Standardized parameter estimates reported.

Model fit indices: $\chi^2(15) = 10.589$, $p = .78$; CFI = 1.000; RMSEA < .001; SRMR = .019. Waist = waist girth. Insulin sensitivity = M. Glucose tolerance = area under the curve for glucose during the oral glucose tolerance test (sign reversed). TG = triglycerides. TC:HDL = total cholesterol-to-HDL-C ratio. DBP = diastolic blood pressure. SBP = systolic blood pressure.

Appendix A

Dutch Eating Behavior Questionnaire (DEBQ)

DIRECTIONS: The following questions relate to your usual eating habits. From the following five selections, please choose the most accurate answer that reflects your eating. Please answer all questions.

1. never
2. seldom
3. sometimes
4. often
5. very often

1. If you have put on weight, do you eat less than you usually do?
2. Do you try to eat less at meal times than you would like to eat?
3. How often do you refuse food or drink offered because you are concerned about your weight?
4. Do you watch exactly what you eat?
5. Do you deliberately eat foods that are slimming?
6. When you have eaten too much, do you eat less than usual the following days?
7. Do you deliberately eat less in order not to become heavier?
8. How often do you try not to eat between meals because you are watching your weight?
9. How often in the evening do you try not to eat because you are watching your weight?
10. Do you take into account your weight with what you eat?
11. Do you have the desire to eat when you are irritated?
12. Do you have a desire to eat when you have nothing to do?
13. Do you have a desire to eat when you are depressed or discouraged?
14. Do you have a desire to eat when you are feeling lonely?
15. Do you have a desire to eat when somebody lets you down?
16. Do you have a desire to eat when you are cross (annoyed or angry)?
17. Do you have a desire to eat when you are approaching something unpleasant to happen?
18. Do you get the desire to eat when you are anxious, worried or tense?
19. Do you have a desire to eat when things are going against you or when things have gone wrong?
20. Do you have a desire to eat when you are frightened?
21. Do you have a desire to eat when you are disappointed?
22. Do you have a desire to eat when you are emotionally upset?
23. Do you have a desire to eat when you are bored or restless?
24. If food tastes good to you, do you eat more than usual?
25. If food smells and looks good, do you eat more than usual?
26. If you see or smell something delicious, do you have a desire to eat it?
27. If you have something delicious to eat, do you eat it straight away?
28. If you walk past the baker do you have the desire to buy something delicious?
29. If you walk past a snack bar or a cafe, do you have the desire to buy something delicious?
30. If you see others eating, do you also have the desire to eat?
31. Can you resist eating delicious foods?
32. Do you eat more than usual, when you see others eating?
33. When preparing a meal are you inclined to eat something?

Appendix B

Three-Factor Eating Questionnaire – Revised 18-item version (TFEQ-R18)

DIRECTIONS: For the following we ask you to rate statements as to how **true or false** they are about your **eating** behaviors. For questions 1 to 13 use the following rating scale.

definitely true (4)
mostly true (3)
mostly false (2)
definitely false (1)

1. When I smell a sizzling steak or juicy piece of meat, I find it very difficult to keep from eating, even if I have just finished a meal.
2. I deliberately take small helpings as a means of controlling my weight.
3. When I feel anxious, I find myself eating.
4. Sometimes when I start eating, I just can't seem to stop.
5. Being with someone who is eating often makes me hungry enough to eat also.
6. When I feel blue, I often overeat.
7. When I see a real delicacy, I often get so hungry that I have to eat right away.
8. I get so hungry that my stomach often seems like a bottomless pit.
9. I am always hungry so it is hard for me to stop eating before I finish the food on my plate.
10. When I feel lonely, I console myself by eating.
11. I consciously hold back at meals in order not to weight gain.
12. I do not eat some foods because they make me fat.
13. I am always hungry enough to eat at any time.

For questions 14 to 18, use the rating scale following each question about your eating habits.

14. How often do you feel hungry?
    only at meal times (1)
    sometimes between meals (2)
    often between meals (3)
    almost always (4)
15. How frequently do you avoid "stocking up" on tempting foods?
    almost never (1)
    seldom (2)
    usually (3)
    almost always (4)
16. How likely are you to consciously eat less than you want?
    unlikely (1)
    slightly likely (2)
    moderately likely (3)
    very likely (4)
17. Do you go on eating binges though you are not hungry?
    never (1)
    rarely (2)
    sometimes (3)
    at least once a week (4)
18. What number would you give yourself on a scale from 1 to 8, where …
    1 = no restraint in eating (eating whatever you want, whenever you want it) and
    8 = total restraint in eating (constantly limiting food intake and never "giving in")

1 2 3 4 5 6 7 8
No Restraint **Total Restraint**

Appendix C

Restraint Scale (RS)

DIRECTIONS: The following questions ask you about how you regulate your body weight.

1. How many pounds over your desired weight were you at your maximum weight? ____ ____ ____ pounds

2. How often are you dieting?

0. Rarely
1. Sometimes
2. Usually
3. Always

3. What is the maximum amount of weight that you ever lost within 1 month? ____ ____ ____ pounds

4. What is your maximum weight gain within a week? ____ ____ pounds

5. In a typical week, how much does your weight fluctuate? ____ ____ pounds

6. Would a weight fluctuation of 5 pounds affect the way you live your life?

0. Not at all
1. Slightly
2. Moderately
3. Very much

7. Do you eat sensibly before others and make up for it alone?

0. Rarely
1. Sometimes
2. Usually
3. Always

8. Do you give too much time and thought to food?

0. Rarely
1. Sometimes
2. Usually
3. Always

9. Do you have feelings of guilt after overeating?

0. Rarely
1. Sometimes
2. Usually
3. Always

10. How conscious are you of what you're eating?

0. Not at all
1. Slightly
2. Moderately
3. Very much

Glossary of Abbreviations and Symbols

λ: standard factor loading estimate

AMDR: Acceptable Macronutrient Distribution Range

ANCOVA: analysis of covariance

AUC: area under the curve

$AUC_{Glucose}$: AUC for glucose during the oral glucose tolerance test

$AUC_{Insulin}$: AUC for insulin during the oral glucose tolerance test

BDI: Beck Depression Inventory

BDI tot: BDI total score

BMI: body mass index

bpm: beats per minute

BSA: body surface area

BUN: blood urea nitrogen

CBC: complete blood count

CES-D: Center for Epidemiological Studies Depression Scale

CFA: confirmatory factor analysis

CFI: comparative fit index

CHD: coronary heart disease

CRH: corticotrophin-releasing hormone

CRP: C-reactive protein

CT: computerized tomography

CV: coefficient of variation

DBP: diastolic blood pressure

DEBQ: Dutch Eating Behavior Questionnaire

DEBQ emo: DEBQ emotional eating subscale

DEBQ ext: DEBQ external eating subscale

DEBQ res: DEBQ restrained eating subscale

DFF: Diet Free Forever

DNA: deoxyribonucleic acid

DRI: Dietary Reference Intake

ECG: electrocardiogram

EBI: Eating Behavior Inventory

EER: Estimated Energy Requirement

EI: Eating Inventory

EPQ: Eating Patterns Questionnaire

FIML: full information maximum likelihood

FLA: Fragenbogen für Latente Adipositas [the Latent Obesity Questionnaire]

FNB: Food and Nutrition Board

GR: glucocorticoid receptor

HAES: Health At Every Size

HDL-C: high-density lipoprotein cholesterol

Ho: Cook-Medley Hostility Scale

HPA: hypothalamo-pituitary-adrenal

IDF: International Diabetes Federation

IES: Intuitive Eating Scale

IL-6: interleukin-6

IMT: intimal-media thickness

kcals: kilocalories

LDL-C: low-density lipoprotein cholesterol

LEP: leptin gene

LEPR: leptin receptors

LSD: lysergic acid diethylamide

M: a measure of insulin sensitivity obtained from the euglycemic hyperinsulinemia clamp test

MARCH: Markers Assessing Risk for Cardiovascular Health (study name)

MC4R: melanocortin 4 receptors

MetS: metabolic syndrome

NCEP ATP III: National Cholesterol Education Program's Adult Treatment Panel III

NHANES: National Health and Nutrition Examination Survey

OGTT: oral glucose tolerance test

PAI-1: plasminogen activator inhibitor-1

PC-1: plasma cell membrane glycoprotein-1

PCP: phencyclidine

POMC: proopiomelanocortin

POMS: Profile of Mood States

POMS ang: POMS Anger-Hostility subscale

POMS anx: POMS Tension-Anxiety subscale

PPAI: Paffenbarger Physical Activity Index

PSS: Perceived Stress Scale

RDA: Recommended Dietary Allowance

RMSEA: root mean square error of approximation

RS: Restraint Scale

SBP: systolic blood pressure

SCID: structured clinical diagnostic interview

SEM: structural equation modeling

SPSS: Statistical Package for the Social Sciences

SRMR: standardized root mean squared residual

STAI: Spielberger's State-Trait Anxiety Inventory

STAXI: Spielberger's State-Trait Anger Expression Inventory

SUGAR: Obesity, Metabolic Syndrome, and Meal-Related Glycemia (study name)

TC:HDL-C: total cholesterol-to- high-density lipoprotein cholesterol ratio

TEE: total energy expenditure

TFEQ: Three-Factor Eating Questionnaire

TFEQ-R18: Three-Factor Eating Questionnaire – Revised 18-item version

TFEQ emo: TFEQ-R18 emotional eating subscale

TFEQ res: TFEQ-R18 cognitive restraint subscale

TFEQ uncon: TFEQ-R18 uncontrolled eating subscale

TG: triglycerides

THC: tetrahydrocannabinol

TIBC: total iron binding capacity

TNF-α: tumor necrosis factor-α

USDA: United States Department of Agriculture

WHR: waist girth-to-hip girth ratio

VITA

Virginia Tai Coryell was born in Fairfax, Virginia, on March 15, 1982. Her parents are Terry Lee Coryell and Kenneth William Coryell. She received her elementary education at Layton Hall Elementary, Mile Creek Elementary, and Center Schools, and her secondary education at Lyme-Old Lyme Middle and Lyme-Old Lyme High Schools in Old Lyme, Connecticut. In August 2000, she entered the College of Arts and Sciences at Indiana University, where she received a Bachelor of Science degree in Psychology and a Certificate in Liberal Arts and Management. In August 2005, she was admitted to the Graduate School of the University of Miami in Coral Gables, Florida. She was granted a Master of Science degree in Health Clinical Psychology in May 2008 and a Doctor of Philosophy degree in Health Clinical Psychology in August 2011.

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