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Energy Density of Food Intake in Overweight Adults

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Doctor of Philosophy

Nursing

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ABSTRACT

Energy Density of Food Intake in Overweight Adults

Daurice Ann Grossniklaus

Background: Abdominal obesity (AO) is associated with cardiometabolic risk. In 2003-2004, more than 50% of adults had AO. Psychological distress, through hypothalamic-pituitary-adrenocortical (HPA) axis activation with increased dietary energy density (ED), may contribute to AO.

Purpose: To examine ED and HPA activation as mediators between psychological factors and AO in overweight adults. This study was guided by the adapted Stress and Coping model.

Methods: Descriptive, cross-sectional design was used to enroll 87 adults, with a mean age of 41.3 $\pm$ 10.2 years; mean body mass index (BMI) of 32.1 $\pm$ 6.1 kg/m^2; 73.6% women; 50.6% African-Americans. Participants completed the Beck Depression Inventory-II (BDI-II), Perceived Stress Scale (PSS) and Three Factor Eating Questionnaire Revised (TFEQR) to measure depressive symptoms, perceived stress and dietary restraint. They completed a weighed three day food record which was analyzed for caloric intake and food and beverage weight, and ED (kilocalories/gram) was calculated. Participants collected saliva samples for cortisol. Height, weight were measured to calculate BMI, and waist circumference (WC), an indicator of AO, was measured. Descriptive statistics and sequential regression were used to predict WC. Three approaches evaluated ED as a mediator between psychological factors and cardiometabolic disease risk.

Findings: Increased depressive symptoms ($p \leq .05$) explained food and beverage ED above that explained by younger age ($p=.06$), male gender ($p=.06$), African-American race ($p \leq .01$) and reported adequate caloric intake ($p \leq .01$). High food and beverage ED ($p \leq .01$) explained WC variance above that explained by older age ($p=.07$), increased food and beverage weight ($p \leq .01$), and increased depressive symptoms ($p \leq .01$). PSS was significant ($p=.04$) explaining 4.0% of WC variance. Increased BDI-II ($p=.05$) explained WC variance above that explained by older age ($p=.23$) and low dietary restraint ($p=.21$). Morning salivary cortisol did not explain variance in food and beverage ED or WC. There was insufficient evidence to conclude food and beverage ED or morning salivary cortisol were mediators.

Conclusions: Increased depressive symptoms independently predicted high food and beverage ED and elevated WC. Food and beverage ED independently predicted elevated WC. Whether a reduction in depressive symptoms alters dietary ED, and if a reduction in dietary ED reduces WC, merit further evaluation.

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CHAPTER I

Introduction

Statement of the Problem

Overweight, a body mass index (BMI) of 25 kg/m^2 or higher, is a public health problem affecting the health of an estimated 66% of U.S. adults aged 20 years or older (Flegal, Carroll, Ogden, & Johnson, 2002). Importantly, abdominal obesity, the central distribution of adipose tissue, is more closely related to insulin resistance than generalized obesity and is associated with hypertension, dyslipidemia, type 2 diabetes, and premature coronary death (Chan, Rimm, Colditz, Stampfer, & Willett, 1994; Cnop et al., 2003; Cnop et al., 2002; Hill, Catenacci, & Wyatt, 2006; Janssen, Katzmarzyk, & Ross, 2002; Larsson et al., 1984; Reaven, 2006; Rexrode, Buring, & Manson, 2001; Walker et al., 1996; Zamboni, Armellini, Cominacini et al., 1994; Zamboni, Armellini, Turcato et al., 1994). Between 1988-2004, the overall age adjusted prevalence of abdominal obesity rose from 29.5 to 42.4% in men and from 47.0 to 61.3% in women (Li, Ford, McGuire, & Mokdad, 2007). Among individuals with a BMI less than 25 kg/m^2, the age adjusted prevalence of abdominal obesity was 1.5% in men and 15.9% in women (Li, Ford, McGuire, & Mokdad, 2007). Importantly, individuals with a normal BMI and elevated waist circumference have a mortality risk 20% higher than individuals with a normal BMI and normal waist circumference (Koster et al., 2008). The prevalence of abdominal obesity increases as weight class increases. For example, among overweight individuals, the age adjusted prevalence of abdominal obesity was 33.9% in men and 79.9% in women (Li, Ford, McGuire, & Mokdad, 2007). Among

obese individuals, the age adjusted prevalence of abdominal obesity was 91.9% in men and 99.5% in women (Li, Ford, McGuire, & Mokdad, 2007). In 2003-2004, more than 50% of adults had abdominal obesity (Li, Ford, McGuire, & Mokdad, 2007). Researchers attribute the dramatic rise in prevalence to a number of factors including decreased physical activity, unhealthy dietary patterns, and chronic stress (Dallman, La Fleur et al., 2004; Hill, Catenacci, & Wyatt, 2006).

Rather than examine unhealthy dietary patterns and the stress response as causal to abdominal obesity, the intent of this study is to examine them as potential biobehavioral mediators of the relationship between psychological distress and abdominal obesity, a marker of cardiometabolic disease risk. To date, most research has focused on how dietary patterns affect BMI and cardiometabolic disease risk (Herman, 1996; Nestle et al., 1998). However, this approach is limited because BMI does not adequately and uniquely signal cardiometabolic disease risk (Ford, Mokdad, & Giles, 2003; Kahn, 2005).

Recent human and animal studies suggest chronic psychological distress in combination with unhealthy diets high in calories and fat may contribute to cardiometabolic disease risk. Furthermore, chronic psychological distress, through its actions on the hypothalamic-pituitary-adrenocortical (HPA) axis, may alter the underlying biology resulting in a re-distribution of body fat to the abdomen (Dallman et al., 2003; Kuo et al., 2007; Rosmond, 2005); Thus, greater understanding and integration of the behavioral and biological mechanisms that together mediate relationships between psychological distress and cardiometabolic disease risk may lead to the creation of innovative, tailored weight loss interventions. Therefore, the purpose

of this study is to examine the mediating roles of an unhealthy diet (represented by an increased dietary energy density) and HPA axis activation (represented by elevated morning salivary cortisol secretion) between psychological distress and abdominal obesity in overweight adults.

Specific Aims and Hypotheses

Each specific aim is proposed to examine a section or pathway of the subsequently described conceptual model.

Specific aim 1: To examine the associations between psychological factors (perceived stress and depressive symptoms) and biobehavioral responses (dietary pattern and HPA activation) in sedentary, overweight adults.

H1: Psychological factors (perceived stress and depressive symptoms) will explain a significant amount of variance in dietary pattern (ED) while controlling for age, gender, race/ethnicity, BMI, reporting adequate caloric intake (RACI) and dietary restraint.

H2: Psychological factors (perceived stress and depressive symptoms) will explain a significant amount of variance in HPA activation (morning salivary cortisol secretion) while controlling for menopause status, race/ethnicity, BMI and dietary restraint.

Specific aim 2: To examine the associations between biobehavioral responses (dietary pattern and HPA activation) and cardiometabolic disease risk (abdominal obesity) in sedentary, overweight adults.

H3: Dietary pattern (ED) will explain a significant amount of variance in abdominal obesity (waist circumference) while controlling for age, gender, race/ethnicity, dietary restraint, perceived stress and depressive symptoms.

H4: HPA activation (salivary cortisol secretion) will explain a significant amount of variation in abdominal obesity (waist circumference) while controlling for menopause status, race/ethnicity, dietary restraint, perceived stress and depressive symptoms.

Specific aim 3: To examine the associations among psychological factors (perceived stress and depressive symptoms), biobehavioral responses (dietary pattern and HPA activation) and cardiometabolic disease risk (abdominal obesity) in sedentary, overweight adults.

H5: Psychological factors (perceived stress and depressive symptoms) will explain a significant amount of variation in abdominal obesity (waist circumference) while controlling for age, gender, race and dietary restraint.

H6: Dietary pattern (dietary energy density) and HPA activation (salivary cortisol secretion) will mediate the relationship between perceived stress and depressive symptoms with abdominal obesity (waist circumference) while controlling for age, gender, race and dietary restraint.

Conceptual Framework

Overview

This study's conceptual basis, adapted from Cohen, Kessler, and Gordon's stress and coping model (Cohen, Kessler, & Gordon, 1995), hypothesizes that individuals who view environmental demands as taxing and coping resources as inadequate perceive

themselves to be under stress. Psychological stress triggers biological and/or behavioral responses that can mitigate or increase cardiometabolic disease risk among susceptible individuals (Cohen, Kessler, & Gordon, 1995).

Dallman and others (2004) extend this concept by proposing a chronic stress model which explains the underlying biological mechanisms that act to mediate the relationship between psychological distress and cardiometabolic disease risk (Dallman, La Fleur et al., 2004). This model hypothesizes that the relationship between psychological factors and cardiometabolic disease risk is mediated by biobehavioral responses, and that cardiometabolic disease risk concurrently provides feedback altering the biobehavioral response. They hypothesize that chronic psychological stress weakens the feedback signals that normally act to shut down the stress response and the release of cortisol, a stress hormone. Cortisol, interacting with other metabolic regulating hormones, may contribute to the redistribution and growth of the adipose tissue. Furthermore, researchers hypothesize that cortisol coupled with high fat, high sugar diets may further exaggerate the growth of the adipose tissue (Kuo et al., 2007). As the adipose increases, it secretes proteins such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-alpha), and plasminogen activation inhibitor-1 (PAI-1) and signaling proteins such as the endocannabinoids that promote inflammation, atherogenesis, and insulin resistance, thus increasing cardiometabolic disease risk (Darmon et al., 2006; Fernandez-Real & Ricart, 2003; Isoldi & Aronne, 2008; Kershaw & Flier, 2004; Senn et al., 2003). As the adipose tissue increases, there is also a decreased synthesis of adiponectin, a protein that offers protection against cardiovascular disease (Cnop et al., 2003; Havel, 2004). Leptin, produced by the adipocytes, is the only adipokine known to

cross the blood brain barrier, and it plays an important role in energy regulation by activating or deactivating pathways which stimulate or inhibit food intake. As fat mass increases, leptin levels increase; however, many obese individuals appear to be leptin resistant (Houseknecht, Baile, Matteri, & Spurlock, 1998; Peters, 2006; Porte, Baskin, & Schwartz, 2002).

This study proposes an amalgamated model derived from stress and coping theories that accounts for individual and environmental demands that predispose individuals to psychological distress (Cohen, Kessler, & Gordon, 1995; Dallman, La Fleur et al., 2004; Dallman et al., 2003). Depending on the individual's coping behaviors and available resources, these factors may contribute to psychological distress. Psychological distress, defined in this study as the co-occurrence of perceived stress and depressive symptoms, may increase cardiometabolic disease risk. This model proposes that psychological distress is associated with unhealthy dietary patterns characterized by increased dietary energy density (ED) and hypothalamic-pituitary-adrenocortical (HPA) activation characterized by alterations in cortisol secretion. Unhealthy dietary patterns and HPA activation, which may direct the distribution of body fat to the abdomen, are viewed as mediators between psychological distress and cardiometabolic disease risk (Cohen, Kessler, & Gordon, 1995; Dallman, La Fleur et al., 2004; Dallman et al., 2003). It is also possible that psychological distress and cardiometabolic disease are linked via the complex interactions of the metabolic regulating hormones and/or other behavioral factors such as smoking, inactivity and alcohol use (Strine et al., 2008). Feedback signals, generated by the adipose tissue, may further alter the biobehavioral mediators (Isoldi & Aronne, 2008). The association

between abdominal obesity and psychological distress may be bidirectional, possibly due to imbalances in neurotransmitters such as serotonin and dopamine, or due to the social stigmatization, discrimination, and other stressors associated with obesity (Isoldi & Aronne, 2008; Tafet & Bernardini, 2003).

In this study, individual characteristics (age, gender, race/ethnicity, overweight, dietary restraint, and physical activity) are the control variables. Perceived stress, depressive symptoms, dietary energy density and salivary cortisol secretion are the independent variables. Abdominal obesity, measured by waist circumference, is the outcome variable. For purposes of this study, the metabolic regulating hormones (glucose, insulin, and leptin) are presented as part of the explanatory model but will not be measured. Figure 1 depicts the conceptual framework guiding the study.

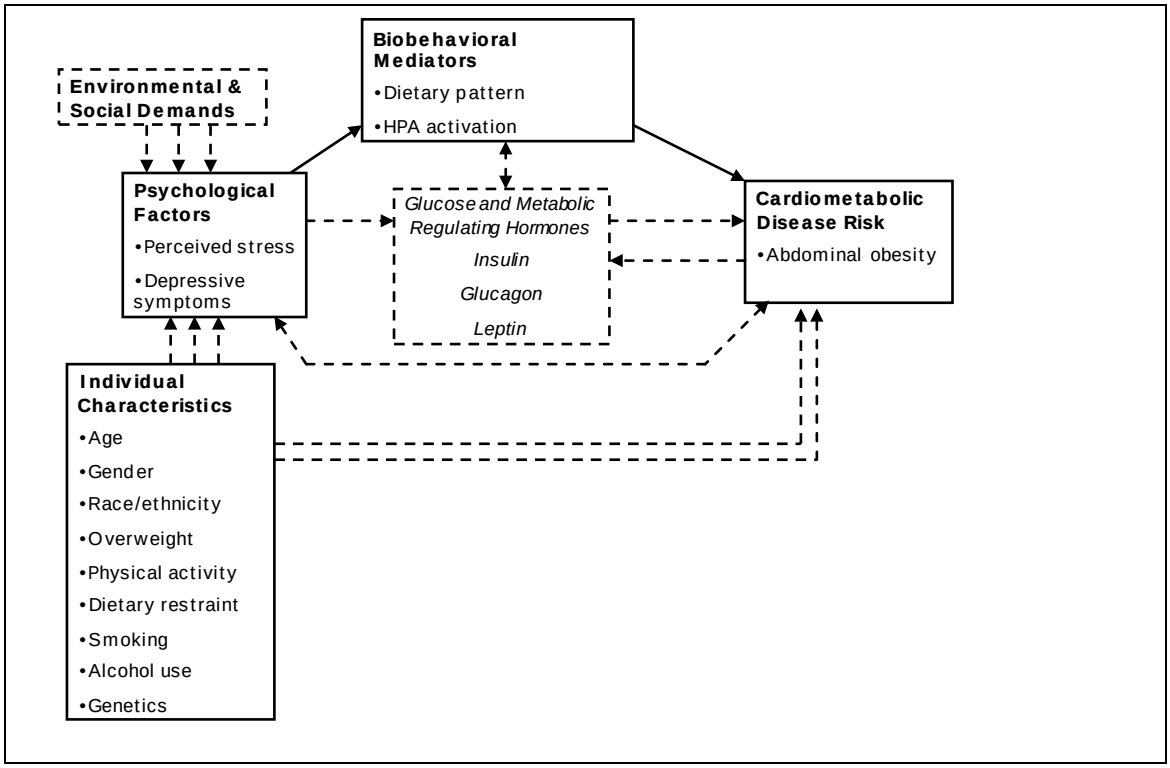

Figure 1. The Adapted Stress and Coping Model

(Cohen, Kessler, & Gordon, 1995; Dallman, La Fleur et al., 2004; Dallman et al., 2003).

Note. Triple dotted lines indicate factors that may predispose an individual to psychological distress. Double dotted lines indicate characteristics that may predispose an individual to abdominal obesity. Double headed arrows and single dotted lines are used to indicate the possible bidirectional relationships and feedback loops. The solid lines represent the two proposed biobehavioral mediators linking psychological distress and abdominal obesity.

Theoretical Assumptions

The theoretical assumptions of the study include:

1. Working adults experience complex environmental demands.
2. Environmental demands and coping behaviors affect the individual's psychological well being.
3. Psychological stress can alter the individual's underlying biology, thereby affecting physiological well being.

Although all adults experience stress, this study focused on working adults, those adults employed for 20 hours or more per week. Because 65% of adults are employed, worksites provide nurses a venue for behavioral interventions in an environment where adults typically consume one or more meals per day (Obesity. Guide to Community Preventive Services Website. Centers for Disease Control and Prevention).

Individual Characteristics

Characteristics that may modify an individual's susceptibility to abdominal obesity include age, gender, race/ethnicity, overweight, physical activity, dietary restraint, smoking, alcohol use and genetic factors. With aging, body weight increases and body composition changes: there is an decrease in the percent of total body weight that is lean and an increase in both subcutaneous and visceral fat (Despres, Lemieux, & Prud'homme, 2001; Gibson, 2005; Ryan, 2000). Resistance training has been shown to decrease fat mass and increase lean body mass in middle aged and older adults (Colado & Triplett, 2008; Levinger, Goodman, Hare, Jerums, & Seliq, 2007; Orsatti, Nahas, Maesta, Nahas-Neto, & Burini, 2008). Women have a higher amount of body fat than

men, and the distribution of body fat shifts to the abdomen following menopause (Gibson, 2005). Hispanic and Caucasian men have greater total visceral adipose tissue than African-American men (Carroll et al., 2008; Despres et al., 2000). Hispanic and white women had greater total visceral adipose tissue than African-American women (Carroll et al., 2008). Although body composition and the amount of body fat are important, where it is distributed plays an important role in determining cardiometabolic disease risk. Abdominal obesity increases cardiometabolic disease risk (Lakka, Lakka, Tuomilehto, & Salonen, 2002).

Behavioral factors also modify cardiometabolic disease risk; for example, physically inactive individuals are at increased risk for insulin resistance (Dunbar & Kacharava, 2005 ; Hu et al., 2003; Knowler et al., 2002; Mannix et al., 2005; Stookey, 2001) and physically inactive postmenopausal women, regardless of their age, race/ethnicity, or BMI, are at increased risk of cardiovascular disease (Manson et al., 2002). Physical activity (PA) decreases total and abdominal obesity (Vessby, 2000), improves insulin sensitivity (NIH, 1998), and improves blood lipid levels by increasing high density lipoproteins (HDL), and lowering total cholesterol and triglyceride levels (Wei, Macera, Hornung, & Blair, 1997; Wilbur, Naftzger-Kang, Miller, Chandler, & Montgomery, 1999). Favorable changes in insulin sensitivity may be due to increased GLUT-4 levels, the insulin regulated transporter that carries glucose into the musle cells. Other possible mechanisms include increased capillary density and enhanced blood flow within the muscle (Ryan, 2000). These favorable changes in lipid profiles may be due, in part, to the effects of PA on lipolytic enzymes which alter the size and composition of lipoproteins (Chatzinikolaou et al., 2008; Williams et al., 1986).

Another behavioral factor is dietary restraint. Individuals who exhibit low dietary restraint may have an increased risk of abdominal obesity: their dietary patterns may differ from those of individuals who consciously monitor their food choices and control their food intake (De Lauzon et al., 2004). Smoking and alcohol use are other behaviors that may play a role in body fat distribution. Although the mechanisms are not well understood, obese smokers have higher waist circumferences than obese non-smokers, and excess alcohol intake is associated with abdominal obesity (Berlin, 2008; Fan et al., 2008).

Finally, body fat and its distribution are controlled, in part, by the complex interaction of multiple genes on different chromosomes (Wajchenberg, 2000). A large number of genes have been identified that control food intake, food preferences, carbohydrate and lipid metabolism, and energy expenditure (Carey, Nguyen, Campbell, Chisholm, & Kelly, 1996; Clement, 2005; De Castro, 1993; Foreyt & Poston, 2002). Independently and collectively these individual characteristics alter cardiometabolic disease risk. Of these, this research focuses on age, gender, race/ethnicity, dietary restraint, and overweight.

Environmental Demands

Another relevant predisposing condition, environmental demands, can contribute to poor health outcomes, particularly among lower socioeconomic individuals (Baum, Garofalo, & Yali, 1999). Time constraints and other environmental factors limit the opportunity for leisure and non leisure physical activity (Isoldi & Aronne, 2008). Time pressures and changes in family structure result in limited time for meal preparation and create a demand for prepackaged and fast foods (Hill, Wyatt,

Reed, & Peters, 2003). Cost, access, and convenience play an important role in food choices (Glanz, Basil, Maibach, Goldberg, & Snyder, 1998). Adverse living situations coupled with limited access and financial resources to purchase healthful food and healthcare (Newby, 2006) expose low income individuals to risk of overweight and its attendant disease.

Psychological Factors

Perceived stress and depressive symptoms may alter food preferences (Benton, 2002; Epel, Lapidus, McEwen, & Brownell, 2001; Greeno & Wing, 1994; Herman & Polivy, 1975; Oliver, Wardle, & Gibson, 2000; Wurtman, 1993). Overweight individuals report stress and depressive symptoms as psychological factors affecting dietary pattern (Benton, 2002; Chambers & Swanson, 2006). Depressive symptoms may also affect adherence to dietary recommendations and physical activity patterns (Ciechanowski, Katon, Russo, & Hirsch, 2003; Farmer et al., 1988; Stunkard, Faith, & Allison, 2003; Ziegelstein, Bush, & Fauerbach, 1998).

Possible Biobehavioral Mediators

Alterations in the underlying biological mechanisms may make psychologically distressed individuals particularly vulnerable to abdominal obesity. These biological mechanisms involve alterations in the normal negative feedback pathway of the hypothalamic-pituitary-adrenocortical (HPA) axis (Casper et al., 1988; Chrousos & Elenkov, 2006; Dallman et al., 2003; McLean, Barr, & Prior, 2001; Rexrode, Buring, & Manson, 2001). Elevated cortisol levels, characteristic of increased HPA axis activity, are associated with increased hunger and the intake of high fat, high carbohydrate foods

(Gluck, Geliebter, Hung, & Yahav, 2004). Additionally, a recent study of 78 overweight adults reported that the cortisol response to awakening (cortisol level at awakening minus the cortisol level 30 minutes after awakening) was positively correlated with sensations of fullness following a standardized test breakfast meal. In other words, individuals with a blunted awakening cortisol response, an indicator of HPA dysregulation, did not feel satiated following the standardized test meal. This sense of satiation is an important predictor of total energy intake such that individuals who feel full consume less total energy (Therrien et al., 2008). Individuals experiencing HPA dysregulation may be susceptible to overeating due to the combined effects of increased hunger coupled with not feeling satiated after eating.

Dietary pattern as characterized by dietary energy density (ED), the ratio of kilocalories to gram of food consumed, is particularly relevant in examining how high fat and carbohydrate foods together may modify cardiometabolic disease risk. High energy dense diets can represent an important health risk due to the excess intake of energy, total and saturated fats and the limited intake of micronutrient rich foods (Cuco, Arija, Marti-Henneberg, & Fernandez-Ballart, 2001; Ledikwe et al., 2006b). Dietary factors may explain as much as 8% of the variance in body adiposity. Although total dietary fat has been positively correlated with body fat mass in adults (Larson et al., 1996; Westerterp et al., 1996), fatty acid profile may be more important than the percentage of calories derived from dietary fat (Huang, Xin, McLennan, & Storlien, 2004). For example, in a large, nationally representative sample, there was a lower prevalence of obesity among individuals who consumed a diet high in fat (greater than 30% of total calories from fat) and more than nine servings per day of fruits and

vegetables as compared to those who consumed diets high in fat with less than nine servings per day of fruits and vegetables. Importantly, individuals with a high fruit and vegetable intake also consumed a lower percent of energy from saturated fat (Ledikwe et al., 2006a). In a study of middle aged adults, saturated fat was positively correlated with fat mass. In contrast, polyunsaturated fat was negatively correlated with fat mass. Of note, dietary fat intake explained only two percent of variance in fat mass (Larson et al., 1996).

Blood Glucose and the Metabolic Regulating Hormones

Energy metabolism is primarily controlled by the actions of insulin and glucagon, hormones produced in the pancreas. Elevated blood glucose, a potent stimulant for insulin secretion, typically occurs after the ingestion of a carbohydrate-rich meal. Insulin and glucagon act in opposition so that blood glucose levels are tightly maintained. Insulin plays an important role in glucose metabolism in the liver, muscle, and adipose tissue. Leptin, the only hormone produced in the adipose tissue that crosses the blood brain barrier, acts in the hypothalamus to control appetite and energy expenditure. Although obese individuals have normal levels of circulating leptin in relation to their fat mass, they are leptin resistant (Champe, Harvey, & Ferrier, 2005). Growth hormone, insulin like growth factor and catecholamines also play a role in energy balance (Ryan, 2000).

Cardiometabolic Disease Risk

Excess abdominal adipose tissue contributes to cardiometabolic disease risk; for instance, insulin resistance is more closely related to abdominal than generalized

obesity (Chan, Rimm, Colditz, Stampfer, & Willett, 1994; Cnop et al., 2003; Cnop et al., 2002; Hill, Catenacci, & Wyatt, 2006; Janssen, Katzmarzyk, & Ross, 2002; Larsson et al., 1984; Reaven, 2006; Rexrode, Buring, & Manson, 2001; Walker et al., 1996; Zamboni, Armellini, Cominacini et al., 1994; Zamboni, Armellini, Turcato et al., 1994). Abdominal obesity has emerged as an independent factor associated with increased risk of hypertension, stroke, coronary heart disease, insulin resistance, and type 2 diabetes (Bjorntorp, 1988; Folsom, Prineas, Kaye, & Munger, 1990; Lakka, Lakka, Tuomilehto, & Salonen, 2002; Larsson et al., 1984; Rexrode, Buring, & Manson, 2001; Walker et al., 1996; Welin, Svardsudd, Wilhelmsen, Larsson, & Tibblin, 1987).

In addition to storing fat, adipose tissue produces and secretes hormones that regulate energy balance (Havel, 2004; Mohamed-Ali, Pinkney, & Coppack, 1998; Porte, Baskin, & Schwartz, 2002). Researchers hypothesize that insulin, cortisol, and adipose hormones interact centrally and peripherally to regulate energy intake and body adiposity (Havel, 2001; La Fleur, Houshyar, Roy, & Dallman, 2005; Reaven, 2006).

Conceptual and Operational Definitions

Individual characteristics.

Age, gender, and race/ethnicity will be determined by self-report. These data will be recorded on the demographic and clinical information form.

Body Mass Index (BMI) is the ratio of weight to height, (kg)/(height)^2. Height and weight will be measured using National Health and Nutrition Examination Survey (NHANES) procedures and recorded on the anthropometric data form (National Health and Nutrition Examination Survey, 2000). BMI will be calculated using the measured information (National Institutes of Health, [NIH], 1998).

Overweight individuals, those with a BMI of 25.0 kg/m^2 and higher, may carry extra body weight but not necessarily body fat (Flegal, Carroll, Ogden, & Johnson, 2002).

Obese individuals, those with a BMI of 30 kg/m^2 and higher, carry extra body fat. Among obese women, body fat content exceeds 30%, and among obese men, body fat content exceeds 25% (Ogden et al., 2006).

Extreme obese are individuals with a BMI greater than or equal to 40 kg/m^2 (Ogden et al., 2006).

Dietary restraint, the conscious processes that regulate food intake to maintain body weight or promote weight loss (De Lauzon et al., 2004; Herman & Polivy, 1975; Rotenberg & Flood, 1999), will be measured using the Three Factor Eating Questionnaire Revised (TFEQR) (Karlsson, Persson, Sjostrom, & Sullivan, 2000). Individuals will be categorized as exhibiting high or low dietary restraint.

Physical activity is bodily movement produced by skeletal muscle contraction resulting in energy expenditure including activity associated with occupation, household work, child/elder care giving, leisure time, conditioning and sports/exercise. Physical activity will be controlled by excluding individuals who self report greater than or equal to 30 minutes or more of physical activity per day on 3 or more days of the week (Sternfeld, Ainsworth, & Quesenberry, 1999).

Psychological factors.

Depressive symptoms are thoughts, feelings and behaviors demonstrating sadness, loss of interest in life, and negative perceptions of self or the future (Beck, Steer, Ball, & Ranieri, 1996; Steer, Ball, Ranieri, & Beck, 1997). These cognitive features can be

accompanied by somatic features like changes in appetite. Depressive symptoms will be measured using the Beck Depression Inventory II (BDI-II) (Beck, Steer, Ball, & Ranieri, 1996).

Perceived stress is the degree to which an individual finds his/her life to be unpredictable, uncontrollable, or overwhelming. It will be measured using the Perceived Stress Scale (PSS) (Cohen, Kamarck, & Mermelstein, 1983).

Proposed biobehavioral mediators.

Dietary energy density is the ratio of calories per weight of food and beverages consumed (kilocalories/gram). Dietary energy density is correlated with the water content of foods: water content explains 85% of the variance in energy density; water and dietary fat together explain 99% of the variance (Drewnowski, 2003; Grunwald, Seagle, Peters, & Hill, 2001). Participants recorded their food and beverage intake for three consecutive days. These dietary data were analyzed using nutrition software to calculate food and beverage ED (Drewnowski, 1998; Ledikwe et al., 2005).

Salivary cortisol levels, quantitative measures of hypothalamic-pituitary-adrenocortical (HPA) axis activity, were measured at awakening and 10:00 p.m. (Rosmond, Dallman, & Bjorntorp, 1998).

Cardiometabolic disease risk.

Abdominal obesity, assessed by measuring waist circumference, refers to the distribution of body fat in the central region between the thorax and pelvis. Cut points that indicate increased risk of cardiometabolic disease are a waist circumference greater than 88 cm for women and greater than 102 cm for men (NIH, 1998). Waist

circumference will be measured using the NHANES protocol procedure (National Health and Nutrition Examination Survey, 2000).

Significance of the Proposed Study

Understanding how individual characteristics, psychological factors and biobehavioral responses interact and alter the health risks posed by abdominal obesity is of great importance, particularly in light of the disability and death associated with subsequent cardiometabolic disease. This study will have particular relevance for clinical practice, the behavior of health professionals and the broader field of public health. First, clarifying these associations may lead to the development and testing of clinical assessment tools and tailored interventions aimed at weight control for at risk populations. Second, this study will contribute to the growing body of literature that supports the use of ED not only as a weight control strategy but, potentially, as a means to reduce abdominal obesity. Third, the findings may inform future research using clinically relevant biomarkers to clarify the underlying metabolic pathways linking psychological distress, dietary energy density, and abdominal obesity. Fourth, the findings may assist clinicians to develop tailored strategies to modify stress and mood related dietary patterns. Fifth, the findings may inform evidence-based guidelines to educate at risk patients. Using energy density as a guide for food selection strategies will potentially modify abdominal obesity. Sixth, the findings may guide the development of clinical trials testing the efficacy of targeting dietary energy density to reduce abdominal obesity. Finally, the findings may guide policy development to increase accessibility to low energy dense foods at worksites and community settings through pricing strategies and subsidies.

This study relates closely to the Centers for Disease Control and Prevention Health Promotion Goal for Healthy People in Every Stage of Life, objective 30, to improve behaviors among adults ages 21-49 years that promote health and well being, and to objective 37, to improve behaviors among older adults and elderly ages 50 and above that promote health and well being (Centers for Disease Control and Prevention, 2007); to the Healthy People 2010, objective 19.1, to increase the proportion of adults at a healthy weight, and objective 19.2, to reduce the proportion of adults who are obese (U.S. Department of Health and Human Services, 2000); to the National Institute of Health Roadmap Initiative to improve our ability through interdisciplinary collaboration to prevent, detect, diagnose and treat disease and disability related to obesity (National Institutes of Health, 2008); and, to the National Institute of Nursing Research strategic plan to integrate biological and behavioral science, promote healthy lifestyles to prevent disease, and identify strategies aimed at long term behavior change (National Institute of Nursing Research, 2006).

Summary

The purpose of this study is to investigate the role of dietary energy density and cortisol secretion as biobehavioral variables that potentially mediate the relationship between psychological distress and abdominal obesity, an indicator of cardiometabolic disease risk. The framework guiding this study is an adapted biobehavioral model incorporating the concepts of individual characteristics, environmental demands, psychological factors, biobehavioral responses, and cardiometabolic disease risk. The specific aims and research questions are posed to help clarify the direct and mediating relationships among the model components. This study will lead to a greater

understanding of linkages among psychological distress, biobehavioral mediators, and cardiometabolic disease outcomes as a basis for intervention development and future research. More specifically, this study will address one unhealthy dietary characteristic, increased dietary energy density, in greater depth and within a biobehavioral context to extend existing knowledge about psychological distress, dietary patterns and subsequent abdominal obesity.

CHAPTER II

Review of the Literature

Introduction

This chapter provides the scientific underpinnings for investigating cortisol secretion and dietary energy density as potential mediators of the relationship between psychological distress and abdominal obesity. First, individual characteristics that may contribute to abdominal obesity are reviewed. Second, the environmental demands that may contribute to psychological distress are reviewed. Third, psychological factors, perceived stress and depressive symptoms, are described. Fourth, the proposed biobehavioral mediators, dietary energy density and cortisol secretion, are reviewed. Finally, a comprehensive review of the health outcome of interest, abdominal obesity, is presented.

Individual Characteristics

Age, Gender, Race/ethnicity and Psychological Distress

Individual characteristics may predispose individuals to psychological distress as well as abdominal obesity. Depression is more common among women than men, and in younger than older adults, but does not differ by race/ethnicity (Carpenter, Hasin, Allison, & Faith, 2000). Some individuals may have a genetic predisposition for both depression and obesity. A candidate gene has been identified on chromosome seven. This gene is of interest because it has been shown to play a role in depression, and it is near the *OB* gene which regulates body weight (Faith, Matz, & Jorge, 2002; Stunkard,

Faith, & Allison, 2003). Adults currently depressed or with a lifetime diagnosis of depression or anxiety are more likely to be obese and physically inactive than adults without (Strine et al., 2008). Medications used for the treatment of depression can contribute to weight gain. Tricyclic antidepressants are associated with the highest incidence of weight gain. Selective serotonin reuptake inhibitors (SSRI) are associated with weight gain; however, the incidence varies between the different medications (Papakostas, 2008).

Age, Gender, Race/ethnicity and Abdominal Obesity

Age, gender, and race/ethnicity may also modify an individual's susceptibility to abdominal obesity. With aging, body weight increases and body composition changes: there is an decrease in the percent of total body weight that is lean and an increase in both subcutaneous and visceral fat (Despres et al., 2000; Gibson, 2005; Ryan, 2000). Resistance training has been shown to decrease fat mass and increase lean body mass in middle aged and older adults (Colado & Triplett, 2008; Levinger, Goodman, Hare, Jerums, & Seliq, 2007; Orsatti, Nahas, Maesta, Nahas-Neto, & Burini, 2008). In normal weight adults, body fat accounts for 26.9% of the total body weight in women compared to 14.7% in men (Gibson, 2005). Storage fat, which includes visceral, subcutaneous and inter/intramuscular fat, accounts for 15% of total body weight in normal weight women compared to 12% in normal weight men (Gibson, 2005). Whereas premenopausal women are more likely to have lower body adiposity, men and postmenopausal women are more likely to have upper body adiposity (Bhasin, 2003; Bhasin, Woodhouse, & Storer, 2003; Despres, Lemieux, & Prud'homme, 2001; Douchi et al., 2003; Ijuin, Douchi, Oki, Maruta, & Nagata, 1999; Reubinoff et al., 1995; Tremollieres, Pouilles, &

Ribot, 1996). Estrogen regulates lipoprotein lipase (LPL) activity in gluteofemoral adipocytes. Thus, during women's reproductive years, lipids are preferentially deposited in the gluteofemoral region. Following menopause and its associated reduction in estrogen, there is decreased LPL activity in the gluteofemoral adipocytes which results in circulating lipids being deposited in the central abdominal region (Olson, Atwood, Grabrick, Vachon, & Sellers, 2001; Samaras & Campbell, 1997).

Race and ethnic differences may also modify an individual's susceptibility to abdominal obesity. Some Asian populations, for instance Polynesians, have less body fat than Caucasians. Asian Indians have more body fat than Caucasians. In contrast, Chinese individuals from Beijing have similar amounts of body fat as Caucasians (Gibson, 2005). Hispanic adults have larger amounts of abdominal fat than African-American adults (Carroll et al., 2008; Wagenknecht et al., 2003). White men have larger amounts of abdominal fat than African-American men (Carroll et al., 2008; Despres et al., 2000; Hill et al., 1999). It is unclear if these racial/ethnic differences hold true for white and African-American women (Conway, Yanovski, Avila, & Hubbard, 1995; Despres et al., 2000; Hill et al., 1999; Lovejoy, de la Bretonne, Klemperer, & Tulley, 1996; Perry et al., 2000); however, these differences could potentially explain disparities in disease prevalence between these groups of women (Despres et al., 2000; Hill et al., 1999; Perry et al., 2000). Emerging evidence suggests that truncal subcutaneous adipose tissue (SAT) may be an important indicator of cardiometabolic disease risk particularly among minority groups. Higher levels of SAT are correlated with insulin resistance in African-American women. In a recent study of middle age and older adults, African-American women had lower visceral adipose tissue compared to

Caucasian women. There was no significant difference between African American women and Caucasian women in regards to truncal subcutaneous adipose tissue (Carroll et al., 2008).

As the obesity epidemic occurred in the United States, the largest relative increase in abdominal obesity was among African-American adults, 20-29 year olds and in overweight individuals who have a higher total body fat than normal weight individuals (Li, Ford, McGuire, & Mokdad, 2007). When adjusted for age, the largest increase in abdominal obesity prevalence was among 40-49 year old men, women 80 years or older, white men and Mexican-American women (Li, Ford, McGuire, & Mokdad, 2007).

Dietary Restraint

Individuals with low dietary restraint may have an increased risk of abdominal obesity due to different dietary patterns compared to individuals with high dietary restraint (De Lauzon et al., 2004). Individuals who consciously restrict food intake exhibit high dietary restraint, and are more likely to consume fish, vegetables, and reduced fat products. In comparison, individuals who do not consciously restrict food intake exhibit low dietary restraint, and are more likely to consume high fat and salty foods (De Lauzon et al., 2004). Dietary restraint style differs by gender. Although women, in general, have a higher degree of dietary restraint than men, they are more prone to emotional eating than men. Emotional eating is associated with the intake of high fat and high carbohydrate snack foods such as cakes and sweets (De Lauzon et al., 2004).

Individual differences in dietary restraint style may explain some variation in stress-related eating. For instance, under highly stressful conditions, individuals with high dietary restraint consume more high fat and sweet foods compared to individuals with low dietary restraint. These high carbohydrate foods are described as sweet suggesting that they may consist of simple rather than complex carbohydrates. In contrast, under highly stressful conditions, individuals with low dietary restraint consume the same amount or even less food (Benton, 2002). Individuals with high restraint may respond differently to depressed mood than those with low restraint. For instance, women with high dietary restraint experiencing depressed mood, consumed excess calories with subsequent weight gain as compared to women with low dietary restraint experiencing depressed mood (Greeno & Wing, 1994; Herman & Polivy, 1975; Rotenberg & Flood, 1999). Dietary restraint style predicts eating behaviors during psychological distress as opposed to everyday eating behaviors (Greeno & Wing, 1994).

Overweight

Overweight is associated with increased risk of cardiometabolic disease (National Institutes of Health, 1998). However, abdominal obesity has emerged as a better predictor of cardiometabolic disease risk than overall obesity (Lakka, Lakka, Tuomilehto, & Salonen, 2002), which may be due to limitations in body mass index as an independent measure of body fatness. Body mass index is accurate in assessing populations; however when it comes to individuals, BMI implies that all individuals with the same BMI, regardless of their age, sex, or ethnicity, have the same degree of body adiposity. In addition, BMI does not differentiate between muscle weight as compared to fat weight. Perhaps the most important limitation is that BMI does not

provide information about the distribution of body fat (Gibson, 2005). Results of a recent study of middle age and older adults shows that BMI does not adequately reflect abdominal fat distribution particularly among different racial and ethnic groups (Carroll et al., 2008).

Although overweight individuals may have an elevated BMI, they do not necessarily have elevated body fat. Percent body fat is the ratio of body fat weight to total body weight. In comparison, obese individuals have an abnormally high proportion of body fat: obese women have a body fat content that exceeds 30%, and obese men have a body fat content that exceeds 25% (National Institutes of Health, 1998). Weight gain that is attributable to an increase in adiposity, results in increases in both visceral and subcutaneous fat (Wajchenberg, 2000).

Physical Activity

Physical activity (PA), bodily movement produced by skeletal muscle contraction resulting in energy expenditure, is associated with a reduced risk for all cause mortality, type 2 diabetes, colon cancer, ischemic stroke, osteoporosis, depression, fall related injuries, and cardiovascular disease (Physical Activity. Guide to Community Preventive Services Website. Centers for Disease Control and Prevention, 2005). PA decreases total and abdominal obesity (Vessby, 2000), improves insulin sensitivity (National Institutes of Health, 1998), improves blood lipid levels by increasing high density lipoproteins (HDL) and lowering cholesterol and triglyceride levels (Wei, Macera, Hornung, & Blair, 1997; Wilbur, Naftzger-Kang, Miller, Chandler, & Montgomery, 1999), and delays or prevents the development of hypertension (Giannuzzi et al., 2003). These favorable changes in lipid profiles may be

due, in part, to the effects of PA on lipolytic enzymes which alter the size and composition of lipoproteins (Williams et al., 1986).

To maintain a stable body weight, energy intake must balance energy expenditure (Centers for Disease Control and Prevention, 2008). Total daily energy expenditure is the kilocalories spent to maintain metabolic functioning in the resting state (basal metabolic rate; [BMR]), the energy used to metabolize food (thermic effect of food), and energy expended through physical activity. The BMR accounts for approximately 60% of the daily energy expenditure and the thermic effect of food accounts for approximately 10% of daily energy expenditure (Shetty, 2005). These two processes can be altered only slightly. Therefore, the amount of energy expended through physical activity is the most amenable to change: increasing physical activity increases energy expenditure (Levine, 2005; Montoye, Kemper, Saris, & Washburn, 1996; Shetty, 2005).

Physical activity contributes to weight loss and weight loss maintenance in overweight individuals (National Institutes of Health, 1998). Low physical fitness levels are correlated with increased visceral adiposity. In men, weight loss achieved through exercise is associated with reduced visceral fat (Wajchenberg, 2000). Similarly, an endurance exercise program has been shown to reduce abdominal obesity in older men (Schwartz et al., 1991). Regular PA may be most beneficial to abdominally obese individuals; for instance, healthy elderly adults achieved a 20% reduction in visceral adiposity with endurance training (Wajchenberg, 2000). Thirty minutes or more of moderate intensity PA most days of the week is recommended to reduce the risk of

cardiovascular and coronary artery disease mortality by 20-30% (Giannuzzi et al., 2003; Pate et al., 1995).

Importantly, PA induced energy deficits do not increase energy intake in lean and overweight individuals (Blundell & King, 1999). Furthermore, moderate PA has been shown to reduce food intake in obese women (Tsofliou, Pitsiladis, Malkova, Wallace, & Lean, 2003). Regarding the effect of physical activity on food selection, there are inconsistent findings. For example, moderate PA does not alter macronutrient intake in overweight men and women (Donnelly et al., 2003). However, moderate physical activity among sedentary men is correlated with increased carbohydrate intake (Wood, Terry, & Haskell, 1985). The intake of high fat foods after exercise counteracts the activity induced energy deficit (Tremblay, Almeras, Boer, Kranenbarg, & Despres, 1994).

Smoking

Although the mechanisms are not well understood, smoking is another behavior that may contribute to increased waist circumference. Smoking increases cardiovascular risk: smoking is associated with higher cholesterol, lower HDL and higher triglyceride levels as well as increased platelet aggregation. Former and current male smokers were more likely to develop type 2 diabetes than male nonsmokers. Older female smokers were more likely to develop type 2 diabetes than older female nonsmokers. Smoking lowers peripheral uptake of glucose thus contributing to insulin resistance, and chemical components of smoke may damage the pancreas and impair beta cell function. Smokers have a BMI that is on average 1 kg/m^2 lower than non-smokers (Berlin, 2008). Similar findings from a large population study which included 2,408 smokers were recently

published: smokers had a lower BMI than that of never smokers (Pisinger, Toft, & Jorgensen, 2009). This difference in BMI may be due to several factors: a lower caloric intake due to nicotine's appetite suppressing effect and increased energy expenditure (Berlin, 2008; Pisinger, Toft, & Jorgensen, 2009). Smoking can increase energy expenditure by as much as 10% (Berlin, 2008). Although the mechanisms are not understood, smoking is associated with increased waist circumference and increased waist hip ratio. Obese smokers have a higher waist circumference than obese non-smokers (Berlin, 2008). Pisinger and others (2009) reported similar findings: current smokers had a higher waist-hip ratio than never smokers. In addition, waist-hip ratio increased as tobacco use increased. Tobacco use was associated with a less healthy lifestyle including lower quality diet, higher caloric intake, increased alcohol use and decreased physical activity (Pisinger, Toft, & Jorgensen, 2009).

Alcohol Use

The relationship between alcohol intake and abdominal obesity is complex and not well understood. Three recent studies with conflicting results highlight the complexity of these associations. In an epidemiologic study of 10,766 Swedish women, the prevalence of metabolic syndrome was 11.6%. The women with metabolic syndrome were older (mean age=56.9 years), had higher BMIs and higher waist hip ratios compared to the women without metabolic syndrome. These women were more likely to smoke, and they reported less frequent alcohol use compared to the women without metabolic syndrome (Qader, Shakir, Nyberg, & Samsioe, 2008).

In a prospective longitudinal study of 245,533 participants in the National Institutes of Health-AARP Diet and Health Study, individuals in the highest quintile of

waist circumference had lower educational levels, were less likely to smoke, were less physically active and reported lower alcohol intake compared to those in the lowest quintile of waist circumference (Koster et al., 2008). There are methodological problems with how data were collected in the study. First, waist circumference was self measured: participants were given pictures and instructions. This is a threat to the internal reliability of the study. Second, alcohol intake data were extracted from the food frequency questionnaire. Alcohol consumption over the past year was evaluated by categorizing intake as 0 to less than 5 grams, 5 to less than 15 grams, and greater than or equal to 15 grams of alcohol per day. These data focus on the average daily intake rather than alcohol consumption patterns (Koster et al., 2008).

Research has focused on average daily alcohol intake rather than the relationship between alcohol consumption patterns and health outcomes. Fan and others (2008) conducted a secondary analysis of data from 1,529 participants in the 1992-2002 NHANES study (Fan et al., 2008). Data were examined to evaluate the relationship between alcohol consumption patterns and components of the metabolic syndrome in adults with no cardiovascular disease. The sample was comprised primarily of non-Hispanic whites (77%) between the ages of 20-84 years (mean=42 years). Forty one percent were high school graduates or less. The majority of men (52%) reported their usual alcohol intake exceeded the U.S. Dietary Guidelines recommendation of two drinks per day, and the majority of women (67%) reported their usual alcohol intake exceeded the U.S. Dietary Guidelines recommendation of one drink per day (Fan et al., 2008). Individuals who reported drinking in excess of the U.S. Dietary Guidelines had an odds ratio of 1.77 for abdominal obesity, and those reporting that their usual quantity

was 3 or more drinks per day had an odds ratio of 1.8 for abdominal obesity (Fan et al., 2008). In other words, men who consume more than the recommended two drinks per day are at increased risk of abdominal obesity as compared to men who consume less than the recommended amount, and women who consume more than the recommended amount of one drink per day are at increased risk of abdominal obesity as compared to women who consume less than the recommended amount. In addition, individuals who reported consuming 3 or more drinks per day had an increased risk of abdominal obesity as compared to those who consume less than 3 drinks per day. Light, frequent drinking was associated with higher levels of high density lipoprotein which may account for the protective effect of light to moderate alcohol intake on cardiovascular health. Of note, alcohol contains seven calories per gram (Rolls & Barnett, 2000) which can increase caloric intake contributing to weight gain and potentially abdominal obesity. Excess alcohol intake was associated with abdominal obesity, impaired glucose function, diabetes, hypertension and high triglycerides (Fan et al., 2008).

Genetic Factors

Some individuals may be at risk for developing obesity due to their genetic makeup. Body fat and its distribution are controlled in part by the complex interactions of multiple genes and may explain 25-40% of individual variation in body mass (Bouchard, 1994, 1997; National Institutes of Health, 1998). These estimates were obtained from comprehensive studies of twins (Bouchard, 1994, 1997). To date, more than 400 obesity related genes have been identified (Newell, Zlot, Silvey, & Arail, 2007). Genetic factors account for 5% of individual variation in subcutaneous fat as compared to 56% of the individual variation in visceral fat (Wajchenberg, 2000).

Polymorphisms in related genes could also predispose the individual to abdominal obesity. For example, a polymorphism of the glucocortocoid receptor gene may contribute to the accumulation of visceral adipose tissue (Wajchenberg, 2000). Complex interactions of multiple genes control food intake, carbohydrate and lipid metabolism, energy expenditure, body composition and fat distribution (Clement, 2005; Wajchenberg, 2000).

Current obesity-related genomics research focuses on the complex interactions among genes, environment and lifestyle. Martinez and others (2003) investigated the interaction between a β_2-adrenoceptor gene polymorphism, dietary macronutrient composition and obesity risk: women with a polymorphism of the β_2-adrenoceptor gene consuming a carbohydrate rich diet (greater than 49% of energy) were 2.5 times as likely to be obese compared to women with no polymorphism consuming a carbohydrate rich diet (Martinez et al., 2003). Further research is needed to better understand the complex interactions among genes, lifestyle and environment.

Environmental Demands

Another relevant predisposing condition, environmental demands, may contribute to poor health outcomes particularly among lower socioeconomic individuals (Baum, Garofalo, & Yali, 1999). In the past twenty years, as the prevalence of overweight has dramatically increased, so has the pace of daily life (Hill, Wyatt, Reed, & Peters, 2003). During this same time, changes in the family structure (large numbers of working women and an increase in the number of single parent families) have placed multiple demands on many adults (Hill, Wyatt, Reed, & Peters, 2003). Furthermore, due to time constraints and other environmental factors, there are fewer opportunities for

leisure and non leisure physical activity (Isoldi & Aronne, 2008). Low income adults face additional environmental challenges such as crowded and noisy living situations, neighborhood crime, and discrimination (Baum, Garofalo, & Yali, 1999). Thus, the nature and degree of stress and environmental demands are varied and increasing in intensity.

Time constraints of working and mid-life adults have resulted in insufficient time for food preparation and increased demand for prepackaged and fast foods (Hill & Peters, 1998; Hill, Wyatt, Reed, & Peters, 2003). Additionally, the nature of the American food supply has changed over time: high fat and carbohydrate foods coupled with larger portion sizes results in excess energy intake (Hill, Wyatt, Reed, & Peters, 2003). Furthermore, cost and access are important considerations in food choices, particularly among low income adults (Glanz, Basil, Maibach, Goldberg, & Snyder, 1998). The availability and selection of food at home, in the workplace, and at social events is determined, in part, by these factors. Eating away from home and consuming fast food, particularly common among young adults, is associated with the consumption of high fat foods, soft drinks, and large portion sizes that may contribute to weight gain (Isoldi & Aronne, 2008; Ledikwe, Ello-Martin, & Rolls, 2005; Stein & Colditz, 2004).

Cost is an important consideration with food selection, particularly among low income adults (Glanz, Basil, Maibach, Goldberg, & Snyder, 1998). A low energy dense diet, comprised primarily of fruits and vegetables, is more expensive compared to a high energy dense diet, comprised primarily of convenience foods. The daily cost associated with the lowest energy density diets were 165% higher than those diets with the highest energy density (Andrieu, Darmon, & Drewnowski, 2006). This price

differential may prevent low income individuals from adopting a low energy density diet. Furthermore, large supermarkets with well supplied produce departments are sparse in low income, urban neighborhoods and limited transportation may be another factor that impedes access. High energy dense diets have also been shown to have the lowest nutrient quality (Andrieu, Darmon, & Drewnowski, 2006; Ledikwe et al., 2006b).

Adverse living situations, coupled with limited financial resources to purchase healthful food and healthcare (Newby, 2006), may further expose low income adults to food insecurity as well as overweight and its attendant cardiometabolic disease risk. Overweight individuals experience discrimination in housing, education, employment, and healthcare limiting their access to social, economic, and health resources. Overweight individuals, particularly obese women, are paid less for the same work and have limited opportunities for promotions and pay raises (Puhl & Brownell, 2003). These complicated social demands are likely to contribute to feelings of stress and depression in working adults and may, in part, explain disparities in cardiometabolic disease risk (Baum, Garofalo, & Yali, 1999; Dallman, La Fleur et al., 2004; Rosmond, 2005).

Psychological Factors

Adults are more likely to feel stressed when they perceive environmental demands as taxing and see themselves lacking resources to cope with these demands (Cohen, Kessler, & Gordon, 1995). Stress can precipitate or exacerbate depressive symptoms and reinforce unhealthy dietary patterns leading to greater psychological distress and potentially chronic illness (Bale, 2006; McEwen, 2005). Obesity may

contribute to depression due to negative body image and social stigma (de Wit, van Straten, van Herten, Penninx, & Cuijpers, 2009). Research does not support a causal pathway between depression and obesity, and the relationship may be cyclical (de Wit, van Straten, van Herten, Penninx, & Cuijpers, 2009) Overweight individuals report stress and depressive symptoms as psychological factors affecting dietary pattern (Chambers & Swanson, 2006).

During negative emotions such as anger, sadness, tension and fear, young women with normal BMI report more intense hunger or a tendency to eat irregularly. Others report an increased desire to eat as a distraction, to relax and to feel better (Macht & Simons, 2000). Other studies have associated perceived stress with cravings for high fat, high carbohydrate foods such as sweets, particularly among women (Benton, 2002; Epel, Lapidus, McEwen, & Brownell, 2001; Greeno & Wing, 1994; Herman & Polivy, 1975; Oliver, Wardle, & Gibson, 2000). Women report more stress induced eating than men (Epel, Lapidus, McEwen, & Brownell, 2001; Greeno & Wing, 1994).

Individuals vary in their response to depressive symptoms, which may include excessive food intake and preferences for high fat and carbohydrate rich foods (Benton, 2002; Wurtman, 1993). Depressive symptoms have been associated with higher BMI in women (Istvan, Zavela, & Weidner, 1992; Palinkas, Wingard, & Barrett-Connor, 1996; Siegel, Hyg, Yancey, & McCarthy, 2000), and may be accompanied by increased appetite and physical inactivity potentially increasing the risk of abdominal obesity (Farmer et al., 1988; Strawbridge, Deleger, Roberts, & Kaplan, 2002; Strine et al., 2008; Stunkard, Faith, & Allison, 2003). Importantly, due to the poor concentration and

low motivation that often accompanies depression, severely depressed individuals may be less able to adhere to dietary recommendations (Ciechanowski, Katon, Russo, & Hirsch, 2003; Fabricatore & Wadden, 2004; Istvan, Zavela, & Weidner, 1992). Although stress and depressive symptoms can lead to weight gain in some individuals, psychological distress can have the opposite effect in others (Benton, 2002; Dallman et al., 2003; Kuo et al., 2007).

Biobehavioral Responses

Hypothalamic-Pituitary-Adrenocortical Activation

Underlying biological mechanisms may make psychologically distressed individuals particularly vulnerable to abdominal obesity. The hypothalamic-pituitary-adrenocortical (HPA) axis plays an important role in glucose metabolism and energy use. Corticotrophin-releasing hormone (CRH) is secreted from the paraventricular nucleus of the hypothalamus. CRH, released into the pituitary portal bloodstream, is transported to the anterior pituitary gland where adrenocorticotropic hormone (ACTH) is produced and released. As ACTH travels through the systemic circulation, it binds with its receptors on the adrenal cortex which then produce and release cortisol into the bloodstream (Akil et al., 1999).

There are two intracellular glucocorticoid receptors: the high affinity type 1 receptor (mineral-corticoid receptor) and the low affinity type II receptor (glucocorticoid receptor). These receptors rest in the cytosol until stimulated by glucocorticoids. Once the glucocorticoid diffuses across the cell membrane, it binds with these receptors and translocates to the nucleus. Within the cell nucleus, the receptors bind with DNA and promote the expression of various genes (Akil et al.,

1999). The role of the two receptors in regulating HPA activity is unclear. However, scientists hypothesize that when cortisol increases due to stress, the mineral-corticoid receptors become saturated and the glucocorticoid receptors mediate the stress response (Akil et al., 1999).

In normal, healthy adults, cortisol is released in a daily rhythm with a peak at approximately 7:00 a.m. and a trough between 7:00 p.m. to midnight. The diurnal pattern consists of an early morning peak (approximately 30-45 minutes after awakening), a rapid decline over the next few hours, followed by a gradual decline over the rest of the day and reaching the lowest levels by bedtime (Kurina, Schneider, & Waite, 2004). Steep declines are considered indicative of a normal diurnal pattern (Stetler & Miller, 2005). The diurnal pattern is influenced, in part, by food intake with increased cortisol secretion around mealtimes. For example, in a recent study of lean and obese adults, blood cortisol levels peaked 40-60 minutes after eating a 1000 calorie meal and returned to baseline levels 100 minutes following the meal. This cortisol response was more pronounced among the abdominally obese individuals compared to lean individuals (Korbonits et al., 1996). Over time, changes in meal timing can alter the daily cortisol rhythm (Akil et al., 1999). The diurnal pattern is also affected by menstrual cycle, contraceptive use, and menopausal status (Duclos, Pereira, Barat, Gatta, & Roger, 2005). The diurnal pattern also affects the responsiveness of the HPA axis to stress: the stress response is initiated and terminated more efficiently at the trough than at the peak (Akil et al., 1999).

Psychological distress activates the hypothalamic-pituitary-adrenocortical (HPA) axis triggering a cascade of events that result in the release of cortisol (Casper et

al., 1988; Chrousos & Elenkov, 2006; Dallman et al., 2003; McLean, Barr, & Prior, 2001; Rexrode, Buring, & Manson, 2001). Increased cortisol provides negative feedback to the system inhibiting further activation of the HPA axis; however, chronic stress may weaken this negative feedback loop (Champe, Harvey, & Ferrier, 2005; Cohen, Kessler, & Gordon, 1995; Dallman, La Fleur et al., 2004).

There are three feedback mechanisms that play a role in mediating the stress response. The fastest of these is rapid feedback which regulates the immediate initiation and termination of the stress response. After cortisol rises in response to stress, this rapid feedback mechanism immediately shuts down the release of adrenocorticotropic hormone (ACTH) once the stressor is removed. Genomic feedback, which can take hours to days for its effects, mediates negative feedback by inhibiting transcription of the corticotrophin-releasing hormone (CRH) and pro-opiomelanocortin (POMC), a precursor of ACTH, genes (Akil et al., 1999). Intermediate feedback, which occurs between the fast and genomic feedback, is thought to act through gene regulation (Akil et al., 1999).

Dallman and colleagues (2004) propose that chronic psychological distress may diminish this negative feedback loop by increasing the expression of corticotrophin-releasing factor mRNA, thus stimulating rather than inhibiting HPA activity with constant or repeated stress exposure (Dallman, Akana et al., 2004; Dallman, La Fleur et al., 2004). This heightened response may lead to an increase in hepatic gluconeogenesis, thereby stimulating insulin secretion (Dallman, Akana et al., 2004; Dallman, La Fleur et al., 2004). Glucocorticoid receptors are more prevalent in the visceral than subcutaneous fat; cortisol, through a process mediated by the release of neuropeptide Y

from sympathetic nerves (neuropeptide Y pathway), may promote the expansion of the visceral adipose tissue by stimulating the proliferation and differentiation of precursors to mature adipocytes (Dallman, La Fleur et al., 2004; Dallman, Pecoraro, & La Fleur, 2005; Darmon et al., 2006; Duclos, Pereira, Barat, Gatta, & Roger, 2005; La Fleur, Akana, Manalo, & Dallman, 2004; Pedersen, Jonler, & Richelsen, 1994).

In the adipose tissue, cortisol and insulin stimulate LPL activity: within the adipose tissue capillary walls, LPL breaks down the triglyceride contained in its carrier lipoprotein. The adipocytes take up the free fatty acids reassembling them into triglyceride for storage (Duclos, Pereira, Barat, Gatta, & Roger, 2005; Qi & Rodrigues, 2006). Glucocorticoid receptor binding and LPL activity is higher in visceral than subcutaneous adipose tissue which may explain the greater metabolic activity of visceral adipose. By promoting triglyceride storage, cortisol may increase cardiometabolic disease risk. In the central nervous system, chronically elevated cortisol may impair receptor mediated transport of insulin into the brain which diminishes insulin's effectiveness in decreasing food intake, thus allowing cortisol to stimulate food intake (Havel, 2001). It is not known if the association between psychological distress and cardiometabolic disease risk is mediated through biological mechanisms, such as the role of cortisol in re-distributing body fat, or through behavioral mechanisms, such as an energy dense diet (Wilson, 2008).

Perceived stress, depressive symptoms, and cortisol secretion.

It is well established that individuals with major depression have HPA axis dysregulation and approximately half of these individuals over secrete cortisol (Cowen, 2002; Harris, Borsanyi, Messari, Stanford, & Brown, 2000); however, it is unclear if

community dwelling adults experiencing less severe forms of psychological distress have altered cortisol secretion (Kurina, Schneider, & Waite, 2004). Kurina and others (2004) investigated the relationship between psychological symptoms and cortisol secretion in a sample of 91 working adults. The sample was 74.7% women, 94% Caucasian, and participants ranged in age from 27 to 58 years old. Based on self reported height and weight, the mean BMI was 24.6 (*SD* 5.5) kg/m^2 for women and 27.6 (*SD* 4.4) kg/m^2 for men. Perceived stress was measured using the 5 item Perceived Stress Scale and psychological symptoms were measured using the Center for Epidemiological Studies Depression scale and four items from the Taylor Anxiety Inventory. For the analysis, scores from these instruments were dichotomized into substantial depressive symptoms (score greater than or equal to 16) versus no depressive symptoms and high anxiety (scores in the top quartile of the sample) versus no anxiety. Stress at work was measured with one question and dichotomized into high work stress versus no work stress. Saliva was collected over two working days for a total of 6 samples each day. Anxiety was the psychological factor associated with altered cortisol secretion; however, the direction of that relationship varied by gender. Working men experiencing severe anxiety had higher average cortisol levels than working men without anxiety (Kurina, Schneider, & Waite, 2004). In contrast, women who reported frequent work stress had lower average cortisol levels lower than women without work stress. The researchers hypothesize that these gender differences are due to different coping styles: women are more likely to interact and talk about their work stress. There was no significant relationship between depressive symptoms and cortisol levels. The researchers attribute the negative findings to within and between individual

variation in cortisol secretion which may have masked the relationship between depressive symptoms and altered cortisol patterns (Kurina, Schneider, & Waite, 2004).

Similarly, in a cross sectional study, Strickland and others (2002) demonstrated that the majority of community dwelling, mildly depressed adult women had morning cortisol levels that were not significantly different from non-depressed women (Strickland et al., 2002). Among the depressed women, those with chronic stress had higher evening cortisol levels than depressed women without chronic stress. Among all women, those experiencing severe, recent life events had higher evening cortisol levels than those without severe, recent life events. The higher evening cortisol levels reflected a blunted cortisol rhythm. These findings are consistent with the hypothesis that HPA dysregulation results in a blunted cortisol response to recent and persistent adversity (Cowen, 2002; Strickland et al., 2002).

Cortisol secretion and abdominal obesity.

The association between cortisol secretion and abdominal obesity is unclear. In one cross-sectional study, obesity was associated with lower cortisol levels (Walker, Soderberg, Lindahl, & Olsson, 2000) whereas in another cross-sectional study, obesity was associated with normal cortisol levels (Stewart, Boulton, Kumar, Clark, & Shackleton, 1999). In a longitudinal study, Travison and others (2007) found that obese men had lower baseline cortisol levels than non-obese men but baseline cortisol levels did not predict changes in BMI or body composition over time (Travison, O'Donnell, Arajuo, Matsumoto, & McKinlay, 2007).

Steptoe and others (2004) conducted a study of 89 men and 83 women ages 47-59 years to examine the association between abdominal obesity and HPA axis

regulation (Steptoe, Kunz-Ebrecht, Brydon, & Wardle, 2004). Their findings showed that while controlling for age and socioeconomic status, an elevated cortisol response to awakening (30 minutes after awakening – wakening value) and a higher average cortisol over the day (30 minutes after waking – minimum evening value) were associated with abdominal obesity, as measured by waist-hip ratio, among middle-aged men but not women (Steptoe, Kunz-Ebrecht, Brydon, & Wardle, 2004).

In a study of 284 men aged 51 years, those employed as manual laborers had higher visceral adiposity as measured by abdominal sagittal diameter, lower cortisol variability (a blunted cortisol rhythm), and higher stress related cortisol secretion than men who were university graduates (Rosmond & Bjorntorp, 2000). There was no significant difference in cortisol secretion over the day among manual laborers, civil servants or university graduates. The authors attributed these differences in anthropometric and cortisol secretion between occupational categories to the financial worries and job insecurities that are a function of lower socioeconomic status and argue that as socioeconomic status improves, the less cortisol secretion is affected by stress (Rosmond & Bjorntorp, 2000). Interestingly, the alterations in the HPA axis function were exaggerated by the length of time in the socioeconomic situation; for instance, stress related cortisol secretion was higher in men who had been working as manual laborers for more than 21 years as compared to those working as manual laborers for 10 years or less (Rosmond & Bjorntorp, 2000). This suggests that chronic exposure affected cortisol secretion, and this could not be due to aging because all of the men were 51 years at the time of the study (Rosmond & Bjorntorp, 2000),

These conflicting findings suggest that the relationship between cortisol secretion and abdominal obesity is complex and not well understood. It is paradoxical that cortisol levels are not consistently elevated in obesity, yet higher cortisol levels have been linked to cardiometabolic complications; for instance, morning cortisol levels are associated with glucose intolerance, hypertension, and dyslipidemia (Reynolds & Walker, 2007). This may be explained by a recently discovered enzyme, 11ß-hydroxysteroid dehydrogenase type 1, that converts cortisone to its active form, cortisol. This enzyme is over expressed by adipocytes thus, generating higher intra-adipose levels of cortisol which could result in the proliferation of adipocytes (Reynolds & Walker, 2007). Because of the high concentration of glucocorticoid receptors in the visceral fat, the effects are likely to be greater than in the subcutaneous fat (Andrew, Phillips, & Walker, 1998; Reynolds & Walker, 2007; Stewart & Tomlinson, 2002).

Dietary Pattern

Unhealthy diets, particularly those high in saturated fat and simple carbohydrates, have emerged as a potential mediator between psychological distress and abdominal obesity. The possible underlying mechanisms have been described in mice: a diet high in sucrose and saturated fats increased the secretion of neuropeptide Y, a potent appetite stimulant (Huang, Xin, McLennan, & Storlien, 2004; Kaga et al., 2001; La Fleur, Akana, Manalo, & Dallman, 2004), and insulin may increase the pleasurable effects of food, possibly through actions on the catecholaminergic system which regulates activity in the reward center of the brain (Dallman, La Fleur et al., 2004; Figlewicz, 2003a, 2003b). Laboratory rats exposed to chronic stress or elevated corticosterone (the animal equivalent of cortisol), in the presence of insulin, preferred

energy dense chow to standard chow. In addition, there was a re-distribution of body fat to the abdomen, and the rats were less insulin sensitive possibly due to the shift in body fat to the abdomen (La Fleur, Akana, Manalo, & Dallman, 2004). Based on animal studies, Kuo and others (2007) propose a possible synergistic relation between stress and high fat, high sugar diets. Independently, stress and high fat, high sugar diets stimulate the release of corticosterone into the plasma, and stress increases its conversion to the active form in the adipose tissue (Kuo et al., 2007). Once activated, cortisol increases the production of neuropeptide Y in the adipose tissue. Additionally, stress may further exaggerate the response through its actions on the autonomic nervous system, which also stimulates the release of neuropeptide Y in the adipose tissue. Neuropeptide Y and its receptors promotes adipose tissue growth through the proliferation and differentiation of adipocytes as well as angiogenesis (Kuo et al., 2007).

Findings from a recent study of non-human primates suggest that chronic stress plays an important role in dietary patterns. Subordinate female macaques, considered to be chronically stressed, have a higher incidence of abdominal obesity than dominant female macaques. A recent study showed that subordinate macaques consumed more calories than did dominant female macaques in the daytime and, interestingly, in the nighttime as well. The macaques consumed more high fat chow compared to low fat chow; however, due to the small sample size, the difference was not statistically significant (Wilson et al., 2008).

Dietary energy density (ED), the ratio of kilocalories per gram of food consumed, is particularly relevant in examining how high fat and carbohydrate foods

together may modify risk of disease. Energy density is a function of both the caloric intake and volume of food and is strongly linked to the water content of foods. The water content of food accounts for 85% of the variance in energy density; fat and water together account for 99% of variance in energy density (Drewnowski, 2003). Dietary fats, the most energy dense macronutrient, contain 9 calories per gram of food which is more than twice the number of calories per gram found in carbohydrates or protein (Drewnowski, 2003). Dietary fats and carbohydrates differ in their roles in regulating energy balance and body composition. Dietary fats in the presence of insulin increase neuropeptide Y secretion in the arcuate nucleus possibly contributing to increased caloric intake, weight gain, and abdominal fat stores (La Fleur, Akana, Manalo, & Dallman, 2004). Dietary fats consumed in excess of energy needs may be stored more efficiently in adipose tissue than carbohydrates (Schaefer et al., 1995).

Four categories are frequently used to categorize the energy density of foods: high energy dense foods have a calories to gram ratio of 4.0-9.0 and include candies, cookies and nuts; medium energy dense foods have a ratio of 1.5 to 4.0 and include meats, cheeses and salad dressing; low energy dense foods have a ratio of 0.6 to 1.5 and include beans, salads, and cereals with milk; very low energy dense foods have a ratio of less than 0.6 and include skim milk, broth based soups, and fruits and vegetables (Rolls & Barnett, 2000). The quality of dietary fat is important. For example, saturated and trans fats such as those found in red meats, high fat dairy products and hard margarines, increase risk of cardiovascular disease. In contrast, unsaturated fats such as those found in fish, seafood, nuts and olive and canola oils, decreases risk of cardiovascular disease (Rolls & Barnett, 2000).

Dietary energy density is also an indicator of diet quality. For example, high energy dense diets are those composed primarily of fast foods, snacks, and desserts whereas low energy dense diets are those composed primarily of fruits and vegetables (Marti-Henneberg et al., 1999). Individuals following a low energy dense diet consumed fewer calories, less fat, and had a higher intake of vitamins A, C, B6, folate, iron, calcium, and potassium than individuals consuming a high energy dense diet. Additionally, these individuals consumed fewer caloric beverages (Ledikwe et al., 2006b).

Energy density varies by gender, age and race/ethnicity. Men consume a more energy dense diet than women, younger adults consume a more energy density diet than older adults, and non Hispanic Blacks consume a more energy density diet than non Hispanic Whites (Ledikwe et al., 2005). Because of these differences, these variables should be controlled during data analysis.

Energy density is an important characteristic in determining energy intake: an increase or decrease in energy density is associated with a corresponding increase or decrease in energy intake. Three possible mechanisms are thought to link dietary energy density with total energy intake and regulation. First, individuals eat in order to maintain a certain volume of food in the stomach; food volume triggers the vagal nerve signaling satiation. Consuming energy dense foods can lead to excess energy intake because the low food volume does not signal fullness (Bell, Roe, & Rolls, 2003; National Academy of Science, Institute of Medicine, & Food and Nutrition Board, 2005; Rolls & Bell, 1999). Second, because energy dense foods are high in fat, they are also very palatable, and taste is an important factor in food choices (Drewnowski, 1998;

Glanz, Basil, Maibach, Goldberg, & Snyder, 1998; McCrory, Saltzman, Rolls, & Roberts, 2006). Highly palatable foods are associated with increased consumption (Drewnowski, 1998, 2003; McCrory, Saltzman, Rolls, & Roberts, 2006; National Academy of Science, Institute of Medicine, & Food and Nutrition Board, 2005). Third, although energy dense foods decrease the rate of gastric emptying, they increase the rate at which energy leaves the stomach. Because energy rich foods are digested more rapidly, blood sugar levels fall quickly and hunger returns (National Academy of Science, Institute of Medicine, & Food and Nutrition Board, 2005). Individuals consume approximately the same weight of food each day, so the concentration of energy per gram of food is an important factor in regulating energy intake (Drewnowski, 2003). Since food weight plays an important role in regulating appetite and energy intake, ED has the advantage over kilocalories alone by conceptualizing the number of kilocalories per gram weight of food. For example, by adding fruits and vegetables to a casserole it is possible to decrease the energy density yet increase the volume of food consumed while maintaining the same caloric intake (Rolls, Ello-Martin, & Tohill, 2004).

Establishing the relationship between ED and cardiometabolic disease risk is challenging due, in part, to researchers using different methods to calculate ED. Grunwald and others (2001) used mathematical models to examine the associations among energy density, macronutrient, and non-nutrient (water and fiber) composition of the diet (Grunwald, Seagle, Peters, & Hill, 2001). Data were obtained from a sample of 32 men and 15 women who provided weighed food records for four consecutive days. Energy density was calculated using two methods. One method included all foods and

beverages, the second included food only. Small changes in non-nutrients, particularly water, generated the largest effects on energy density. These findings reflect the importance of understanding how non-nutrients can impact the calculation of ED and potentially attenuate relationships between ED and cardiometabolic disease risk (Grunwald, Seagle, Peters, & Hill, 2001).

In a small sample of adults (n=89), Cox and Mela (2000) used six different techniques to calculate energy density comparing the dietary ED of lean individuals (n=46, BMI of 20-25 kg/m^2) to the dietary ED of obese individuals (n=43. BMI equal to or greater than 30 kg/m^2) (Cox & Mela, 2000). Participants recorded their daily intake for 4 consecutive days. Depending on the calculation method used, differences between weight classification and associations with ED varied. Cox and Mela (2000) reported obese individuals consumed a more energy dense diet than lean individuals when all food, milk and alcoholic beverages were included in the calculation (Cox & Mela, 2000). Further, a significant difference between obese and lean individuals was found when all dry matter (excludes water and water composition) was included in the calculation (Cox & Mela, 2000). There was a strong, positive correlation between the calculation method that included the grams of protein, carbohydrate and fat with the percent of dietary energy derived from fat ($r=.73$, $p<0.001$) among the obese individuals (Cox & Mela, 2000).

In a third study, Ledikwe and colleagues (2005) present a comprehensive analysis of eight different methods of calculating energy density providing a sound scientific rationale for the inclusion of various beverages for each calculation method (Ledikwe et al., 2005). Data were collected from a large (n=9288) nationally

representative sample of U.S. adults. Dietary data were obtained during two interviewer administered 24 hour recalls. These two 24 hour recalls were administered 3-10 days apart. Each of the eight calculation methods used a different strategy to adjust for beverage intake. The highest estimates of energy density were obtained for the calculation method including solid foods only and those including solid foods and liquid meal replacements. In contrast, the lowest estimate of energy density was obtained using the calculation method including food and all beverages. Regardless of the method used, day to day variations were higher within individuals than between individuals. Within-individual variation is of concern in that it may weaken associations with the outcome variable. It was particularly large in the calculation method including food and energy containing beverages; therefore, non-significant associations in studies that use this method of calculation may be misleading. Depending on the calculation method used, important relationships may be missed (Ledikwe et al., 2005). Taken together, findings from these three studies highlight the importance of a priori decisions about the method used to calculate ED with particular attention to decisions about the inclusion and exclusion of different types of beverages (Cox & Mela, 2000; Grunwald, Seagle, Peters, & Hill, 2001; Ledikwe et al., 2005).

A number of cross sectional studies have examined the relationship between ED, energy intake, and BMI. Few researchers have examined the relationship between ED and waist circumference. Due to the lack of a standard method of calculating ED, these correlations must be considered in the context of which foods and beverages were included or excluded during the calculation of ED.

Poppitt and Prentice (1996) conducted a secondary analysis of datasets of three epidemiological studies: the Cambridge Family Food Survey, the Medical Research Council National Survey of Health and Development (NSHD) and the Leeds Nutritional Survey (Poppitt & Prentice, 1996). In each of these studies, seven day food diaries were collected, and all recorded food and beverages including alcohol were included analyzed. An increase in ED was accompanied by an increase in energy intake among the women in the Cambridge study ($R^2 = .35$), among the men in the NSHD ($R^2 = .08$), and among the women in the NSHD ($R^2=0.27$). These data are cross sectional in nature and are not adjusted for physical activity or body size (Poppitt & Prentice, 1996). None of the three studies examined the relationship of ED with body mass index or the relationship of ED with abdominal obesity.

Marti Henneberg (1999) examined changes in ED across the lifespan in a cross sectional study of 895 Spanish individuals (Marti-Henneberg et al., 1999). The weight of all energy containing solid foods and beverages excluding alcohol were used to calculate energy density. The researchers found a positive correlation between ED and BMI in adult men but not adult women. As ED increased, BMI increased in men age 21-30 years ($r=.43$, $p<0.001$) and 41-50 years ($r=.29$, $p<0.05$). However, physical activity was not controlled in the analysis (Marti-Henneberg et al., 1999). The researchers did not examine the relationship between ED and abdominal obesity.

In a cross sectional study of 5,783 Chinese adults ages 20-59 years with a mean BMI of 22.2 kg/m^2, a trend toward a significant difference between the ED of lean individuals compared to overweight individuals ($p=0.06$) was observed (Stookey, 2001). All solid food and beverages including milk, coconut juice, sugarcane juice, and

alcohol were included in the calculation of energy density. After adjusting for covariates including age and physical activity, for each kilocalorie per gram increase in ED the likelihood of overweight increased by 26% (Stookey, 2001). There was a strong positive correlation between ED and water content of food ($r=.91$, $p<0.05$) and between ED and grams of dietary fat ($r=.17$, $p<0.05$) (Stookey, 2001). Although dietary fat played a role in determining ED, the researchers did not report the quality of dietary fat. The researchers did not examine the relationship between ED and abdominal obesity (Stookey, 2001). These epidemiologic data linking dietary energy density with elevated BMI are consistent with metabolic studies (Poppitt & Prentice, 1996).

Cuco and colleagues (2001), in a study of 572 adults with a mean age of 43 (*SD* 10.1) years and a mean BMI of 26.1 (*SD* 3.0) kg/m^2, examined the relationship between the ED and nutritional content of diets (Cuco, Arija, Marti-Henneberg, & Fernandez-Ballart, 2001). Solid foods, milk, and alcoholic beverages were included in the calculation of energy density. Men and women consuming the most energy dense diets reported a higher energy intake, and they reported a higher fat intake than those consuming the least energy dense diets. Men consuming high energy dense diets had a 5.2 % higher intake of energy from fat and a 1.3% higher intake of energy from saturated fat compared to men consuming low energy dense diets (Cuco, Arija, Marti-Henneberg, & Fernandez-Ballart, 2001). Women consuming high energy dense diets had a 2.3% higher intake of energy from fat and a 1.3% higher intake of energy from saturated fat than women consuming low energy dense diets (Cuco, Arija, Marti-Henneberg, & Fernandez-Ballart, 2001). Based on these findings, Cuco and colleagues (2001) raised concerns about the effect of high ED diets on cardiometabolic disease risk

(Cuco, Arija, Marti-Henneberg, & Fernandez-Ballart, 2001). Importantly, dietary fat intake is positively correlated with body fat mass in adults (Larson et al., 1996; Westerterp et al., 1996). Additionally, among men, a high intake of saturated fats is correlated with abdominal obesity (Ward et al., 1994).

A recently released cross sectional study by Mendoza et al. (2007) examined the associations among ED, BMI, and abdominal obesity in a nationally representative sample of 6,988 adults (Mendoza, Drewnowski, & Christakis, 2007). Foods only were included in the ED calculation. Energy density was correlated with higher BMI among women (beta=0.44, 95% CI [0.14, 0.73], and a trend towards significance in men (beta=0.37, 95% CI [-0.007, 0.74] p=0.05). Energy density was correlated with elevated waist circumference in women (beta=1.11, 95% CI [0.42, 1.80] and men (beta=1.33, 95% CI [0.46, 2.19) (Mendoza, Drewnowski, & Christakis, 2007). These cross-sectional study findings suggest higher energy density diets may contribute to elevated waist circumference.

Only one longitudinal cohort study examined the relationship between ED and cardiometabolic disease risk. In a prospective seven year cohort study, healthy individuals consuming a diet high in fruits, vegetables, whole grains, and low fat dairy products had comparatively smaller increases in BMI and waist circumference than those consuming primarily a meat and potatoes diet (Newby et al., 2003).

In summary, data from metabolic studies provide evidence demonstrating that an increase in ED is accompanied by an increase in energy intake (Poppitt & Prentice, 1996). A similar pattern was observed in community studies: men and women consuming high energy dense diets have a higher energy intake (Cuco, Arija, Marti-

Henneberg, & Fernandez-Ballart, 2001; Poppitt & Prentice, 1996). In one study, overweight men have been shown to consume a more energy dense diet than lean men (Marti-Henneberg et al., 1999). Obese individuals consume a more energy dense diet than lean individuals (Cox & Mela, 2000; Ledikwe et al., 2006a). High ED may represent an important health risk due to the excess intake of energy, total and saturated fats, and the limited intake of micronutrient rich foods (Cuco, Arija, Marti-Henneberg, & Fernandez-Ballart, 2001; Ledikwe et al., 2006b) Findings from a recently published study suggest ED may contribute to abdominal obesity in men and women (Mendoza, Drewnowski, & Christakis, 2007). Data from one longitudinal cohort study suggests low ED may, over time, decrease the risk of abdominal obesity (Newby et al., 2003). However, knowledge is limited to data from correlation studies; no longitudinal intervention studies examined the associations among ED, body mass index, and abdominal obesity. Energy density alone may not produce sustained changes in body adiposity; it may be the interactions among ED and the metabolic hormone regulators that are critical in determining body adiposity and cardiometabolic disease risk (Havel, 2001).

Glucose and the Metabolic Regulating Hormones

Glucose, Insulin and Glucagon

Elevated blood glucose, a potent stimulant for insulin secretion, typically occurs after the ingestion of a carbohydrate-rich meal. Energy metabolism is largely controlled by the actions of insulin and glucagon. Insulin, produced by the beta cells of the pancreatic islets of Langerhans, exerts anabolic effects resulting in the synthesis of glycogen (storage form of glucose), triglycerides, and protein. In contrast, glucagon,

produced by the alpha cells of the pancreatic islets of Langerhans, activates hepatic glycogenolysis and gluconeogenesis to maintain blood glucose levels. The pancreas releases relative amounts of insulin and glucagon so that the rate of glucose production in the liver equals the use of glucose in the peripheral tissues (Champe, Harvey, & Ferrier, 2005).

Insulin plays an important role on glucose metabolism in the liver, muscle, and adipose tissue. By binding with high affinity receptors in the cell membrane it recruits insulin-sensitive glucose transporters (GLUT) from the intracellular storage pool to the cell membrane. Five different glucose transporters have been identified and each is tissue specific (McGrane, 2006). For example, GLUT-4 transports glucose into the adipose tissue and skeletal muscle (Champe, Harvey, & Ferrier, 2005). By increasing the number of glucose transporters at the cell membrane, insulin facilitates glucose uptake into the tissues. In the liver, insulin decreases glucose production by inhibiting gluconeogenesis and glycogenolysis. In muscle and adipose, it promotes glycogen synthesis and storage. In the adipose, insulin also plays a role in lipid metabolism: it inhibits the activity of hormone sensitive lipase (HSL) which prevents the breakdown of triglycerides, and it increases activity of LPL which promotes triglyceride storage (Champe, Harvey, & Ferrier, 2005).

Leptin

Scientists identified leptin as one of the most important hormones produced in the adipose tissue; it is the only adipose hormone known to cross the blood-brain barrier and act in the central nervous system (Zhang et al., 1994). Normally, leptin acts within the hypothalamus to inhibit food intake and increase energy expenditure. However,

obese individuals are resistant to the appetite suppressing effects of leptin (Houseknecht, Baile, Matteri, & Spurlock, 1998).

The normal leptin pathway.

During energy balance, leptin is secreted in proportion to fat mass. Depending on the fat cell mass, adiposity signals promote or inhibit food intake. In a fasting state (12-48 hours) or during weight loss, leptin gene expression is reduced. Reduced expression of leptin and insulin activates the neuropeptide Y and agouti-related protein pathways and inhibits the pro-opiomelanocortin/cocaine-and amphetamine-regulated transcript (POMC/CART) pathway resulting in increased food intake (Houseknecht, Baile, Matteri, & Spurlock, 1998; Peters, 2006). In contrast, in a fed state, leptin and insulin activate the POMC/CART pathway and inhibit neuropeptide Y and agouti-related protein secretion resulting in decreased appetite (Peters, 2006; Porte, Baskin, & Schwartz, 2002).

Factors such as gender, quality and quantity of food intake, meal pattern, physical activity and hormones influence the production of leptin. Women have leptin levels 3-4 times higher than men after adjusting for body mass index. This is not attributed to differences in reproductive hormones because pre-and post-menopausal women have similar plasma leptin levels. Energy intake alters leptin production. High caloric intake increases leptin production. In contrast, fasting or restricted caloric intake decreases leptin production. Foods, such as high fat meals and fructose containing beverages, which do not stimulate insulin secretion result in lower leptin levels. For example, fasting leptin levels were lower in postmenopausal women who consumed high fat, low carbohydrate meals than women who consumed low fat, high carbohydrate

meals (Koursari, C et al. 2003). Although leptin secretion follows a diurnal pattern with a peak at approximately midnight, the peak is affected by meal patterns with concentrations remaining low for 4-6 hours following a meal. Insulin increases the expression and secretion of leptin (Kershaw & Flier, 2004; Margetic, Gazzola, Pegg, & Hill, 2002). Physical activity plays a role in leptin production and secretion. In a small study of eight postmenopausal women with a mean BMI of 26.4 (*SD* 2.3) kg/m^2, a high carbohydrate diet in combination with an hour of walking was associated with lower fasting and postprandial leptin levels (Koutsari, Karpe, Humphreys, Frayn, & Hardman, 2003). Individuals participating in twice weekly moderate physical activity have leptin levels 25.3% (*SD* 11.3%) lower than sedentary individuals, and those who participate in vigorous physical activity have leptin levels 40.4% (*SD* 13.2%) lower than sedentary individuals (Beasley et al., 2009). The effect of cortisol on leptin secretion is not well understood. Zakrzewska and colleagues (1999) report that cortisol exerts an inhibitory effect whereas Havel and colleagues (2004) report that cortisol stimulates leptin secretion (Havel, 2004; Zakrzewska et al., 1999).

Recent animal research linked chronic stress, elevated corticosterone (equivalent of cortisol) and reduced leptin with depressed behavior. Rats exposed to chronic stress had impaired leptin production and secretion. When exogenous leptin was administered to the rats, antidepressant behavioral effects were seen. These data suggest that leptin, in the hippocampus and through the serotonin system, may exert antidepressant effects, and this has lead to the hypothesis that leptin insufficiency or resistance may contribute to increased vulnerability to depression among obese individuals (Lu, 2007; Lu, Kim, Frazer, & Zhang, 2006). In a study of 72 adults, women diagnosed with major

depression had lower cerebrospinal fluid (CSF) leptin levels than women without major depression while adjusting for BMI (Westling, Ahren, Traskman-Bendz, & Westrin, 2004). In contrast, there was no significant difference in CSF leptin levels between men with or without major depression (Westling, Ahren, Traskman-Bendz, & Westrin, 2004). These findings suggest leptin dysregulation may occur in depressed women (Westling, Ahren, Traskman-Bendz, & Westrin, 2004). In a case control study, eight men and 12 women diagnosed with major depressive disorder with a mean age of 45 (*SD* 3) years and mean BMI of 27.0 (*SD* 1.0) kg/m^2 were compared to 10 healthy men and 10 healthy women with a mean age of 40 (*SD* 2) years and mean BMI of 26.0 (*SD* 1.0) kg/m^2. There was no significant difference in plasma leptin between those with and without major depressive disorder compared to the healthy controls. However, there were significantly lower levels of brain leptin among the individuals with depression (Eikelis et al., 2006). In contrast, the findings of a large epidemiologic study of 510 women ages 20-78 years underscore the complexities of these relationships. Women with a lifetime history of a depressive disorder had higher serum leptin levels (mean leptin=16.37, range=14.70-18.23 ng/m), independent of BMI, than women with no history of depressive disorder (mean leptin=14.46, range=13.79-15.16 ng/ml)(Pasco et al., 2008). Furthermore, over a five year time period, the likelihood of developing a depressive disorder increased 2.5 times for each unit increase in plasma leptin level regardless of BMI (Pasco et al., 2008).

Leptin and the central nervous system.

Leptin crosses the blood brain barrier through a saturable transport system. After leptin binds with its receptor, phosphorylation of the intracellular domain is initiated creating a docking site for the Signal Transducers and Activators of Transcription

(STAT) protein. The STAT protein translocates into the nucleus where it regulates gene transcription (Houseknecht, Baile, Matteri, & Spurlock, 1998; Porte, Baskin, & Schwartz, 2002).

Within the ventromedial hypothalamus there are different regions involved in energy regulation including the arculate nucleus. For several hours following a meal, insulin crosses the blood brain barrier via a receptor mediated transport system. Receptors for leptin and insulin are highly prevalent in the arculate nucleus. Within the arculate nucleus are two groups of neurons involved in energy regulation. The first set of neurons responds to negative energy balance, triggering the secretion of agouti-related protein and neuropeptide Y, a very potent appetite stimulant. These peptides act to stimulate eating behavior. The second set of neurons exerts opposing effects including the stimulation of pro-opiomelanocortin/cocaine neurons (POMC) and cocaine-amphetamine related transcript (CART) neurons which act to inhibit eating behavior and increase energy expenditure (Houseknecht, Baile, Matteri, & Spurlock, 1998; Peters, 2006).

Leptin resistance.

Clinical trials that tested the efficacy of leptin supplementation in overweight individuals demonstrated that leptin supplementation was ineffective. In positive energy balance, serum leptin levels increase dramatically and, theoretically, act to inhibit the expression of neuropeptide Y and agouti-related protein and stimulate the POMC/CART pathway, thereby decreasing food intake (Houseknecht, Baile, Matteri, & Spurlock, 1998; Peters, 2006). Although obese individuals secrete adequate amounts of leptin, they appeared to be “leptin resistant” (Houseknecht, Baile, Matteri, & Spurlock,

1998). There are a number of possible explanations for leptin resistance. Leptin circulates in the blood in the bound form. Therefore, defects in binding could create leptin resistance. It is possible that there are defects in transporting leptin across the blood brain barrier. For example, although obese individual have higher plasma concentrations of leptin, their cerebrospinal fluid levels are comparable to that of lean individuals suggesting possible defects in the transport system. It is possible that neurons would be able to use the leptin if it could be delivered to the cells. In addition, there may be proximal or distal signaling defects. Finally, there could be defects in the target tissue such as defects in synthesis or secretion of leptin (Chehab, Qiu, & Ogus, 2004; Houseknecht, Baile, Matteri, & Spurlock, 1998).

Advances in biotechnology led to the discovery that adipose tissue is an active endocrine organ involved in the regulation of lipid and glucose metabolism and the regulation of body weight (Havel, 2004; Mohamed-Ali, Pinkney, & Coppack, 1998; Porte, Baskin, & Schwartz, 2002). Since the discovery of leptin and other adipose hormones, biological research into the causes of cardiometabolic disease risk has increased dramatically. This research holds the promise of improving our understanding of the biological basis of cardiometabolic disease risk and the development of innovative, tailored weight loss interventions.

Cardiometabolic Disease Risk

Excess abdominal adipose tissue contributes to cardiometabolic disease risk. Insulin resistance, a reduced sensitivity to insulin, is more closely related to abdominal than generalized obesity (Chan, Rimm, Colditz, Stampfer, & Willett, 1994; Cnop et al., 2003; Cnop et al., 2002; Hill, Catenacci, & Wyatt, 2006; Janssen, Katzmarzyk, & Ross,

2002; Larsson et al., 1984; Reaven, 2006; Rexrode, Buring, & Manson, 2001; Walker et al., 1996; Zamboni, Armellini, Cominacini et al., 1994; Zamboni, Armellini, Turcato et al., 1994). Abdominal obesity has emerged as an independent factor associated with increased risk of hypertension, stroke, coronary heart disease, insulin resistance, and type 2 diabetes (Bjorntorp, 1988; Folsom, Prineas, Kaye, & Munger, 1990; Lakka, Lakka, Tuomilehto, & Salonen, 2002; Larsson et al., 1984; Rexrode, Buring, & Manson, 2001; Walker et al., 1996; Welin, Svardsudd, Wilhelmsen, Larsson, & Tibblin, 1987).

Between 1988 and 2004, data from a nationally representative survey demonstrated that the age-adjusted mean waist circumference increased in men from 96.0 to 100.4 cm and, in women from 89.0 to 94.0 cm (Li, Ford, McGuire, & Mokdad, 2007). Between 1999 and 2004, the largest relative increases in abdominal obesity were among younger adults ages 20-29 years and African-American adults (Li, Ford, McGuire, & Mokdad, 2007). During this same period, the largest increase in the age adjusted prevalence of abdominal obesity was among 40-49 year old men, women 80 years or older, white men, and Mexican American women (Li, Ford, McGuire, & Mokdad, 2007). Also between 1999 and 2004, the largest relative increase in abdominal obesity was among overweight individuals as compared to obese individuals (Li, Ford, McGuire, & Mokdad, 2007).

Abdominal adipose tissue, considered to be more metabolically active than subcutaneous adipose tissue, is connected to the liver via the portal circulation which delivers free fatty acids directly into the liver. Elevated levels of free fatty acids impair insulin signaling and inhibit glucose transport into skeletal muscle, thus contributing to

insulin resistance (Boden & Shulman, 2002). Although the mechanisms are not well understood, insulin resistance is due in part to abnormalities in the insulin signaling pathway which disrupt the links between receptor activation and cellular processes. Additionally, it may be that obese individuals have fewer insulin receptors in skeletal muscle, liver and adipose than lean individuals (Guyton & Hall, 2006). Insulin resistance, a physiologic state that increases cardiometabolic disease risk, occurs when adipose and muscle tissues do not respond normally to insulin and are unable to effectively uptake glucose into the cells. This results in hyperglycemia, thus increasing the demand for insulin. Once the pancreatic ß-cells are unable to meet the increasing demand for insulin, hyperglycemia worsens, and type 2 diabetes develops (Reaven, 2006).

Scientists identified adipose tissue hormones that offer protection from cardiometabolic disease such as adiponectin, and those that may contribute to the development of insulin resistance and cardiometabolic disease such as interleukin-6, tumor necrosis factor-alpha, and plasminogen activator inhibitor-1 (Greenberg & Obin, 2006). Recently, researchers discovered that the endocannabinoid system, a signaling system between cells, plays a role in energy balance and metabolism (Matias, Cristino, & Di Marzo, 2008).

Adiponectin

Adiponectin protects against cardiovascular disease and regulates lipid and carbohydrate metabolism. Low adiponectin levels are associated with low high density lipoproteins (HDL) levels, high triglyceride levels, and insulin resistance (Cnop et al., 2003; Havel, 2004). In the adipose tissue, insulin increases adiponectin synthesis. Small

adipocytes secrete adiponectin and insulin sensitizing hormone. Conversely, large adipocytes, associated with obesity, undersecrete insulin sensitizing hormone and oversecrete insulin resistance hormone contributing to insulin resistance (Meier & Gressner, 2004). In contrast, cortisol inhibits adiponectin synthesis thereby exposing individuals to increased risk of insulin resistance and cardiovascular disease (Fallo et al., 2004). Additionally, adiponectin exerts anti-inflammatory, antifibrotic, and antiatherogenic effects on the endothelial cells of the blood vessels (Kubota et al., 2002; Meier & Gressner, 2004).

Other adipose hormones, interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-alpha), and plasminogen activator inhibitor-1 (PAL-1), play a role in insulin resistance and cardiometabolic disease risk. Secreted primarily by the adipose tissue, IL-6 inhibits insulin signaling (Senn et al., 2003), leptin signaling, and decreases adiponectin secretion (Fernandez-Real & Ricart, 2003; Kershaw & Flier, 2004). Tumor necrosis factor-alpha acts to inhibit glucose uptake and fatty acid oxidation, and it increases the expression of genes involved in de novo synthesis of cholesterol and fatty acids. Finally, TNF-alpha interferes with insulin signaling, which diminishes the uptake of glucose by multiple tissues (Kershaw & Flier, 2004). Plasminogen activator inhibitor-1 inhibits fibrinolysis contributing to the development of artherosclerosis (Darmon et al., 2006).

Endocannabinoid System

The endocannabinoid system (ECS), a signaling system between cells, plays a role in energy balance and metabolism and includes two endocannabinoids, anandamide and 2-arachidonoylglycerol, and the cannabinoid receptor, CB_1 (Matias, Cristino, & Di

Marzo, 2008). The ECS is thought to be overactive in obesity. In genetically obese mice, a deficiency in leptin signaling is associated with elevated hypothalamic endocannabinoid levels. Furthermore, mice fed a high fat diet had more ECS activity in the liver than mice fed standard chow (Matias, Cristino, & Di Marzo, 2008).

Diet may play a role in endocannabinoid synthesis. Due to the availability of phospholipids precursors, the omega-6-polyunsaturated fatty acids may increase endocannabinoid production (Isoldi & Aronne, 2008). Yet, the omega-3-polyunsaturated fatty acids may act to suppress endocannabinoid production (Isoldi & Aronne, 2008). Additionally, other factors may affect endocannabinoid production: glucocorticoids may stimulate endocannabinoid synthesis in the hypothalamus (Matias, Cristino, & Di Marzo, 2008).

The ECS acts centrally by stimulating the appetite system in the hypothalamus and by reinforcing the pleasure pathways in the nucleus accumbens. The ECS acts within the adipose tissue to increase the expression of LPL, thus promoting fat accumulation and reducing the synthesis of adiponectin. The ECS acts within the muscle to decrease glucose uptake and within the liver to increase de novo fatty acid synthesis. Through central and peripheral mechanisms, the ECS acts to promote weight gain and insulin resistance (Isoldi & Aronne, 2008; Matias, Cristino, & Di Marzo, 2008)

Summary

Modifiable and non-modifiable individual characteristics predispose individuals to cardiometabolic disease risk. Complicated social demands, particularly among low income adults, may contribute to feelings of stress and depression further increasing

their cardiometabolic disease risk. Experiments using animal models lay the foundation for understanding the effects of chronic psychological distress on cardiometabolic disease risk. However, little research has systemically investigated HPA activation and dietary patterns as possible biobehavioral responses mediating psychological distress and cardiometabolic disease risk in overweight, sedentary adults.

It is unclear if HPA activation is altered among working adults experiencing psychological distress and if these alterations in cortisol secretion contribute to cardiometabolic disease risk. Establishing this relationship is challenging due to the lack of standardized cortisol sampling protocols making it difficult to interpret available evidence. Dietary energy density is an important determinant of energy intake and a recent study was the first to establish a link between food energy density and abdominal obesity in adults. The relationship between dietary energy density with cardiometabolic disease risk has been difficult to establish due, in part, to the lack of a standardized method for calculating dietary energy density.

Although previous research has established that hospitalized individuals with major depression have altered HPA activation and as many as half over secrete cortisol, the associations among depressive symptoms, perceived stress and HPA activity in community dwelling, working adults is unclear. Importantly, this study examined these associations in community dwelling, working adults prior to the onset of major depressive disorder.

Gender, age and race/ethnicity are non-modifiable individual characteristics associated with high energy dense diets. Access and socioeconomic status are environmental factors that play a role in determining dietary energy density. Previous

research focused on how behaviors such as smoking, alcohol use and physical inactivity link psychological distress with cardiometabolic risk. To date, no scientific data exists to explain the role psychological distress may play in determining dietary energy density. This study provides a greater understanding of how these complex biobehavioral responses interact with psychological factors to modify cardiometabolic disease risk as well as provides a foundation to guide future intervention research aimed at modifying dietary energy density in the context of changing psychological states.

CHAPTER III

Methodology

Introduction

This chapter describes the research design and methodology including a description of the intended sample and recruitment strategies. The study procedures are delineated along with a description of study instruments and relevant reliability and validity data. Findings from the pilot study are presented. Data analysis techniques are discussed and limitations addressed.

Research Design

Design

This prospective, cross-sectional, correlation study examined the associations among psychological factors, biobehavioral responses, and cardiometabolic disease risk in overweight, sedentary adults. Individual characteristics including age, gender, race/ethnicity, overweight, reporting adequate caloric intake (RACI) and dietary restraint were controlled during the analysis to better understand the contribution of potentially modifiable factors (perceived stress, depressive symptoms and dietary energy density) to waist circumference variance. The outcome variable was waist circumference, a marker of abdominal obesity. Participants completed the demographic and clinical information form, Beck Depression Inventory-II (BDI-II), Perceived Stress Scale (PSS), and the Three Factor Eating Questionnaire Revised (TFEQR) at the beginning of the 3 day study period and anthropometric measures were obtained.

Participants collected two saliva samples upon awakening and at 10:00 p.m. on the first week day of the study. They completed a weighed three day food record, weighing and recording their food and beverage intake at home for three consecutive days including one weekend day.

Setting: The data were collected during home visits or, if convenient for the participants, at the research office at the Emory University School of Nursing. Participants completed the food record in their homes over the span of three days. Anthropometric measurements were obtained during the first visit. Computer access for data entry and analysis was provided at the School of Nursing. Data records were maintained in a locked cabinet and locked room at the School of Nursing.

Setting and Sample

Participants were recruited from community organizations in the greater Atlanta metropolitan area. The recruitment sites included a large healthcare agency, a university, neighborhood churches and small businesses. The sample included community dwelling adult men and women who lived or worked in the greater Atlanta area, met the study inclusion and exclusion criteria, were willing to participate, and provided written informed consent.

A convenience sample of 91 sedentary, overweight adults of any race and ethnicity between the ages of 18-65 years old who met the study criteria were invited to participate. The inclusion criteria were: individuals age 18-65 years; BMI of 24.9 – 50 kg/m^2; sedentary lifestyle (self report of less than 30 minutes or more of moderate intensity physical activity three or more days per week)(Centers for Disease Control and

Prevention, 2003); and English fluency (able to give a return verbal explanation of the study).

The exclusion criteria were: pregnant or recently given birth within the past 3 months; currently breastfeeding; history of type 1 or type 2 diabetes; individuals, who in the last 4 weeks, were or had been engaged in weight loss activities including weight loss medications (fenfluramine, sibutramine, orlistat); individuals who were being treated with steroids, valproic acid, phenothiazines, or antidepressants; or individuals who were being treated with highly active retroviral therapy.

Rationale for inclusion/exclusion criteria.

The rationale for inclusion/exclusion criteria are that adults between the ages of 18-65 are more likely to have multiple environmental and occupational demands and social roles (employee, care giver to children, grandchildren or parents) that increase perceived stress; adults with documented BMI of 24.9 – 50.0 will ensure that participants have the health condition of interest. Fluency in English is necessary to comprehend and complete the study questionnaires, particularly the weighed three day food record. Sedentary individuals are selected to control for the effects physical activity may have on abdominal obesity. Pregnant and breastfeeding women or those who have recently given birth have unique dietary fat requirements, and they are in a potential stage of weight gain or loss. Individuals with type 1 or type 2 diabetes also have unique nutritional needs. Individuals who were or had been in the last 4 weeks engaged in weight loss activities, including weight loss medications were excluded because the dynamic nature of weight loss could bias study results (Caro & Dananberg, 2006). Individuals who were being treated with steroids, valproic acid, phenothiazines,

antidepressants, and/or highly active retroviral therapy were excluded due to the confounding effect of these medications on appetite and the associated weight gain (Centers for Disease Control and Prevention, 2008).

Sample Size

Power Analysis and Sample Size (PASS) software was used to determine sample size based on the sixth hypothesis. A multiple regression power analysis procedure was conducted using an R^2 of 0.12 accounted for by four predictor variables and an R^2 of 0.10 accounted for by the four control variables (age, gender, race/ethnicity, and dietary restraint). The predictor variables include perceived stress, depressive symptoms, ED, and salivary cortisol. A sample size of 83 was needed to achieve a power of .80 with an alpha of .05. (Appendix A). Ninety one participants were recruited to allow for 10% attrition in completing the three day food record. Ten percent attrition was estimated based on a drop out rate of 11% from a study examining psychosocial determinants of fat intake (De Bourdeaudhuij, Brug, Vandelanotte, & Van Oost, 2002).

Recruitment

After approval from the Emory University Institutional Review Board was obtained (Appendix B), multiple active and passive strategies were used to recruit potential participants (Denzmore, Dilorio, & McCarty, 2005). Flyers were posted in community settings and participants were recruited from community wellness fairs held within a 20 mile radius of Emory University. Potential participants were asked to contact the principal investigator (PI) by telephone. Once contacted, a screening

procedure was followed to ensure that participants met inclusion and exclusion criteria. Interested participants were met at their home, workplace, or in the research office in the School of Nursing, as they preferred.

Pilot Study

Background and Purpose

Dietary patterns, including food and caloric beverage intake, fast food consumption, and meal skipping may vary by gender (Kant & Graubard, 2006). Among adults, sugar-sweetened soft drinks, pizza and hamburgers which are proxies for fast food consumption, were associated with weight gain (Bes-Rastrollo et al., 2006). The relative importance of caloric beverage consumption in weight gain compared to related factors is not well documented (Flood, Roe, & Rolls, 2006). The purpose of the pilot study was to explore associations among psychological factors, dietary patterns and abdominal obesity among overweight African-American adults enrolled in a longitudinal intervention study to reduce cardiometabolic disease risk.

Conceptual Model

The conceptual model guiding the study was the adapted stress and coping model (Cohen, Kessler, & Gordon, 1995; Dallman, La Fleur et al., 2004; Dallman et al., 2003) which proposes that dietary patterns mediate the relationship between psychological factors and cardiometabolic disease risk.

Sample and Methods

This was a secondary analysis of baseline data from a large lifestyle intervention study. Data from 15 participants collected prior to intervention were analyzed for this pilot study. All participants (n=15) were diagnosed with hypertension and two or more parameters of metabolic syndrome: fasting blood glucose > 100 to < 126 mg/dl; waist circumference > 102 cm for men and > 88 cm for women; triglycerides $\geq$ 150 mg/dl; and HDL cholesterol < 50 mg/dl for women and < 40 mg/dl for men.

Participants recorded their dietary intake for three consecutive days using a standardized food record. Food records were analyzed using a computer program (Food Processor SQL) to determine total daily caloric intake from food and daily caloric intake from beverages. Dietary pattern was obtained from the food record. Demographic and anthropometric data were obtained during an initial visit prior to the intervention. Anthropometrics, including height, weight, and waist circumference, were measured using standardized NHANES procedures by the principal investigator (National Health and Nutrition Examination Survey, 2000). Measured height and weight were used to calculate BMI. The Beck Depression Inventory-II was used to assess depressive symptoms, and the Perceived Stress Scale was used to assess perceived stress (Beck, Steer, & Brown, 1996; Cohen, Kamarck, & Mermelstein, 1983). Due to the small sample size, non-parametric statistical tests were performed. Bivariate correlations between terms were analyzed using Spearman's rho. Gender differences in dietary patterns were analyzed using the Mann Whitney U statistic.

Results

The sample included seven men and eight women with a mean age of 51.1 (range 38-65) years, mean waist circumference: men 107.5 (*SD* 11.8) cm, women 113.6 (*SD* 10.4) cm; mean BMI: men 34.6 (*SD* 5.3) kg/m^2, women 40.2 *(SD* 5.5) kg/m^2. Overall, the mean daily caloric intake was 1756.9 (*SD* 583.4) kilocalories/day, and the mean caloric intake of beverages: 317.1(*SD* 271.5) kilocalories/day. Among men, the mean daily caloric intake was 1925.19 (*SD* 242.30) kilocalories/day, and the mean caloric intake of beverages was 319.47 (*SD* 166.43) kilocalories/day. Among women, the mean daily caloric intake was 1609.71 (*SD* 759.79) kilocalories/day, and the mean caloric intake of beverages was 314.97 (*SD* 351.73) kilocalories/day. The mean BDI-II score was 9.2 (*SD* 10.1) indicating minimal depressive symptoms, and the mean PSS score was 20.73 (*SD* 2.9) indicating low levels of perceived stress.

There was a strong positive correlation between waist circumference and BMI ($r=.817$, $p < .01$). There were no significant differences between men and women on average number of meals skipped ($z=-.245$, $p=.87$) or fast food consumption ($z=.000$, $p=.10$). There was a significant positive correlation between PSS score and grams of carbohydrates consumed in beverages ($r=.598$, $p=.02$). There was a trend toward a significant correlation between PSS score and caloric beverage intake ($r=.505$, $p=.06$). There was a significant positive correlation between the average grams of carbohydrates consumed as beverages and average daily caloric intake ($r=.774$, $p\leq.01$), and a significant negative correlation between caloric beverage consumption and average number of meals skipped ($r=-.667$, $p\leq.01$). There was a significant positive correlation between caloric beverage consumption and average number of meals purchased at

restaurants (r=.531, p=.04). However, there was no significant correlation between perceived stress and waist circumference (r=-.437, p=.10), no significant correlation between caloric beverage consumption and waist circumference (r=-.178, p=.53), and no significant correlation between grams of carbohydrates consumed and waist circumference (r=-.26, p=.36).

Conclusions and Implications

Men and women did not differ on caloric beverage intake, average number of meals skipped, or on average numbers of meals purchased at restaurants. As perceived stress increased, a higher number of grams of carbohydrates in the form of beverages were consumed. As the grams of carbohydrates in beverages increased, the average daily caloric intake increased. As the number of meals skipped increased, the caloric beverage consumption decreased. In addition, a higher caloric beverage intake was reported by individuals purchasing more meals at restaurants. These findings suggest that caloric beverage consumption is important in determining overall energy intake, may be positively related to perceived stress, and may contribute to weight gain. For these reasons, individuals should be encouraged to limit the intake of beverages with added sugar (American Heart Association Nutrition Committee et al., 2006).

These findings confirm the importance of determining a priori the calculation method for dietary energy density and describing the total dietary pattern including both foods and beverages. Although beverages may provide a substantial amount of energy, their high water content results in a relatively low energy density value, potentially diminishing the strength of the relationships between ED and the outcome variables. Due to the large amount of individual day to day variation, these relationships may be

weaker when beverages are included in the calculation (Ledikwe et al., 2005). In this study, water and caloric beverage intake were collected with the same rigor as food intake, and this study examined the relationship between beverage consumption on ED and health outcomes (B.C. Tohill, personal communication, October 11, 2006).

Variables and Measures

The data collection instruments are described, and the relevant reliability and validity information is provided. Study instruments and relevant protocols are included in Appendix C.

Participant Screening Form

A standardized recruitment script and participant screening form were developed to identify adults eligible to participate in the study.

Anthropometric Measurement Form

This form was used to record measured height, weight, waist and hip circumference according to the NHANES protocol (National Health and Nutrition Examination Survey, 2000) and to calculate BMI. Height was measured using the SECA 214 portable stadiometer which measures up to 188 centimeters with graduations of one millimeter. Height was measured with no shoes and recorded in centimeters. Weight was measured in kilograms using the Health-o-Meter 320KL medical scale which has the capacity of 181.5 kilograms with graduations of 0.1 kilograms. Weight was measured in street clothing with no shoes. Waist circumference was measured three times over one layer of clothing. Using the SPSS mean function, the mean of the three measurements was calculated. Waist circumference was measured using the Lufkin

W606PM Executive Diameter Steel tape which has the capacity of 200 centimeters with graduations of one millimeter. The NHANES measures of waist circumference have reliabilities of .99 (Sonnenschein, Kim, Pasternack, & Toniolo, 1993; Wang et al., 2003), and measures of height and weight have reliabilities of .97 (National Health and Nutrition Examination Survey, 2000). Measured height and weight were used to calculate BMI.

The PI had formal coursework on nutritional assessment and anthropometric measurements including laboratory demonstrations and practice. Additionally, the PI viewed the NHANES anthropometric training video, received training by a research nutritionist, and practiced measuring overweight individuals under the supervision of a nutritionist. Based on three measurements obtained by the PI and the nutritionist, the interrater correlation coefficients for weight, height, and waist circumference were r=.999, r=.991 and r=.990, respectively. Based on three measurements, the intrarater correlation coefficients for weight, height, and waist circumference were r=.999, r=.992; and r=.964, respectively. The principal investigator collected all anthropometric measurements on all study participants following the procedure for anthropometric measurements described in Appendix C.

Demographic and Clinical Data Forms

These forms were completed by the participant and used to collect information regarding self reported age, gender, race/ethnicity, health risks related to overweight (NIH, 1998), and duration, frequency, and intensity of physical activity. A health history form was used to collect information regarding current and past health problems. A medication form and a vitamin and supplementation form were used to

collect information regarding the use of prescription medications and vitamins or other supplements. These data were used to describe the clinical characteristics of the sample.

Weighed Three-Day Food Record

This form was used by the participants to record detailed information about every item the participant ate or drank for three consecutive days including one weekend day. These data were used to estimate the kilocalories and gram weight of food and beverages consumed. The weighed three day food record is the gold standard in dietary assessment (Gibson, 2002; Gibson, 2005). To minimize errors, visual illustrations and photographs, written and oral instructions, and examples of serving size were provided. Participants were asked to record details about food purchased or eaten away from home, including name of restaurant, brand of food, and quantity consumed. Participants were asked to record information about how food was prepared including method of cooking, salt and dietary fats added during preparation and/or at the table. Participants were loaned a digital food scale and asked to weigh and record the weight of food prior to and after eating at home. During the first visit, they were instructed and gave a return demonstration on how to use the food scale and keep the food record. The Soehnle 67000 digital scale was used to measure the gram weight of food and beverages. It has a maximum capacity of 2,000 grams with 1 gram graduations. Participants were asked to practice weighing their food at home. Participants were contacted by phone on the first day to problem solve any difficulties encountered using the digital scale, weighing the food, or recording food weight. Once the participant completed the three days of recording, a second visit was scheduled, and a standardized procedure was followed to review the food record for completeness and

accuracy. A variety of measurement aids including photographs of serving sizes, measuring devices including cups, spoons and rulers, and food models were used to ensure accurate estimations. Participants were asked to rate on a 10 point Likert scale to what degree the three days of recording represented their usual eating behaviors.

Estimates of mean caloric intake obtained from three day food records are considered to be more accurate than estimates obtained from food frequency questionnaires or 24 hour dietary recalls (De Castro, 1994). To accurately estimate, three days of reporting are required to obtain a correlation coefficient of .7 between observed and actual energy densities (Ledikwe et al., 2005). Previous research has shown that three days of recording yields accurate information to estimate energy density (Ledikwe et al., 2005). Under- or over-reporting across all food types will have little impact on energy density calculations. However, under-reporting of high energy dense foods or over-reporting of low energy density foods may mask associations (Ledikwe et al., 2005). Reporting of mean caloric intake is higher for three day food records as compared to food frequency or dietary recalls suggesting food records would yield the most accurate information (Gibson, 2005).

The food records were analyzed for calories, fats, carbohydrates, protein, alcohol and weight of foods using the Food Processor SQL Nutrition Analysis software from ESHA Research, Salem, Oregon. This software was available at the Emory University General Clinical Research Center where the food records were analyzed by the study dietitian. This database contains more than 29,000 food items and tracks 133 nutritional components. Energy density was calculated for each day and using the SPSS mean function, the mean energy density for the three study days was calculated. Energy

density of food and beverages combined, beverages alone and food alone were calculated. The food and beverage energy density variable was tested in linear regression models, and are reported in Chapter IV. Results related to beverage energy density and food energy density are reported in the Appendix.

Three Factor Eating Questionnaire Revised (TFEQR)

This is an 18 item self-report questionnaire adapted for an obese population that measures the extent of dietary cognitive restraint (consciously restricting food intake to control body weight or promote weight loss). Dietary restraint was considered a dichotomous variable; a median split was performed and individuals were classified as exhibiting either a high or low level of dietary restraint. Alpha coefficients range from .76-.8 (Karlsson, Persson, Sjostrom, & Sullivan, 2000). Subscale scores correlate well with factors comprising the original scale providing evidence of construct validity in an obese population: the six item cognitive restraint subscale (r=.92), the nine item uncontrolled eating subscale (r=.95) and the three item emotional eating subscale (r=.92) (Karlsson, Persson, Sjostrom, & Sullivan, 2000).

The Beck Depression Inventory II (BDI-II)

The BDI-II is a 21 item self-report questionnaire that assesses depressive symptoms over the prior 2 weeks with higher scores indicating more severe symptoms. In a clinical sample of 500 subjects, the instrument had acceptable reliability coefficients (r= .92); test-retest (1 week interval) correlation was r=.93. The BDI-II has a positive correlation (r=.71) with the Hamilton Rating Scale for Depression (Beck, Steer, Ball, & Ranieri, 1996; Steer, Ball, Ranieri, & Beck, 1997) providing evidence of

construct validity. The BDI-II was selected because it is useful in adults with chronic disease risk factors. The subscales distinguish cognitive and somatic dimensions of depressive symptoms. In this study, the total score was used in the analysis.

Perceived Stress Scale (PSS)

The PSS is a 10-item questionnaire that measures the degree to which participants find their lives to be unpredictable or uncontrollable during the past month. The instrument is sensitive to stress due to environmental and social demands such as ongoing life situations, specific events, and future events. Alpha coefficients range from .84, .85 and .86 indicating the instrument is internally reliable (Cohen, Kamarck, & Mermelstein, 1983). Test-retest reliability within 2 days was .85 and after 6 weeks dropped to .55 (Cohen, Kamarck, & Mermelstein, 1983). Correlations between PSS and number of life events ranged from .20 to .39; the impact of life events ranged from .24 to .49 providing evidence of construct validity (Cohen, Kamarck, & Mermelstein, 1983). Correlations between the Center for Epidemiologic Studies Depression scale and physical symptoms were .31 and .38 providing additional evidence of construct validity (Cohen, Kamarck, & Mermelstein, 1983). The PSS was selected because of its sensitivity to stress due to ongoing life situations. Assessing perceived stress during a month time period, captures a more representative picture of chronic stress than instruments that assess perceived stress over a shorter period (Cohen, Kamarck, & Mermelstein, 1983).

Cortisol Secretion

Morning salivary cortisol levels were used to assess HPA activity. A 1 milliliter sample of saliva was collected using a sampling device called a Salivette manufactured by Sarstedt (Rommelsdorf, Germany). The salivette consists of a small cotton swab inside a centrifuge tube. Participants were taught to collect samples according to the manufacturers' guidelines, and were instructed not to eat, drink, smoke, brush teeth, or chew gum for 30 minutes prior to using the salivette. Participants were instructed to lightly chew on the cotton swab for three to four minutes to ensure adequate saturation. Participants were instructed to collect two saliva samples on the first study weekday. These samples were to be collected upon awakening before getting out of bed and at 10:00 p.m. The expected salivary cortisol values are 0.18-0.95 µg/dl for 8:00 a.m. and 0.005-0.17 µg/dl for 11:00 p.m. (Salimetrics, 2006). Participants were provided with the materials, taught how to collect the specimen, and asked to store the samples in their refrigerator. Participants completed a morning and evening sample collection card.

During the second study visit, the PI collected the two samples from each participant and reviewed the information on the sample collection card for completeness and accuracy. In the event that there was an inadequate amount of saliva collected, participants were asked to repeat the collection. Samples were taken to the General Clinical Research Center laboratory where they were labeled with the participant's study identification number and the date and time of collection. The samples were then centrifuged at 1,000 times gravity for two minutes and stored at minus 20 degrees Celsius. The minimum volume recovered was 1 milliliter of saliva. The analysis was performed at the Yerkes National Primate Research Center Biomarkers Core Laboratory

using a commercially prepared kit produced by Diagnostic Systems Laboratories (Webster, Texas). Salivary cortisol was measured by enzyme immunoassay with an average intra-assay coefficient of variation 8.7% for low and 5.2% for high concentration; average inter-assay coefficient of variation was 2.9% for low and 4.4 % for high concentration. These coefficients of variation were deemed acceptable. Saliva cortisol represents the unbound, free hormone and reflects the free fraction of cortisol in plasma. Correlations between salivary and plasma cortisol are reported to be strongly correlated ($r=.91$, $p<0.0001$) providing construct validity for this measure (Salimetrics, 2006).

Procedure

During the initial appointment, the study was explained to participants and they were given an opportunity to ask questions. See Figure 2 for a depiction of the study procedure. The participant's understanding of the study was elicited by asking for a return explanation. Participants then signed the informed consent and HIPAA documents. A copy of the consent and HIPAA documents was given to each participant. Using a standardized procedure, the participant was taught how to use the food scale, how to estimate the portion size for food that could not be weighed, how to keep the food record, and how to collect and store the saliva samples. In addition to the verbal instruction, participants were given a booklet that included instructions with pictures. Participants were loaned a digital food scale so that they could weigh their food and beverages at home for three consecutive days including one weekend day. Anthropometric measurements (height, weight, and waist circumference) were obtained.

On the first study day, the participant was contacted to clarify any questions. At the completion of the three day recording period, the second study visit was completed. During the second visit, the study questionnaires, food record and saliva samples were collected. A standardized procedure was used to review all data with the participant. Any incomplete items on the questionnaires were clarified and completed. The saliva sample card was reviewed to ensure samples were collected and handled appropriately. A standardized procedure was used to review the food record to ensure completeness and accuracy in estimating items that were composite foods or any items that were not weighed. Using a standardized protocol, details of the food record including food preparation techniques were clarified. Participants were asked to compare and rate how similar each day of recording was to their usual intake. At the conclusion of the second visit, participants received a $25.00 gift card.

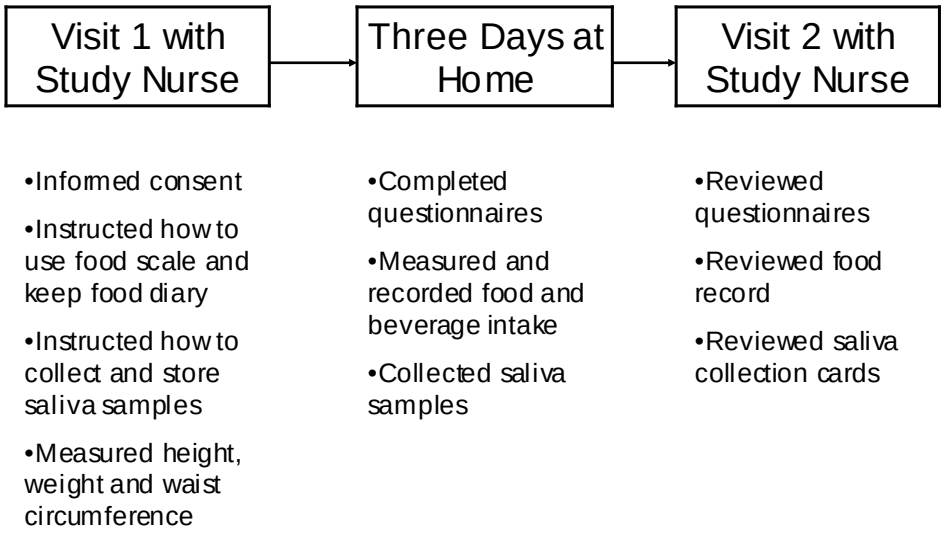

Figure 2. Study Procedure

At the conclusion of the second visit, saliva samples were taken to the General Clinical Research Center and were labeled with participant identification number, date and time of sample collection, and were processed. The BDI-II questionnaire was reviewed for completeness and scored. If the BDI-II score indicated moderate depressive symptoms with a response of two or higher on items two and nine or a total score greater than or equal to 20 and less than 29, a standardized procedure for the BDI-Level I Alert was followed which included contacting the participant within 48 hours, making a referral, and notifying the academic advisor within 48 hours. If the BDI-II score indicated severe depression with a response of three on item nine or greater than or equal to two on both items two and nine, or if the total score was greater than or equal to 29, then the procedure for BDI-II Level II Alert was followed which included contacting the participant as soon as possible, making a referral for emergency psychiatric assistance, and contacting the academic advisor within 24 hours. In addition, a one week follow up phone call was completed to determine if the participant had gotten help and how he/she was doing.

The study dietitian reviewed each food record for completeness and for clarifying information. If the dietitian needed clarifying information, the PI contacted the participant to obtain the additional information. Once the food record was analyzed, the participant was mailed an individualized report which showed how his/her intake compared to the recommendations for someone of similar age, gender, and BMI.

Data Analysis

Data Management Plan

Data were analyzed using the most current version of Statistical Program for Social Sciences (SPSS)-Personal Computer (PC) software. Nutrient analysis data were saved in an Excel file which was then entered into SPSS using a double data entry procedure. All other data was collected on teleforms, scanned and saved in an Excel file which was imported into SPSS and combined with the nutrient analysis data into one dataset.

Prior to data collection, a detailed code book was written to ensure consistent data entry; all responses for continuous variables were entered in the same form with the same degree of precision. Levels were assigned for each categorical variable and each level was defined. Missing values in categorical data were assigned to the ninth level which was labeled “unknown”. Missing continuous data were assigned a period. During data analysis, missing values were imputed using the missing value analysis in SPSS. Cross classification checks were performed to ensure that participants responded in a manner that was internally consistent. A record was kept to track when errors were found and how they were corrected.

Each participant was assigned a unique identification (ID) number (beginning with the number 111) which was maintained with the data. After scanning the teleforms, data were compared visually with paper documentation to ensure accurate entry into the database. Frequency distributions, with particular attention to the lowest and highest values, were checked for all continuous variables to identify outliers.

Outlying and implausible data points were compared to the original data source to verify that they were valid. Internal data consistency checks were run when appropriate.

For each variable, a frequency distribution was used to determine the amount of missing data. The TFEQR was the only questionnaire with missing values. Two participants were missing TFEQR values. One of the participants was missing values for three items and was excluded from the TFEQR analysis. The other participant was missing a value for one item. A SPSS missing value analysis was performed and the missing value was replaced with the imputed value.

Psychometric properties of each instrument were assessed by examining the internal consistency reliability (Cronbach's alpha) for each of instruments. The data distribution was examined to identify variables with limited variance, skewness, or ceiling and floor effects, indicating that data may need to be transformed prior to analysis. BDI-II scores were right skewed and underwent square root transformations for linear regression modeling.

A detailed procedure was written regarding the analysis and coding of the weighed three day food records. For each day of recording, all foods and beverages were entered by meal and analyzed for energy (kilocalories), weight (grams), total fats (grams), saturated fats (grams), total protein (grams), total carbohydrates (grams), total sugars (grams) and fiber (grams). Foods consumed were separated from beverages and then reanalyzed for energy, weight, total fats, saturated fats, protein, carbohydrates, sugars and fiber. Each item on the food record was categorized as a food or beverage by two independent coders. Foods were defined as solid and liquid items that would typically be chewed, eaten with a utensil, or consumed to replace a meal and included

soft and liquid foods such as ice cream, soups, broths, and liquid meal replacements. Beverages were defined as liquids that would typically be drank including juice, milk, smoothies, milkshakes, soft drinks, alcoholic drinks, and water. Items such as sugar, creamer, and lemon juice that are added to liquids and consumed as a drink were classified as beverages. For example, if lemon juice was added to iced tea, it was classified as a beverage; if lemon juice was drizzled on fish, the lemon juice was classified as a food. If there was a classification discrepancy between the two coders, the original food record was reviewed, and the discrepancy discussed until agreement was reached.

Dietary energy density was examined in three ways: first, as the mean energy density value for all foods and beverages consumed (food and beverage ED); second, as the mean energy density value for beverages consumed (beverage ED); and third, as the mean energy density value for food only (food ED). All participants reported food intake for all three days of recording. One participant did not report beverage intake for one study day; therefore, that participant was excluded from analysis involving food and beverage energy density and beverage energy density.

The total energy density from food and beverages for each day was divided by the total weight of food and beverages reported to determine the daily food and beverage energy density value. Using the SPSS mean function, the day one, day two and day three food and beverage ED values were averaged to derive a mean food and beverage ED value for each participant. Total energy intake from beverages for each day was divided by the total weight of beverages reported to determine the daily beverage energy density values. Using the SPSS mean function, day one, day two and

day three beverage ED values were averaged to derive a mean beverage ED value for each participant. Total energy intake from food for each day was divided by the total weight of food reported to determine the daily food energy density values. Using the SPSS mean function, the day one, day two and day three food ED values were averaged to derive a mean food ED value for each participant. To simplify reporting, the results of the analysis related to food and beverage energy density are reported in the body of Chapter IV. Results of the analysis related to beverage energy density and food energy density are reported in Appendix E.

Using the SPSS mean function, the estimated caloric intake from day one, day two and day three of recording were averaged to derive a mean caloric intake value for each participant. For each participant, the Harris-Benedict equation, which takes age, gender, height, weight, and activity level into account, was used to estimate energy expenditure (Harris & Benedict, 1919). This estimated energy expenditure value indicates the caloric intake needed to maintain current body weight. For the Harris-Benedict equation calculation, the participants were assumed to be inactive (Harris & Benedict, 1919). The estimated mean caloric intake from the three day food record was compared to the estimated energy expenditure value calculated using the Harris-Benedict equation (Harris & Benedict, 1919). Participants with an estimated caloric intake equal to or greater than the estimated energy expenditure value were considered to have reported adequate caloric intake. Participants with an estimated caloric intake less than the estimated energy expenditure value were considered to have reported an inadequate caloric intake. To assess the nature of underreporting, the macronutrient

composition, food group patterns and energy density values of those reporting adequate caloric intake were compared to those reporting inadequate caloric intake.

In order to adjust for reporting inadequate caloric intake, a continuous variable (reporting adequate caloric intake [RACI]) was created. This continuous variable was calculated as the ratio of the estimated energy expenditure value to the reported mean caloric intake. The larger the RACI value, the greater the discrepancy between the estimated energy expenditure and the estimated mean caloric intake. A RACI equal to one indicated that the estimated energy expenditure value was equal to the reported mean caloric intake. A value greater than one indicated the participant reported an inadequate caloric intake. In contrast, a value less than one indicated the participant reported an adequate caloric intake and consumed more calories than his/her estimated energy expenditure.

In addition to describing the dietary energy density values, two other aspects of the dietary pattern were characterized. The macronutrient composition was characterized including the quality and quantity of dietary fat intake. The food group (grains, vegetables, fruits, milk, and meat/beans) pattern was characterized based on the Dietary Guidelines for Americans which take age, gender, height, weight and physical activity into account (U. S. Department of Health and Human Services & U.S. Department of Agriculture, 2005). The food group pattern analysis reports the ounce equivalent for grains and meat/beans and the cup equivalent for vegetables, fruits and milk.

For all questionnaire data, instruments were scored according to the specific procedures; subscale and total scores were calculated from the individual items. Initial

data analysis included descriptive statistics of sample characteristics, examination of the distribution of the data, type and extent of missing data, assessment of the psychometric properties of instruments, and determination that statistical assumptions were met. Preliminary review of the salivary cortisol data revealed a wide variation in the collection time for the 10:00 p.m. cortisol sample. Due to the large variability, the evening value and the cortisol change over the day were omitted from the analysis. The morning salivary cortisol level was used in sequential linear regression models.

Underlying assumptions of sequential linear regression were examined; normality was visually investigated using frequency distributions and scatter plots as well as statistical indices of skewness and kurtosis. Based on this evaluation, BDI-II scores, BMI, beverage ED values and mean waist circumference underwent a square root transformation for sequential linear regression modeling. For purposes of sequential linear regression modeling, race and TFEQR were categorized as dichotomous variables and labeled African-American or Caucasian and high or low dietary restraint, respectively.

Data Analysis Procedures

This study examined dietary pattern and HPA activation as potential mediating variables between psychological factors and cardiometabolic disease risk. Mediating variables attempt to explain the association between a predictor and outcome variable. In order to determine if a variable acts as a mediator, three conditions must be satisfied. First, there must be a significant correlation between the predictor variable and the mediating variable. Second, the mediating variable must be significantly associated with the outcome variable while holding the predictor variable constant. Third, the predictor

variable must be significantly correlated with the outcome variable (Baron & Kenny, 1986; Lindley & Walker, 1993). The correlations between perceived stress, depressive symptoms, dietary energy density and abdominal obesity, and the correlations between perceived stress, depressive symptoms, salivary cortisol and abdominal obesity will be evaluated in this manner. To test each of the hypotheses, sequential linear regression was conducted with the dependent variable of interest. Models were constructed entering variables that were significant at the alpha .05 level. Variables that were not significant at an alpha of .20 or below were excluded. If the model was significant, then a reduced model was tested, and variables in the full model with an alpha of .20 or below were entered into the reduced model. These analyses identified individual characteristics that are important in explaining these relationships.

Specific aim 1.

Specific aim 1: To examine the associations between psychological factors (perceived stress and depressive symptoms) and biobehavioral responses (dietary pattern and cortisol secretion) in sedentary, overweight adults.

H1: Psychological factors (perceived stress and depressive symptoms) will explain a significant amount of variance in dietary pattern (dietary energy density) while controlling for age, gender, race/ethnicity, BMI, reporting adequate caloric intake (RACI) and dietary restraint.

First, data from all participants were examined using Pearson's correlations to determine if there were significant correlations between the independent variables (PSS and BDI-II scores) and the potential mediating variable (food and beverage ED). Second, sequential linear regression modeling was used to determine the amount of

variation in the potential mediating variable (food and beverage ED) explained by the independent variables (PSS and BDI-II scores) while holding individual characteristics constant (age, gender, race, BMI, RACI and TFEQR score). Individual characteristics were first entered individually into the model in a block to control for their influence on the mediating variable. The PSS and BDI-II scores were then individually entered as a block to determine their contribution to the model.

Variables in the full model with an alpha of .20 or below were entered to determine the amount of food and beverage energy density variance explained by the reduced model. If a significant relationship was demonstrated between the independent and mediating variable, the first condition was satisfied and it was deemed appropriate to continue testing the mediating model. If a significant relationship was not demonstrated between the independent and mediating variables, no further testing of the mediating model was warranted (Baron & Kenny, 1986; Kachigan, 1991).

H2: Psychological factors (perceived stress and depressive symptoms) will explain a significant amount of variance in HPA activation (morning salivary cortisol value) while controlling for menopause status, race/ethnicity, BMI and dietary restraint.

First, Pearson's correlations were used to determine if there were significant correlations between the independent variables (PSS and BDI-II scores) and the potential mediating variable (morning salivary cortisol value). Second, sequential linear regression models were used to determine the amount of variation in the potential mediating variable (morning salivary cortisol value) explained by the independent variables (PSS and BDI-II scores) while holding individual characteristics constant (menopause status, race/ethnicity, BMI and TFEQR score). Individual characteristics

were entered into the model as a block to control for their influence on the mediating variable. PSS and BDI-II scores were then entered as a block to determine their contribution to the model. If a significant relationship was demonstrated between the independent and mediating variable, the first condition was satisfied and it was deemed appropriate to continue testing the mediating model. If a significant relationship was not demonstrated between the independent and mediating variables, no further testing of the mediating model was warranted (Baron & Kenny, 1986; Kachigan, 1991).

Specific aim 2.

Specific aim 2: To examine the associations between biobehavioral responses (dietary pattern and HPA activation) and cardiometabolic disease risk (abdominal obesity) in sedentary, overweight adults.

H3: Dietary pattern (dietary energy density) will explain a significant amount of variance in abdominal obesity (waist circumference) while controlling for age, gender, race/ethnicity, dietary restraint, perceived stress and depressive symptoms.

Pearson's correlations were used to determine if there was a significant association between the potential mediating variable (food and beverage energy density) and the outcome variable (waist circumference). The procedures described by Mendoza and others (2007) using food energy density to predict waist circumference informed the analysis of this hypothesis (Mendoza, Drewnowski, & Christakis, 2007). First, they standardized the energy density values so that each unit of standardized energy density was equal to a one standard deviation change. They controlled for age, gender, race, education, income and physical activity. Additionally, they controlled for the mean daily total amount of food in grams. They tested a series of linear regression models,

stratified by sex, to evaluate the association between the standardized energy density variable and waist circumference (Mendoza, Drewnowski, & Christakis, 2007). In this analysis, the food and beverage energy density value was centered and standardized. In order to compute this value, the mean food and beverage energy density was subtracted from the observed values and then divided by the standard deviation. By centering and standardizing the food and beverage energy density, the correlation between the food and beverage energy density value and food and beverage weight was removed.

Sequential linear regression modeling was used to determine the amount of variation in the outcome variable (waist circumference) explained by the potential mediating variable (food and beverage energy density) while holding individual characteristics (age, gender, race/ethnicity, BMI, TFEQR, PSS and BDI-II scores) constant. Individual characteristics were entered into the model as a block to control for their influence on the mediating variable. The food and beverage ED value was then entered to determine its contribution to the model. A significant F change indicates that the food and beverage energy density value explained additional variance in waist circumference above that accounted for by the individual characteristics including the food and beverage weight.

If a significant relationship was demonstrated between the mediating and outcome variable, the second condition was satisfied and it was deemed appropriate to continue testing the mediating model. If a significant relationship was not demonstrated between the mediating and outcome variables, no further testing of the mediating model was warranted (Baron & Kenny, 1986; Kachigan, 1991).

H4: HPA activation (morning salivary cortisol value) will explain a significant amount of variance in cardiometabolic disease risk (abdominal obesity) while controlling for menopause status, race/ethnicity, PSS and BDI-II scores.

First, Pearson's correlation was used to determine if there was a significant association between the potential mediating variable (morning salivary cortisol value) and the outcome variable (waist circumference). Second, sequential linear regression modeling was used to determine the amount of variation in the outcome variable (waist circumference) explained by the potential mediating variable (morning salivary cortisol value) while holding individual characteristics constant. Individual characteristics were entered into the model as a block to control for their influence on the mediating variable. The morning salivary cortisol value was then entered to determine its contribution to the model.

If a significant relationship was demonstrated between the mediating and outcome variable, the second condition was satisfied and it was deemed appropriate to continue testing the mediating model. If a significant relationship was not demonstrated between the mediating and outcome variable, no further testing of the mediating model was warranted (Baron & Kenny, 1986; Kachigan, 1991).

Specific aim 3.

Specific aim 3: To examine the associations among psychological factors (perceived stress and depressive symptoms), biobehavioral responses (dietary pattern and HPA activation) and cardiometabolic disease risk (abdominal obesity) in sedentary, overweight adults.

H5: Psychological factors (perceived stress and depressive symptoms) will explain a significant amount of variation in abdominal obesity (waist circumference) while controlling for age, gender, race and dietary restraint.

First, Pearson's correlations will determine a significant association between the independent variables (PSS and BDI-II scores) and the outcome variable (waist circumference). Second, simple linear regression modeling was used to determine the amount of variation in the outcome variable (waist circumference) explained by perceived stress scores and depressive symptom scores. In a simple linear regression model, perceived stress scores were entered to determine their contribution in explaining waist circumference variance. In a second simple linear regression model, depressive symptom scores were entered to determine their contribution in explaining waist circumference variance. Third, a sequential linear regression model was used to determine the amount of waist circumference variance explained by the addition of perceived stress and depressive symptoms while holding individual characteristics constant. Individual characteristics (age, gender, race and dietary restraint) were entered into the model as a block to control for their influence on waist circumference variable. The perceived stress and depressive symptom scores were then entered to determine their contribution to the model. In the full model, if the F change statistic and model were significant, a reduced model was tested.

If a significant relationship exists between the independent and outcome variable, the third condition was satisfied and it was deemed appropriate to continue testing the mediating model. If a significant relationship did not exist between the

independent and outcome variables, no further testing of the mediating model was warranted (Baron & Kenny, 1986; Kachigan, 1991).

H6: Dietary pattern (dietary energy density) and HPA activation (morning salivary cortisol value) mediate the relationship between perceived stress and depressive symptoms with abdominal obesity (waist circumference) while controlling for age.

Pearson's correlations will determine a significant association between the independent variables (PSS and BDI-II scores), the mediating variables (dietary energy density and morning salivary cortisol value) and the outcome variable (waist circumference).

If the three underlying conditions of the mediating model were satisfied, three additional statistical approaches were used to determine if the mediating variable (morning salivary cortisol levels or food and beverage energy density values) explained variation in the outcome variable (waist circumference). Complete mediation occurs when the independent variable no longer affects the dependent variable while controlling for the mediator. Partial mediation occurs when the path from the independent to the dependent variable is reduced in absolute size, but it is still different from zero while controlling for the mediator. First, the Baron and Kenny (1986) approach evaluates a series of simple linear regression models to test the direct and total effects of the independent, mediating and outcome variables (Baron & Kenny, 1986; Preacher & Hayes, 2004). Second, the Sobel approach evaluates the indirect effect and tests for significance using the normal distribution. If the result is significant, it demonstrates that the total effect of the dependent variable on the independent variable is significantly reduced when the mediating variable is added to the model indicating

partial mediation (Preacher & Hayes, 2004). Third, the indirect effects are assessed with the bootstrap sampling distribution technique. This is a non-parametric approach that makes no assumptions about the shape of the distribution. A confidence interval is calculated, and if zero does not fall in the confidence interval, the test is significant confirming the mediation model (Preacher & Hayes, 2004).

Finally, a sequential linear regression model was tested to examine the associations among the independent, mediator, and outcome variables. Perceived stress and depressive symptom scores were individually entered in a block. Morning salivary cortisol levels and food and beverage energy density values were then individually entered in a block to determine the amount of variation in waist circumference explained by the mediating variables.

Additional Analysis

To better understand the dietary pattern of individuals with depressive symptoms, an additional analysis was conducted. First, a food and non-alcoholic beverage energy density value was calculated. To calculate this value, the kilocalories in alcoholic beverages were subtracted from the kilocalories in food and beverages. Second, the grams in alcoholic beverages were subtracted from the grams in food and beverages. This was done for each of the three study days. The food and nonalcoholic beverage energy density value was calculated for each study day. Using the SPSS mean function, the mean food and non-alcoholic beverage energy density values for day one, day two and day three were calculated.

A sequential linear regression model was used to determine the amount of total variance in food and non-alcoholic beverage energy density explained by the addition of

depressive symptom scores. Individual characteristics were entered as a block to control for their influence on food and non-alcoholic beverage energy density. The depressive symptom scores were then added to the model to determine their contribution in explaining food and non-alcoholic beverage energy density (Kachigan, 1991).

Summary

This was a prospective, cross-sectional, correlation study investigating dietary pattern and HPA activation as biobehavioral mediators between psychological factors and cardiometabolic disease risk in sedentary, overweight adults. A convenience sample of 91 working adults was enrolled in the study. Sedentary adults between the ages of 18-65 years old with a BMI of 24.9-50 kg/m^2 were eligible to participate.

A description of the pilot study, examining the associations among perceived stress, depressive symptoms and dietary pattern, was provided. Results of the pilot study demonstrated a preliminary trend in the relationships between perceived stress and dietary pattern, concepts of specific aim 1.

Data were analyzed using the most current version of SPSS-PC software. Teleforms were used to scan all questionnaires into the database. Dietary pattern analysis data were entered using double data entry. The data management plan delineated the procedures used to identify and resolve outliers and missing values. Linear regression models were tested to examine dietary pattern and HPA activation as potential mediators between psychological factors and cardiometabolic disease risk. Three statistical approaches were used to test indirect effects in simple mediation models.

CHAPTER IV

Results

Introduction

This chapter presents the results of the data analysis. First, sample demographics and clinical characteristics are presented. Second, descriptive statistics for the main study variables are presented, followed by findings related to the specific aims and hypotheses. Data were analyzed using the Statistical Program for the Social Sciences-PC (SPSS), version 15.0 software (Chicago, Illinois). The distribution, skewness and possible outliers of each variable were investigated to ensure that there were no measurement or recording errors.

Description of the Sample

Demographic Characteristics of the Sample

Participants were recruited from a variety of sources including community health fairs and through recruitment flyers posted in community settings. The majority were self-referral (n=83, 80.2%), and less than a quarter (n=18, 19.8%) were recruited from health fairs. Of these, 91 adults consented to participate in the study. Four participants were excluded from data analysis: two participants were unable to complete the study activities, and two participants were considered extreme outliers due to underreporting of their caloric intake. In total, 87 participants comprised the final study sample.

Demographic characteristics of the enrolled sample are described in Table 1. The sample was composed primarily of women with a mean age of 41.3 (*SD* 10.2) years. The ethnic composition was primarily non Hispanic/Latino, and the racial composition was primarily African-American. The majority of participants were well educated and graduates of college or graduate and professional schools, and only a few had only a high school diploma, GED or technical/vocational training. The majority of participants were employed full time, and more than a third were professionals or executives. More than a quarter had a total combined family income of $40,000-$59,999.

Clinical Characteristics of the Sample

Cardiometabolic risk factors.

Overall, the mean BMI was 32.13 (*SD* 6.09) kg/m^2. The mean BMI was 30.98 (*SD* 4.54) kg/m^2 for men and 32.54 (*SD* 6.54) kg/m^2 for women. The majority of participants (n=50, 57.5%) were classified as obese with a BMI greater than or equal to 30 kg/m^2. The majority of participants were relatively healthy with a small number reporting a history of hypertension (n=18, 20.7%), high cholesterol (n=13, 14.9%), chest pain (n=5, 5.7%). None of the participants reported a history of stroke or heart failure. A few of the participants reported conditions that limited their ability to participate in physical activity (n=5, 5.7%).

Other health conditions.

A small number of participants reported other health conditions including a history of asthma (n=5, 6.2%), anemia (n=13, 16.0%), arthritis (n=7, 8.6%), migraine headaches (n=11, 13.6%), gastric reflux (n=12, 14.8%), irritable bowel (n=2, 2.5%),

pneumonia (n=3, 3.7%), seizures (n=1, 1.2%), sleep apnea (n=3, 3.7%). No participants reported a history of cancer, glaucoma, sickle cell or tuberculosis. The majority of participants (n=53, 64.6%) were not currently taking any medications, and 53.7% (n=44) reported vitamin or supplement use.

Table 1

Demographic Characteristics (N=87)

Individual characteristic	Mean *(SD)*/N *(%)*	Range
Age in years – *M (SD)*	41.3 (10.2)	20-64
Men	38.4 (9.9)	21-64
Women	42.4 (10.1)	20-64
Gender – *n (%)*		
Male	23 (26.4)	
Female	64 (73.6)	
Ethnicity – *n (%)*		
Non Hispanic/Latino	84 (96.6)	
Hispanic/Latino	3 (3.4)	
Race – *n (%)*		
White	43 (49.4)	
African-American	44 (50.6)	
Education – *n (%)*		
High school graduate or GED	7 (8.0)	
Technical/vocational school	2 (2.3)	
Completed some college	27 (31.0)	
College graduate	27 (31.0)	
Post graduate or professional school	24 (27.6)	
Total Combined Family Income – *n (%)*		
Less than $19,999	3 (3.4)	
$20,000-$39,999	13 (14.9)	
$40,000-$59,999	25 (28.7)	
$60,000-$79,999	14 (16.1)	
$80,000 and greater	21 (24.1)	
Don’t know	3 (3.4)	
Prefer not to answer	8 (9.2)	
Type of work – *n (%)*		
Professional/executive	33 (37.9)	
Management/administration	19 (21.8)	
Clerical/Sales	18 (20.7)	
Skilled labor	6 (6.9)	
Semi-skilled labor	2 (2.3)	
Unskilled labor	2 (2.3)	
Not applicable	7 (8.0)	

Individual Behavioral Characteristics of the Sample

Dietary restraint.

Of the participants, 45.3% (n=39) were categorized as exhibiting high dietary restraint. Of these, 12 men and 27 women exhibited high restraint. Overall, there were no significant differences on age, gender, race, education, income or waist circumference between participants exhibiting high versus low restraint. Overall, individuals reporting high dietary restraint had significantly lower BMIs (t=-2.589, p=.01), less depressive symptoms (t=-2.844, $p \leq .01$), and less perceived stress (t=-3.323, $p \leq .01$) than individuals reporting low dietary restraint. An interesting gender difference was observed. Among men, there were no significant differences on BMI, depressive symptoms, perceived stress or waist circumference between those with high compared to low restraint. In contrast, women with high restraint had lower BMIs (U=300.0, z=-2.583, p=.01), less depressive symptoms (U=278.5, z=-2.888, $p \leq .01$), less perceived stress (U=324.0, z=-2.253, p=.02) and lower waist circumference (U=345.5, z=-1.951, p=.05) than women with low restraint.

Smoking and reported alcohol intake.

Overall, the majority of participants denied currently smoking (n=82, 94.3%), and the majority of participants did not report alcohol intake (n=58, 66.7%). The mean age of those reporting alcohol intake was 43.5 years compared to 40.2 years and this difference was not significant (U=663.0, z=-1.604, p=.11). There was a significant difference on race (Chi square=4.506, p=.04) with nineteen (44.2%) whites and ten (22.7%) African-Americans reporting alcohol intake.

Individuals who reported alcohol intake had significantly lower BMIs (U=608.0, z=-2.098, p=.04) than those who did not report alcohol intake. There was no significant difference on waist circumference (U=664.0, z=-1.594, p=.11) between those who did and did not report alcohol intake. Among those reporting alcohol intake, the mean alcohol servings per day was 1.04 (*SD* .53) for men (n=7) and .92 (*SD* .72) for women. The majority (n=22, 75.9%) did not exceed the recommendation of two drinks per day for men and one drink per day for women. Among individuals reporting alcohol intake, the mean kilocalories of alcohol consumed was 148.15 (*SD* 154.87) kilocalories, and the mean weight of alcohol consumed was 262.10 (*SD* 386.23) grams.

There were significant differences in the macronutrient composition and dietary patterns of those who reported alcohol intake compared to those who did not report alcohol intake. In terms of macronutrient composition, those who reported alcohol intake had a lower intake of total carbohydrates (U=626.0, z=-1.936, p=.05) and lower intake of total sugars (U=586.0, z=-2.296. p=.02) than those who did not report alcohol intake. Individuals who reported alcohol intake and adequate caloric intake (n=15) had higher intakes of total fats (U=39.0, z=-2.2880, p≤.01), total carbohydrates (U=35.0, z=-3.055, p≤.01), dietary fiber (U=52.0, z=.021, p=.02), total sugars (U=23.0, z=-3.579, p≤.01), protein (U=34.0, z=-3.099, p≤.01) and kilocalories (U=10.0, z=-4.146, p≤.01) than those who reported alcohol intake and inadequate caloric intake (n=14). In terms of dietary pattern, those who reported alcohol intake consumed fewer cups of fruit (U=593.0, z=-2.240, p=.03) than those who did not report alcohol intake. Individuals who reported alcohol intake and adequate caloric intake (n=15) had a higher intake of

grain (U=54.0, z=-2.226, p=.03) and a higher intake of vegetables (U=56.0, z=-2.139, p=.03) than those who reported alcohol intake and inadequate caloric intake (n=14).

Of the 36 participants who reported adequate caloric intake, there was no significant difference in kilocalories, total fat, saturated fat, total carbohydrates, cholesterol, dietary fiber, total sugars and protein between those who reported alcohol intake (n=15) compared to those who did not report alcohol intake (n=21). There was a trend toward a significant difference in cups of vegetables and ounces of beans consumed. Those reporting adequate caloric intake and alcohol intake had a higher intake of vegetables (U=100.50, z=-1.829, p=.07) and a higher intake of beans (U=102.0, z=-1.781, p=.08) than those reporting adequate caloric intake and no alcohol intake. These findings are consistent with the trend in significance toward those reporting adequate caloric intake and no alcohol intake consuming higher energy dense foods and beverages (U=101.0, z-1.813, p=.07) than those reporting adequate caloric intake and alcohol intake.

Those who reported alcohol intake had a higher beverage energy density than those who did not report alcohol intake (U=560.0, z=-2.434, p=.015); however, there were no significant differences in food and beverage energy density (U=682.0, z=-1.320, p=.19), food energy density (U=788.0, z=-.477, p=.63) or food and non-alcoholic beverage energy density (U=729.0, z=-.891, p=.37). These two groups did not differ on reporting adequate caloric intake (U=740.0, z=-.91, p=.36), dietary restraint (U=776.0, z=-.463, p=.64), depressive symptoms (U=840.0, z=-.009, p=.99), perceived stress (U=800.0, z=-.370, p=.71) or morning salivary cortisol level (U=632.5, z=-.718, p=.47).

Environmental Factors

Environmental factors that may affect access to high quality, low energy dense foods are described. In general, households were composed of 2.8 (*SD* 1.6) people. Of these, the mean number of adults per household was 2.0 (*SD* 1.2), the mean number of children per household was .8 (*SD* 1.1) and the mean number of wage earners per household was 1.9 (*SD* .08). The majority of participants lived less than 5 miles away from a grocery store (n=78, 89.7%) and either owned a car or had access to a car (n=86, 98.9%). The majority of participants (n=68, 78.2%) shopped primarily at supermarkets or grocery stores, and 14.9% (n=13) shopped at stores that sell in bulk. In contrast, a small number of participants shopped primarily at convenience stores (n=3, 3.4%) or farmer's markets (n=3, 3.4%).

Psychological Factors

Perceived stress.

Overall, the mean PSS score was 16.47 (*SD* 7.19, range 0-33) suggesting that the participants perceived their levels of stress to be similar to that of other working adults. The majority of participants (n=85, 97.7%) scored 30 or less indicating minimal levels of stress. The mean PSS score was 16.13 (*SD* 6.65, range 4-32) for men and was 16.59 (*SD* 7.42, range 0-33) for women. Men and women did not differ on mean PSS score (U=672.5, z=-.612, p=.54).

Depressive symptoms.

The mean BDI-II score was 8.67 (*SD* 8.34, range 0-42, median 6.0). The majority of participants (n=68, 78.2%) scored less than or equal to 13 indicating no or minimal depressive symptoms, ten (11.5%) scored 14 through 19 indicating mild

depressive symptoms, five (5.7%) scored 20 through 28 indicating moderate depressive symptoms, and four (4.6%) scored 29-63 indicating severe depressive symptoms. The mean BDI-II score was 6.91 (*SD* 7.27, range 0-32) for men and 9.31 (*SD* 8.65, range 0-42) for women. Men and women did not differ on mean BDI-II score (U=620.0, z=-1.12, p=.26). A higher percent of women (n=17, 26.6%) reported mild, moderate or severe depressive symptoms as compared to men (n=2, 8.7%).

Dietary Pattern

Reported adequate caloric intake.

Less than half of all participants (n=36, 40.4%) reported an adequate caloric intake based on age and gender standards. Of the 36 reporting adequate caloric intake, the majority were women (n=28, 77.8%) and minorities (n=20, 55.6%). The mean age of those reporting adequate caloric intake was 41.9 (*SD* 10.47) years with a mean BMI of 31.41 (*SD* 5.36) mg/k^2 and mean waist circumference of 103.16 (*SD* 12.77) cm. The mean PSS was 15.8 (*SD* 6.37) for those reporting adequate caloric intake compared to the mean of 16.94 (*SD* 7.74) for those reporting inadequate caloric intake, and this difference was not significant (t=.724, p=.47). The mean BDI-II score was 7.25 (*SD* 5.77) for those reporting adequate caloric intake compared to the mean of 9.69 (*SD* 9.68) for those reporting inadequate caloric intake, and this difference was not significant (t=1.349, p=.15).

Among men, eight (43.8%) reported adequate caloric intake with a mean age of 43.75 (*SD* 9.93) years, mean BMI of 30.04 (*SD* 4.85), and mean waist circumference of 105.18 (*SD* 11.27) cm. Men reporting adequate caloric intake did not differ significantly compared to men reporting inadequate caloric intake on mean age (U=38.0, z=-1.421,

p=.16), BMI (U=48.0, z=-.775, p=.44), or waist circumference (U=49.0, z=-.710, p=.48). Among women, 28 (42.4%) reported adequate caloric intake with a mean age of 41.40 (*SD* 10.73) years, mean BMI of 31.8 (*SD* 5.52) mg/k^2 and a mean waist circumference of 102.58 (*SD* 13.29) cm. Women reporting adequate caloric intake did not differ significantly on mean age (U=470.0, z=-.454, p=.65), BMI (U=494.0, z=-.135, p=.89), or waist circumference (U=498.5, z=-.074, p=.94) compared to women reporting inadequate caloric intake.

Among the 36 individuals reporting adequate caloric intake, 22 (61.1%) exhibited high restraint. Those with high restraint reporting adequate caloric intake had significantly lower BMIs (U=92.0, z=-2.012, p=.05) compared to those with low restraint reporting adequate intake. Among those reporting adequate caloric intake, there were no significant differences in macronutrient composition, food group pattern, or dietary energy density values between those with high as compared to low restraint.

Among the 36 individuals reporting adequate caloric intake, seven (19.5%) reported mild, moderate or severe depressive symptoms. The seven individuals with depressive symptoms reported a different macronutrient composition and food group pattern compared to those reporting an adequate caloric intake without depressive symptoms. Those with adequate caloric intake and depressive symptoms reported a lower intake of protein (U=47.0, z=-2.178, p=.03), a lower intake of meat and beans (U=52.0, z=-1.979, p=.05), and fewer cups of vegetables (U=37.0, z=-2.578, p=.01) compared to those with adequate caloric intake and no depressive symptoms. There was no significant difference in energy density values between those reporting adequate

caloric intake with depressive symptoms compared to those reporting adequate caloric intake with no depressive symptoms (U=76.0, z=-1.019, p=.33).

Macronutrient composition.

In the overall group (n=87), there were significant differences in the macronutrient composition between those who reported adequate caloric (n=36, 41.4%) versus inadequate caloric intake (n=51, 58.6%). Men reporting an adequate caloric intake (n=8, 34.8%) reported a significantly higher intake of calories, total fat, saturated fat, total carbohydrates, and total sugars compared to men reporting an inadequate caloric intake (n=15, 65.2%). Women reporting an adequate caloric intake (n=28, 43.8%) reported a significantly higher intake of calories, total fat, saturated fat, total carbohydrates, cholesterol, total sugars and protein compared to women reporting an inadequate caloric intake (n=36, 56.2%). See Appendix D for complete data tables (Table 21) comparing macronutrient intake by reported adequate versus inadequate caloric intake.

For those participants reporting adequate caloric intake, their macronutrient intake was compared to the RDA standards for age and sex (National Academy of Science, Institute of Medicine, & Food and Nutrition Board, 2005). All (n=36) participants reporting adequate caloric intake exceeded the recommended 130 grams of carbohydrate per day which is the average minimal usage of glucose by the brain (National Academy of Science, Institute of Medicine, & Food and Nutrition Board, 2005). Although the majority of participants (n=28, 77.8%) reporting adequate caloric intake exceeded the recommended total protein per day intake, almost a quarter (n=8, 22.2%) did not consume the recommended amount of protein (National Academy of

Science, Institute of Medicine, & Food and Nutrition Board, 2005). The majority of participants (n=26, 72.2%) reporting adequate caloric intake reported consuming less than the recommended amount of dietary fiber for their age and gender (National Academy of Science, Institute of Medicine, & Food and Nutrition Board, 2005). When compared with the Dietary Guidelines for Americans, the majority of participants (n=22, 61.6%) reporting adequate caloric intake complied with the recommendation to consume less than 300 mg of dietary cholesterol per day, and exceeded the recommendation for total fat (n=26, 72.2%) and saturated fat (n=23, 63.9%) (U. S. Department of Health and Human Services & U.S. Department of Agriculture, 2005).

Food group pattern.

Individuals reporting an adequate caloric intake consumed a different food group pattern than those reporting an inadequate caloric intake. Those reporting an adequate caloric intake consumed significantly more ($t=-4.347$, $p \leq .01$) grain than did those reporting inadequate caloric intake. Among men, those reporting adequate caloric intake (n=8, 34.8%) consumed a significantly higher ($U=28.0$, $z=-2.066$, $p=.04$) amount of vegetables than men reporting inadequate caloric intake. Among women, those reporting adequate caloric intake (n=28, 41.2%) consumed a significantly higher ($U=208.0$, $z=-4.006$, $p \leq .01$) amount of grain than women reporting inadequate caloric intake (n=36, 58.8%). (Table 2).

Table 2

Food Group Pattern by Reported Adequate and Inadequate Caloric Intake (N=87)

Food group (equivalent)	Adequate intake n Mean *(SD)*	Inadequate intake n Mean *(SD)*	T Statistic	Mann Whitney U	z Score
Grain (ounce)					
All	36	51			
	8.2 (3.0)	5.4 (2.7)	-4.347**		
Men	8	15			
	9.4 (4.3)	7.0 (2.3)		35.0	-1.614
Women	28	36			
	7.9 (2.6)	4.9 (2.5)		208.0	-4.006**
Meat & beans (ounce)					
All	36	51			
	5.6 (2.9)	5.7 (4.0)	.089		
Men	8	15			
	5.4 (3.2)	8.4 (6.0)		39.0	-1.356
Women	28	36			
	5.6 (2.8)	4.6 (2.1)		401.0	-1.394
Vegetable (cup)					
All	36	51			
	1.9 (1.5)	1.7 (.8)	-.837		
Men	8	15			
	3.1 (2.1)	1.7 (.9)		28.0	-2.066*
Women	28	36			
	1.5 (1.2)	1.6 (.8)		443.5	-.819
Fruit (cup)					
All	36	51			
	1.2 (1.1)	.7 (.9)	-1.942		
Men	8	15			
	1.0 (1.3)	.6 (.9)		46.0	-.912
Women	28	36			
	1.2 (1.1)	.8 (.8)		383.0	-1.641
Milk (cup)					
All	36	51			
	1.3 (1.0)	1.0 (.9)	-1.505		
Men	8	15			
	1.9 (1.5)	1.1 (.9)		45.0	-.968
Women	28	36			
	1.1 (.8)	1.0 (.9)		412.5	-1.239

Note. *p≤.05, **p≤.01.

Biobehavioral Mediators

Dietary energy density (ED).

For all participants, the mean energy density of solid foods and beverages was 0.75 (*SD* .22) kilocalories per gram (kcal/g), whereas the mean beverage energy density was 0.15 (*SD* .10) kcal/g, and the mean food energy density was 1.84 (*SD* .43) kcal/g. (Figure 2). There were no significant differences between the energy density of food and beverages, beverages, or food reported by men and women. African-Americans reported consuming more energy dense food and beverages (t=-2.198, p=0.03), and more energy dense foods (t=-2.408, p=.02) than whites. African-Americans and whites did not differ on energy density of beverages indicating the difference was accounted for by food. (Table 3).

Energy density values differed between those who reported adequate versus inadequate caloric intake. Energy density values for food and beverages (t=-3.671, $p \leq .01$) and beverages (t=-2.953, $p \leq .01$) were higher for those reporting an adequate compared to inadequate caloric intake. Regarding gender, there were no significant differences in energy density values between men reporting an adequate caloric intake (n=8, 34.8%) compared to men reporting an inadequate caloric intake (n=15, 65.2%). In contrast, energy density values for food and beverages (U=205.0, z=-4.046, $p \leq .01$) and beverages (U=348.0, z=-2.111, p=.04) were significantly higher for women reporting an adequate (n=28, 43.8%) compared to women reporting inadequate caloric intake (n=36, 56.2%). Regarding race, whites reporting adequate caloric intake (n=20, 46.5%) reported higher energy density values for food and beverages (U=120.0, z=-2.678, $p \leq .01$) and beverages (U=124.0, z=-2.581, p=.01) than whites reporting inadequate

caloric intake (n=23, 53.5%). Energy density values of food and beverages (U=118.0, z=-2.462, p=.01) were higher for African-Americans reporting an adequate intake (n=16, 37.2%) compared to African-Americans reporting an inadequate intake (n=27, 62.8%). (Table 4).

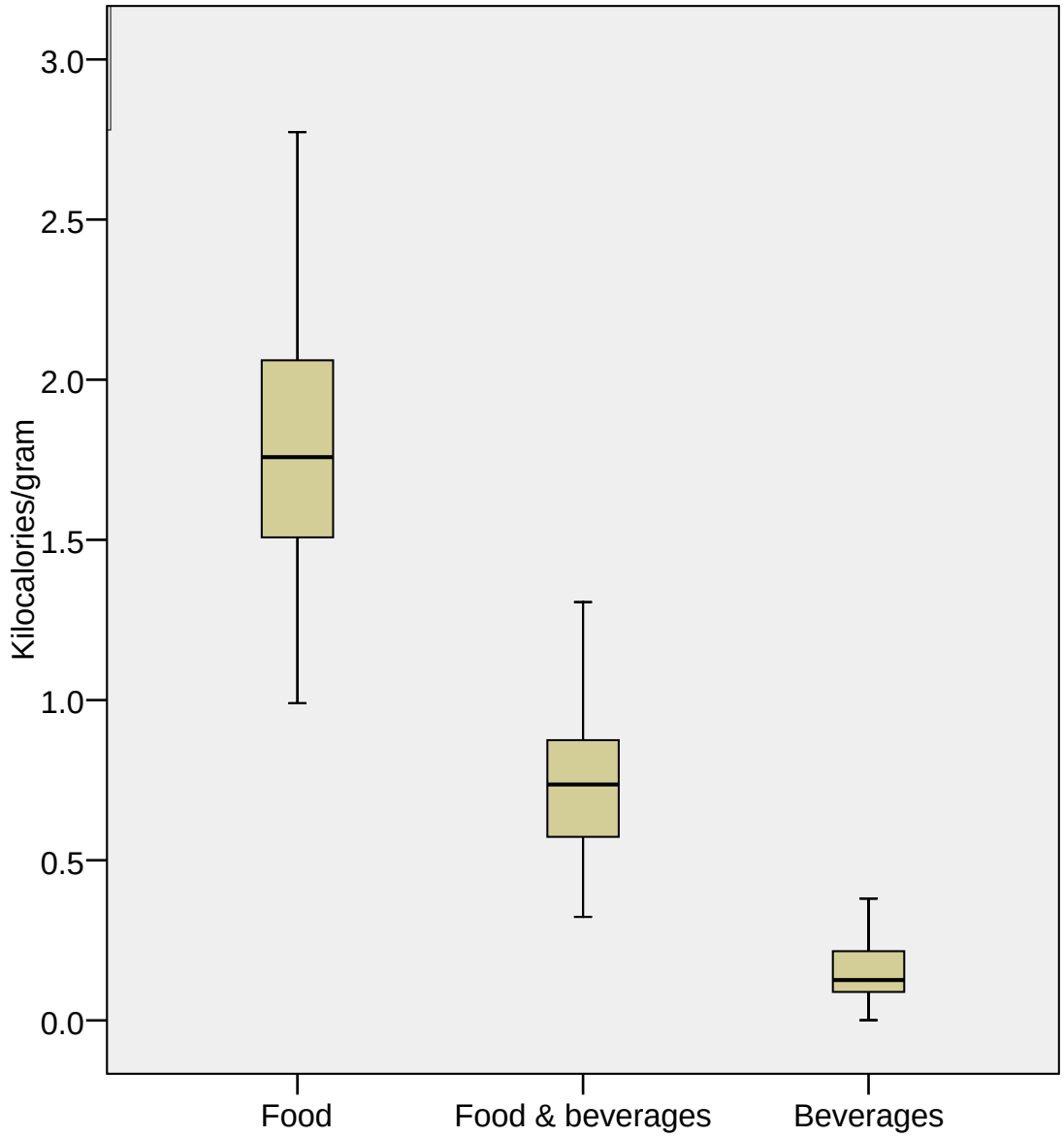

Figure 3. Boxplot of Energy Density Values of Sample

Note. Whiskers represent lowest and highest scores. Open circles represent outliers.

Table 3

Dietary Energy Density Values by Gender and Race

Dietary Energy Density Values	Mean *(SD)*	Range
Food and beverages		
All (N=86)	.75 (.22)	.32-1.31
Men (n=22)	.80 (.25)	.32-1.25
Women (n=64)	.74 (.21)	.41-1.31
Beverages		
All (N=86)	.15 (.10)	0-.50
Men (n=22)	.17 (.11)	0-.38
Women (n=64)	.15 (.09)	0-.50
Food		
All (N=87)	1.84 (.43)	.99-2.96
Men (n=23)	1.95 (.49)	.99-2.96
Women (n=64)	1.80 (.41)	.99-2.77
Food and beverages		
Whites (n=43)	.70 (.19)	.32-1.08
African-Americans (n=43)	.81 (.24)	.41-1.31
Beverages		
Whites (n=43)	.15 (.11)	0-.50
African-Americans (n=43)	.16 (.08)	.02-.37
Food		
Whites (n=43)	1.73 (.42)	.99-2.77
African-Americans (n=44)	1.95 (.43)	.99-2.96

Table 4

Dietary Energy Density Values by Adequate and Inadequate Caloric Intake

Dietary Energy Density Value	Adequate Intake n Mean *(SD)*	Inadequate Intake n Mean *(SD)*	T Statistic	Mann Whitney U	z Score
Food & beverages					
All (N=89)	36	50			
	.85 (.20)	.69 (.21)	-3.671**		
Men	8	14			
(n=23)	.85 (.24)	.77 (.26)		47.00	-.614
Women	28	36			
(n=66)	.85 (.19)	.65 (.18)		205.00	-4.046**
Beverages					
All (N=89)	36	50			
	.19 (.11)	.13 (.08)	-2.953**		
Men	8	14			
(n=23)	.22 (.11)	.14 (.10)		32.00	-1.638
Women	28	36			
(n=66)	.18 (.11)	.12 (.06)		348.0	-2.111*
Food					
All (N=89)	36	51			
	1.92 (.42)	1.78 (.44)	-1.502		
Men	8	15			
(n=23)	2.09 (.59)	1.87 (.42)		50.00	-.645
Women	28	36			
(n=66)	1.87 (.36)	1.75 (.44)		387.00	-1.583
Food & beverages					
Whites	20	23			
(n=43)	.79 (.19)	.63 (.14)		120.00	-2.678**
AA	16	27			
(n=43)	.92 (.19)	.74 (.24)		118.00	-2.462*
Beverages					
Whites	20	23			
(n=43)	.20 (.13)	.10 (.06)		124.00	-2.581*
AA	16	27			
(n=43)	.18 (.08)	.15 (.08)		179.00	-.930
Food					
Whites	20	23			
(n=43)	1.81 (.42)	1.65 (.41)		180.00	-1.217
AA (n=44)	16	28			
	2.06 (.40)	1.89 (.44)		158.00	-1.610

Note. AA = African Americans. *p≤.05. **p≤.01.

There were no significant differences in the energy density values for food and beverages, beverages, or food reported by men with an adequate intake (n=8, 22.2%) compared to women with an adequate intake (n=28, 77.8%). African-Americans with an adequate intake (n=16, 44.4%) reported significantly higher food energy density values (U=99.0, z=-1.942, p=.05) than whites with an adequate intake (n=20, 55.6%).

Morning salivary cortisol level.

The 8:00 am salivary cortisol reference value ranges from .18-.95 µg/dl and 81.0% (n=64) had values within the reference range. Overall (n=79), the mean morning salivary cortisol level was .68 (*SD* .45, range .08-2.21) µg/dl. Due to variability in collection times, evening salivary cortisol levels were not reported (See page 88). Premenopausal women did not differ on mean morning salivary cortisol level as compared to postmenopausal women (U=255.0, z=-1.409, p=.16). Men did not differ on mean morning salivary cortisol level as compared to women (U=477.5, z=-1.460, p=.14). There was no significant difference on mean morning salivary cortisol level between those with depressive symptoms compared to those without (U=484.5, z=-.754, p=.45). (Table 5).

Table 5

Morning Salivary Cortisol Values by Gender and Depressive Symptoms (N=79)

Morning salivary cortisol value	Mean (*SD*) µg/dl	Range µg/dl
All (n=79)	.68 (.45)	.08-2.21
Men (n=21)	.60 (.50)	.08-2.0
Women (n=58)	.70 (.43)	.15-2.21
Premenopausal (n=42)	.75 (.46)	.15-2.21
Postmenopausal (n=16)	.59 (.32)	.18-1.33
Without depressive symptoms (n=61)	.71 (.48)	.08-2.21
With depressive symptoms (n=18)	.56 (.26)	.21-1.08

Waist Circumference

Overall, the mean waist circumference was 103.2 (14.8) cm. The mean waist circumference was 103.4 (*SD* 12.7) cm for men, and it was 103.2 (*SD* 14.9) cm for women. A waist circumference greater than 102 cm for men and greater than 88 cm for women represent an increased risk for cardiometabolic disease. Overall, the majority were at increased risk for cardiometabolic disease. The majority of women were at increased risk compared to less than half of the men. (Table 6). Those with waist circumference risk (n=58) had a mean morning cortisol level significantly higher (U=403.0, z=-2.286, p=.02) compared to those without waist circumference risk (n=21). (Table 7).

Table 6

Waist Circumference by Gender

Waist circumference (cm)	Mean *(SD)*	Range
All (n=87)	103.2 (14.3)	78.27-139.97
Men (n=23)	103.38 (12.7)	89.30-139.97
Women (n=64)	103.19 (14.9)	78.27-139.70
Increased cardiometabolic risk	N *(%)*	
Overall	64 (73.6%)	
Men (>102 cm)	10 (43.5%)	
Overweight	2 (20.0 %)	
Obese	8 (80.0 %)	
Women (>88cm)	54 (84.8%)	
Overweight	18 (33.3%)	
Obese	36 (66.6%)	

Table 7

Salivary Cortisol Values by At Risk Waist Circumference

Morning salivary cortisol value	Mean (*SD*) µg/dl	Range µg/dl	Mann Whitney U µg/dl	z Score
Not at risk WC (n=21)	.52 (.40)	.08-1.73	403.0	-2.286*
At risk WC (n=58)	.73 (.45)	.15-2.21		

Note. WC = Waist circumference. *p≤.05.

An important pattern emerged when examining salivary cortisol levels by waist circumference risk and depressive symptoms. Among the 58 individuals with waist circumference risk, fifteen had mild, moderate or severe depressive symptoms. Individuals with waist circumference risk and depressive symptoms had a significantly lower morning cortisol level (U=180.5, z=-2.522, p=.01) than those individuals with waist circumference risk and no depressive symptoms. (Table 8).

Table 8

Salivary Cortisol Values by At Risk Waist Circumference and Depressive Symptoms

Morning salivary cortisol value	Mean (*SD*) µg/dl	Mann Whitney U µg/dl	z Score
At risk WC and no depressive symptoms (n=43)	.81 (.48)	180.5	-2.522*
At risk WC and depressive symptoms (n=15)	.49 (.21)		

Note. WC = Waist circumference. *p≤.01.

Reliability of Study Instruments

The Cronbach's alpha for the Three Factor Eating Questionnaire-Revised was .76 demonstrating the internal consistency reliability of this instrument in measuring the concept of dietary restraint in this sample. The Cronbach's alpha for the Beck Depression Inventory II was.91 demonstrating the internal consistency reliability of this instrument in measuring the concept of depressive symptoms in this sample. The Cronbach's alpha for the Perceived Stress Scale was.89 demonstrating the internal consistency reliability of this instrument in measuring the concept of perceived stress in this sample. These were all deemed adequate.

In the following section, the findings related to each specific aim and hypotheses are reported. Bivariate correlations between individual characteristics, psychological factors, biobehavioral mediators and the outcome variable are presented in Table 9. The food and beverage energy density values were used to test the hypotheses and are presented in the body of the dissertation. Results related to hypothesis testing of the

beverage energy density and the food energy density values are presented in Appendix E.

Specific Aim 1

The first specific aim examined the associations between psychological factors (perceived stress and depressive symptoms) and biobehavioral responses (dietary pattern and cortisol secretion) in sedentary, overweight adults.

Hypothesis 1

Psychological factors (perceived stress and depressive symptoms) will explain a significant amount of variation in dietary pattern (food and beverage energy density) while controlling for age, race, gender, BMI, reporting adequate caloric intake (RACI) and dietary restraint.

Psychological factors and food and beverage energy density.

Examining data from the 87 participants, there were significant bivariate correlations between age and reporting adequate caloric intake with food and beverage energy density. Younger age and those reporting adequate caloric intake reported higher food and beverage energy density values than older adults and those reporting inadequate caloric intake. There were no significant bivariate correlations between BMI, TFEQR, PSS or BDI-II with food and beverage energy density. (Table 9).

A sequential linear regression model was used to determine the amount of total variance in food and beverage energy density explained by the addition of psychological factors while holding individual characteristics constant. The individual characteristics of age, gender, race, BMI, reporting adequate caloric intake (RACI), and

TFEQR were entered as a block to control for their influence on food and beverage energy density. PSS and BDI-II scores were then entered and the F change statistic trended toward significance. The full model was significant with the adjusted R^2 of .349, which indicates that these variables explained approximately 34.9% of the total variance in food and beverage energy density. BDI-II score was a significant independent predictor of food and beverage energy density. (Table 10). Examination of the collinearity diagnostics showed that the variance inflation factor values were below 10 and the tolerance statistic values were above .2 for all variables indicating that there was no collinearity within the data (Field, 2005).

Table 9

Correlation Matrix (Pearson's and Point by Serial Correlations)

Variable	Gender	Age	BMI	TFEQR	RACI	PSS	BDI-II	ED	Weight	Cortisol£	WC
Race	.14	-.16	.05	.06	.18	.01	.07	.23	-.36**	-.12	-.07
Gender	-	.17	.11	.17	-.03	.03	.14	-.11	-.25*	.10	-.01
Age		-	-.01	-.13	-.01	-.31**	-.09	-.27*	.06	.24**	.09
BMI			-	.26*	.10	.23*	.31**	.14	.15	-.04	.86*
TFEQR				-	.23*	.35**	.37**	-.03	-.05	-.22*	.16
RACI					-	.14	.18	-.42**	-.33**	-.16	.01
PSS						-	.60**	.11	-.01	-.17	.23**
BDI-II							-	.16	-.19	-.08	.25**
ED								-	-.48**	.002	.15
Weight									-	-.002	.23*
Cortisol£										-	-.004

Note. BMI = Body Mass Index. TFEQR = Three Factor Eating Questionnaire Revised. RACI = Reported Adequate Caloric Intake. PSS = Perceived Stress Scale. BDI-II = Beck Depression Inventory-II. ED = Food and beverage energy density. Weight = Food and beverage weight. Cortisol = Morning Salivary Cortisol Value. WC = Waist Circumference. *p≤.05. **p≤.01. £n = 79.

Table 10

Regression Model for the Association between Psychological Factors and Food and Beverage Energy Density (N=85)

	Model 1			Model 2		
Variable	*B*	SE *B*	ß	*B*	SE *B*	ß
Age	-.004	.002	-.21*	-.004	.002	-.20*
Race	.13	.04	.30**	.13	.04	.30**
Gender	-.08	.05	-.15	-.09	.05	-.18
RACI	-.41	.08	-.48**	-.43	.08	-.50**
BMI	.08	.04	.19*	.06	.04	.14
TFEQR	-.02	.04	-.05	-.04	.04	-.08
PSS				.001	.004	-.02
BDI-II				.03	.02	.23*
R^2		.372			.411	
Adjusted R^2		.324			.349	
R^2 change		.372			.039	
F for change in R^2		7.705**			2.506	
F (*p-value* total model)		7.705 (*p* = .00)			6.628 (*p* = .00)	

Note. RACI = Reported Adequate Caloric Intake. BMI = Body Mass Index. TFEQR = Three Factor Eating Questionnaire Revised. PSS = Perceived Stress Scale. BDI-II = Beck Depression Inventory-II. *B*= beta coefficient. *$p \leq .05$. **$p \leq .01$.

The full model was reduced to include those variables that were significant or trended toward significance (alpha .20 or below) in explaining food and beverage energy density. In the reduced model, age, gender, race, and RACI were entered as a block. The BDI-II scores were then entered to determine the amount of total variance in food and beverage energy density explained by the addition of BDI-II score while holding age, gender, race and RACI constant. The F change statistic and the reduced model were significant with an adjusted R^2 of .353 indicating that 35.3% of the total variance in food and beverage energy density can be explained by these variables. In the reduced model, age (t=-1.924, p=.06) and gender (t=-1.889, p=.06) trended toward significance. The standardized betas indicate that RACI (t=-5.733, $p \leq .01$) is inversely related while race (t=3.348, $p \leq .01$) and BDI-II (t=2.645, p=.01) are positively related to food and beverage energy density. Increased depressive symptoms explained 5.3% of food and beverage energy density variance above that accounted for by younger age, male gender, African-American race, and reporting adequate caloric intake. (Table 11). A post hoc power analysis demonstrated that the sample size of 86 achieved 75% power to detect an R^2 of .05 attributed to one independent variable using an F-test with an alpha of .05. The variables tested are adjusted for an additional three control variables with an R^2 of .34.

In order to test food and beverage energy density as a biobehavioral mediator between psychological factors and waist circumference, there must be a significant correlation between the predictor and mediating variable. In the adjusted model, there was a significant correlation, and further testing of the mediating model was warranted.

Table 11

Reduced Model for the Association between Psychological Factors and Food and Beverage Energy Density (N=86)

	Model 1			Model 2		
Variable	*B*	SE *B*	ß	*B*	SE *B*	ß
Age	-.004	.002	-.20*	-.004	.002	-.17
Race	.13	.04	.31**	.13	.04	.31**
Gender	-.07	.05	-.14	-.09	.05	-.17
RACI	-.41	.08	-.47**	-.45	.08	-.52**
BDI-II				.04	.01	.24**
R^2		.338			.391	
Adjusted R^2		.305			.353	
R^2 change		.338			.053	
F for change in R^2		10.341**			6.995*	
F (*p value* total model)		10.341 (*p* = .00)			10.284 (*p* = .00)	

Note. RACI = Reported Adequate Caloric Intake. BDI-II = Beck Depression Inventory-II. *B*= beta coefficient. * $p \leq .05$. ** $p \leq .01$.

Hypothesis 2

Hypothesis 2: Psychological factors (perceived stress and depressive symptoms) will explain a significant amount of variance in HPA activation (morning salivary cortisol value) while controlling for menopause status, race, BMI, and dietary restraint.

Psychological factors and morning cortisol.

Examining the data from the 79 participants with morning salivary cortisol data, there was a significant negative correlation between TFEQR with morning salivary cortisol level. There were no significant bivariate correlations between race, BMI, PSS or BDI-II scores with morning salivary cortisol level. (Table 9). Premenopausal status (r=.17, p=.09) trended toward a significant correlation with morning salivary cortisol level, and postmenopausal status was not significantly correlated with morning salivary cortisol level (r=-.08, p=.48).

A sequential linear regression model was used to determine the amount of variance in morning salivary cortisol level explained by psychological factors while holding individual characteristics constant. Menopause status, race, BMI and TFEQR scores were entered into the regression model as a block to control for their influence on morning salivary cortisol value. The PSS and BDI-II scores were then entered. Dietary restraint was significant, and menopause status trended toward significance (t=1.938, p=.06). The F change statistic and the model were not significant. A reduced model was not tested because the variables of interest, perceived stress and depressive symptoms, were not significant. Table 12.

Table 12

Regression Model for the Association between Psychological Factors and Morning Salivary Cortisol Value (N=78)

	Model 1			Model 2		
Variable	*B*	*SE B*	ß	*B*	*SE B*	ß
Pre	.13	.07	.26*	.13	.07	.26
Post	.06	.08	.09	.05	.08	.08
Race	-.07	.06	-.13	-.07	.06	-.14
BMI	.03	.06	.05	.03	.06	.05
TFEQR	-.17	.06	-.33**	-.15	.06	-.30*
PSS				-.004	.005	-.12
BDI-II				.009	.03	.05
R^2		.157			.166	
Adjusted R^2		.098			.082	
R^2 change		.157			.009	
F for change in R^2		2.677*			.379	
F (*p-value* for total model)		2.677 (*p* = .03)			1.988 (*p* = .07)	

Note. Pre = Premenopausal women. Post = Men and postmenopausal women. BMI = Body Mass Index. TFEQR = Three Factor Eating Questionnaire Revised. PSS = Perceived Stress Scale. BDI-II = Beck Depression Inventory-II. *B*= beta coefficient. *$p \leq .05$.

The variables of menopause status, race, BMI, dietary restraint, depressive symptoms and perceived stress did not contribute to explaining variance in morning salivary cortisol level. In order to test HPA activation as a biobehavioral mediator between psychological distress and abdominal obesity, there must be a significant correlation between the predictor variable and the mediating variable. In this evaluation, there were no significant correlations between depressive symptoms or perceived stress with morning salivary cortisol level. Further testing of the morning salivary cortisol level as a mediating variable was not warranted; however, the relationship between morning salivary cortisol with waist circumference will be further explored in specific aim 2.

Specific Aim 2

The second specific aim examined the associations between biobehavioral responses (dietary energy density and cortisol secretion) and cardiometabolic disease risk (abdominal obesity) in sedentary, overweight adults.

Hypothesis 3

Hypothesis 3: Dietary pattern (food and beverage energy density) will explain a significant amount of variance in abdominal obesity (waist circumference) while controlling for age, gender, race, dietary restraint, food and beverage weight (grams), perceived stress and depressive symptoms.

Food and beverage energy density and waist circumference.

There were no significant bivariate correlations between age, gender, race, dietary restraint with waist circumference. There was a significant bivariate correlation

between PSS with waist circumference (r=.23, p≤.01) indicating that increased perceived stress was associated with a larger waist circumference. The BDI-II scores were significantly correlated with waist circumference (r=.25, p≤.01) indicating that increased depressive symptoms was associated with a larger waist circumference. There was no significant correlation between food and beverage energy density with waist circumference. (Table 9).

A sequential linear regression model was used to determine the amount of total variance in waist circumference explained by the addition of the food and beverage energy density value while holding individual characteristics and psychological factors constant. Age, gender, race, food and beverage weight (grams), TFEQR, PSS and BDI-II scores were entered in a block to control for their influence on waist circumference. The food and beverage energy density values were then entered. The F change statistic and the full model were significant. The adjusted R^2 of .200 indicates that these variables explained 20.0% of the total variance in waist circumference. Food and beverage energy density values were significant independent predictors of waist circumference. (Table 13). Examination of the collinearity diagnostics showed that the variance inflation factor values were below 10 and the tolerance statistic values were above .2 for all variables indicating that there was no collinearity within the data (Field, 2005).

Table 13

Regression Model for the Association between Food and Beverage Energy Density and Waist Circumference (N=85)

	Model 1			Model 2		
Variable	*B*	SE *B*	ß	*B*	SE *B*	ß
Age	.01	.008	.15	.02	.008	.23*
Race	.04	.16	.03	.006	.15	.004
Gender	-.01	.18	-.009	.15	.17	.09
TFEQR	.14	.16	.10	.22	.15	.16
PSS	.01	.01	.11	.01	.01	.11
BDI-II	.10	.07	.22	.08	.06	.18
Food & beverage weight (grams)	.00	.00	.26*	.00	.00	.47**
Food & beverage energy density				.29	.09	.42**
R^2		.163			.276	
Adjusted R^2		.087			.200	
R^2 change		.163			.113	
F for change in R^2		2.147*			11.825**	
F(*p-value* for total model)		2.147 (p = .05)			3.621 (p=.001)	

Note. TFEQR = Three Factor Eating Questionnaire Revised. PSS = Perceived Stress Scale. BDI-II = Beck Depression Inventory-II. *B*= beta coefficient. *p≤.05. **p≤.01.

The full model was reduced to include those variables that were significant or trended toward significance (alpha .20 or below) in explaining waist circumference. In the reduced model, age, food and beverage weight and BDI-II scores were entered as a block. Food and beverage energy density values were then entered to determine the amount of total variance in waist circumference explained by the addition of the food and beverage energy density value while holding age, food and beverage weight and BDI-II scores constant. The F change statistic was significant indicating that food and beverage energy density explained variance in waist circumference above that accounted for by age, food and beverage weight, and depressive symptoms. The reduced model was significant with an R^2 of .200 indicating that 20.0% of the total variance in waist circumference can be explained by these variables. Age trended toward significance in the model (t=1.823, p=.07). The standardized betas indicate that food and beverage weight (t=3.918, $p \leq .01$), BDI-II scores (t=2.983, $p \leq .01$) and food and beverage energy density (t=3.129, $p \leq .01$) are positively correlated with waist circumference. High food and beverage energy density explained 11.3% of waist circumference variance beyond that explained by older age, higher food and beverage weight and increased depressive symptoms. (Table 14). A post hoc power analysis demonstrated that the sample size of 86 achieved 89% power to detect an R^2 of .092 attributed to one independent variable using an F-test with an alpha of .05. The variables tested are adjusted for an additional three control variables with an R^2 of .145.

In order to determine if dietary energy density acts as a mediator between psychological distress and abdominal obesity, there must be a significant correlation between the mediator and outcome variable while controlling for the independent

variables. Based on these evaluations, there was a significant correlation between food and beverage energy density with waist circumference while controlling for perceived stress and depressive symptoms. Continued evaluation of food and beverage energy density as a mediating variable is warranted.

Table 14

Reduced Regression Model for the Association between Food and Beverage Energy Density and Waist Circumference (N=86)

	Model 1			Model 2		
Variable	*B*	SE *B*	ß	*B*	SE *B*	ß
Age	.007	.007	.10	.01	.007	.19
BDI-II	.15	.05	.31**	.14	.05	.30**
Food & beverage weight (grams)	.00	.00	.28*	.00	.00	.44**
Food & beverage energy density				.25	.08	.36**
R^2		.145			.237	
Adjusted R^2		.114			.200	
R^2 change		.145			.092	
F for change in R^2		4.637**			9.791**	
F(*p-value* for total model)		4.637 (p < .01)			6.298 (p < .01)	

Note. BDI-II = Beck Depression Inventory-II. *B*= beta coefficient. *p≤.05. **p≤.01.

Hypothesis 4

Hypothesis 4: Cortisol secretion (morning salivary cortisol value) will explain a significant amount of variation in abdominal obesity (waist circumference) while controlling for menopause status, race, dietary restraint, perceived stress and depressive symptoms.

Morning salivary cortisol level and waist circumference.

There was a trend toward a significant bivariate correlation between men and postmenopausal women with waist circumference (r=.207, p=.06), and there was a significant correlation between BDI-II and waist circumference (r=.25, p≤.01). There was no significant bivariate correlation between morning salivary cortisol value and waist circumference. (Table 9).

To determine the total amount of variance in waist circumference explained by the addition of the morning salivary cortisol value while holding individual characteristics constant, a linear regression model was tested. Menopause status, race, TFEQR, PSS and BDI-II scores were entered into the regression model as a block to control for their influence on waist circumference. The morning salivary cortisol values were then entered into the model. The F change statistic was significant, and the full model trended toward significance; however, the variable of interest, morning salivary cortisol level, was not significant. (Table 15).

Table 15

Regression Model for the Association between Morning Salivary Cortisol Value and Waist Circumference (N=78)

	Model 1			Model 2		
Variable	*B*	*SE B*	ß	*B*	*SE B*	ß
Pre	-.30	.18	-.22	-.33	.18	-.25
Post	.15	.22	.09	.14	.22	.08
Race	-.10	.15	-.07	-.08	.15	-.06
TFEQR	.06	.16	.04	.10	.17	.07
PSS	.01	.01	.07	.01	.01	.08
BDI-II	.12	.06	.26	.12	.06	.26
Morning salivary cortisol level				.26	.31	.10
R^2	.166			.174		
Adjusted R^2	.095			.092		
R^2 change	.166			.008		
F for change in R^2	2.354*			.705		
F (*p-value* for total model)	2.354 (*p* = .04)			2.110 (*p* = .05)		

Note. Pre = Premenopausal women. Post = Men and postmenopausal women. TFEQR = Three Factor Eating Questionnaire Revised. PSS = Perceived Stress Scale. BDI-II =Beck Depression Inventory II. *B*= beta coefficient. * $p \leq .05$.

In order to determine if HPA activation mediates the relationship between psychological factors and abdominal obesity, there must be a significant correlation between the mediator variable and the outcome variable while controlling for the independent variables. In this evaluation, there was not a significant correlation between morning salivary cortisol level with waist circumference while controlling for perceived stress and depressive symptoms. Further testing of morning salivary cortisol level as a mediating variable is not warranted.

Specific Aim 3

The third specific aim examined the associations between psychological factors (perceived stress and depressive symptoms), biobehavioral responses (dietary pattern and cortisol secretion), and cardiometabolic disease risk (abdominal obesity) in sedentary, overweight adults.

Hypothesis 5

Hypothesis 5: Psychological factors (perceived stress and depressive symptoms) will explain a significant amount of variance in abdominal obesity (waist circumference) while controlling for age, gender, race and dietary restraint.

Psychological factors and waist circumference.

A simple linear regression model was used to determine the amount of variance in waist circumference explained by perceived stress. In a simple linear regression model, PSS scores ($F_{1,85}$=4.550, p=.04) were significantly correlated with waist circumference explaining 4.0% of the total variance. A simple linear regression model was used to determine the amount of variance in waist circumference explained by

depressive symptoms. In a simple linear regression model, BDI-II scores ($F_{1,85}$=5.616, p=.02) were significantly correlated with waist circumference explaining 5.1% of the total variance.

A sequential linear regression model was used to determine the total amount of variance in waist circumference explained by the addition of perceived stress and depressive symptoms while holding individual characteristics constant. Age, gender, race and TFEQ were entered as a block to control for their influence on waist circumference. PSS and BDI-II scores were then entered. Although the overall model was not significant (F=1.667, p=.14), the F change statistic (F=2.638, p=.08) trended toward significance, and a reduced model was tested. (Table 16).

Table 16

Regression Model for the Association between Psychological Factors and Waist Circumference (N=86)

	Model 1			Model 2		
Variable	*B*	*SE B*	ß	*B*	*SE B*	ß
Age	.01	.01	.12	.01	.01	.17
Gender	-.07	.18	-.04	-.11	.17	-.07
Race	-.07	.15	-.05	-.06	.15	-.05
TFEQR	.29	.15	.21	.17	.16	.12
PSS				.01	.01	.15
BDI-II				.07	.07	.15
R^2		.053			.112	
Adjusted R^2		.006			.045	
R^2 change		.053			.059	
F for change in R^2		1.136			2.638	
F (*p-value* for total model)		1.136 (*p* = .35)			1.667 (*p* = .14)	

Note. TFEQR = Three Factor Eating Questionnaire Revised. PSS = Perceived Stress Scale. BDI-II = Beck Depression Inventory II. *B*= beta coefficient.

The full model was reduced to include those variables that trended toward significance in explaining waist circumference. In the reduced model, age and TFEQR were entered as a block. The BDI-II scores were then entered to determine the amount

of total variance in waist circumference explained by the addition of BDI-II scores while holding age and dietary restraint constant. The F change statistic was significant indicating that depressive symptoms explained additional variance in waist circumference above that accounted for by age and dietary restraint. The reduced model was significant with an adjusted R^2 of .058 indicating that 5.8% of the total variance in waist circumference can be explained by these variables. Although age (t=1.218, p=.23) and dietary restraint (t=1.252, p=.21) were not independent predictors of waist circumference, BDI-II scores (t=1.972, p=.05) independently predicted waist circumference. Increased depressive symptoms explained 4.3% of waist circumference variance beyond that explained by older age and low dietary restraint. (Table 17). A post hoc power analysis demonstrated that the sample size of 86 achieved 51.3% power to detect an R^2 of .043 attributed to one independent variable using an F-test with an alpha of .05. The variables tested are adjusted for two control variables with an R^2 of .048. Examination of the collinearity diagnostics showed that the variance inflation factor values were below 10 and the tolerance statistic values were above .2 for all variables indicating that there was no collinearity within the data (Field, 2005).

Table 17

Reduced Regression Model for the Association between Psychological Factors and Waist Circumference (N=86)

	Model 1			Model 2		
Variable	*B*	*SE B*	ß	*B*	*SE B*	ß
Age	.01	.01	.12	.01	.01	.13
TFEQR	.29	.15	.20	.19	.16	.14
BDI-II				.10	.05	.22*
R^2		.048			.091	
Adjusted R^2		.025			.058	
R^2 change		.048			.043	
F for change in R^2		2.097			3.888	
F (*p-value* for total model)		2.097 (*p* = .13)			2.742 (*p* = .05)	

Note. TFEQR = Three Factor Eating Questionnaire Revised. BDI-II = Beck Depression Inventory-II. ß = Beta Coefficient. *p≤.05.

In order to test dietary pattern as a mediator between psychological factors and abdominal obesity, there must be a significant correlation between the predictor variable and the outcome variable. In the sequential linear regression model, depressive symptoms were significant in explaining waist circumference variance, and continued testing of food and beverage energy density as a mediator is warranted.

Hypothesis 6

Hypothesis 6: Dietary pattern and cortisol secretion mediate the relationship between perceived stress and depressive symptoms with abdominal obesity while controlling for race and BMI.

Psychological factors, biobehavioral mediators and waist circumference.

There were no significant bivariate correlations between psychological factors, food and beverage energy density, morning salivary cortisol level, and waist circumference. (Table 9). Three conditions must be met to test dietary pattern as a mediator between psychological factors and abdominal obesity. First, there must be a significant correlation between the independent and mediator variables. In the adjusted analysis, BDI-II scores were significant predictors of food and beverage energy density. PSS was not a significant predictor. Second, the mediating variable must be associated with the outcome variable while holding the independent predictors constant. In the adjusted analysis, the food and beverage energy density value was a significant predictor of waist circumference while holding BDI-II scores constant. PSS was not a significant predictor. Third, there must be a significant correlation between the independent and outcome variables. The third condition was met: there was a significant unadjusted correlation between depressive symptoms with waist circumference. Although there was a significant correlation between perceived stress with waist circumference, further testing of perceived stress as a mediating variable was not warranted because the first two conditions were not met.

Three additional statistical analyses were conducted to evaluate the potential mediating role of food and beverage energy density. The results are presented in Table

17. First, the Baron and Kenny approach evaluates a series of simple linear regression models to test the direct and total associations among the independent, mediating and outcome variables without controlling for any covariates. In this model, there was a significant relationship between depressive symptoms and waist circumference. Depressive symptoms did not have a significant bivariate relationship with food and beverage energy density. Food and beverage energy density did not have a significant bivariate relationship with waist circumference while controlling for depressive symptoms. There was no significant bivariate relationship between depressive symptoms and waist circumference while controlling for food and beverage energy density (Preacher & Hayes, 2004). The Baron and Kenny approach indicates that food and beverage energy density did not statistically mediate the relationship between psychological factors with waist circumference.

Second, the Sobel test examines the indirect relationships and tests for significance using the normal distribution. The Sobel test statistic is the ratio of the t-test statistic for the relationship between the independent variables and the mediator and the t-test statistic for the relationship between the mediator and the outcome variable. The Sobel test statistic is converted to a z-score which is compared to the critical value. If the z-score is greater than or equal to 1.96 or -1.96, the total relationship between the predictor variable and the outcome variable is significantly reduced when the mediator is added to the model indicating the variable mediated the relationship. In this evaluation, the total relationship between depressive symptoms and waist circumference was not significantly reduced when the food and beverage energy density value was added to the model indicating that food and beverage energy density did not statistically

mediate the relationship between psychological factors with waist circumference (Preacher & Hayes, 2004).

Third, the indirect effects were assessed using the non-parametric bootstrap sampling distribution approach (Preacher & Hayes, 2004). Because this approach makes no assumptions about the normality of the data, it was deemed appropriate to use (Preacher & Hayes, 2004). This analysis also demonstrated that food and beverage energy density did not statistically mediate the relationship between psychological factors with waist circumference. (Table 18).

Table 18

Evaluation of Food and Beverage Energy Density as a Mediator

Method of Analysis				
Tests for direct and total effects using simple linear regression models				
	Coefficient	Standard error	T-statistic	*P*-value
b(YX)	.1178	.0505	2.3341	.02
b(MX)	.0237	.0162	1.4591	.15
b(YM.X)	.3482	.3395	1.0255	.31
b(YX.M)	.1095	.0511	2.1446	.03
Tests for indirect effect using the normal distribution				
	Value	Standard error	Z-statistic	*P*-value
Sobel	.0082	.0113	.7318	.46
Tests for indirect effect using the bootstrap approach				
	Mean	Standard error	LL 95% CI	UL 95% CI
Bootstrap	.0091	.0104	-.0057	.0352

Note. X=BDI-II scores. M=Food and beverage energy density. Y=Waist circumference. LL = Lower limit. UL = Upper limit. CI = Confidence Interval.

Three conditions must be met to test morning salivary cortisol level as a mediator between psychological factors and abdominal obesity. First, there must be a significant correlation between the independent and mediator variables. In the adjusted analysis, there was no significant correlation between PSS and BDI-II scores with morning salivary cortisol level. Second, the mediating variable must be associated with the outcome variable while holding the independent predictors constant. The second

condition was not met; there was no significant correlation between morning salivary cortisol level with waist circumference. Third, there must be a significant correlation between the independent and outcome variables (Baron & Kenny, 1986). The third condition was met; there was a significant correlation between depressive symptoms with waist circumference, and there was a significant correlation between perceived stress with waist circumference. However, because the first two conditions were not met, further testing of the mediating model was not warranted. In conclusion, morning salivary cortisol level did not mediate the relationship between PSS and BDI-II scores with waist circumference.

Finally, sequential linear regression was used to examine the associations among the independent, mediators and outcome variables. First, PSS and BDI-II scores were entered into the model. Morning salivary cortisol and food and beverage energy density values were then entered. The F change statistic and the model were not significant. This model confirms that together morning salivary cortisol and food and beverage energy density values do not account for additional variance in waist circumference. (Table 19).

Table 19

Regression Model for the Association between Psychological Factors, Biobehavioral Mediators and Waist Circumference (N=78)

	Model 1			Model 2		
Variable	*B*	*SE B*	ß	*B*	*SE B*	ß
PSS	.01	.01	.06	.01	.01	.06
BDI-II	.11	.06	.23	.10	.07	.21
Morning salivary cortisol level				.05	.30	.02
Food & beverage energy density				.36	.34	.12
R^2		.074			.088	
Adjusted R^2		.049			.038	
R^2 change		.074			.014	
F for change in R^2		2.991			.554	
F (*p-value* for total model)		2.991 (*p* = .06)			1.755 (*p* = .15)	

Note. PSS = Perceived Stress Scale. BDI-II = Beck Depression Inventory II. *B*= beta coefficient.

Additional Analyses

To better understand the dietary pattern of individuals with depressive symptoms, an additional analysis was conducted. Increased depressive symptoms are linked to high alcoholic beverage consumption (Strine et al., 2008). To better understand the differences in food and beverage energy density between those with

depressive symptoms compared to those without, a food and nonalcoholic beverage energy density value was calculated.

A sequential linear regression model was used to determine the amount of total variance in food and nonalcoholic beverage energy density explained by the addition of BDI-II scores. Age, gender, race and RACI were entered as a block to control for their influence on food and nonalcoholic beverage energy density. The BDI-II scores were then added to the model. The F change statistic and the model were significant with an adjusted R^2 of .369 indicating the 36.9% of the total variance in food and nonalcoholic beverage energy density is explained by these variables. Age trended toward significance (t=-1.851, p=.07). The standardized betas indicate that race (t=3.375, $p \leq .01$) and BDI-II scores (t=2.692, $p \leq .01$) were positively related and gender (t=-2.343, p=.02) and RACI (t=-5.938, $p \leq .01$) were negatively related to food and nonalcoholic beverage energy density. Increased depressive symptoms explained 5.4% of food and nonalcoholic beverage energy density above that accounted for by younger age, male gender, African-American race and reporting adequate caloric intake. BDI-II scores independently predicted food and nonalcoholic beverage energy density. (Table 20). Post hoc power analysis demonstrated that the sample size of 85 achieved 76% power to detect an R^2 of .054 attributed to one independent variable using an F-test with an alpha of .05. The variables tested are adjusted for an additional three control variables with an R^2 of .353.

Table 20

Regression Model for the Association between Depressive Symptoms and Food and Nonalcoholic Beverage Energy Density (N=85)

	Model 1			Model 2		
Variable	*B*	SE *B*	ß	*B*	SE *B*	ß
Age	-.004	.002	-.19*	-.004	.002	-.17
Race	.13	.04	.31**	.14	.04	.31**
Gender	-.09	.05	-.18	-.11	.05	-.21*
RACI	-.43	.08	-.48**	-.47	.08	-.53**
BDI-II				.04	.01	.24**
R^2		.353			.406	
Adjusted R^2		.321			.369	
R^2 change		.353			.054	
F for change in R^2		11.033**			7.248**	
F (*p-value* total model)		11.033 (*p* = .00)			10.957 (*p* = .00)	

Note. RACI = Reported Adequate Caloric Intake. BDI-II = Beck Depression Inventory II. *B*= beta coefficient. *$p \leq .05$. ** $p \leq .01$.

Summary

The purpose of this study was to examine food and beverage energy density and morning salivary cortisol levels as biobehavioral mediators between psychological factors and waist circumference. There was inadequate evidence to conclude that food

and beverage energy density mediated this relationship. The results demonstrated that increased depressive symptoms, younger age, male gender, African-American race, and reporting adequate caloric intake were associated with high food and beverage energy density. Depressive symptoms independently predicted food and beverage energy density. After removing alcoholic beverages from the food and beverage energy density value, depressive symptoms continued to independently predict food and nonalcoholic beverage energy density. Perceived stress was not a significant predictor of food and beverage energy density or food and nonalcoholic beverage energy density.

Increased food and beverage energy density accounted for waist circumference variance above that explained by older age, higher food and beverage weight and increased depressive symptoms. High food and beverage energy density independently predicted increased waist circumference. Perceived stress was not an independent predictor.

In the unadjusted analysis, increased depressive symptoms and perceived stress were significant predictors of increased waist circumference. Results of a sequential linear regression model suggest that increased depressive symptoms accounted for waist circumference variance beyond that explained by older age and low dietary restraint. However, this hypothesis was underpowered, and these results should be considered preliminary.

Three statistical approaches were used to test food and beverage energy density as a mediator. None of these statistical tests were significant, demonstrating that food and beverage energy density did not mediate the relationship between psychological factors and waist circumference. Correlations among depressive symptoms, perceived

stress, food and beverage energy density and waist circumference are depicted in Figure 4.

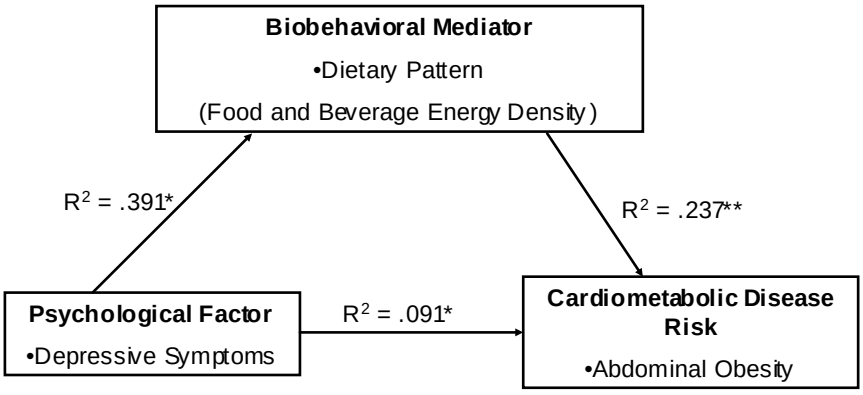

Figure 4. Correlations among Depressive Symptoms, Food and Beverage Energy Density and Waist Circumference.
Note. *p≤.05. **p≤.01.

There was inadequate evidence to conclude that morning salivary cortisol level mediated the relationship between psychological factors with waist circumference. There was no significant relationship between perceived stress or depressive symptoms with morning salivary cortisol level. In addition, there was no significant relationship between morning salivary cortisol level with waist circumference. Correlations among depressive symptoms, perceived stress, morning salivary cortisol level and waist circumference are depicted in Figure 5. Finally, the variables of food and beverage energy density and morning salivary cortisol level did not explain additional variance in waist circumference above that accounted for by increased depressive symptoms and perceived stress.

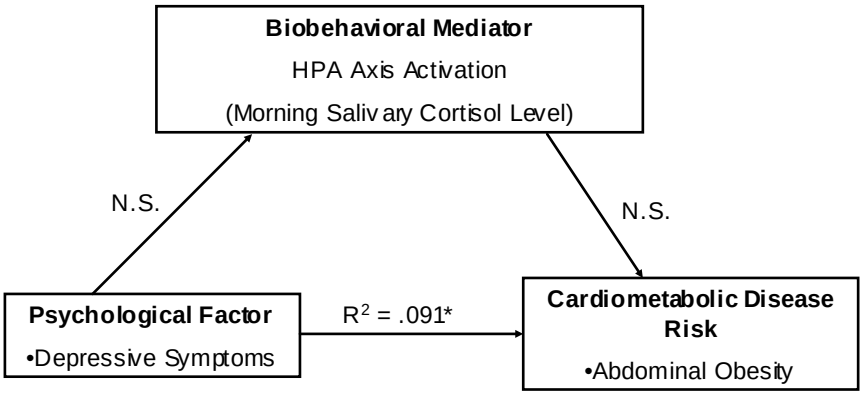

Figure 5. Correlations among Psychological Factors, Morning Salivary Cortisol and Waist Circumference.

Note. *p≤.05.

CHAPTER V

Discussion

Introduction

This prospective, correlation study examined the associations among psychological factors, biobehavioral responses and cardiometabolic risk. Depressive symptoms and perceived stress were the psychological factors examined. Dietary pattern and cortisol secretion were conceptualized as biobehavioral mediators between psychological factors and abdominal obesity, a cardiometabolic risk factor.

This chapter presents the findings in the context of the theoretical framework, examines study strengths and limitations, and provides recommendations for future research and clinical practice.

Summary of Results

Demographic and Clinical Characteristics

Eighty-seven middle aged, working adults participated in this study. The study sample was comprised primarily of women and African-Americans. The majority of participants were relatively healthy, and relatively few reported conditions that limit their ability to participate in physical activity. The majority of participants were classified as obese with a BMI greater than or equal to 30 kg/m^2. The most commonly reported cardiometabolic risk factor was hypertension, and it was reported by a quarter of the participants. The majority of participants were not currently taking any medications. The majority of participants were well educated and employed full time as

professionals/executives, managers/administrators, or clerical/sales. The sample reflected relatively healthy overweight men and obese women with elevated waist circumference at risk for cardiometabolic disease. By enrolling participants who are working full time, these data can be used to inform the development and design of worksite interventions. By intervening at worksites, nurses can gain access to 65% of adults in an environment where they typically consume one or more meals per day (Obesity. Guide to Community Preventive Services Website. Centers for Disease Control and Prevention).

Behavioral Characteristics

Most participants were categorized as exhibiting low dietary restraint. These individuals had higher BMIs, more depressive symptoms and more perceived stress than did the individuals with high dietary restraint. Among women, those with low restraint had higher BMIs, more depressive symptoms, more perceived stress and higher waist circumference than women with high restraint.

The majority of participants were non-smokers and did not report alcohol intake. There were not enough smokers to conduct meaningful analyses based on this variable. More whites reported alcohol intake compared to African-Americans. Individuals reporting alcohol intake consumed higher energy dense beverages than those who did not report alcohol intake. There was no significant difference in waist circumference between those reporting alcohol intake compared to those who did not. The majority of participants did not consume alcohol in excess of the recommendations; thus, this finding is consistent with the literature. For example, Fan and others (2008) reported an increased risk of abdominal obesity only among individuals consuming alcohol in

excess of the U.S. Dietary Guidelines (Fan et al., 2008). In contrast, Koster and others (2008) reported that individuals in the highest quintile of waist circumference had a lower alcoholic beverage intake than those in the lowest quintile of waist circumference (Koster et al., 2008). The data related to alcohol consumption and abdominal obesity is complex, and in future studies it may be important to assess alcohol consumption patterns over time in addition to daily alcohol intake.

Environmental Factors

Access and cost have been identified as environmental factors that contribute to poor quality diets (Glanz, Basil, Maibach, Goldberg, & Snyder, 1998). In this study, adults lived close to supermarkets and had cars giving them access to fruits and vegetables. However, cost may have been a factor for some participants since 18.3% reported earning less than $40,000 per year. Time pressures, limited time for meal preparation and a demand for prepackaged and fast foods also contribute to poor quality diets (Hill, Wyatt, Reed, & Peters, 2003). For the majority of participants, full time work responsibilities may have limited time for grocery shopping and meal preparation.

Psychological Factors

The majority of participants reported low levels of perceived stress and no depressive symptoms. However, 19 (21.8%) were considered to have mild, moderate or severe depressive symptoms. Although the prevalence of depressive symptoms in this sample is similar to the 20-25% depression prevalence reported in patients undergoing coronary bypass surgery (Parashar & Vaccarino, 2007) and the 20% depression prevalence associated with chronic disease (Krogh, Peterssen, Timmermann, Saltin, &

Nordentoft, 2007), it was four times higher than the three to five percent prevalence reported in the general population (Krogh, Peterssen, Timmermann, Saltin, & Nordentoft, 2007). Obese individuals are 20% more likely to have depression than non-obese individuals (Lu, 2007). Although depression is more prevalent among women, there were no significant differences between men and women on perceived stress or depressive symptoms in this study.

Dietary Pattern

Less than half of the participants reported an adequate caloric intake based on age and gender standards. Of those reporting adequate caloric intake, the majority were women and African-American. The individuals reporting adequate caloric intake did not differ significantly on mean age, BMI, waist circumference, PSS or BDI-II scores from those reporting inadequate caloric intake. Among those reporting adequate caloric intake (N=36), the majority exceeded the Dietary Guidelines for Americans recommendations for total and saturated fats and did not consume the recommended amount of dietary fiber for their age and gender (U. S. Department of Health and Human Services & U.S. Department of Agriculture, 2005).

Reporting adequate caloric intake was an important variable in the analysis of dietary patterns. Among all participants, there were significant differences in macronutrient composition, food group patterns, and energy density values between those who reported an adequate compared to inadequate caloric intake. Men reporting adequate caloric intake had a higher caloric intake and consumed more total fat, total carbohydrates, dietary fiber, and total sugars than men reporting inadequate caloric intake. Men reporting adequate caloric intake also consumed more cups of vegetables

than men reporting inadequate caloric intake. Women reporting adequate caloric intake had a higher caloric intake and consumed more total fat, saturated fat, cholesterol, total carbohydrates, dietary fiber and protein than women reporting inadequate caloric intake. Women reporting adequate intake consumed more grains than did women reporting inadequate intake.

The majority of participants did not consume the recommended amount from the fruit, vegetables or milk groups. In a review of published studies, Rolls and others (2004) found evidence of an inverse association between fruit and vegetable intake and body weight (Rolls, Ello-Martin, & Tohill, 2004). Although the majority of studies were cross-sectional, there was sufficient evidence to recommend replacing energy rich foods with fruits and vegetables to help regulate body weight. Fruits and vegetables play an important role in determining energy intake because they are high in water and fiber and low in calories so that the kilocalorie per gram ratio is low. Additionally, they may help promote weight loss and body weight regulation because of their appetite suppressing effect (Rolls, Ello-Martin, & Tohill, 2004).

Of concern, the majority of participants did not consume the recommended amount of milk products. A recent study suggests that adequate dairy intake, possibly due to the combined effects of calcium, phosphorous, and magnesium, plays a role in weight regulation and possibly body fat distribution and cardiovascular disease (Melanson, Donahoo, Dong, Ida, & Zemel, 2005). Although the mechanisms are not well understood, researchers propose three potential pathways. First, calcium may promote the formation of fatty acid soaps in the gut reducing fat absorption; second, high calcium intake may prevent fat deposits by tipping the balance in favor of lipolysis

over lipogenesis; third, high calcium intakes may increase the rate of fat breakdown. Slower rates of fat breakdown are associated with weight gain (Melanson, Donahoo, Dong, Ida, & Zemel, 2005). In addition, diets rich in fruit, milk products and vegetables have a lower energy density. Importantly, these same foodstuffs are the major contributors to food volume across all age groups (Marti-Henneberg et al., 1999).

Among the individuals reporting adequate caloric intake, those with high dietary restraint had lower BMIs than those with low dietary restraint. Among the individuals reporting adequate caloric intake, those with depressive symptoms reported different dietary patterns than those with no depressive symptoms. Those with adequate intake and depressive symptoms reported a lower intake of protein and a lower intake from the meat and beans and the vegetable groups. However, they did not differ on dietary energy density values.

Men reporting adequate intake did not differ on the energy density of food and beverages, beverages, or food from men reporting inadequate intake. Women reporting adequate intake consumed more energy dense food and beverages and more energy dense beverages than women reporting inadequate intake. African-Americans with an adequate intake reported significantly higher energy density values for food and beverages than did whites with an adequate intake. In this study, individuals reporting adequate caloric intake reported the highest energy dense diets exceeding the recommendations for total and saturated fat intake and consuming less than the recommended amount of dietary fiber. These findings support the concern about the poor nutritional quality of high energy dense diets due to excess dietary fat and inadequate fiber intake (Cuco, Arija, Marti-Henneberg, & Fernandez-Ballart, 2001).

The finding that this sample comprised primarily of well educated working adults with access to financial resources had overall poor dietary quality is of concern.

HPA Activation

The majority of participants had morning salivary cortisol levels within the reference range. Men and women did not differ on morning salivary cortisol levels. Premenopausal women did not differ on morning cortisol compared to postmenopausal women. Individuals with waist circumference risk and depressive symptoms had a lower morning cortisol as compared to individuals with waist circumference risk and no depressive symptoms.

Findings

Specific Aim 1

The first specific aim examined the associations between psychological factors (perceived stress and depressive symptoms) and biobehavioral responses (dietary pattern and cortisol secretion) in sedentary overweight adults.

Hypothesis 1.

The first hypothesis was that psychological factors will explain a significant amount of variation in dietary pattern while controlling for age, gender, race, BMI, reporting adequate caloric intake and dietary restraint. In a reduced model, depressive symptoms did account for variance in food and beverage energy density above that explained by age, gender, race and reporting adequate caloric intake. Younger age, male gender, African American race, reporting adequate caloric intake and increased

depressive symptoms were associated with the intake of high energy dense foods and beverages.

Hypothesis 2.

The second hypothesis was psychological factors will explain a significant amount of variation in cortisol secretion while controlling for menopause status, race, dietary restraint and BMI. Race, BMI, depressive symptoms and perceived stress were not significant in explaining variation in morning salivary cortisol level. Dietary restraint was negatively correlated with morning salivary cortisol level. There is no theoretical basis for this correlation, and it is thought to be spurious. Premenopausal status trended toward a significant relationship with higher morning cortisol levels, and this contradicts findings from a recent study (Kalleinen et al., 2008). These contradictory findings may be specific to this sample: of the 42 premenopausal women, 10 (23.8%) had morning cortisol levels above the reference range which may act to pull the mean higher. In comparison, of the 16 postmenopausal women, 2 (12.5%) had morning cortisol levels above the reference range. Perceived stress and depressive symptoms were not associated with high morning cortisol levels. A single measure of morning salivary cortisol may not be adequately sensitive to detect alterations in HPA activation related to stress and depressive symptoms.

Specific Aim 2

The second specific aim examined the associations between biobehavioral responses (dietary energy density and cortisol secretion) and cardiometabolic disease risk (abdominal obesity) in sedentary, overweight adults.

Hypothesis 3.

The third hypothesis was that dietary pattern (food and beverage energy density) will explain a significant amount of variance in abdominal obesity (waist circumference) while controlling for age, gender, race, dietary restraint, food and beverage weight, perceived stress and depressive symptoms. In a reduced model, food and beverage energy density did explain variance in waist circumference above that accounted for by age, food and beverage weight and depressive symptoms. Higher food and beverage energy density values explained elevated waist circumference variance beyond that explained by older age, higher food and beverage weight and increased depressive symptoms.

Hypothesis 4.

The fourth hypothesis was that cortisol secretion (morning salivary cortisol value) will explain a significant amount of variation in cardiometabolic disease risk (abdominal obesity) while controlling for menopause status, race, dietary restraint, perceived stress and depressive symptoms. Morning salivary cortisol level, a manifestation of HPA axis function, did not contribute to explaining variance in waist circumference while controlling for individual characteristics and psychological factors.

Specific Aim 3

The third specific aim examined the associations between psychological factors (perceived stress and depressive symptoms), biobehavioral responses (dietary pattern and cortisol secretion) and cardiometabolic disease risk (abdominal obesity) in sedentary, overweight adults.

Hypothesis 5.

The fifth hypothesis was that psychological factors (perceived stress and

depressive symptoms) will explain a significant amount of variance in abdominal obesity (waist circumference) while controlling for age, gender, race and dietary restraint. In unadjusted correlations, increased perceived stress and increased depressive symptoms were associated with increased waist circumference. Increased depressive symptoms explained waist circumference variance beyond that accounted for by older age and low dietary restraint.

Hypothesis 6.

The sixth hypothesis was that biobehavioral responses (dietary pattern and cortisol secretion) mediate the relationship between psychological factors (perceived stress and depressive symptoms) with abdominal obesity (waist circumference) while controlling for age, gender, race and dietary restraint. Based on these analyses, there is insufficient evidence to conclude that dietary pattern mediated the relationship between psychological factors with abdominal obesity. Additionally, there is insufficient evidence to conclude that morning salivary cortisol level mediated the relationship between psychological factors with abdominal obesity. Finally, there were no significant correlations among perceived stress, depressive symptoms, food and beverage energy density and morning salivary cortisol level with waist circumference.

Implications for Theoretical Framework

This study proposed an amalgamated model derived from stress and coping theories that accounts for individual and environmental demands predisposing individuals to psychological distress (Cohen, Kessler, & Gordon, 1995; Dallman, La Fleur et al., 2004; Dallman et al., 2003). Psychological distress, the co-occurrence of perceived stress and depressive symptoms, may lead to HPA activation and unhealthy

dietary patterns. Cortisol secretion and high energy dense diets were viewed as biobehavioral mediators between psychological distress and cardiometabolic disease risk.

Psychological Factors and Biobehavioral Mediators

The study results provide limited support to the proposed relationship between psychological factors and biobehavioral mediators. In this study, increased depressive symptoms in combination with younger age, male gender and African-American race were correlated with higher food and beverage energy density. Perceived stress was not correlated with food and beverage energy density. The study results did not support the proposed relationship between psychological factors and HPA activation: depressive symptoms and perceived stress were not correlated with morning salivary cortisol level.

Psychological factors and dietary pattern.

Individual characteristics associated with the intake of high energy dense food and beverages include younger age, male gender, and African-American race. This finding is consistent with the literature. In a nationally representative sample, Ledikwe and others (2005) showed that younger age, male gender and non Hispanic blacks consumed more energy dense foods than older adults, women and non Hispanic whites (Ledikwe et al., 2005).

In contrast to the literature (Cox & Mela, 2000), BMI did not contribute to explaining food and beverage energy density. This finding seems counterintuitive and may be due to the problem of reporting inadequate caloric intake: obese participants underreported their caloric intake, and obese men, in particular, reported similar caloric

intake as overweight men. Underreporting, particularly by obese individuals, is well established in the literature (Gibson, 2005).

According to the literature, individuals with low dietary restraint have different dietary patterns than those with high restraint which may predispose them to waist circumference risk (De Lauzon et al., 2004). In this study, there was no association between dietary restraint with food and beverage energy density. This lack of association may be due to the problem of not reporting adequate caloric intake: individuals with low dietary restraint were less likely to report adequate caloric intake which would weaken any potential relationship.

Perceived stress did not account for additional variance in food and beverage energy density above that explained by individual characteristics and depressive symptoms. These findings are not consistent with research that has demonstrated an association between chronic stress and the intake of high fat chow in rats (La Fleur, Akana, Manalo, & Dallman, 2004), chronic stress and higher caloric intake in subordinate compared to dominant macaques (Wilson et al., 2008), and novel stress with a preference for sweet foods in women (Epel, Lapidus, McEwen, & Brownell, 2001).

These findings may be due, in part, to participants reporting relatively low levels of stress. On average, participants reported their levels of stress to be comparable to that of other working adults. Furthermore, the majority of participants were well-educated professionals suggesting that they have access to resources, such as health information, that could mitigate the negative effects of stress. Additionally, participants were relatively healthy and did not experience any limitations in physical activity due to their

health. Although these individuals experienced environmental demands similar to that of other working adults, access to resources and good health may mitigate the effects of stress (Cohen, Kessler, & Gordon, 1995).

In comparison, depressive symptoms did account for additional variance in food and beverage energy density above that explained by individual characteristics. Younger adults, men, African-Americans, individuals who reported adequate caloric intake and those with increased depressive symptoms reported consuming high energy dense food and beverages. Participants with increased depressive symptoms consumed higher energy dense foods and beverages than did those without depressive symptoms; for each gram of food and beverage consumed, individuals with depressive symptoms consumed more kilocalories than did individuals with no depressive symptoms. Individuals with depressive symptoms may compensate for feelings of stress and low mood by consuming not only highly palatable, sweet foods but also sweetened beverages. Sweetened beverages, in contrast to solid food, may not satisfy appetite and may contribute to excess energy intake. Of concern, caloric soft drinks, fruit juice and fruit punch consumption are correlated with weight gain (Bellisle & Drewnowski, 2007). Additionally, other health behaviors that may increase cardiometabolic risk are linked to sweetened soft drink consumption: those consuming the highest amounts of sugar sweetened soft drinks are less physically active and more likely to be smokers than those consuming the least (Bellisle & Drewnowski, 2007). Previous research has shown that individuals with depressive symptoms are more likely to drink alcoholic beverages (Strine et al., 2008); however, the finding that depressive symptoms were associated with higher food and nonalcoholic beverage intake suggests further research

regarding the relationship between depressive symptoms and caloric, non-alcoholic beverage consumption is warranted. The study results lend support to the proposed relationship between depressive symptoms and food and beverage energy density.

Psychological factors and HPA activation.

The study results did not support the proposed relationship between psychological factors and HPA activation. There was no significant relationship between depressive symptoms and morning cortisol level or between perceived stress and morning cortisol level. The negative findings may be because the majority of participants (n= 68, 78.2%) were not depressed and not stressed (n=85, 97.7%). Additionally, the negative findings may be due to the salivary cortisol sampling protocol.

This negative finding is consistent with a population based study conducted by Alderling and others (2006). Of the 529 adults participating in the study, 348 were women (Alderling, Theorell, de la Torre, & Lundberg, 2006). The authors did not report or adjust for menopausal status in the analysis. Demand-control at work was assessed using the 11-item Demand-Control-Support Questionnaire. The analyses were adjusted for BMI, medication use, smoking and alcohol consumption. There was no significant difference in the mean morning salivary cortisol concentration, sampled at the time of awakening, between men with high job demands compared to men with low job demands. There was no significant difference in morning cortisol, sampled at the time of awakening, between women with high job demands compared to low job demands. However, women with high stress jobs had higher cortisol levels 30 minutes after awakening than did women with low stress jobs. Alderling and others (2006) suggest

that women may be more susceptible to job stress immediately after awakening as they begin to anticipate these daily stressors (Alderling, Theorell, de la Torre, & Lundberg, 2006). These findings suggest that a salivary cortisol sampling protocol that includes samples collected at awakening and 30 minute after awakening may be a more sensitive measure of HPA activity.

Biobehavioral Mediators and Cardiometabolic Disease Risk

The study results do support the proposed relationship between dietary pattern and abdominal obesity. The study results do not support the proposed relationship between HPA activation and abdominal obesity.

Dietary pattern and abdominal obesity.

In this study, there was a significant correlation between the mediating variable, food and beverage energy density, and the outcome variable, waist circumference while controlling for individual and psychological factors. Additionally, food energy density was correlated with waist circumference while controlling for individual and psychological factors. Appendix E.

Dietary patterns associated with increased waist circumference are not well understood, and the role of beverage consumption is particularly perplexing. A recent study linked food energy density with abdominal obesity and cardiometabolic disease risk. Mendoza and others (2007) showed that food energy density was correlated with elevated waist circumference in men and women (Mendoza, Drewnowski, & Christakis, 2007). Food energy density was also associated with elevated fasting insulin levels and the metabolic syndrome: participants were 10% more likely to have metabolic

syndrome with each standard deviation increase in food energy density (Mendoza, Drewnowski, & Christakis, 2007).

Mendoza and others (2007) did a secondary analysis of data from the NHANES study which provided a large nationally representative sample (Mendoza, Drewnowski, & Christakis, 2007). Diet was assessed primarily with an interview and the use of a multiple pass computer assisted recall method. A challenge in linking dietary energy density and disease risk is calculating the dietary energy density value. Mendoza and others (2007) calculated energy density values of food only and excluded all beverages (Mendoza, Drewnowski, & Christakis, 2007). Their rationale was that beverages have a lower energy density than food, and this may disproportionately impact individual energy density values (Mendoza, Drewnowski, & Christakis, 2007). Although the statistical analyses conducted by Mendoza and others (2007) informed the procedures used in this study, there were also differences (Mendoza, Drewnowski, & Christakis, 2007). Mendoza and others (2007) stratified the sample by gender for multivariate linear regression analysis (Mendoza, Drewnowski, & Christakis, 2007). In this study, due to the small sample size, gender was controlled in linear regression analyses. In both studies a standardized dietary energy density variable with each unit of dietary energy density representing a 1-standard deviation change was used in linear regression models. The gram weight of food and beverages was entered as a covariate in the linear regression model. In addition to controlling for age, gender, race, income and total weight of food in grams, Mendoza and others (2007) also controlled for physical activity which was measured by self report as a categorical variable with 4 levels (Mendoza, Drewnowski, & Christakis, 2007). In comparison, physical activity was

controlled in this study by excluding individuals who reported 30 minutes or more of physical activity on three or more days of the week. BMI was not controlled in either analysis.

HPA activation and abdominal obesity.

Although there were null findings in the linear regression model testing the relationship between morning cortisol and abdominal obesity, the finding that individuals with waist circumference risk had significantly higher morning cortisol levels than those without waist circumference risk is important. Of concern, previous research has shown that higher morning cortisol levels are predictive of severity of cardiometabolic disease complications (Reynolds & Walker, 2007).

The finding that morning cortisol, a manifestation of HPA axis function, did not contribute to explaining waist circumference variance is consistent with findings from a study conducted by Steptoe and others (2004) which examined the relationship between HPA axis dysregulation and abdominal obesity (Steptoe, Kunz-Ebrecht, Brydon, & Wardle, 2004). Their sample was composed of 89 men and 83 women aged 47-59 years. Among men, the mean BMI was 25.6 (*SD* 3.3) kg/m^2, and the mean waist-hip ratio was .91 (*SD* .07). Among women, the mean BMI was 25.4 (*SD* 4.0) kg/m^2, and the mean waist-hip ratio was .80 (*SD* .11). Salivary cortisol samples were collected multiple times over a day and five cortisol parameters were analyzed which included cortisol on waking, cortisol response to waking (30 minutes after waking – waking value), cortisol day mean, cortisol evening minimum (lowest value recorded between 8:00-8:30 p.m. and 10:00-10:30 p.m.) and cortisol change over the day. The cortisol change over the day was calculated as the difference between cortisol measured 30 minutes after

awakening and the minimum evening value. They assessed abdominal obesity using measured waist and hip circumferences to calculate waist-hip ratio (Steptoe, Kunz-Ebrecht, Brydon, & Wardle, 2004).

Consistent with this study, there was no significant relationship between cortisol on waking and abdominal obesity among men or women. Additionally, there was no significant relationship between cortisol change over the day (waking – evening value) and abdominal obesity in men or women. However, the cortisol response to waking value (30 minutes after waking – waking value) was positively correlated with waist-hip ratio in men while controlling for age, socioeconomic status, alcohol intake and time of waking. In addition, the cortisol change over the day (30 minutes after waking – minimum evening value) was positively correlated with waist-hip ratio in men while controlling for age, socioeconomic status, alcohol intake and time of waking (Steptoe, Kunz-Ebrecht, Brydon, & Wardle, 2004).

Steptoe and others (2004) did not show a significant relationship between HPA axis dysregulation and abdominal obesity in women. All the women were postmenopausal with 28.9% using hormone replacement therapy (Steptoe, Kunz-Ebrecht, Brydon, & Wardle, 2004). Controlling for the use of hormone replacement therapy did not alter their findings. They offer several possible explanations for their negative findings. One explanation was that men tend to have larger visceral fat deposits than women, and since glucocorticoid receptors are more abundant in the visceral fat, the impact of HPA axis dysregulation was weakened among the women (Steptoe, Kunz-Ebrecht, Brydon, & Wardle, 2004). Although this explanation is consistent with the literature that reports men have larger visceral fat deposits than

women and glucocorticoid receptors are dense in the visceral adipose (Bjorntorp, 1991), the argument would be stronger if they had triangulated their anthropometric data with another method of assessing body composition such as dual x-ray absorptionmetry (DXA). Another explanation offered by the researchers was that men may be more susceptible to environmental exposures, thus increasing cortisol following awakening (Steptoe, Kunz-Ebrecht, Brydon, & Wardle, 2004). This explanation contradicts that of Alderling and others (2006) who argue that women may be more susceptible to environmental exposures immediately following awakening (Alderling, Theorell, de la Torre, & Lundberg, 2006). However, neither group of researchers collected a stress or environmental exposure measure simultaneously with cortisol sampling. Alderling and others (2006) used an 11 item questionnaire to measure demand control at work, and this was administered at a time separate from the collection of the salivary cortisol samples (Alderling, Theorell, de la Torre, & Lundberg, 2006). In comparison, Steptoe and others (2004) did not include a measure of perceived stress or environmental exposures (Steptoe, Kunz-Ebrecht, Brydon, & Wardle, 2004). These conflicting explanations underscore the complexity of the associations among these dynamic processes.

In contrast, Travison and others (2007) reported a negative correlation between morning cortisol levels and abdominal obesity (Travison, O'Donnell, Arajuo, Matsumoto, & McKinlay, 2007). In a longitudinal study of 999 men ages 40-79 years with a mean BMI of 27.7 (*SD* 4.4), serum cortisol samples and body composition (waist circumference, waist hip ratio, and bioimpedance) measurements were obtained twice with a 5 year interval between the assessments. Twenty-seven percent of the men had a

normal BMI, 47% were overweight and 26% were obese. At each time point, two non-fasting blood samples were collected within 4 hours of awakening to assess morning cortisol levels. The two blood samples were drawn within a 30 minute time interval and pooled to smooth episodic secretion. At baseline, obese men had lower morning cortisol levels than non-obese men, and there was a weak negative correlation between waist circumference and waist hip ratio with morning cortisol level; as waist circumference increased, morning cortisol level decreased (Travison, O'Donnell, Arajuo, Matsumoto, & McKinlay, 2007).

Methodological differences in specimen collection make it difficult to directly compare study results. Travison and others (2007) conducted the analysis on serum as compared to saliva (Travison, O'Donnell, Arajuo, Matsumoto, & McKinlay, 2007). The samples were collected over a wide time interval following awakening making it difficult to interpret the values. Pooling the samples further complicates interpretation. Additionally, these were non-fasting samples making it unclear how food intake may have affected cortisol levels (Travison, O'Donnell, Arajuo, Matsumoto, & McKinlay, 2007). Travison and others (2007) hypothesize that the over expression of 11ß-hydroxysteroid dehydrogenase enzyme in the adipose tissue would increase the conversion of the inactive form, cortisone, into its active form, cortisol. This would increase glucocorticoid exposure at the tissue level, yet it would have very little impact on the circulating cortisol concentrations (Travison, O'Donnell, Arajuo, Matsumoto, & McKinlay, 2007).

The work by Kurina and others (2004) demonstrates the variability in diurnal cortisol secretion pattern highlighting the challenge in establishing relationships

between psychological distress and cortisol levels (Kurina, Schneider, & Waite, 2004). They studied 91 working parents to determine the relationship between psychological distress and cortisol secretion patterns. Examining 24 possible correlations between measures of psychological distress and cortisol pattern, only two correlations were significant. In addition to random variation, Kurina and others (2004) argue that variability in diurnal cortisol pattern may be due to genetic differences in baseline cortisol secretion, exposure to environmental factors that affect secretion and sensitivity to these environmental exposures (Kurina, Schneider, & Waite, 2004). Activity, eating, sleeping and reproductive factors are environmental exposures that affect cortisol secretion. Also, participants in their study reported relatively low levels of stress, thereby limiting the ability to detect a true relationship. It may be that the noise created by these other factors, overpowers any true relationship between psychological distress and diurnal cortisol pattern. Because the effect size appears small, a larger sample may have been needed to detect a true relationship (Kurina, Schneider, & Waite, 2004). Among the women participants, there was an inverse relationship between work stress and average cortisol levels which was not observed in the men. Kurina and others (2004) attribute this to a possible down regulation of the HPA axis in response to stress that may occur in some individuals. Additionally, they attribute this finding to gender differences in coping with stress; for instance, women are more likely to participate in nurturing and networking activities than men (Kurina, Schneider, & Waite, 2004).

Our negative findings add information about HPA axis dysregulation and abdominal obesity in a more diverse population than that of the study conducted by Steptoe and others (2004) (Steptoe, Kunz-Ebrecht, Brydon, & Wardle, 2004). In this

study, the participants had higher BMIs with a mean BMI of 30.98 kg/m^2 for men, and the mean BMI of 32.54 kg/m^2 for women. In comparison, in the study by Steptoe and others (2004), the mean BMI was 25.6 (*SD* 3.3) kg/m^2 for men, and the mean BMI was 25.4 (*SD* 4.0) kg/m^2 for women (Steptoe, Kunz-Ebrecht, Brydon, & Wardle, 2004). Additionally, the participants in this study were primarily African American and younger with a mean age of 38.4 (*SD* 9.9) years for men and a mean age of 42.4 (*SD* 10.1) years for women. In comparison, in the study by Steptoe and others (2004), the sample was composed primarily of whites and older adults with the mean age of 52.5 (*SD* 2.6) years for men and 51.9 (*SD* 2.7) years for women (Steptoe, Kunz-Ebrecht, Brydon, & Wardle, 2004).

Dallman and others (2004) argue that abdominal obesity is related to activation of the HPA axis in response to chronic stress, our findings do not lend support to this regarding morning salivary cortisol level at awakening (Dallman, Akana et al., 2004). To better understand the relation between HPA function and abdominal obesity, cortisol response to awakening (awakening value – 30 minutes after awakening) or cortisol change over the day may be better indicators than cortisol measured only at awakening. Additionally, a larger sample size may have been needed to detect the proposed relationship.

Psychological Factors and Cardiometabolic Disease Risk

The study results provide support of the proposed relationship between psychological factors and cardiometabolic disease risk. The study results show a significant correlation between depressive symptoms with waist circumference and between perceived stress with waist circumference. The study results do not support the

food and beverage energy density or cortisol secretion as mediators between depressive symptoms, perceived stress and waist circumference. However, important associations among these variables have been identified and merit further study. For example, depressive symptoms are related to food and beverage energy density as well as waist circumference. Although food and beverage energy density may not mediate the relationship, it may be an independent factor associated with elevated waist circumference.

Psychological factors, biobehavioral mediators and disease risk.

The evidence does not support that food and beverage energy density mediates the relationship between psychological factors and waist circumference. However, individuals with depressive symptoms consumed higher energy dense foods and beverages than those without depressive symptoms, and they consumed higher energy dense foods and nonalcoholic beverages than those without depressive symptoms. High energy dense diets are associated with poor nutritional quality suggesting that there may be important nutritional differences between those with depressive symptoms compared to those without (Cuco, Arija, Marti-Henneberg, & Fernandez-Ballart, 2001; Ledikwe et al., 2006b; Marti-Henneberg et al., 1999). Poor dietary quality may increase the risk for cardiometabolic disease among individuals who are more likely to drink alcohol, smoke, and be physically inactive (Strine et al., 2008).

The finding that there was no significant difference in morning cortisol between those with and without depressive symptoms may be due to differences in the number of participants in each group. Among participants with morning cortisol data, 18 participants reported mild, moderate or severe depressive symptoms compared to 61

participants who did not. This unequal distribution may have masked a true relationship. This finding is consistent with the literature. Strickland and others (2002) reported no significant relationship between depression and cortisol concentrations in women (Strickland et al., 2002). In a study of 453 women, 94 of these women were diagnosed with depression: 48 were mildly depressed, 37 were moderately depressed and 9 were severely depressed. Saliva samples were collected at 9:00 a.m. and 11:00 p.m. on two consecutive days. These samples were collected during the follicular phase of the menstrual cycle or during the pill-free days for women using oral contraceptive pills. Social adversity and vulnerability to depression was assessed using the Life Events and Difficulties Schedule and the Self Evaluation and Social Support Scales. Although the women with depression did not have significantly different morning or evening cortisol levels than those without depression, a subgroup analysis revealed interesting findings. Depressed women with high psychosocial difficulties, as measured by the Life Events and Difficulties Schedule, had higher evening cortisol levels than depressed women with low psychosocial difficulties. Although half of the women reported mild depression, the most severely depressed women did not have significantly higher morning cortisol levels. The null findings may be due, in part, to the lack of variability in depressive symptoms. The researchers conclude that depression among community dwelling adults is not associated with sustained elevated cortisol levels (Strickland et al., 2002).

Methodological Issues

During the study period, several issues related to the methodology were addressed. First, it was difficult to recruit men. Additional recruitment strategies were

initiated including aggressive follow up with men expressing interest at health fairs and community events as well as posting flyers in venues frequented by men. Snowball sampling techniques were also used: men participants were asked to tell their male friends, family members and co-workers about the study. Women participants were asked if they had a male friend or family member who might be interested in participating. Despite these efforts, men were not well represented. Second, it was difficult to recruit low income individuals. The principal investigator participated in numerous community health fairs many of which were held in shopping malls and churches in low income neighborhoods. Despite aggressive efforts to recruit and enroll participants, only 19.8% were recruited through health fairs.

A challenge in dietary assessment is related to variability in dietary intake. For most nutrients there is greater within-person variability than between-person variability, and large within-person variability reduces the strength of correlations with the outcome variable (Gibson, 2005). Between-person variability measures the differences between individuals in their usual daily intake. A large, representative sample minimizes between- person variability. Additionally, age and gender differences contribute to between-person variation (Gibson, 2005). The small sample size and the use of convenience sampling may have contributed to between-person variation in usual daily intake.

Within-person variability measures the day to day variation in the dietary intake of the individual, and it depends, in part, on how similar an individual's food choices are on a day-to-day basis (Gibson, 2005). Food and beverage energy density values were higher on day three than on day one indicating that individual's food and beverage

choices varied from the first to last study day. Increasing the number of measurement days minimizes within-person variability (Gibson, 2005). In this study, three days of weighing and recording were deemed appropriate to minimize within-person variability without excessive participant burden. Seasonal effects can increase variability; however, seasonal effects are considered to be relatively small in industrialized countries (Gibson, 2005).

Although the weighed three day food record is the gold standard in dietary assessment, teaching participants how to weigh and measure their food and beverages, estimate portion sizes and complete the food record as well as reviewing the weighed food record after it was completed were time intensive activities. This method of dietary assessment placed a burden on participants, the majority of whom had full time job responsibilities and long commutes. Using these face to face visits to collect two multiple pass, 24 hour recalls may have resulted in a higher percentage of participants reporting adequate caloric intake and better estimates of dietary energy density values.

Post hoc power analyses showed that the study was underpowered. The study was originally powered to test hypothesis 6. The sample size was calculated to detect a 12% change when testing four independent variables while holding four variables constant. The power analysis underestimated the amount of variation explained by the control variables and overestimated the amount of variation explained by the independent variables. Because the study was underpowered, the study findings are considered preliminary.

Study Limitations

This study employed convenience sampling to enroll participants. The study was biased toward relatively well educated professionals earning relatively high incomes. The study activities required highly motivated participants, and people who chose to enroll in this study may be different from non-participants. Two incentives were offered to participants; a dietary pattern analysis completed by the study dietitian and a $25 gift card. Many individuals were motivated to participate in order to receive this dietary analysis suggesting that individuals who were concerned and wanted to learn about their dietary quality were more likely to enroll. Despite best efforts, it was difficult to enroll men. Of the men, the majority did not have waist circumference risk making it difficult to generalize these findings to men with at risk waist circumference.

In order to minimize the effect of reporting inadequate caloric intake, a protocol was used to standardize the level of detail and description of food and beverage intake. Memory and measurement aids were used to ensure accuracy, and the time interval between recording and review with the principal investigator was minimized (Gibson, 2005). Despite these rigorous methods, the majority (n=51, 58.6%) of participants reported inadequate caloric intake which has the potential to weaken associations and mask significant findings. Briefel and others (1997) examined NHANES III data to estimate the extent of underreporting caloric intake. The sample was comprised of 7,769 adults aged 20 years or above, and dietary data were collected using a 24 hour recall method. A ratio of estimated energy expenditure to estimated basal metabolic rate was used to evaluate underreporting caloric intake. In the sample, 18% of men and 28% of women underreported caloric intake (Briefel, Sempos, McDowell, Chien, & Alaimo,

1997). Among those underreporting caloric intake, 47% were overweight (Briefel, Sempos, McDowell, Chien, & Alaimo, 1997). Methodological differences make it difficult to compare the extent of underreporting between this study and that of Briefel and others (1997). Dietary assessment methods and sample characteristics differed. For example, this sample included only overweight individuals.

Reporting inadequate caloric intake can be attributed to multiple factors. First, participants may have under-recorded: participants may not have written down everything they ate and drank. Second, participants may have underestimated portion sizes. Estimating composite foods was particularly challenging and may have resulted in underestimating quantities of multiple components. Third, participants may have under-consumed. Participants may have consumed less food and/or beverages during the three days of reporting. Finally, weighing and writing down all food and beverages consumed as well as how they were prepared may have created a recording burden (Gibson, 2005).

In this study, the mean energy density values were relatively low which may be explained, in part, by the problem of not reporting adequate caloric intake. Several other factors may help explain the low mean energy density values. For example, men with adequate intake reported a high intake of vegetables. Because of the low calorie per gram ratio, high vegetable intake will drive down the mean energy density values. Conversely, because of the high calories per gram ratio, high fat foods will drive up the mean energy density values. The majority of participants did not report adequate caloric and dietary fat intake which may have acted to lower the mean energy density value. Because beverages have a low calorie per gram ratio, non-caloric beverage intake drives

down the mean energy density value. A large number of participants consumed either large quantities of water and other non-caloric beverages which will lower the mean beverage energy density value. Significant correlations or trends toward associations would likely be stronger if a larger percent of the sample had reported adequate caloric intake.

Only one day of salivary cortisol data were collected limiting the ability to generalize the findings related to morning salivary cortisol levels. Salivary cortisol data were missing on nine participants (10.3%) due to inadequate collection. Although participants did not report difficulties collecting the saliva samples, it is possible that participants may not have collected the sample immediately upon awakening as instructed. Due to the large variability in the time that the evening sample was collected, data were not usable, and the diurnal pattern of cortisol secretion could not be assessed.

Limited data were collected on other behaviors that may influence waist circumference. Physical activity was controlled by including only sedentary adults; however, among sedentary adults there is variation in physical activity that was not quantified. For example, some participants had jobs which required standing much of the day as compared to participants who had jobs sitting much of the day. Additionally, some participants may have taken short 20 minute walks twice a week as compared to participants who did not walk for leisure. Although this represents a narrow window of activity, there was variability in energy expenditure between participants that was not quantified. In addition, data were only collected on current smoking and reported alcohol intake. These data would be strengthened if information on past smoking

behaviors were collected. Additionally, alcohol intake data would be strengthened if information on alcohol consumption patterns were collected.

Measured waist circumference was used to assess abdominal obesity, and these anthropometric data were not triangulated with other methods of assessing body composition. For instance, abdominal sagittal diameter is another method of assessing abdominal visceral adipose tissue, and it is preferred over the waist-hip ratio because it more strongly correlates with metabolic risk (Pouliot et al., 1994). Bioimpedance, another method of assessing total body water and estimating fat-free mass and body fat, is safe and relatively inexpensive. Careful participant instruction is necessary as the estimates are affected by hydration status, physical activity, food and beverage intake and body position (Gibson, 2005). Computerized tomography (CT), magnetic resonance imaging (MRI) and dual energy X-ray absorptiometry (DEXA) are more precise methods of determining body fat distribution (Gibson, 2005). No clinical laboratory data were collected which limits the ability to determine if there were correlations between dietary energy density or morning cortisol with metabolic profiles.

Study Strengths

Enrolling a large number of African-American women provided insight into the biobehavioral responses of a population at high risk of cardiometabolic disease. Additionally, very little questionnaire data were missing ensuring efficient use of available data and minimizing biased estimates (Fitzmaurice, 2008). Finally, three approaches (macronutrient composition, food group pattern and dietary energy density values) were used to examine dietary pattern providing a comprehensive assessment of dietary quality. Dietary energy density added to the understanding of the associations

between psychological factors and cardiometabolic disease risk beyond that explained by macronutrient composition and food group pattern.

Recommendations for Research

Research is needed to better understand how to identify individuals prone to depressive symptoms, and to determine if an improvement in depressive symptoms alters food and beverage energy density. Short, reliable instruments aimed at assessing depressive symptoms need to be tested, and nursing interventions aimed at reducing depressive symptoms need to be developed and tested in clinical trials. It is suggested that future researchers use a short screening tool to identify and enroll individuals experiencing depressive symptoms allowing for greater variability in depressive symptoms among participants. This study examined the role of chronic stress in working adults. It may be that a measure of chronic stress does not adequately capture the kind of stress that affects eating behaviors and dietary patterns. In future studies, it may be important to measure more specific types of stress, such as work or job related stress in working adults. Additionally, research is needed to understand how the intensity and duration of stress may affect eating behaviors and dietary patterns.

The finding that women with high dietary restraint had significantly lower BMIs and waist circumferences than women with low dietary restraint is of great interest. Research has shown that individuals who have been successful in maintaining weight loss use more behavioral strategies to control their body weight and pay more attention to fluctuations in their body weight (McGuire, Wing, Klem, & Hill, 1999). Further research is needed to determine if dietary restraint is a potentially modifiable characteristic.

The finding that individuals with depressive symptoms consumed high energy dense beverages suggests further research regarding the relationship between depressive symptoms and beverage consumption is warranted. Studies are needed to describe the type and quantity of beverages consumed by overweight adults experiencing depressive symptoms, and to test if interventions aimed at reducing depressive symptoms alters the intake of high energy dense beverages.

After experiencing a myocardial infarction, men participating in behavioral as compared to cardiac counseling experienced improvements in depressive symptoms (Mendes de Leon, Powell, & Kaplan, 1991). The behavioral counseling sessions were conducted by psychologists and psychiatrists, and focused on identifying negative symptoms such as anger and depression, practicing behavioral changes, and exploring healthier alternatives. In addition to improved mood, reduced stress and gains in self efficacy, there was a 44% reduction in the incidence of re-infarction among the men participating in the behavioral as compared to the cardiac counseling program (Mendes de Leon, Powell, & Kaplan, 1991). A nursing intervention incorporating components of the behavioral counseling program and focusing on improving self management behaviors needs to be developed and tested in a more diverse population and among relatively healthy adults prior to the onset of cardiometabolic disease.

A recent study highlights the important relationship between physical activity and mood: young men engaging in low levels of physical activity experienced less depressive symptoms than inactive young men. Additionally, women engaging in moderate levels of physical activity experienced less depressive symptoms than inactive young women (Augestad, Slettemoen, & Flanders, 2008). A nursing intervention that

incorporates a tailored physical activity component needs to be developed and tested to determine if a physical activity program tailored to meet individualized intensity, frequency and duration goals improves depressive symptoms.

Psychological distress is frequently accompanied by poor sleep (Srinivasan et al., 2009). Interestingly, duration of nighttime sleep has emerged as important factor linked to elevated BMI (Taheri, Lin, Austin, Young, & Mignot, 2004; Vorona et al., 2005). Short nighttime sleep has been shown to adversely affect appetite regulating hormones, increase sensations of hunger and preference for sweet, salty and starchy foods (Spiegel, Tasali, Penev, & Van Cauter, 2004; Taheri, Lin, Austin, Young, & Mignot, 2004). Future studies should explore this novel and underappreciated relationship as the quality and duration of nighttime sleep are potentially modifiable factors.

In addition to an intervention aimed at improving depressive symptoms, dietary messages should be developed and tested in at risk groups, such as younger adults, men and African-Americans, and innovative approaches to deliver these messages need to be tested. For instance, tailored messages that can be delivered electronically may be the most effective approach to reaching young adults. The dietary messages as well as the mode of delivery should be tested in clinical trials.

Modifying dietary energy density may be an appropriate strategy to reduce waist circumference. Nursing interventions aimed at older adults and those experiencing depressive symptoms that focus on modifying energy density by replacing energy dense foods with high volume foods such as fruits and vegetables and replacing caloric beverages with non-caloric beverages need to be tested. Interventions aimed at

increasing the intake of low fat milk products to recommended amounts need to be tested to determine the effect of adequate dairy intake on waist circumference. Factors that contribute to inadequate intake of milk products need to be better understood so that appropriate interventions can be developed.

The lack of variability in perceived stress scores may have masked the relationship between perceived stress and dietary energy density. In future studies, a short reliable instrument, such as the perceived stress scale, could be used as a screening tool in order to enroll individuals experiencing a wider spectrum of perceived stress scores. Despite the negative findings, there may be more to learn about the role of HPA activation and cortisol secretion in understanding the relationships between psychological distress and abdominal obesity. In future studies, researchers should collect at least three salivary cortisol samples over a minimum of two days in order to examine the awakening response and diurnal pattern of cortisol secretion as well as assess the consistency of the daily pattern. In future studies, triangulating salivary cortisol data with another measure may provide a better understanding of the biological responses to stress and depressive symptoms. Heart rate variability, the small beat-to-beat differences in heart rate which are controlled by the balance between the parasympathetic and sympathetic nervous systems, may be a useful biological measure. Low heart rate variability has been linked to anxiety, depression and cardiovascular disease (Gorman & Sloan, 2000; Moses, Luecken, & Eason, 2007).

To better understand the role that behavioral factors may play in body weight regulation and potentially fat deposition in the abdomen, future studies should include information related to current and past smoking behaviors, and information related to

average daily alcohol intake as well as alcohol consumption patterns. Physical activity patterns, including frequency, duration and intensity of physical activity, need to be assessed; however, measuring physical activity in a sedentary population is challenging. First, few instruments query light and moderate intensity activities such as child care, housework and occupational activities. Second, few tools capture intermittent or incidental activities resulting in a floor effect. Third, walking, which is considered to be the most important physical activity to measure in this group is also the least reliably recalled activity. Walking is difficult for participants to quantify because it occurs across settings. Additionally, many people have difficulty understanding questions about speed, pace or intensity of walking (Tudor-Locke & Myers, 2001). Measuring physical activity in midlife and minority women is another challenge, and instruments currently available do not adequately capture physical activity in these women. This may be due, in part, to the type of activities queried. Measuring activities that are relevant to women, such as physical activity associated with volunteer work or religious services should provide a more accurate estimate of physical activity. Because women fulfill multiple roles within the family and culture, activities associated with household responsibilities such as elder care or child care need to be estimated. Many women are engaged in multiple activities simultaneously; therefore, estimating the total time of physical activity rather than estimating the amount of each activity separately may be more effective (Masse et al., 1998). Studies are needed to evaluate the psychometric properties of various physical activity measures in this population.

In the future, methods that capture the dynamic nature of body weight regulation and body fat distribution are needed. Typically, dietary assessment focuses on 24 hour

time intervals. Using this approach, researchers potentially lose information about within the day variation of energy intake and expenditure (Deutz, Benardot, Martin, & Cody, 2000). For example, the weighed food record could be used to collect information about the timing and duration of meals and snacks. Dietary data combined with information about the timing, intensity and duration of physical activity could be used to better understand how energy deficits and surpluses over the course of the day may affect dietary patterns, mood states, body composition and energy metabolism. This information could be obtained from a 24 hour physical activity recall conducted by a trained interviewer using a standardized protocol. This approach has been used with elite athletes to quantify energy deficits and surpluses and body composition. Among elite athletes, greater within day energy deficits were associated with a higher percentage of body fat (Deutz, Benardot, Martin, & Cody, 2000). The reliability and validity of this method needs to be evaluated in overweight adults to better understand how food intake and energy expenditure over the day are related to mood and body composition.

The study results demonstrated a relationship between depressive symptoms and abdominal obesity. However, the biologic mechanism proposed did not mediate the relationship. It is possible that other biobehavioral pathways mediate the relationship between depressive symptoms and abdominal obesity. One possible pathway involves inflammation. Depression has been correlated with increased inflammation in clinical and community based samples (Howren, Lamkin, & Suls, 2009). In obesity, enlarged adipocytes and macrophages store lipids and secrete proinflammatory proteins such as tumor necrosis factor-alpha, interleukin 6 and C-reactive protein. Leptin increases the

expression of interleukin 6 which promotes the release of C-reactive protein. These biomarkers of inflammation are elevated in abdominal obesity and are predictors for the development of cardiometabolic disease (Lee & Pratley, 2005). Sedentary behavior may also play a role as it promotes inflammation (Howren, Lamkin, & Suls, 2009).

To better understand this possible mechanism, a descriptive study that examines inflammation, antioxidant intake and supplementation and physical activity as biobehavioral mediators between depressive symptoms and abdominal obesity is warranted. Dietary assessment of antioxidant rich foods and supplement use could be triangulated with antioxidant biomarkers such as serum beta carotene levels, serum alpha tocopherol levels, serum ascorbic acid levels and glutathione (Gibson, 2005). Biomarkers of inflammation such as tumor necrosis factor-alpha, interleukin 6 or C-reactive protein would be collected, and physical activity could be measured using an accelerometer or pedometer (Howren, Lamkin, & Suls, 2009). Abdominal obesity would be measured using two anthropometric measurements such as waist circumference and sagittal diameter and computerized tomography (Gibson, 2005). The findings would clarify if the relationship between increased depressive symptoms and elevated waist circumference has a behavioral component mediated by diet and/or physical activity, and/or if it has a biologic component mediated through the inflammatory response.

The study results also demonstrated important associations among depressive symptoms, food and beverage energy density and abdominal obesity. For this reason, a clinical trial that examines the effectiveness of a nursing intervention directed at reducing depressive symptoms coupled with an intervention directed at reducing dietary

energy density is warranted. The transtheoretical model and stages of change theory would provide the basis of the intervention and would incorporate processes of change including self liberation, commitment to act on change, helping relationships, counterconditioning, reinforcement management and stimulus control. Overweight individuals would be enrolled and randomly assigned to either the usual care or the intervention group. Participants would be followed for 12 months, and measurements would be collected at baseline and every four months. Potential measurements would include mood, dietary and physical activity assessments. Outcome variables would include abdominal obesity and cardiometabolic disease risk. Measures of outcome variables would include anthropometry such as waist circumference, sagittal diameter and metabolic and lipid profiles.

The nursing intervention would use counseling to teach participants how to reduce depressive symptoms with behavioral strategies which would include increasing physical activity. The behavioral counseling component would be delivered over the telephone by a nurse interventionist, and it would focus on identifying healthier coping behaviors, problem solving how to replace unhealthy coping behaviors, identifying relationships that offer acceptance and support for behavior change, removing cues that reinforce problem behaviors and adding cues to engage in healthy behaviors. Increasing physical activity would be a counterconditioning strategy by replacing unhealthy behaviors with a healthier alternative. Behavioral counseling would focus on benefits gained through physical activity and enlisting others to support efforts to increase physical activity. Behavioral counseling would also focus on replacing energy dense foods with high volume, healthier alternatives such as fruits, vegetables and low fat

dairy products. Findings from such a study would clarify the effectiveness of nursing interventions aimed at reducing depressive symptoms and reducing dietary energy density on abdominal obesity and cardiometabolic risk.

Interventions need to be developed and tested aimed at building competencies in nurses to assess abdominal obesity and teach healthy dietary patterns. This intervention should focus on teaching nurses how to assess abdominal obesity as well as identifying appropriate intervention strategies for at risk individuals. The efficacy of these interventions should be tested in clinical trials and the effectiveness evaluated in a variety of worksites as well as clinical settings.

Recommendations for Clinical Practice

The findings from this study have implications for clinical nursing practice. Nurses should be aware of the definitions of overweight, and the risk obesity imposes on cardiometabolic health. Nurses should use measured height and weight to calculate BMI and identify individuals who are overweight (Cameron & Zimmet, 2008). In service and continuing education programs directed at teaching nurses about anthropometric measurements and basic nutrition are needed to build the capacity of the nursing work force. Once trained, nurses in clinical settings and worksites can collaborate with dietitians and other professionals to provide basic instruction and resources to help overweight individuals decrease the energy density of their diet and improve dietary quality. Basic instruction should include replacing non-nutritious caloric beverages with non-caloric and nutritious caloric beverages, consuming low fat milk and dairy products, decreasing saturated fats, consuming lean proteins, replacing simple with complex carbohydrates, and increasing fruit and vegetable intake. These

strategies will decrease dietary energy density, improve nutritional quality and potentially decrease abdominal obesity (Rolls, Drewnowski, & Ledikwe, 2005). Relevant patient resources are offered through the U.S. Department of Agriculture including My Pyramid which provides individualized dietary recommendations based on age, gender, height, weight and physical activity level (U.S. Department of Agriculture, 2005). In addition, nurses can help overweight individuals identify emotional states that may trigger unhealthy dietary patterns as well as explore and incorporate healthier dietary patterns into their daily lives. Obese individuals should be referred to a dietitian for nutrition counseling.

Because individuals who maintain weight loss use more behavioral strategies to manage their weight, nurses may be able to teach effective strategies. For example, research has shown that eating a piece of whole fruit, as opposed to drinking fruit juice, at the beginning of a meal improves satiety and decreases energy intake (Flood-Obbagy & Rolls, 2008). In addition, research has shown that consuming low energy dense soup or salad at the onset of the meal, decreases meal energy intake (Flood & Rolls, 2007; Rolls, Roe, & Meengs, 2004). On the other hand, research has shown some behavioral strategies are not effective. For example, using a smaller dinner plate has been advocated as an effective strategy to lose weight; however, research has shown that using a smaller dinner plate did not decrease meal energy intake (Rolls, Roe, Halverson, & Meengs, 2007). Highly restrained individuals who use rules to manage their body weight may be responsive to and benefit from instruction about behavioral strategies that are scientifically sound.

Nurses should be aware of the role that depressive symptoms can play in cardiometabolic risk. Mild to moderate depressive illnesses are common and, these are often undetected and untreated. In many clinical settings, mood and affect are not assessed or treated until they interfere with activities of daily living; however, early identification of and intervention for those at risk for depressive symptoms may improve cardiometabolic outcomes (Strickland et al., 2002). Nurses in workplaces and other community settings play an important role in early identification of individuals predisposed to depressive symptoms and referral to qualified mental health professionals. Nurses, in clinic settings, can play an important role in coordinating medical and mental health care for these individuals.

Finally, these findings have implications for worksite interventions. Importantly, working adults with cardiometabolic risk factor clusters missed 179% more work days and spent 147% more days in bed than adults without cardiometabolic risk factor clusters resulting in $17.3 billion in lost productivity to American businesses (Sullivan, Ghushchyan, Wyatt, Wu, & Hill, 2007). Worksite interventions have the potential to improve employee health and job performance while increasing business productivity.

Worksites offer a venue for occupational health nurses to provide educational and behavioral interventions in an environment where adults typically consume at least one meal per day (Obesity. Guide to Community Preventive Services Website. Centers for Disease Control and Prevention). Obese individuals report that they learn about weight loss strategies from friends and family and turn to commercially available weight loss products (Thomas, Hyde, Karunaratne, Kausman, & Komesaroff, 2008). Education for employees provided by qualified professionals about scientifically sound

strategies such as reducing dietary energy as a healthy, long term weight management strategy could be delivered at the worksite in a variety of formats such as via company intranet, at point of purchase for foods, at wellness fairs and health screenings or in face to face instruction (Ello-Martin, Roe, Ledikwe, Beach, & Rolls, 2007; Ledikwe et al., 2007; Rolls, Drewnowski, & Ledikwe, 2005). In addition, obese individuals report that although they may receive instruction, they lack the long term guidance and support needed to successfully change their dietary behavior ((Thomas, Hyde, Karunaratne, Kausman, & Komesaroff, 2008). Long term interventions aimed at supporting employees in their attempts to initiate and sustain dietary behavior change could be delivered at worksites. Obese individuals report that interventions that involve their social support networks help them feel accepted and supported in behavior change (Thomas, Hyde, Karunaratne, Kausman, & Komesaroff, 2008).The social support offered in the worksite may motivate individuals to initiate as well as sustain their efforts in dietary behavior change.

In addition to interventions aimed at employees, nurses can work with dietitians to assess the nutritional environment of the worksite and develop strategies aimed at improving the nutritional quality of food offered (Drewnowski & Rolls, 2005). For example, this may involve evaluating foods offered in vending machines and working with vendors and administrators to make low energy snack items available at a competitive price (French et al., 2001; French, Story, & Jeffery, 2001). Nurses can work with the management and human resource personnel to develop policies aimed at creating a work culture that makes low energy dense food choices, easy and convenient for the employee (Drewnowski & Rolls, 2005).

Summary

This descriptive, correlation study was undertaken to examine the associations among psychological distress, biobehavioral mediators and cardiometabolic disease risk. An adaptation of the Stress and Coping Model (Cohen, Kessler, & Gordon, 1995; Dallman, La Fleur et al., 2004; Dallman et al., 2003) provided the theoretical basis for the study. Results showed that depressive symptoms, in combination with younger age, male gender, and African-American race, were associated with the intake of high energy dense food and beverages. Increased depressive symptoms explained variance in food and beverage energy density above that accounted for by younger age, male gender and African-American race. Depressive symptoms in combination with younger age, African-American race, male gender, and reporting adequate caloric intake were associated with higher food and nonalcoholic beverage intake. Perceived stress was not correlated with dietary energy density. Neither depressive symptoms nor perceived stress were correlated with morning salivary cortisol levels.

Older age, increased depressive symptoms and high energy dense food and beverages were associated with increased waist circumference. High energy dense food and beverages explained variance in waist circumference above that accounted for by increased depressive symptoms and older age. There were significant bivariate correlations between depressive symptoms and perceived stress with increased waist circumference. Increased depressive symptoms explained waist circumference variance above that accounted for by older age and low dietary restraint. Increased depressive symptoms independently predicted elevated waist circumference. Although, there was insufficient evidence to conclude that dietary pattern mediated the relationship between

psychological factors and waist circumference, the relationship between food and beverage energy density with waist circumference merits further study. Food and beverage energy density independently predicted elevated waist circumference. Additionally, there was insufficient evidence to conclude that morning salivary cortisol level mediated the relationship between psychological factors with waist circumference. Study findings provided support for several of the proposed relationships in the theoretical framework and implications for future research and clinical nursing practice were identified.

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APPENDIX A

Multiple Regression Power Analysis

Page/Date/Time 1 3/21/2007 3:57:22 p.m.

Numeric Results

				Ind. Variables Tested		Ind. Variables Controlled	
Power	N	Alpha	Beta	Cnt	R^2	Cnt	R^2
0.80163	83	0.05000	0.10000	4	0.19837	4	0.12000

Power is the probability of rejecting a false null hypothesis.

N is the number of observations on which the multiple regression is computed.

Alpha is the probability of rejecting a true null hypothesis. It should be small.

Beta is the probability of accepting a false null hypothesis. It should be small.

Cnt refers to the number of independent variables in that category.

R^2 is the amount that is added to the overall R-Squared value by these variables.

Ind. Variables Tested are those variables whose regression coefficients are tested against zero.

Ind. Variables Controlled are those variables whose influence is removed from experimental error.

Summary Statements

A sample size of 116 achieves 80% power to detect an R-Squared of 0.12000 attributed to 4 independent variable(s) using an F-Test with a significance level (alpha) of

0.01000. The variables tested are adjusted for an additional 4 independent variable(s) with an R-Squared of 0.10000.

APPENDIX B

IRB Approval Letter

EMORY UNIVERSITY

Institutional Review Board

FROM: Susan M. Ray, MD
Vice Chair
Emory University IRB

TO: Daurice Grossniklaus
Principal Investigator

CC: Townsend Jill Nursing - Main
Dunbar Sandra Nursing - Main

DATE: March 1, 2007

RE: **Notification of Expedited Approval**
IRB00001568
Energy Density of Food Intake in Overweight Adults

This is your notification that your above referenced study was reviewed and APPROVED under the Expedited review process per 45 CFR 46.110(4) and 21 CFR 56.110. The approval is valid from **3/1/2007 until 2/28/2008**. Thereafter, continued approval is contingent upon the submission of a continuing review request that must be reviewed and approved by the IRB prior to the expiration date of this study.

Any reportable events (serious adverse events, breaches of confidentiality, protocol deviation or protocol violations) or issues resulting from this study should be reported immediately to the IRB and to the sponsoring agency (if any). Any amendments (changes to any portion of this research study including but not limited to protocol or informed consent changes) must have IR

B approval before being implemented.

All correspondence and inquiries concerning this research study must include the IRB ID, the name of the Principal Investigator and the Study Title.

Sincerely,

Susan M. Ray, MD
Vice Chair
Emory University Institutional Review Board
This letter has been digitally signed

Emory University
1256 Briarcliff Road, NE Room 307N - Atlanta, Georgia 30306
Tel: 404.712.0720 - Fax: 404.727.1358 - Email: irb@emory.edu - Web: http://www.emory.edu/irb
An equal opportunity, affirmative action university

Amendment 1 Approval

EMORY UNIVERSITY

Institutional Review Board

FROM: Colleen DiIorio, PhD
Chair
Emory University IRB

TO: Daurice Grossniklaus
Principal Investigator

CC: Townsend Jill Nursing - Main
Dunbar Sandra Nursing - Main

DATE: August 5, 2007

RE: **Notification of Amendment Approval**
AM1_IRB00001568
Amendment 1 for IRB Study #IRB00001568
Energy Density of Food Intake in Overweight Adults

This is your notification that your above referenced amendment was reviewed and APPROVED by the IRB on **8/2/2007**.

Changes to Consent Form(s): main ICF version dated 7/30/2007, HIPAA and revocation letter versions dated 7/13/2007.

Changes to Protocol Document(s): Protocol version dated 7/31/2007.

Changes to Advertisements: Telephone screening form dated 7/31/2007;
Recruitment flyer (undated) and tearoffs entitled: Dietary Patterns and Heart Health Study.

Changes to study sites: Added Emory Hospital (non-GCRC),

The Emory Clinic, Emory Satellite, Crawford W. Long, GCRC-Emory, and Emory University.

Changes to study enrollment: Increased total target to 91 subjects.

Changes to funding or funding sources: Added National Institute of Nursing Research.

Other changes: Added to Study Population: Students-college, Patients,
Employees, and Women. Added Specimen Collection Card to Research Instruments.

All correspondence and inquiries concerning this research study must include the IRB ID, the name of the Principal Investigator and the Study Title.

Sincerely,

Colleen DiIorio, PhD
Chair
Emory University Institutional Review Board
This letter has been digitally signed

Emory University
1256 Briarcliff Road, NE Room 307N - Atlanta, Georgia 30306
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Amendment 2 Approval

EMORY UNIVERSITY | Institutional Review Board

FROM:	Clifford J. Gunthel, MD Vice Chair Emory University IRB
TO:	Daurice Grossniklaus, MEd Principal Investigator
CC:	Townsend Jill Nursing - Main
	Dunbar Sandra Nursing - Main
DATE:	August 27, 2007
RE:	**Notification of Amendment Approval**
	AM2_IRB00001568
	Amendment 2 for IRB Study #IRB00001568 Energy Density of Food Intake in Overweight Adults

This is your notification that your above referenced amendment was reviewed and APPROVED by the IRB on **08/25/2007**.

Changes to Consent Form(s): ICF revision dated 8-12-07

Changes to Protocol Document(s): Protocol revision dated 8-15-07

Changes to Advertisements: Telephone script revision 8-15-2007

Changes to Study Team members: Added Margaret Pedersen

All correspondence and inquiries concerning this research study must include the IRB ID, the name of the Principal Investigator and the Study Title.

Sincerely,

Clifford J. Gunthel, MD
Vice Chair
Emory University Institutional Review Board
This letter has been digitally signed

Emory University
1256 Briarcliff Road, NE Room 307N - Atlanta, Georgia 30306
Tel: 404.712.0720 - Fax: 404.727.1358 - Email: irb@emory.edu - Web: http://www.emory.edu/irb
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Amendment 3 Approval

EMORY UNIVERSITY | Institutional Review Board

FROM: Colleen DiIorio, PhD
Chair
Emory University IRB

TO: Daurice Grossniklaus
Principal Investigator

CC: Townsend Jill Nursing - Main
Dunbar Sandra Nursing - Main

DATE: January 3, 2008

RE: **Notification of Amendment Approval**
AM3_IRB00001568
Amendment 3 for IRB Study #IRB00001568
Energy Density of Food Intake in Overweight Adults

This is your notification that your above referenced amendment was reviewed and APPROVED by the IRB on **01/02/2008**.

Changes to Consent Form(s)
Changes to Protocol Document(s)
Changes to Study Team members
Changes to funding or funding sources
Other changes

All correspondence and inquiries concerning this research study must include the IRB ID, the name of the Principal Investigator and the Study Title.

Sincerely,

Colleen DiIorio, PhD
Chair
Emory University Institutional Review Board
This letter has been digitally signed

Emory University
1599 Clifton Road, 5th Floor - Atlanta, Georgia 30322
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2008 Continuing Review Approval

EMORY UNIVERSITY | Institutional Review Board

FROM: Sarah Freeman, PhD
Designated Reviewer
Emory University IRB

TO: Daurice Grossniklaus
Principal Investigator

CC: Townsend Jill Nursing - Main
Dunbar Sandra Nursing - Main

DATE: February 4, 2008

RE: **Notification of Continuing Review Expedited Approval**
CR00000577

IRB00001568
Energy Density of Food Intake in Overweight Adults

This is your notification that your above referenced Continuing Review was reviewed and APPROVED under the Expedited review process per 45 CFR 46.110 and 21 CFR 56.110. The approval is valid from **1/25/2008** until **1/24/2009**. Thereafter, continued approval is contingent upon the submission of a continuing review request that must be reviewed and approved by the IRB prior to the expiration date of this study.

Any reportable events (serious adverse events, breaches of confidentiality, protocol deviation or protocol violations) or issues resulting from this study should be reported immediately to the IRB and to the sponsoring agency (if any). Any amendments (changes to any portion of this research study including but not limited to protocol or informed consent changes) must have IRB approval before being implemented.

All correspondence and inquires concerning this research study must include the IRB ID, the name of

the Principal Investigator and the Study Title.

Sincerely,

Sarah Freeman, PhD
Designated Reviewer
Emory University Institutional Review Board
This letter has been digitally signed

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2009 Continuing Review Approval

EMORY UNIVERSITY | Institutional Review Board

FROM: Aryeh Stein, PhD
Co-Chair
Emory University IRB

TO: Daurice Grossniklaus
Principal Investigator

CC: Townsend Jill Nursing - Main

Dunbar Sandra Nursing - Main

DATE: January 15, 2009

RE: **Notification of Continuing Review Expedited Approval**

CR2_IRB00001568

IRB00001568

Energy Density of Food Intake in Overweight Adults

This is your notification that your above referenced Continuing Review was reviewed and APPROVED under the Expedited review process per 45 CFR 46.110 and 21 CFR 56.110. The approval is valid from **2/12/2009 until 2/11/2010**. Thereafter, continued approval is contingent upon the submission of a continuing review request that must be reviewed and approved by the IRB prior to the expiration date of this study.

Any reportable events (serious adverse events, breaches of confidentiality, protocol deviation or

protocol violations) or issues resulting from this study should be reported immediately to the IRB and to the sponsoring agency (if any). Any amendments (changes to any portion of this research study including but not limited to protocol or informed consent changes) must have IRB approval before being implemented.

All correspondence and inquires concerning this research study must include the IRB ID, the name of the Principal Investigator and the Study Title.

Sincerely,

Aryeh Stein, PhD
Co-Chair
Emory University Institutional Review Board
This letter has been digitally signed

Emory University

1599 Clifton Road, 5th Floor - Atlanta, Georgia 30322

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APPENDIX C

Telephone Screening and Verbal Permission Form

Principal Investigator: Daurice A. Grossniklaus, RN, MS, MEd

Other Investigators: Dr. Sandra B. Dunbar, RN, DSN, FAAN

Sponsor's Name: Southern Nurses Research Society; National Institute of Nursing Research, NIH (pending)

Telephone Script

PI Initiated Contact:

Hello, I am Daurice Grossniklaus, a doctoral student, calling from the Emory University School of Nursing. I am calling you today to see if you would like to take part in a study. I would like to tell you more about the study and make sure you are able to take part. [Go to section titled For All Potential Participants].

Participant Initiated Contact:

You may be calling because you are interested in learning more about the dietary patterns and heart health study. I would like to tell you more about the study and make sure you are able to take part. [Go to section titled For All Potential Participants].

For All Potential Participants:

You are being asked to take part because you are between the ages of 18-65 years old and may have some factors that make you at risk for heart disease and diabetes. People who are overweight are at a greater chance of heart problems such as heart attack and stroke. The purpose of this study is to look at how stress, mood, attitudes toward eating and dietary patterns are related to heart health and weight around the abdomen (waist

circumference). About 91 adults in the metropolitan Atlanta area will be asked to participate.

To take part, the study nurse will meet with you either at your home or at the SON for about an hour. During this time, you will tell you about the study and answer any questions you might have. You will then be asked to sign the informed consent. You will be asked to complete questionnaires about your stress, mood and attitudes toward eating. We will measure your height, weight, waist and hip circumferences. We will teach you how to weigh and measure your food which you will do for three days. We will lend you a food scale to weigh your food. We will also ask you to take a sample of your saliva from your mouth twice during the study-you will do this by lightly chewing on a cotton swab. We will use the saliva samples to measure a stress hormone called cortisol. Once you have completed the three days of recording your food, you will meet with the study nurse again for about 30 minutes. During this time, the study nurse will review your food record, collect the food scale and saliva samples. Your food record will be analyzed and you will receive written information about how your dietary pattern compares with the recommended pattern for someone of your age and sex.

Before I tell you more about the study, please let me know if you are pregnant, breastfeeding or have recently had a baby (within the past 3 months) because you will not be able to take part (If yes, thank you and end phone call).

Does the study sound like something you would like to take part in?

_____Yes

_____No. Thank you for your time.

Now, I would like to ask you a few questions so that I can be sure that you are eligible to take part in the study.

This should only take a few minutes and what you tell me will be kept confidential. We will not share your personal information without your permission to do so. Do you have about 10 minutes to answer some questions? (If no, schedule a time to call back).

Call back date/time and phone number: _____

1. Are you enrolled in any other studies that might make it difficult for you to take part in this study?
   a. Yes (Thank you and end call)
   b. No (Continue)
2. Do you have any problems filling out questionnaires?
   a. Yes (Thank you and end call)
   b. No (Continue)
3. Do you take part in physical activity for 30 minutes or more, three or more days per week?
   a. Yes (Thank you and end call)
   b. No (Continue)
4. Are you between the ages of 18 and 65 years old?
   a. No (Thank you and end call)
   b. Yes (Continue)
5. How tall are you? _____
6. Are you between ___ pounds (BMI of 25) and less than ___ pounds (BMI of 40)? [Use self reported height from #6, refer to the Body Mass Index table]

You can not be included if you have any of the following conditions or take certain medications. If you have any of these conditions, please tell me that you will not be able to take part. If you have been told by your doctor that you have diabetes or high blood sugar, you have had bariatric surgery or liposuction in the past or other abdominal surgery in the past year. You can not be included if you take any of the following medications. If you take any of these medications, please tell me that you do not be able to take part. These medications include insulin or oral medications for your blood sugar, medications for the treatment of depression, bipolar disorder, mental disorders, or

steroids used for the treatment of allergies, arthritis (inflammation/joint pain), immunosuppression (such as organ transplant), cancer chemotherapy (such as lymphoma), or weight loss medications such as sibutramine or orlistat.

a. I will not be able to take part (Thank you and end call)
b. I will be able to take part (Continue)

Thank you for answering our questions.

You are eligible to take part in the study. I will send you a packet that contains the consent form and directions to the School of Nursing for your visit. I will call you again to review the information and answer any questions that you might have. We will review the consent form together at your appointment and I will answer any questions you have regarding the consent form then.

Record name: Record phone number:

Date and time of phone call:

Verbal permission to send packet

I have explained the study and have verbal permission from the participant to send the packet of information and to schedule the participant's visit. He/she is aware that if he/she decides to take part, he/she will sign a written consent form at the first appointment and will be given a copy of the consent at that time.

Person obtaining verbal permission: Date: Time:

What day and time would be convenient to schedule your first appointment?

Date: Time: Location:

I will call you the day before to confirm your appointment.

Anthropometric Measurements Form

Participant ID# ☐☐☐

Date (mm/dd/yy): ☐☐ / ☐☐ / ☐☐

Record the following information for each participant:

1. Height: ☐☐☐.☐ centimeters
2. Weight: ☐☐☐.☐ kilograms
3. Waist circumference: First measurement: ☐☐☐.☐ centimeters
4. Waist circumference: Second measurement: ☐☐☐.☐ centimeters
5. Waist circumference: Third measurement: ☐☐☐.☐ centimeters
6. Average waist circumference: ☐☐☐.☐ centimeters
7. Hip circumference: First measurement: ☐☐☐.☐ centimeters
8. Hip circumference: Second measurement: ☐☐☐.☐ centimeters
9. Hip circumference: Third measurement: ☐☐☐.☐ centimeters
10. Average hip circumference: ☐☐☐.☐ centimeters
11. Use the following formula to calculate BMI:
    a. BMI = [(Weight in kilograms) / (Height in cm) x (Height in cm)]
12. BMI ☐☐.☐ kg/m^2
13. Comments:

Procedures for Anthropometric Measurements

Calibration Procedure

Weight (Health-o-Meter 320KL Digital scale)

The calibration procedure should be completed prior to each participant appointment. A rough calibration should be performed daily:

1. Step on scale and weight yourself, noting weight.
2. Add a calibrated weight and check to make sure the displayed weight increases accordingly.
3. Record this information on the Quality Assurance Checklist.

If the scale is not accurate, contact the service representative for recalibration.

Height (SECA 214 Portable stadiometer)

To calibrate stadiometer follow this procedure:

1. Place the calibration rod on the floor of the stadiometer.
2. Place the horizontal bar of the stadiometer firmly against the top of the calibration stick. The display should read 91.4 cm.
3. Note that the stadiometer was recalibrated to the 91.4 cm on the Quality Assurance Checklist.

Measuring and Recording Guidelines

Weight

Weight will be measured in kilograms and recorded to the nearest 0.1 kg.

Height

1. All measurements should be taken in centimeters and recorded to the nearest tenth of a centimeter.

Waist circumference

1. All measurements should be taken in centimeters and recorded to the nearest tenth of a centimeter.

Measurement Procedure

Weight

1. Participants should wear lightweight clothing without shoes.
2. Adults will stand still in the center of the scale platform facing the recorder, hands at side, and looking straight ahead.

Height

1. Participants will be asked to more or remove hair ornaments, jewelry, buns, braids, and corn rolls from the top of the head in order to measure stature properly.
2. Participant should not wear shoes.
3. The participant will stand on the center of the platform with heels of both feet together and toes pointed slightly outward at approximately a 60 degree angle.
4. Make sure the body weight is evenly distributed and both feet are flat on the floor. Check the position of the heels, buttocks, shoulder blades, and the back of the head for contact with the vertical backboard.
5. Depending on the overall body shape of the individual, all points may not touch. In such a case, make sure the participant's trunk is vertical above the waist and the arms and shoulders are relaxed.
6. The head should be aligned in the Frankfort horizontal plane. The head is aligned when the horizontal line from the ear canal to the lower border of the

orbit of the eye is parallel to the floor and perpendicular to the vertical backboard.

7. Many people will assume this position naturally, but for some it may be necessary to make a minor adjustment. If required, gently tilt the head up or down until proper alignment is achieved with eyes looking straight ahead.
8. Once correctly positioned, lower the headboard and instruct the participant to take a deep breath and stand as tall as possible. A deep breath will allow the spine to straighten, yielding a more consistent and reproducible stature measurement.
9. Position the headboard firmly on top of the head with sufficient pressure to compress the hair.
10. Record the measurement.
11. Then have the participant relax and step away from the stadiometer.
12. Some participants may have conditions that interfere with specific procedures for measuring stature. In these cases, it is important to get the best measurement possible according to the protocol. Then record, “not straight” in the comment section of the Anthropometric Measurement form.

Waist circumference

1. Participant should wear light weight clothing. Lower the pants or underclothing slightly.
2. To locate the bony landmark, while the participant stands, palpate the upper hip area to locate the right ileum.

3. Just above the uppermost lateral border of the right ileum, a horizontal mark is drawn and then crossed with a vertical mark on the midaxillary line.
4. An inelastic measuring tape is then placed around the trunk, at the level of the mark on the right side, making sure that it is on the level horizontal plane on all sides.
5. The tape is then tightened slightly without compressing the skin and underlying subcutaneous tissues.
6. Make the measurement at the end of the normal expiration.

Ongoing Quality Assurance Procedures for Maintaining Reliable Measurements

Interrater reliability

To ensure consistent measurements, the PI and General Clinical Research Center (GCRC) nutritionist will measure the weight, height and waist circumference of two overweight individuals. The interrater correlation coefficient will be calculated for each measure. If the level of agreement is less than .90, additional practice supervised by the nutritionist will be completed. After this additional practice, the PI and nutritionist will measure weight, height, and waist circumference on two overweight individuals. The interrater correlation coefficient will be calculated. This information will be documented in the procedure manual.

Intrarater reliability

To ensure consistent measurements, the PI under the supervision of the General Clinical Research Center (GCRC) nutritionist will measure the weight, height and waist circumference twice on two overweight individuals. The intrarater correlation coefficient will be calculated for each measure. If the level of agreement is less than

.90, additional practice supervised by the nutritionist will be completed. After this additional practice, the PI and nutritionist will measure weight, height, and waist circumference on twice on two overweight individuals. The interrater correlation coefficient will be calculated. This information will be documented in the procedure manual.

Demographic and Clinical Data Form

Participant ID # ☐☐☐

Date (mm/dd/yy): ☐☐ / ☐☐ / ☐☐

Please check the answer that best describes you:

1. What is your date of birth? ☐☐ / ☐☐ / ☐☐☐☐
   month / day / year

2. What is your age? ☐☐ Years

3. What is your gender? ☐ Male ☐ Female

4. What is your ethnicity?
   ☐ Hispanic / Latino
   ☐ Not Hispanic / Latino

5. What is your race?
   ☐ American Indian / Alaska Native
   ☐ Asian
   ☐ Native Hawaiian or other Pacific Islander
   ☐ Black or African American
   ☐ White

6. What is your marital status?
   ☐ Married
   ☐ Domestic partner
   ☐ Single
   ☐ Divorced / separated
   ☐ Widowed

7. What category best describes the amount of education you have?
   ☐ 6th grade or less
   ☐ 9th grade or less
   ☐ Partial high school
   ☐ High school graduate or GED
   ☐ Technical / vocational school
   ☐ Completed some college
   ☐ College graduate
   ☐ Post graduate or professional school

8. What is your current employment status?
   ☐ Full-time
   ☐ Part-time
   ☐ Retired
   ☐ Medical leave / disability
   ☐ Not currently employed

9. Which category best describes the type of work you do?
   - ☐ Professional / executive
   - ☐ Business management / administration
   - ☐ Clerical / sales
   - ☐ Skilled labor
   - ☐ Semi-skilled labor
   - ☐ Unskilled labor
   - ☐ Not applicable

10. Please write your job title in the space provided: (Be specific: for example; registered nurse, cashier, machine operator, etc).

11. How many people are currently living in your household, including yourself?
    - ☐☐ Number of people

12. Of these people, how many are children (18 years or less)?
    - ☐☐ Number of people

13. Of these people, how many are adults (more than 18 years old)?
    - ☐☐ Number of people

14. Of these people, how many bring income into the household?
    - ☐☐ Number of people

15. Which of the categories best describes your total combined family income for the past 12 months? This should include all income (before taxes) from all sources, wages, rent from properties, social security, disability, and/or veterans benefits, workman's compensation, help from relatives (including child payments and alimony) and so on.
    - ☐ Less than $19,999
    - ☐ $20,000 through $39,999
    - ☐ $40,000 through $59,999
    - ☐ $60,000 through $79,999
    - ☐ $80,000 and greater
    - ☐ Don't know
    - ☐ Prefer not to answer

16. Where do you usually shop for food items?
    - ☐ Convenience stores such as QT
    - ☐ Stores that sell in bulk such as Sam's Club, Costco, or BJ's
    - ☐ Supermarket or grocery stores such as Walmart, Publix, Kroger, or Ingles
    - ☐ Farmer's markets such as the DeKalb Farmer's Market
    - ☐ Target or Dollarstores

17. How close is the nearest store where you can buy fruits and vegetables?
    - ☐ Less than 5 miles
    - ☐ 5-9 miles
    - ☐ 10-14 miles
    - ☐ 15-19 miles
    - ☐ 20 miles or more

18. Do you own a car or have access to a car?
    - ☐ Yes
    - ☐ No

19. Do you have any of the following health conditions?

    a. High blood pressure ☐ Yes ☐ No

    b. High levels of fat in the blood (lipid or cholesterol) ☐ Yes ☐ No

    c. Chest pain ☐ Yes ☐ No

    d. Heart failure ☐ Yes ☐ No

    e. Arthritis ☐ Yes ☐ No

    f. Any conditions that limit your ability to participate in physical activity? ☐ Yes ☐ No

    g. Stroke ☐ Yes ☐ No

    h. Sleep apnea ☐ Yes ☐ No

For women participants only:

20. Do you have regular periods? ☐ Yes ☐ No

21. Have you been through the change of life? ☐ Yes ☐ No

22. During a typical day, how many minutes do you take part in light physical activity (easy or no effort at all; such as laundry, bathing, stretching/flexibility exercises)? ☐☐☐ Number of minutes

23. During a typical week, how many days do you take part in light physical activity? ☐ Number of days

24. During a typical day, how many minutes do you take part in moderate physical activity (harder than light, some increase in breathing or heart rate; such as mowing, walking, dancing, low impact aerobics)? ☐☐☐ Number of minutes

25. During a typical week, how many days do you take part in moderate physical activity? ☐ Number of days

26. During a typical day, how many minutes do you take part in intense physical activity (all out effort, large increase in breathing or heart rates; such as tennis, soccer, step aerobics, running/jogging)? ☐☐☐ Number of minutes

27. During a typical week, how many days do you take part in intense physical activity? ☐ Number of days

Health History Form

Do you have any of the following health conditions?

1. Asthma ☐ Yes ☐ No Year Diagnosed: ☐☐☐☐

2. Anemia or low blood iron ☐ Yes ☐ No Year Diagnosed: ☐☐☐☐

3. Arthritis ☐ Yes ☐ No Year Diagnosed: ☐☐☐☐

4. Cancer ☐ Yes ☐ No Year Diagnosed: ☐☐☐☐

   a. Type of cancer ☐☐☐☐☐☐☐☐☐☐

5. Frequent headaches or migraines ☐ Yes ☐ No Year Diagnosed: ☐☐☐☐

6. Gastric reflux ☐ Yes ☐ No Year Diagnosed: ☐☐☐☐

7. Glaucoma ☐ Yes ☐ No Year Diagnosed: ☐☐☐☐

8. Gout ☐ Yes ☐ No Year Diagnosed: ☐☐☐☐

9. High blood pressure ☐ Yes ☐ No Year Diagnosed: ☐☐☐☐

10. High cholesterol ☐ Yes ☐ No Year Diagnosed: ☐☐☐☐

11. Irritable bowel syndrome ☐ Yes ☐ No Year Diagnosed: ☐☐☐☐

12. Pneumonia ☐ Yes ☐ No Year Diagnosed: ☐☐☐☐

13. Seizures or blackouts ☐ Yes ☐ No Year Diagnosed: ☐☐☐☐

14. Sickle cell anemia ☐ Yes ☐ No Year Diagnosed: ☐☐☐☐

15. Sinus problems ☐ Yes ☐ No Year Diagnosed: ☐☐☐☐

16. Sleep apnea ☐ Yes ☐ No Year Diagnosed: ☐☐☐☐

17. Tuberculosis ☐ Yes ☐ No Year Diagnosed: ☐☐☐☐

18. Ulcer disease ☐ Yes ☐ No Year Diagnosed: ☐☐☐☐

Medications

1. Are you currently taking any medications? ☐ Yes ☐ No

2. Medication name: ☐☐☐☐☐☐☐☐☐☐☐☐☐☐

Dose: How often:

3. Medication name: ☐☐☐☐☐☐☐☐☐☐☐☐☐☐

Dose: How often:

4. Medication name: ☐☐☐☐☐☐☐☐☐☐☐☐☐☐

Dose: How often:

5. Medication name: ☐☐☐☐☐☐☐☐☐☐☐☐☐☐

Dose: How often:

Vitamin or Other Supplements

1. Do you take vitamins or other supplements? ☐ Yes ☐ No

2. Record the amount, time, or frequency of vitamin or other supplement use.

Date	Vitamin, Mineral, or Herbs	Amount	Time or Frequency (How many times a day)

Three Day Food Record

Date of Record: _____

Day of Week: _____

Participant ID ☐☐☐

DAY 1

Remember to write down everything you eat and drink.

Time of meal	Food item. Be specific Write one item per line	Amount specify number of grams	Cooking Method	Type of fat used when cooking	Amount of fat added during cooking	Butter, margarine or oil added at table	Brand/ Restaurant

Date of Record: _____

Day of Week: _____

Participant ID ☐☐☐

DAY 2

Remember to write down everything you eat and drink.

Time of meal	Food item. Be specific Write one item per line	Amount specify number of grams	Cooking Method	Type of fat used when cooking	Amount of fat added during cooking	Butter, margarine or oil added at table	Brand/ Restaurant

Date of Record: _____

Day of Week: _____

Participant ID ☐☐☐

DAY 3

Remember to write down everything you eat and drink.

Time of meal	Food item. Be specific Write one item per line	Amount specify number of grams	Cooking Method	Type of fat used when cooking	Amount of fat added during cooking	Butter, margarine or oil added at table	Brand/ Restaurant

How to Keep a Three-Day Food Record

We need you to keep a food record. A food record is a list of everything you eat, drink or taste. Here are some tips on keeping a food record.

- Weigh all the food you eat for three days in a row. Write down the weight of the food in ounces.
- Write down all of the beverages you DRINK for three days.
- Make sure that you write down:
    - Breakfast, lunch, dinner or other meals
    - Snacks
    - Nibbling
    - Juices, water or drinks, including water taken with pills
- Don’ t forget to write down:
    - Seasonings, spices, mustards, ketchup, mayonnaise, dressing or other condiments added to foods.
- Be sure you know much of each ingredient you use in mixed dishes or on sandwiches.
- So that you don’t forget anything, write down what you eat or drink as soon as you eat or drink it.
- Remember to note whether weights are for cooked or raw portions.
- To help you with portion sizes of foods, use the pictures on the following pages.

Try to accurately estimate the amount of food you ate or beverage you drank!

Recommended dates for recording:

Three Factor Eating Questionnaire Revised (TFEQR)

Participant ID ☐☐☐

Date (mm/dd/yy) ☐☐ / ☐☐ / ☐☐

Please check the answer that best describes your eating pattern:

1. I deliberately take small helpings as a means of controlling my weight.
   - ☐ Definitely True
   - ☐ Mostly True
   - ☐ Mostly False
   - ☐ Definitely False
2. I consciously hold back at meals in order not to gain weight.
   - ☐ Definitely True
   - ☐ Mostly True
   - ☐ Mostly False
   - ☐ Definitely False
3. I do not eat some foods because they make me fat.
   - ☐ Definitely True
   - ☐ Mostly True
   - ☐ Mostly False
   - ☐ Definitely False

4. I frequently avoid “stocking up” on tempting foods?
   - ☐ Almost Never
   - ☐ Seldom
   - ☐ Usually
   - ☐ Almost Always
5. Are you likely to consciously eat less than you want?
   - ☐ Unlikely
   - ☐ Slightly likely
   - ☐ Moderately likely
   - ☐ Very likely
6. When I smell a sizzling steak or a juicy piece of meat, I find it very difficult to keep from eating, even if I have just finished a meal.
   - ☐ Definitely True
   - ☐ Mostly True
   - ☐ Mostly False
   - ☐ Definitely False
7. Sometimes when I start eating, I just can’t seem to stop.
   - ☐ Definitely True
   - ☐ Mostly True
   - ☐ Mostly False
   - ☐ Definitely False
8. Being with someone who is eating often makes me hungry enough to eat also.
   - ☐ Definitely True
   - ☐ Mostly True
   - ☐ Mostly False
   - ☐ Definitely False

9. When I see a real delicacy, I often get so hungry that I have to eat right away.
   - ☐ Definitely True
   - ☐ Mostly True
   - ☐ Mostly False
   - ☐ Definitely False

10. I get so hungry that my stomach often seems like a bottomless pit.
    - ☐ Definitely True
    - ☐ Mostly True
    - ☐ Mostly False
    - ☐ Definitely False

11. I am always hungry so it is hard for me to stop eating before I finish the food on my plate.
    - ☐ Definitely True
    - ☐ Mostly True
    - ☐ Mostly False
    - ☐ Definitely False

12. I am always hungry enough to eat at any time.
    - ☐ Definitely True
    - ☐ Mostly True
    - ☐ Mostly False
    - ☐ Definitely False

13. I go on eating binges though I am not hungry.
    - ☐ Never
    - ☐ Rarely
    - ☐ Sometimes
    - ☐ At least once a week

14. When I feel anxious, I find myself eating.
- ☐ Definitely True
- ☐ Mostly True
- ☐ Mostly False
- ☐ Definitely False

15. When I feel blue, I often overeat.
- ☐ Definitely True
- ☐ Mostly True
- ☐ Mostly False
- ☐ Definitely False

16. When I feel lonely, I console myself by eating.
- ☐ Definitely True
- ☐ Mostly True
- ☐ Mostly False
- ☐ Definitely False

17. I often feel hungry.
- ☐ Definitely True
- ☐ Mostly True
- ☐ Mostly False
- ☐ Definitely False

18. I usually eat whatever I want, whenever I want it.
- ☐ Definitely True
- ☐ Mostly True
- ☐ Mostly False
- ☐ Definitely False

Beck Depression Inventory-II

Participant ID ☐☐☐

Date (mm/dd/yy) ☐☐ / ☐☐ / ☐☐

Instructions: This questionnaire consists of 21 groups of statements. Please read each group of statements carefully, and then pick out the one statement in each group that best describes the way you have been feeling during the past 2 weeks, including today. Mark the box beside the statement you have picked. If several statements in the group seem to apply equally well, mark the highest number for that group Be sure that you do not choose more than one statement for any group, including item 16 (Changes in sleep patterns) or item 18 (Changes in appetite).

1. Sadness

☐ 0 I do not feel sad.

☐ 1 I feel sad much of the time.

☐ 2 I am sad all of the time.

☐ 3 I am so sad or unhappy that I can't stand it.

2. Pessimism

☐ 0 I am not discouraged about my future.

☐ 1 I feel more discouraged about my future that I used to be.

☐ 2 I do not expect things to work out for me.

☐ 3 I feel my future is hopeless and will only get worse.

3. Past Failure

- ☐ 0 I do not feel like a failure.
- ☐ 1 I have failed more than I should have.
- ☐ 2 As I look back, I see a lot of failures.
- ☐ 3 I feel like I am a total failure as a person.

4. Loss of Pleasure

- ☐ 0 I get as much pleasure as I ever did from the things I enjoy.
- ☐ 1 I don't enjoy things as much as I used to.
- ☐ 2 I get very little pleasure from the things I used to enjoy.
- ☐ 3 I can't get any pleasure from the things I used to enjoy.

5. Guilty Feelings

- ☐ 0 I don't feel particularly guilty.
- ☐ 1 I feel guilty over many things I have or should have done.
- ☐ 2 I feel quite guilty most of the time.
- ☐ 3 I feel guilty all of the time.

6. Punishment Feelings

- ☐ 0 I don't feel I am being punished.
- ☐ 1 I feel I may be punished.
- ☐ 2 I expect to be punished.
- ☐ 3 I feel I am being punished.

7. Self-Dislike

- ☐ 0 I feel the same about myself as ever.
- ☐ 1 I have lost confidence in myself.
- ☐ 2 I am disappointed in myself.
- ☐ 3 I dislike myself.

8. Self-Criticalness

- ☐ 0 I don’t criticize or blame myself more than usual.
- ☐ 1 I am more critical of myself than I used to be.
- ☐ 2 I criticize myself for all of my faults.
- ☐ 3 I blame myself for everything bad that happens.

9. Suicidal Thoughts or Wishes

- ☐ 0 I don’t have any thoughts of killing myself.
- ☐ 1 I have thoughts of killing myself but I would not carry them out.
- ☐ 2 I would like to kill myself.
- ☐ 3 I would kill myself if I had the chance.

10. Crying

- ☐ 0 I don’t cry anymore than I used to.
- ☐ 1 I cry more than I used to.
- ☐ 2 I cry over every little thing.
- ☐ 3 I feel like crying, but I can’t.

11. Agitation

☐ 0 I am no more restless or wound up than usual.

☐ 1 I feel more restless or wound up than usual.

☐ 2 I am so restless or agitated that it's hard to stay still.

☐ 3 I am so restless or agitated that I have to keep moving or doing something.

12. Loss of Interest

☐ 0 I have not lost interest in other people or activities.

☐ 1 I am less interested in other people or things than before.

☐ 2 I have lost most of my interest in other people or things.

☐ 3 It's hard to get interested in anything.

13. Indecisiveness

☐ 0 I make decisions about as well as ever.

☐ 1 I find it more difficult to make decisions than usual.

☐ 2 I have much greater difficulty in making decisions than I used to.

☐ 3 I have trouble making any decisions.

14. Worthlessness

☐ 0 I do not feel that I am worthless.

☐ 1 I don't consider myself as worthwhile and useful as I used to.

☐ 2 I feel more worthless as compared to other people.

☐ 3 I feel utterly worthless.

15. Loss of Energy

- ☐ 0 I have as much energy as ever.
- ☐ 1 I have less energy that I used to have.
- ☐ 2 I don't have enough energy to do very much.
- ☐ 3 I don't have enough energy to do anything.

16. Changes in Sleeping Pattern

- ☐ 0 I have not experienced any change in my sleeping pattern.
- ☐ 1a I sleep somewhat more than usual.
- ☐ 1b I sleep somewhat less than usual.
- ☐ 2a I sleep a lot more than usual.
- ☐ 2b I sleep a lot less than usual.
- ☐ 3a I sleep most of the day.
- ☐ 3b I wake up 1-2 hours early and can't get back to sleep.

17. Irritability

- ☐ 0 I am no more irritable than usual.
- ☐ 1 I am more irritable than usual.
- ☐ 2 I am much more irritable than usual.
- ☐ 3 I am irritable all the time.

18. Changes in Appetite

- ☐ 0 I have not experienced any change in my appetite.
- ☐ 1a My appetite is somewhat less than usual.
- ☐ 1b My appetite is somewhat greater than usual.
- ☐ 2a My appetite is much less than usual.
- ☐ 2b My appetite is much greater than usual.
- ☐ 3a I have no appetite at all.
- ☐ 3b I crave food all the time.

19. Concentration Difficulty

- ☐ 0 I can concentrate as well as ever.
- ☐ 1 I can’t concentrate as well as usual.
- ☐ 2 It’s hard to keep my mind on anything for very long.
- ☐ 3 I find I can’t concentrate on anything.

20. Tiredness or Fatigue

- ☐ 0 I am no more tired or fatigued than usual.
- ☐ 1 I get more tired or fatigued more easily than usual.
- ☐ 2 I am too tired or fatigued to do a lot of the things I used to do.
- ☐ 3 I am too tired or fatigued to do most of the things I used to do.

21. Loss of Interest in Sex

- ☐ 0 I have not noticed any recent change in my interest in sex.
- ☐ 1 I am less interested in sex than I used to be.
- ☐ 2 I am much less interested in sex now.
- ☐ 3 I have lost interest in sex completely.

Perceived Stress Scale

Participant ID ☐☐☐

Date (mm/dd/yy) ☐☐ / ☐☐ / ☐☐

Instructions: The questions in this scale ask you about your feelings and thoughts during the last month. In each case, you will be asked to indicate by checking how often you felt or thought a certain way.

	Never	Almost Never	Some-times	Fairly Often	Very Often
1. In the last month, how often have you been upset because of something that happened unexpectedly?					
2. In the last month, how often have you felt that you were unable to control the important things in your life?					
3. In the last month, how often have you felt nervous and “stressed”?					
4. In the last month, how often have you felt confident about your ability to handle your personal problems?					
5. In the last month, how often have you felt that things were going your way?					
6. In the last month, how often have you found that you could not cope with all the things that you had to do?					
7. In the last month, how often have you been able to control irritations in your life?					

8. In the last month, how often have you felt that you were on top of things?

9. In the last month, how often have you been angered because of things that were outside of your control?

10. In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?

Saliva Collection Cards

Morning Sample

Participant ID ☐☐☐

Date (mm/dd/yyyy) ☐☐ / ☐☐ / ☐☐☐☐

Do not eat, drink, chew gum, brush your teeth, or smoke for a half hour before swabbing your mouth.

1. What day did you swab your mouth? ☐☐ / ☐☐ / ☐☐☐☐

2. What time did you swab your mouth? ☐☐ : ☐☐

3. What time did you wake up this morning? ☐☐ : ☐☐

4. How many hours did you sleep last night? ☐☐ : ☐☐ hours and minutes

5. Did an alarm clock wake you up? ☐ Yes ☐ No

6. Did you wake up on your own? ☐ Yes ☐ No

Any problems?

Evening Sample – 10:00 p.m.

Participant ID ☐☐☐

Date (mm/dd/yyyy) ☐☐ / ☐☐ / ☐☐☐☐

Do not eat, drink, chew gum, brush your teeth, or smoke for a half hour before swabbing your mouth. Remember to swab your mouth at 10:00 p.m. or at bedtime, if earlier.

1. What day did you swab your mouth?	☐☐ / ☐☐ / ☐☐☐☐
2. What time did you swab your mouth?	☐☐ : ☐☐
3. Did you take a nap today?	☐ Yes ☐ No
4. If so, how long did you sleep?	☐☐ : ☐☐ hours and minutes
5. Did you participate in physical activity today?	☐ Yes ☐ No
6. If so, how long did you participate in physical activity today?	☐☐ : ☐☐ hours and minutes
7. If so, how intense was this physical activity?	☐ Light (easy or no effort at all; laundry, bathing, stretching/flexibility exercises) ☐ Moderate (harder than light; some increase in breathing or heart rate; mowing, walking, dancing, low impact aerobics) ☐ Intense (all out effort, large increase in breathing or heart rate; tennis, soccer, step aerobics, running/jogging)

Any problems?

APPENDIX D

Data Table

Table 21

Differences in Macronutrient Intake by Adequate Versus Inadequate Caloric Intake

Macronutrient	Adequate Intake n Mean *(SD)*	Inadequate Intake n Mean *(SD)*	T value	Mann-Whitney U	z Score
Calories (kcal)					
All	36	51			
	2456.34 *(582.21)*	1728.67 *(368.74)*	-6.620**		
Men	8	15			
	2989.36 *(518.72)*	2023.90 *(308.66)*		8.00	-3.357**
Women	28	36			
	2304.05 *(511.28)*	1605.65 *(321.26)*		93.00	-5.562**
Total fat (g)					
All	36	51			
	97.77 *(36.74)*	65.75 *(21.58)*	-4.689**		
Men	8	15			
	121.91 *(32.28)*	78.12 *(23.85)*		12.00	-3.098**
Women	28	36			
	90.87 *(35.46)*	60.60 *(18.56)*		225.00	-3.776**
Saturated fat (g)					
All	36	51			
	30.27 *(11.97)*	20.98 *(8.87)*	-4.161**		
Men	8	15			
	36.91 *(10.93)*	23.22 *(8.65)*		19.00	-2.647*
Women	28	36			
	28.38 *(11.74)*	20.04 *(8.91)*		260.00	-3.302**

Macronutrient	Adequate Intake n Mean *(SD)*	Inadequate Intake n Mean *(SD)*	T value	Mann-Whitney U	z Score
Cholesterol (mg)					
All	36	51			
	282.78 *(145.18)*	248.97 *(141.41)*	-1.086		
	36				
Men	8	15			
	326.25 *(221.68)*	354.42 *(176.69)*		51.00	-0.581
Women	28	36			
	270.36 *(117.74)*	205.04 *(96.59)*		334.00	-2.301*
Total carbohydrates (g)					
All	36	51			
	295.69 *(83.61)*	213.35 *(69.28)*	-5.009**		
Men	8	15			
	342.52 *(108.24)*	236.15 *(53.75)*		19.00	-2.647**
Women	28	36			
	282.32 *(72.04)*	203.86 *(73.39)*		207.00	-4.019**
Dietary fiber (g)					
All	36	51			
	21.59 *(9.43)*	16.88 *(7.54)*	-2.587*		
Men	8	15			
	24.18 *(13.82)*	16.97 *(7.37)*		38.00	-1.420
Women	28	36			
	20.85 *(7.95)*	16.84 *(7.71)*		362.00	-1.922
Sugars (g)					
All	36	51			
	110.03 *(53.14)*	79.44 *(36.15)*	-3.881**		
Men	8	15			
	139.45 *(64.83)*	81.28 *(43.90)*		23.00	-2.388*
Women	28	36			
	113.19 *(49.11)*	78.67 *(33.07)*		291.00	-2.883**

Macronutrient	Adequate Intake n Mean *(SD)*	Inadequate Intake n Mean *(SD)*	T value	Mann-Whitney U	z Score
Protein (g)					
All	36	51			
	86.85 *(20.41)*	73.63 *(25.53)*	-2.578*		
Men	8	15			
	102.88 *(27.02)*	94.83 *(28.99)*		43.00	-1.097
Women	28	36			
	82.28 *(15.90)*	64.80 *(17.96)*		216.00	-3.898**

Note. *p≤.05. **p≤.01

APPENDIX E

Additional Analyses Related to Specific Aim 1

Two additional analyses related to specific aim 1 are presented. The first examines the relationship between psychological factors and beverage energy density. The second examines the relationship between psychological factors and food energy density.

Psychological Factors and Beverage Energy Density

There were no significant bivariate correlations between age, race, gender, BMI, TFEQR, PSS or BDI-II and beverage energy density. There was a significant bivariate correlation between reporting adequate caloric intake (RACI) and beverage energy density (r=-.241, p=.02) indicating that those reporting adequate caloric intake reported a higher beverage energy density than those reporting inadequate caloric intake.

A sequential linear regression model was used to determine the amount of total variance in beverage energy density explained by the addition of psychological factors while holding individual characteristics constant. Age, race, gender, reporting adequacy, BMI and TFEQR were entered in the model as a block to control for their influence on beverage energy density. PSS and BDI-II were then entered to determine their contribution to the model.

The model containing age, race, gender, RACI, BMI, TFEQR, PSS and BDI-II was significant (p=.04) in explaining beverage energy density. The adjusted R^2 of .104 indicates that 10.4% of the total variance in beverage energy density was explained by these variables. (Table 22).

Table 22

Regression Model for the Association between Psychological Factors and Beverage Energy Density (N=86)

Variable	Model 1			Model 2		
	B	SE *B*	ß	*B*	SE *B*	ß
Age	.000	.001	-.002	.000	.001	.02
Race	.05	.03	.18	.05	.03	.18
Gender	-.04	.03	-.13	-.05	.03	-.16
BMI	-.02	.03	-.07	-.04	.03	-.13
TFEQR	-.02	.03	-.08	-.04	.03	-.13
RACI	-.13	.06	-.25*	-.15	.06	-.28*
PSS				.000	.003	-.02
BDI-II				.03	.01	.30*
R^2		.116			.188	
R^2 Adjusted		.049			.104	
R^2 change		.116			.072	
F for change in R^2		1.729			2.233*	
F (*p-value* for total model)		1.729 (p=.13)			2.233 (p=.03)	

Note. BMI = Body Mass Index. TFEQR = Three Factor Eating Questionnaire Revised. RACI = Reporting Adequate Caloric Intake. PSS = Perceived Stress Scale. BDI-II = Beck Depression Inventory II. *B*= beta coefficient. *p≤.05.

A reduced model was tested that included the variables that were significant or trended toward significance. In the reduced model, gender, race, and reporting adequate caloric intake were added as a block to control for their influence on beverage energy density. BDI-II was then added to the model. The F change statistic and the model were significant with an adjusted R^2 of .105 indicating that approximately 10.5% of the total variance in beverage energy density is explained by these variables. Race (t=1.683, p=.10) and gender (t=-1.695, p=.09) trended toward significance. The standardized betas indicate that race and BDI-II scores (t=2.048, $p \leq .05$) were positively related while reporting adequate caloric intake (t=-2.992, $p \leq .01$) were inversely related to beverage energy density. Increased depressive symptoms explained an additional 4.4% of beverage energy density above that accounted for by African-American race, male gender and reporting adequate caloric intake. (Table 23). A post hoc power analysis demonstrated that the sample size of 87 achieved 52% power to detect an R^2 of .044 attributed to one independent variable using an F-test with an alpha of .05. The variables tested are adjusted for an additional three control variables with an R^2 of .103.

Table 23

Reduced Regression Model for the Association between Psychological Factors and Beverage Energy Density (N=87)

	Model 1			Model 2		
Variable	*B*	*SE B*	ß	*B*	*SE B*	ß
Gender	-.04	.03	-.15	-.05	.03	-.18
Race	.05	.03	.18	.05	.03	.18
RACI	-.15	.06	-.28*	-.17	.06	-.32**
BDI-II				.02	.01	.22*
R^2		.103			.146	
Adjusted R^2		.070			.105	
R^2 change		.103			.044	
F for change in R^2		3.163*			4.195*	
F (*p-value* for total model)		3.163 (*p* = .03)			3.512 (*p* = .01)	

Note. RACI = Reporting Adequate Caloric Intake. BDI-II = Beck Depression Inventory II. *B*= beta coefficient. *$p \leq .05$. ** $p \leq .01$.

Psychological factors and food energy density.

There were significant bivariate correlations between age (r=-.312, p≤.01), race (r=.253, p=.02) and reporting adequate caloric intake (r=-.237, p=.03) with food energy density. There were no significant bivariate correlations between gender, BMI, TFEQR, PSS or BDI-II with food energy density.

A sequential linear regression model was used to determine the amount of total variance in food energy density explained by the addition of psychological factors when holding individual characteristics constant. Age, race, gender, RACI, BMI and TFEQR were entered as a block to control for their influence on food energy density. PSS and BDI-II scores were then entered to determine their contribution to the model. The full model was significant with an adjusted R^2 of .228 indicating that these variables explained approximately 22.8% of the total variance in food energy density. Younger age, African American race and reporting adequate caloric intake contributed significantly to the model. Because the variables of interest, PSS and BDI-II scores, did not contribute significantly to explaining food energy density, a reduced model was not tested. (Table 24).

In summary, increased depressive symptoms explained an additional 4.4% of the variance in beverage energy density above that accounted for by race, gender and reporting adequate caloric intake. Together these variables explained 10.5% of the total variance in beverage energy density. Depressive symptoms and perceived stress did not explain additional variance in food energy density above that accounted for by younger age, African American race and reporting adequate caloric intake.

Table 24

Regression Model for the Association between Psychological Factors and Food Energy Density (N=86)

	Model 1			Model 2		
Variable	*B*	*SE B*	ß	*B*	*SE B*	ß
Age	-.01	.004	-.27**	-.01	.005	-.29**
Race	.25	.09	.29**	.24	.09	.28**
Gender	-.14	.10	-.14	-.15	.10	-.16
BMI	.07	.08	.08	.04	.09	.05
TFEQR	-.14	.09	-.16	-.15	.09	-.18
RACI	-.44	.18	-.26*	-.47	.18	-.27**
PSS				-.007	.008	-.12
BDI-II				.07	.04	.23
R^2		.271			.300	
Adjusted R^2		.216			.228	
R^2 change		.271			.029	
F for change in R^2		4.894**			1.620	
F (*p-value* for total model)		4.894 ($p < .01$)			4.133 ($p < .01$)	

Note. BMI = Body Mass Index. TFEQR = Three Factor Eating Questionnaire Revised. RACI = Reporting Adequate Caloric Intake. PSS = Perceived Stress Scale. BDI-II = Beck Depression Inventory II. *B*= beta coefficient. *p≤ .05. **p≤.01.

Additional Analyses Related to Specific Aim 2

Two additional analyses related to specific aim 2 are presented. The first examines the relationship between beverage energy density and waist circumference. The second examines the relationship between food energy density and waist circumference.

Beverage Energy Density and Waist Circumference

There was no significant bivariate correlation between beverage energy density with waist circumference. A sequential linear regression model was used to determine the amount of total variance in waist circumference explained by the addition of the beverage energy density value while holding individual characteristics and psychological factors constant. Age, gender, race, TFEQR, beverage energy density, PSS and BDI-II scores were entered in a block to control for their influence on waist circumference. The beverage energy density value was then entered to determine its contribution to the model. The F change statistic and the model were not significant indicating that these variables do not explain waist circumference variance.

Food energy density and waist circumference.

There was no significant bivariate correlation between food energy density with waist circumference. A sequential linear regression model was used to determine the amount of total variance in waist circumference explained by the addition of food energy density while holding individual characteristics and psychological factors constant. Age, gender, race, food weight, TFEQR, PSS and BDI-II scores were entered in a block to control for their influence on waist circumference. The food energy density

values were then entered to determine their contribution to the model. The F change statistic and model were significant. (Table 25).

Table 25

Regression Model for the Association between Food Energy Density and Waist Circumference (N=86)

	Model 1			Model 2		
Variable	*B*	SE *B*	ß	*B*	SE *B*	ß
Age	.01	.007	.17	.02	.008	.27*
Race	.06	.15	.05	.006	.14	.004
Gender	-.03	.16	-.02	.07	.16	.05
TFEQR	.10	.15	.07	.18	.15	.14
PSS	.01	.01	.14	.02	.01	.18
BDI-II	.11	.05	.23	.09	.06	.18
Food weight (grams)	.001	.000	.36**	.001	.000	.50**
Food energy density				.24	.08	.34**
R^2		.226			.300	
Adjusted R^2		.156			.227	
R^2 change		.226			.074	
F for change in R^2		3.249**			8.120**	
F(p-value for total model)		3.249 ($p < .01$)			4.117 ($p < .01$)	

Note. TFEQR = Three Factor Eating Questionnaire Revised. PSS = Perceived Stress Scale. BDI-II = Beck Depression Inventory II. *B*= beta coefficient. *$p \leq .05$. ** $p \leq .01$.

The full model was reduced to include those variables that were significant or trended toward significance (alpha of .20 or below) in explaining food energy density. In the reduced model, age, food weight (grams) and BDI-II scores were entered as a block. Food energy density values were then entered to determine the amount of total variance in waist circumference explained by the addition of the food energy density value while holding age, food weight, and BDI-II scores constant. The F change statistic and the reduced model were significant with an adjusted R^2 of .223 indicating that 22.3% of the total variance in waist circumference can be explained by these variables. Age trended toward significance (t=1.902, p=.06). The standardized betas indicate that food weight (t=4.509, $p \leq .01$), BDI-II scores (t=3.426, $p \leq .01$) and food energy density (t=2.445, $p \leq .05$) were positively related to waist circumference. Higher food energy density explained an additional 5.4% of waist circumference variance above that accounted for by older age, higher food weight and increased depressive symptoms. In this model, higher food energy density independently predicted increased waist circumference. (Table 26). A post hoc power analysis demonstrated that the sample size of 87 achieved 66% power to detect an R^2 of .054 attributed to 1 independent variable using an F-test with an alpha of .05. The variables tested are adjusted for an additional 3 control variables with an R^2 of .205.

In summary, beverage energy density did not explain additional variation in waist circumference while controlling for age, gender, race, dietary restraint, perceived stress and depressive symptoms. Higher food energy density explained an additional 5.4% of the variance in waist circumference above that accounted for by older age,

higher food weight and increased depressive symptoms. Together these variables explained 22.3% of the total variance in waist circumference.

Table 26

Reduced Regression Model for the Association between Food Energy Density and Waist Circumference (N=87)

	Model 1			Model 2		
Variable	*B*	SE *B*	ß	*B*	SE *B*	ß
Age	.007	.007	.11	.01	.007	.19
BDI-II	.16	.05	.33**	.16	.05	.33**
Food weight (grams)	.001	.000	.37**	.001	.000	.50**
Food energy density				.19	.08	.28*
R^2		.205			.259	
Adjusted R^2		.177			.223	
R^2 change		.205			.054	
F for change in R^2		7.152**			5.980*	
F(p-value for total model)		7.152 (p < .01)			7.181 (p < .01)	

Note. BDI-II = Beck Depression Inventory II. *B*= beta coefficient. *p≤.05. ** p≤.01