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Psychosocial predictors of visceral adiposity

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ABSTRACT

Psychosocial factors are thought to influence health through primarily direct physiological mechanisms or the alteration of health related behaviors. Three factors hypothesized to negatively impact health include arousal, life stress, and depressive symptomatology. One recent theorist suggests that the interaction between psychological stress and stress hormones on the neuroendocrine system may result in adverse changes to body composition, most notably the increased deposition of visceral adipose tissue (Bjorntorp, 1993). The current study prospectively examined the relationship between self-reported stressful life events, depressive symptoms and trait arousal on the deposition of visceral fat, as measured by computerized tomography (CT). Subjects were obtained from a sample of middle-aged males and females (n = 120). Stress measures included the Weekly Stress Inventory (WSI), a life-events measure of minor stressors, and the Life Events Survey (LES) a measure of major life events. Depression symptoms were measured with the Center for Epidemiological Studies Depression Scale (CES-D). Stress and depression were assessed at baseline, 6 and 12 months, and the CT images of visceral fat were obtained at baseline and 12 months. Trait arousal was measured with the Arousal Predisposition Scale at baseline. Arousal, stress and depression scores over 12 months were then standardized and averaged, and entered into a hierarchical multiple regression model in order to predict changes in visceral adiposity from baseline to 12 months. The model was significant in predicting visceral fat, accounting for 16.9% of the variance. Further examination of the model indicated the presence of a significant 3-way interaction between arousal, stress and depression, such that visceral fat was predicted by the interaction of low

arousal, high stress and high depression. When the interaction terms were added to the regression analysis as additional steps, the model continued to be significant, accounting for 20.9% of the variance. Interestingly, these models were significant in predicting visceral adiposity despite the fact that the relationships observed were not all in the expected directions. These findings have implications for both researchers and clinicians, who may wish to incorporate more specific psychosocial measures and interventions in the study and treatment of overweight and obesity.

INTRODUCTION

> "The simple fact is that more people die in the United States of too much food than of too little."

As reflected by former United States Department of Agriculture Secretary Daniel Glickman, in his testimony to the Louisiana Department of Health and Hospitals' Ad Hoc Committee on Obesity in 1999, there exists a growing consensus that overweight and obesity have reached epidemic proportions (LADHH, 1999). Obesity is a condition that significantly raises the risk of health complications from a variety of serious diseases, including hypertension, diabetes, heart disease, and breast, prostate and colon cancers. Overweight and obesity contribute substantially to the causes of preventable illness, and subsequently take an economic toll on rising health care costs, resulting in increased expenditures within both the private and public health care systems. The rates of obesity prevalence are climbing in all sectors of the population, and obesity rates among lower income, female and minority populations are alarming. Since overweight and obesity lead to increased morbidity and mortality, the increasing prevalence rates of this disease demonstrate an enormous public health problem, both within Louisiana and the country as a whole (LADHH, 1999).

Additional evidence has suggested that one's pattern of body composition and fat distribution can further predict significant health risk, above that accounted for by obesity alone. Specifically, an abdominal, visceral pattern of body fat deposition has been demonstrated to be the strongest predictor of morbidity and mortality in obese subjects, and an independent predictor of both cardiovascular disease and diabetes (Arcaro, Zamboni, Rossi, Turcato, Covi, Armellini, Bosello & Lechi, 1999; Peeke & Chrousos, 1995). Visceral obesity has also been associated with the Metabolic

Syndrome, a syndrome marked by multiple endocrine abnormalities, including hypertension and problems with essential insulin and lipid regulation (Bouchard, Bray & Hubbard, 1990).

Several mechanisms are hypothesized to influence the deposition of visceral adipose tissue, and one of the more interesting theories posited involves the interaction of psychological stress and stress hormones on the neuroendocrine system and resulting metabolic changes in the body (Bjorntorp, 1993). This paper reviews the literature on obesity, body fat distribution, stress and its effects on these systems, and evidence for the theory referred to by some as the "Civilization Syndrome." A study designed to test this theory in an adult sample of middle-aged males and females is then presented.

Overweight and Obesity

Estimates published by the National Heart, Lung and Blood Institute indicate that 97 million adults in the United States are overweight or obese, a condition that significantly raises the risk of health complications from a variety of serious diseases. Similarly, the Centers for Disease Control has recently emphasized the deleterious effects of obesity by highlighting several factors, including the increase in obesity-related comorbid illnesses and health complications associated with the disease, as well as from the economic implications of obesity (NHLBI, 1998; CDC, 2000). Overweight and obesity contribute substantially to the costs of preventable illness; for example, obesity-related diseases account for approximately 80% of the national healthcare budget, or approximately $100 billion (Wolf & Colditz, 1998).

Classification of overweight and obesity has typically been based on a percentage of ideal weight, or a calculation of body mass index (BMI), a mathematical

formula which adjusts body weight to height. In most national surveys to date, a BMI of greater than 27 kg/m^2, corresponding to approximately 120% of ideal body weight, has been the defining range for obesity (Bray, 1998a). Using this criteria, a previous report of the National Health and Nutrition Examination Survey indicated that 34% of American adults were obese (Kuczmarski, Flegal, Campbell & Johnson 1994). In 1998, newer obesity standards were adopted worldwide, which have reestablished the criteria for overweight and obesity. "Overweight" is now defined as a BMI of 25-29, and "obese" is now defined as a BMI greater than 30. According to these new criteria, 65% of American adults aged 20 years and older are now overweight or obese (Flegal, Carroll, Ogden & Johnson, 2002).

The incidence of obesity is higher in women than men, with prevalence rates of 27.5% and 33.4% respectively. The disorder is significantly higher in non-white (36.6%) versus white populations (28.7%) (Flegal et al., 2002). Prevalence rates of obesity are even higher in certain subgroups of the population, such as ethnic minorities and individuals with low socioeconomic status, income and educational levels (Flegal et al., 2002; LADHH, 1999). The prevalence of obesity and overweight also tends to increase in both men and women between the ages of 20 and 50 (Bray, 1998a). In addition, while genetics certainly plays a role in the expression of overweight and obesity, the more than 200% increase in prevalence rates in the past 15 years clearly reflects environmental rather than genetic influences (LADHH, 1999).

Conversely, overweight and obesity are risk factors for several other comorbid health conditions, co-occurring with diabetes (95.6%), hypertension (84.1%) and high cholesterol (76.5%) (Mokdad, Ford, Bowman, Dietz, Vinicor, Bales & Marks, 2003).

For example, researchers have indicated that blood pressure, dyslipidemia and other risk factors such as smoking can only account for half of the excess risk of cardiovascular disease (Bray, 1998a). As such, obesity has been shown to be an independent risk factor for cardiovascular disease, and one which is linked to the other cardiovascular risk factors (Manson, Stampfer, Hennekens & Willett, 1987).

This rise in the incidence of obesity has paralleled the rising incidence of obesity-related diseases in the United States, and rates of diabetes, cardiovascular disease and other obesity-related disorders can be expected to continue increasing over the next 15-20 years (Bray, 1998b). The leading causes of death in the United States include coronary artery disease, cancer and cerebrovascular disease, diseases which are all associated with high-risk health states like obesity. Recognizing obesity's fundamental position, McGinnis and Forge (1993) identified diet and physical activity patterns as their 2nd leading "Actual" cause of death, contributing significantly to 5 of the top 10 causes of death in the United States (heart disease, cancer, stroke, COPD and arteriosclerosis).

Data showing that weight loss can improve the risk factors associated with obesity is substantial (NHLBI, 1998; WHO, 1998). In a well-controlled study, Sjostrom and colleagues published two-year data on changes in HDL cholesterol, total cholesterol, triglycerides, insulin, glucose, and blood pressure (Sjostrom, Rissanen, Andersen, Boldrin, Golay, Koppeschaar & Krempf, 1998). These researchers found a nearly linear relationship between the change in body weight and the change in the relative risk factor. The exception was total cholesterol, in which a change of more than

20 kg was required before total cholesterol began to fall. For other risk factors, weight losses of >5% were associated with beneficial changes.

Body Fat Distribution

Evidence is accumulating which suggests that it is not simply the *presence* of excess body weight that is crucial, but that the specific *patterning* of body fat is, in fact, more significant with regard to serious health complications. Specifically, an intraabdominal, visceral pattern of fat distribution is associated with greater health risks than either subcutaneous abdominal or gluteofemoral patterns (Rexrode, Carey, Hennekens, Walters, Colditz, Stamfer, Willett & Manson, 1998; Seidell, Hans, Feskins & Lean, 1997; Donahue, Abbott, Bloom, Reed & Yano, 1987). This pattern of intraabdominal visceral obesity is typically observed in subjects with higher waist-hip ratios (> 1.0), whereas the gluteofemoral pattern is seen in subjects with low waist-hip ratios (< 1.0) (Lemieux, Prud'homme, Bouchard, Tremblay & Depres, 1996; Ljung, Anderssen, Bengstsson, Bjorntorp & Marin, 1996).

Centralized body fat distribution has been shown to be the strongest predictor of morbidity and mortality in obese subjects, and an independent predictor of cardiovascular disease, diabetes, vascular damage and endothelial dysfunction (Arcaro, Zamboni, Rossi, Turcato, Covi, Armellini, Bosello & Lechi, 1999; Peeke & Chrousos, 1995). In addition, while there is a direct correlation between BMI and overall mortality, which begins to increase at BMI's greater than 25, risk factors independently increase with waist circumference size, often used as an indirect measure of abdominal obesity (NHLBI, 1998). Studies have suggested that men with waist sizes above 40"

and women with waist sizes above 35" have substantially higher rates of obesity-related health complications (Lemieux, Prud'homme, Bouchard, Tremblay & Depres, 1996).

In several large epidemiological studies, high waist-hip ratios have been associated with a number of adverse health outcomes, including heart disease, premature death, stroke, Type 2 diabetes mellitus, and increased smoking and alcohol consumption. (Rosmond & Bjorntorp, 1999; Rosmond & Bjorntorp, 1998; Lloyd, Wing & Orchard, 1996; Rosmond, Lapidus, Marin & Bjorntorp, 1996; Marin, Darin, Amemiya, Andersson, Jern & Bjorntorp, 1992; Georges, Mueller & Wear, 1993; Wing, Matthews, Kuller, Meilahn & Plantinga, 1991).

In addition, the presence of a high visceral adipose/total adipose tissue ratio has been demonstrated to accurately differentiate between male patients with and without a history of coronary artery disease (Tirkes, Gottleib, Voci, Waldman, Masetta & Conover, 2002). Likewise, researchers using regression designs have revealed visceral adiposity to be a significant predictor of a number of adverse health outcomes, including higher levels of fasting blood glucose (r^2 = .28), triglycerides (r^2 = .16), low-density lipoproteins (r^2 = .16), total cholesterol (r^2 = .12), and apolipoprotein B (r^2 = .12) (Hernandez, Monter, Zamora, Cardosa, Posadas, Torres & Posadas, 2002).

Measurement of Body Fat Distribution

As noted previously, the current standard for assessing overweight and obesity is the body mass index (BMI), which is a mathematical calculation of weight in kilograms/height^2 in meters. While the body mass index is often used as a crude measure of body fat composition, it is not ideal, because it does not assess the relative contributions of fat mass versus lean body mass, or the placement of fat in different

body compartments. Measures of waist-hip ratio (WHR) and waist circumference (WC) or abdominal saggital diameter are improvements over the use of BMI in estimating body fat distribution, and various studies have indicated that WHR > 1, and WC > 40" for men and WC > 35" for women are associated with an increased risk of adverse health outcomes (Ljung, Anderssen, Bengstsson, Bjorntorp & Marin, 1996; Lemieux, Prud'homme, Bouchard, Tremblay & Depres, 1996). However, the use of WHR and WC are also less than optimal because they do not account for the differences between abdominal visceral adipose tissue (VAT) versus subcutaneous adipose tissue (SAT).

Technological advances in imaging techniques have led to the advent of more direct measures of body composition over the past 5-10 years. Specifically, X-ray, computer-assisted tomography, and magnetic resonance imaging technologies are being utilized in order to obtain precise assessments of body composition. Dual-energy X-ray absorptionmetry (DEXA) is currently considered to be the state-of-the-art in body composition measurement, and is used to obtain accurate measurements of total body mass, lean body mass, fat mass and bone mass on entire bodies or body regions, such as the arm, leg or trunk. While this technology is excellent for assessing differences between muscle, bone and fat mass, it cannot make fine-grained distinctions between the *locations* of fat, muscle or bone distribution. Therefore, like the waist-hip ratio and waist circumference, DEXA cannot account for the differences between abdominal visceral adipose tissue (VAT) and subcutaneous adipose tissue (SAT) (Park, Heymsfield & Gallagher, 2002).

Computer-assisted tomography (CT), a well-established technology in other domains, has recently experienced an increase in use for the assessment of adipose tissue. Cross-sectional CT scans between the L2-L3 lumbar vertibrae and the top of the iliac crest are typically used in the assessment of both visceral (VAT) and total adipose tissue (TAT). In these analyses, VAT is equal to the sum of total intraperitoneal and retroperitoneal adipose tissue, and TAT equals the sum total of visceral and subcutaneous adipose tissue (see Figure 1) (Tirkes, Gottleib, Voci, Waldman, Masetta & Conover, 2002). The biggest limitation to using CT is that this technique is difficult to use in determining total fat mass, or body composition over larger areas. For this reason, it is often used in conjunction with DEXA imaging for this purpose. Norms for assessment of CT adipose tissue are available, and abdominal fat areas are typically adjusted for age, gender and total fat mass in all analyses (Enzi, Gasparo, Biondetti, Fiore, Semisa & Zurlo, 1986).

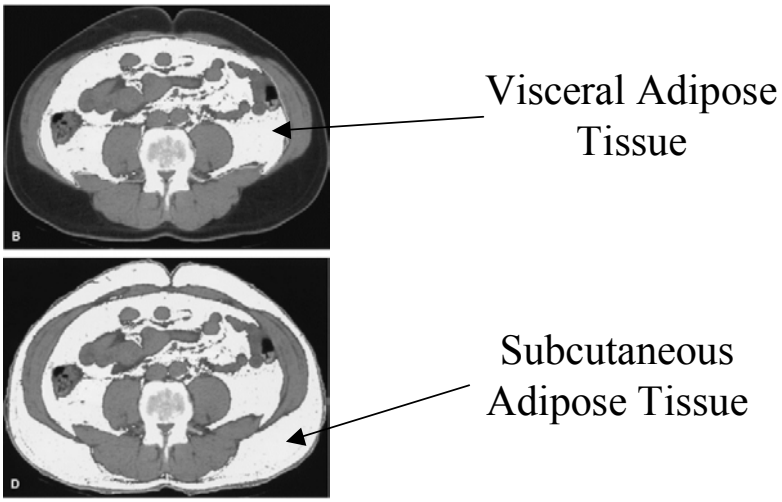

Figure 1. CT Image of Visceral and Subcutaneous Adipose Tissue

The newest imaging technology used in the assessment of abdominal adiposity is magnetic resonance imaging (MRI). MRI is capable of calculating total fat mass, nonabdominal, abdominal subcutaneous and visceral fat masses, and in this way is an improvement over both DEXA and CT (Janssen, Heymsfield, Allison, Kotler & Ross, 2002). However, MRI is often prohibitively expensive, and for this reason few studies have been published using this technology.

Factors Influencing Body Fat Distribution

Several factors are known or hypothesized to influence the deposition of visceral adipose tissue, the most obvious being energy balance in terms of increased caloric intake or decreased energy expenditure (Sorensen, 1995). Undoubtedly, visceral adipose tissue increases as total fat mass increases. However, causal factors contributing to the differential deposition of visceral adipose tissue are less clear.

Age, gender and race are all associated with known differences in central adiposity (Janssen, Katzmarzyk & Ross, 2002; NHLBI, 1998). Whereas men tend to have more of a centralized body fat distribution, premenopausal women tend to have a gluteofemoral pattern (Janssen, Katzmarzyk & Ross, 2002). As women enter menopause body fat distribution changes to a more central pattern. Researchers have hypothesized that decreasing levels of circulating estrogen play a role, since hormone replacement therapy reverses this effect (Simkin-Silverman & Wing, 2000). However, the question of whether estrogen directly affects body fat distribution, or whether it produces changes in dietary intake and physical activity patterns is still unclear (Lovejoy, Smith & Rood, 2001; Poehlman, Toth & Gardner, 1995). Several studies have also noted racial differences in the distribution of visceral adipose tissue, with

African-Americans observed to have significantly smaller VAT depots compared to Caucasians, even after controlling for total adiposity (Hill, Sidney, Lewis, Tolan, Scherzinger & Stamm, 1999; Lovejoy, de le Bretonne, Klemperer & Tulley, 1996; Conway, Yanovski, Avila & Hubbard, 1995).

In a series of descriptive analyses using data from large epidemiological studies, high waist-hip ratios have been associated with a number of psychosocial factors, including depression, anxiety, anger, stress, poor coping, poor social support, low SES, low education, and increased smoking and alcohol consumption. (Rosmond & Bjorntorp, 1999; Rosmond & Bjorntorp, 1998; Lloyd, Wing & Orchard, 1996; Rosmond, Lapidus, Marin & Bjorntorp, 1996; Marin, Darin, Amemiya, Andersson, Jern & Bjorntorp, 1992; Georges, Mueller & Wear, 1993; Wing, Matthews, Kuller, Meilahn & Plantinga, 1991). Smoking and excessive alcohol intake also appear to independently contribute to the differential deposition of visceral adipose tissue, with several studies demonstrating higher VAT depots among smokers and alcoholics (Janssen, Katzmarkzyk & Ross, 2002; Visser, Launer, Deurenberg & Deeg, 1999; Kvist, Hallgren, Jonsson, Pettersson, Sjoberg, Sjostrom & Bjorntorp, 1992; Larsson, Svardsudd, Wilhelmsen, Bjorntorp & Tibblin, 1984). In these studies nicotine and ethanol are presumed to adversely effect both cortisol secretion and insulin regulation, leading to increased visceral fat deposition.

The most comprehensive theory regarding the differential deposition of visceral adipose tissue involves the interaction of psychological stress and stress hormones on the neuroendocrine system. Specifically, this theory hypothesizes that psychological stress and its subsequent pattern of stress hormone release results in profound metabolic

changes in the body over time, leading to the deposition of visceral fat (Bjorntorp, 1993). However, prior to evaluating that theory, a review of stress, physiological arousal, and their impact on health outcomes, weight gain and body fat distribution is needed.

Overview of the Concept of Stress

While the origins of the concept of stress date back to Hippocrates, the construct has been marked by broad variations in the physiological, behavioral and psychological elements actually used to define stress. For example, in the 14th century the term *stress* described the social hardship and economic adversity prevalent at the time. As interest in stress physiology spread to the United States during the early 1900s, William Cannon's research on biobehavioral survival mechanisms and resultant theory of "fight or flight" led to the development of the concept of *homeostasis,* which he defined as "the coordinated physiological process which maintains . . . steady states in the organism" (1939).

In the early 20th century, Hans Selye began his pioneering research, focusing on the behavioral and physiological aspects of stress. Selye's seminal work eventually led to an interest in the systematic study of stress (Everly, 1989). As a result of Selye's endeavors, professionals in many scientific disciplines began to recognize the importance of behavioral factors in the study of stress. Selye posited that a "general adaptation syndrome" (GAS) occurs within an organism when confronted by "diverse nocuous agents" (1936). This view of stress defines the concept as the “nonspecific result of any demand upon the body.” The effect of these demands on the body produce a biological syndrome that is marked by a triad of physiological changes in the adrenal

glands, the lymphatic system, and the stomach and upper intestinal tract. These biological indicators become evident in a stereotyped fashion subsequent to exposure to any type of somatic or psychological stress, including blood loss, fatigue, pain, ingestion of toxins, emotional arousal, fear, concentration, and great elation (Selye, 1982).

The GAS response thus occurs in three discrete stages: alarm, resistance, and exhaustion. The common responses of the body to various types of stressors led Selye to distinguish "eustress", or positive stress, from harmful or negative stress, or "distress". This distinction is evident only in the nature of the stressor itself, however, and not in the body's response to any particular stressor. Contemporary theorists continue to include Selye's GAS among the most highly regarded descriptions of the stress response (Everly, 1989).

Building on the foundations created by the basic sciences, social scientists quickly became interested in the stress concept. Social scientists adopted the term *stress* to describe social demands and disruptions (Lazarus & Folkman, 1984). In 1966 Lazarus suggested that stress be considered a subdiscipline within psychology. Additional developments included the recognition of stress as a contributing factor in psychosomatic illness in the first edition of the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 1968) as well as in the emergence of journals dedicated to the study of stress, such as the *Journal of Human Stress, Psychophysiology,* and the *Journal of Traumatic Stress.* Today, stress has become a household term, popularized by such expositions as the 1969 U.S. Surgeon General's warning about the deleterious effects of stress on health (Everly, 1989).

Definition of Stress

The diversity of opinion surrounding the definition of stress has created disagreements among stress researchers, preventing “stress” from becoming a universally defined or accepted construct. Some researchers have argued that the concept of stress is too broad and ambiguous to adequately define (Engel, 1985). For example, Ader has urged researchers to discard the term as a descriptive label, and instead focus efforts toward uncovering the mechanisms subsumed under stress (1980). Despite this criticism, investigators have attempted to define the nature of stress, primarily described in terms of stimulus, response or interactional theories.

Stimulus Theories

Cannon's work on homeostasis was the first to identify stress as a stimulus, comprised of any event that prepares the organism for the "fight or flight" response (1939). This approach highlights the objective nature of stress, and applies the term “stressor” to the specific internal, external, psychological and biological events which produce the stress response (Lazarus & Folkman, 1984; Everly, 1989). Elliott and Eisdorfer (1982) have described four types of stress, defined by the frequency, intensity and duration of the precipitating stressor: (1) acute, time-limited; (2) stressor sequences; (3) chronic, intermittent; and (4) chronic. While stimulus definitions of the stress response may provide a useful taxonomy, the prevalent view among stress researchers is that individual differences in stress appraisal are important considerations as well (Lazarus & Folkman, 1984).

Response Theories

In contrast to the stimulus approach, other stress theorists have defined stress as the response an organism makes to environmental changes. Selye defined stress as the "nonspecific response of the body to any demand" (1974). In a similar vein, Everly discussed stress in a biological framework, defining it as a "physiological response that serves as a mechanism of mediation, linking any given stressor to its target-organ effect or arousal" (1989). Lacey also noted the importance of specificity in the response mechanism (1950). Specificity refers to the notion that different individuals will respond to the same stressor with differing physiological reactions.

The primary hypothesis of the physiological representation involves the sympatho-adrenomedullary (SAM) and hypothalamo-pituitary-adrenocortical (HPA) systems as mediators of stress responses. When an organism is able to adequately cope with a stressful stimulus, electrochemical changes in the brainstem mobilize the SAM axis to release the catecholamines epinephrine and norepinephrine via the adrenal medulla (Jemmott & Locke, 1984). These neurochemical changes ready the organism for the "fight or flight" response described by Cannon (1939). Selye referred to this increased metabolic activity to mobilize stress resources as a *catatoxic* response, while a *syntoxic* response would occur if no coping resources were available (1982). During the syntoxic response, passive tolerance behaviors such as hypervigilance and withdrawal activate the HPA pathways, resulting in cortisol and corticosteroid release by the adrenal cortex (Jemmott & Locke, 1984).

Stress-induced activation of the HPA axis results in a series of neuroendocrine changes referred to as the “stress response” or “stress cascade”. This response is

described as regulatory in nature, such that it permits the organism to make the physiological and metabolic changes necessary to maintain homeostasis. In humans, this response is initiated with the release of corticotropin-releasing hormone (CRH) from the hypothalamus in response to a discrete stressor. Adrenocorticotropin hormone (ACTH) release by the pituitary gland is then stimulated, which acts on the adrenal cortex to release the glucocorticoids cortisol and corticosteroid into the bloodstream. Under normal conditions, the glucocorticoids then act in a negative feedback loop to terminate release of CRH (Miller & O'Callaghan, 2002).

These neuroendocrine changes parallel the biobehavioral stress response (alarm, resistance, exhaustion) described by Selye as the General Adaptation Syndrome (1936). During the alarm phase, the sympathetic nervous and HPA systems are stimulated. Hyperarousal of these systems occur during the resistance phase, as the body's homeostatic mechanisms attempt to compensate for the physiological effects of the stressor. Finally, if the organism is unsuccessful in coping with the stressor, exhaustion occurs. Both psychological and physiological symptoms of exhaustion may be manifested, with illness and eventual death occurring with sustained application of the stressor (Everly, 1989; Selye, 1982).

Although the hormones of the SAM and HPA axes have received the most attention, additional hormones have also been established as producing physiological reactions to stress (Baum, Grunberg & Singer, 1982). Stress responses have been associated with elevated levels of growth hormone and prolactin in the pituitary gland, as well as increased secretions of the natural opiates beta, endorphin and enkephalin.

Interactional Theories

Investigators have criticized stimulus and response definitions on several levels, with one major criticism constituting the lack of attention paid by these models to individual differences. Stemming from these critiques, interactional descriptions of the stress response emerged, which focus on the relationship between individual and environmental variables in mediating the stress response. For example, Wolff first pointed out that stress is a "dynamic" state dependent on the interaction between an organism and its aversive external environment (1953).

Lazarus expanded the interactionist theory, creating a transactional model of stress (1966). In the transactional model, stress is the "particular relationship between the person and environment that is appraised by the person as [. . .] exceeding his or her resources and endangering his or her well-being" (Lazarus & Folkman, 1984). This view of stress emphasizes the cognitive variables that mediate a person's response to their environment. The perception of the event or situation, and the individual's efforts to manage the stress situation, are defined in terms of two interacting processes: appraisal and coping (Lazarus, 1966; Lazarus & Folkman, 1982).

Appraisal refers to the cognitive process which connotes meaning to the stressful situation for the individual. Specifically, situations are appraised in terms of their expected or potential outcomes, i.e.- positive, negative or neutral. Negative situations can be interpreted one of three ways: threat situations which are anticipated to produce harm; harm-loss situations which are evaluated as having already produced harm; and challenge situations which have the potential for either harm or gain (Lazarus, DeLongis, Folkman & Gruen, 1985). Thus, appraisals can be influenced by a

variety of factors, including learning history, personality variables, and the availability of internal or external resources.

Coping usually refers to a variety of methods implemented by the individual in an effort to manage stressful situations. Problem-focused coping includes strategies that enable the individual to prevent stressful events from occurring, or which enable the individual to successfully avoid or resolve any difficulties which do occur. Emotion-focused coping includes strategies which moderate stress-induced emotions and related physiological arousal.

An individual's ability to utilize these coping strategies can alter biological functioning, and thus affect health outcomes via a variety of mechanisms, such as influencing neuroendocrine stress responses, contributing to changes in health or risk behaviors, or altering the individual's cognitive or behavioral response to illness (Holroyd & Lazarus, 1982). The transactional model thus suggests that individuals "actively define and shape stressful transactions by means of their cognitive appraisals and their coping responses" (Cameron & Meichenbaum, 1982).

Measurement of Stress

Not surprisingly, differences over stress semantics have extended into the measurement domain. With no uniform definition of stress, researchers have encountered difficulty reaching a consensus about appropriate stress measurements. The primary types of stress that have been examined in the literature include major life events, minor life events, and chronic stress. Laboratory simulated stressors and physiological measures of stress responding have additionally been used as objective

measures of stress. However, the most generalizable and commonly used measures of stress are subjective, self-report questionnaires.

Traditionally, researchers have utilized laboratory methods such as noxious physical stimuli (e.g., electric shock) or frustrating psychological tasks (e.g., mental arithmetic) to assess the physiological effects of stress (Baum, Grunberg, & Singer, 1982). However, these procedures have often been plagued with methodological and ethical concerns, and criticized for their limited generalizability because they can only simulate, rather than replicate, naturally occurring stress (Brantley & Jones, 1993). Blood and urinary assays are often used to assess corticosteroid and catecholamine levels, which improve the validity of stress assessment when used in conjunction with other stress measures (Baum, Grunberg, & Singer, 1982; Brantley, Dietz, McKnight, Jones & Tulley, 1988). However, the use of biochemical measures alone is not recommended, because they are susceptible to several confounding events outside the realm of stress, such as caffeine ingestion or exercise (Baum, Grunberg, & Singer, 1982).

Life-events research stemmed from the stimulus view of stress, and has provided the most consistent point of reference in stress measurement. Thomas Holmes pioneered life events research by constructing the Schedule of Recent Experiences (Hawkins, Davis & Holmes, 1957). Soon thereafter, Holmes and Rahe set the standard for life-events scales with the Social Readjustment Rating Scale (SRRS; 1967). The SRRS, a 43-item self-report questionnaire, assesses major life events and estimates the amount of life change in *life change units* (LCUs). However, opinions regarding the LCU measures have been mixed. In a review of LCU measures, face validity, simplicity,

concordant ratings from heterogenous samples, and predictive validity in regard to psychiatric or physical illnesses were noted as positive characteristics of these instruments (Horowitz, Schaefer, Hiroto, Wilner, and Levin, 1977; Miller, 1989). Nevertheless, critics have voiced concern over the psychometric properties of LCU scales and the possibility of compromised recall due to the time interval between event occurrence and scale administration (Horowitz et al., 1977; Monroe, 1982).

Explication of life-events assessment raises two additional issues: weighted versus subjective ratings of life change, and the desirability (or pleasantness) of events. Sarason, Johnson, and Siegel addressed these issues in their construction of the Life Experiences Survey (LES; 1978). This 57-item scale instructs subjects to rate item desirability and degree of impact on a 7-point Likert-type scale ranging from *Extremely negative (* - 3) to *Extremely positive (* + 3). The scale has been demonstrated to possess good psychometric properties, and renders three scores: positive, negative, and total.

Important distinctions about the nature of stressful life events have emerged from life-events research. Traditionally, stress research has focused on major life events, such as the death of a loved one, or job loss. However, more contemporary stress theorists have begun to study the impact of minor life events, termed *daily stressors* or *hassles,* on health and behavior, because of the frequency with which they occur relative to major stressors (Brantley & Jones, 1989; DeLongis, Coyne, Dakof, Folkman & Lazarus, 1982). Examples of minor life events include having an argument with a spouse, getting caught in traffic, or running out of spending money. Kanner and colleagues first directed attention to minor stressors with the Hassles Scale, a 117-item questionnaire measuring the severity and frequency of minor stressors over the past

month on 3-point Likert-type scale (1981). Similarly, the same research group developed the Uplifts Scale, an index of desirable minor life events (Kanner, Coyne, Schaefer & Lazarus, 1981).

Extending the focus on minor stressors, Brantley, Waggoner, Jones and Rappaport published the Daily Stress Inventory (DSI; 1987). Minimizing the problem of temporal remoteness, this 58-item questionnaire measures the frequency and impact of minor stressors likely to occur on a daily basis. The DSI possesses good psychometric properties, and has been validated against other self-report instruments, as well as daily endocrine measures of stress, specifically urinary cortisol and vanillylmandelic acid (a metabolite of epinephrine and norepinephrine) (Brantley, Dietz, McKnight, Jones & Tulley, 1988).

Likewise, the Weekly Stress Inventory was developed in order to conduct assessments over longer intervals (WSI; Brantley, Jones, Boudreaux & Catz, 1997). The WSI is an 87-item questionnaire assessing the frequency and impact of minor stressors likely to have been experienced in the past week. Items are rated on an 8-point Likert scale ranging from *Did not occur (0*) to *Extremely stressful (7*). The scale renders two scores, an event score, which is the total number of events endorsed, and an impact score, which is the sum of the subjective ratings of distress of the items endorsed. Norms for the WSI are available, and the instrument has demonstrated both good psychometric properties and concurrent validity with the DSI. In a recent longitudinal study, psychological assessments across a 6-month period provided a stable indicator of minor stress in a sample of adults recruited from primary care medical clinics (Scarinci, Ames, & Brantley, 1999).

Dissatisfied with event-specific measures, a group of researchers constructed the Perceived Stress Scale (PSS; Cohen, Kamarck & Mermelstein, 1983). The PSS is consistent with cognitive-based, interactionist stress theories and measures a respondent's appraisal of the global stress level in his or her life. The developers have reported the PSS to have adequate reliability and validity. An abbreviated phone-interview version is available. Additionally, the authors purport that the predictive power of the PSS is greater than life-events scores. However, opponents of this approach have argued that the PSS contains confounds with outcome measures that are greater than the confounds associated with minor life-event scales (Lazarus, DeLongis, Folkman & Gruen, 1985).

Stress and Physiological Arousal

Arousal has been described in theories of personality, performance, motivation and attention, and has been used to identify changes in the responsiveness of subjects to various types of environmental conditions. (Strelau & Eysenck, 1987; Eysenck & Eysenck, 1985; Strelau, 1985). It has been hypothesized that individuals who exhibit higher levels of arousal may also have an increased susceptibility to stress. For example, measurements of arousal have been associated with increased autonomic lability, or the inability to habituate to repeated autonomic stimulation, such as repeated exposure to environmental stressors (Lacey & Lacey, 1958). Similarly, the term arousability refers to individual differences in the predisposition toward arousal, and has often been described as a trait variable (Coren, 1990).

Stressors are capable of producing both central and peripheral physiological arousal, as evidenced by studies examining the effects of the stress response on the

sympathetic nervous, cardiovascular and neuroendocrine systems via measurements of electrodermal, electromyograph and hormonal indices. Physiological changes occurring during stress-induced arousal include increased heart rate, spleen contraction, glycogen-glucose transfer and release by the liver, increased blood flow to the brain and muscular system, increased respiration, and pupil dilation (Cox, 1978). These physiological adaptations are thought to increase the organism's resources for responding to threatening stimuli, and are suggestive of Selye's *catatoxic* stress response, described previously.

Similarly, the effect of arousal on the neuroendocrine system is marked by a variety of hormonal responses, most commonly the increased secretion of epinephrine and norepinephrine, adrenocorticotropin hormone, cortisol and corticosteriod. The release of glucocorticoids result in increased production of glucose and urea, release of free-fatty acids into the blood stream, suppression of immune system functions, and increased production of ketones (Everly & Rosenfield, 1981). These responses are thought to promote adaptation to stressors of an extended, chronic nature, and are suggestive of the previously explicated *syntoxic* stress response described by Selye.

Assessment of Physiological Arousal

Measurements of arousal have been assessed by electrodermal activity (EDA), as well as changes in electromyogram (EMG) and biochemical indices (Lacey & Lacey, 1958). Electrophysiological assessment involves the use of EDA and EMG equipment to detect acute changes in the electrical activity of target organs, as well as the heart rate, blood pressure and temperature of the subject. One common method of EDA assessment is measurement of skin conductance, whereas assessment of EMG is often

taken via bipolar recording of frontalis muscle activity (Venables & Christie, 1980). Use of biochemical assays to evaluate the amount or presence of catecholamine and/or glucocorticoids in bodily fluids is another method of assessing physiological arousal, and common measures include salivary cortisol, 24-hour urinary cortisol, urinary catecholamines, or serum free-cortisol (Cox, 1978).

Several limitations to using electrophysiological and biochemical indices of arousal exist, and are comparable to the limitations described previously in the discussion of stress measurement. Most significantly, both methods are suitable primarily for measuring transitory arousal states rather than chronic arousal, because of the acute nature of physiological assessment and the highly fluctuating, cyclical nature of stress hormone release. These reasons, in addition to issues regarding cost and ease of administration, have led to the development of several self-report measures of subjective physiological arousal.

Self-report measures of both state and trait arousal have been developed by various researchers, the first of which was the Autonomic Perception Questionnaire (APQ; Mandler, Mandler & Uviller, 1958). While this instrument has been shown to correlate significantly with self-reported anxiety and to reflect individual differences in autonomic reactivity, it does not correlate well with electrophysiological measures of arousal, and is limited by a lack of adequate reliability and validity data.

Thayer attempted to improve validity with electrophysiological measures of arousal by developing the Activation-Deactivation Adjective Check List (AD-ACL; Thayer, 1967). This measure assesses self-reported activation and deactivation, such as feeling excited or drowsy, as well as cognitive dimensions of arousal, like feeling

anxious. This instrument has been extensively validated with physiological measures of arousal, including heart rate, skin conductance, muscle action potential and blood flow volume. While the AD-ACL demonstrates good reliability and validity properties, it does not assess specific bodily systems associated with arousal, and appears to be most useful in assessing state, rather than trait, arousal.

Waters and colleagues (1984) attempted to improve upon these measures by developing the Autonomic Nervous System Response Inventory (ANSRI), a measure assessing specific physiological responses to memories of distinct emotional situations. This measure possesses adequate reliability and validity data, and has been well validated against electrophysiological measures of arousal, but like the others, is intended to assess only state arousal.

While researchers previously have attempted to assess trait arousal using various personality scales, use of these measures is limited by a lack of psychometric data and validity indicators (Stern & Higgins, 1969; Hastrup & Katkin, 1976). One exception in this area is the Arousal Predisposition Scale, an empirically derived scale developed by Coren (APS; 1988; 1990; 1993). This scale was developed as a measure of trait arousal, and item selection was based on ability of the item to predict sleep disturbance, a physiological index of arousal. Norms for the APS are available, and the instrument has been demonstrated to possess good validity in studies using the Activation-Deactivation Adjective Check List, and both electrodermal and electromyogram measures of arousal. The APS has also been shown to differentiate between high and low arousability in subjects reporting stress-related physical symptoms (Hicks, Conti & Nellis, 1992).

Stress and Health Outcomes

As previously noted, life-events research has stemmed from stimulus theories of stress, a view which suggests that stressful life events impact certain illnesses (Brown & Harris, 1989). Researchers and clinicians have therefore used a variety of measures to assess the impact of stress on psychiatric and medical populations. Notably, research has indicated that the effect of minor stressors on the progression of physical and psychological illness may be greater than the influence of major stressors (Brantley & Jones, 1993; DeLongis, Coyne, Dakof, Folkman & Lazarus, 1982).

Psychological factors, including life stress and coping, are believed to affect health primarily through direct physiological mechanisms or the alteration of health related behaviors. In a review of the literature, Brantley and Garrett (1993) summarize the proposed models of stress and illness which include: a) changes in physiological functioning, b) increased high-risk behavior, c) decreased resistance to disease, d) neurological hypersensitivity or e) inadequate coping. Investigations examining the specific relationship between stress and illness have consistently reported correlations between psychological distress and symptom presentation of both acute and chronic illness, with the most consistent evidence found for infectious diseases, cancer, cardiovascular disease, and chronic conditions, such as diabetes, asthma and gastrointestinal disorders (McEwen & Stellar, 1993).

The evidence linking stress to cardiovascular disease has been indirect, with the most consistent associations found among stress, personality and behavioral variables, such as hostility and Type A behavior pattern, and intermediary factors, such as severity of underlying coronary disease (Kop, 1999; Rosenman, 1996). However, one recent

longitudinal study has reported that minor stressors were found to be more important than major life events in the development of cardiovascular disease risk factors in young adults, particularly when coupled with poor coping and Type A personality features (Twisk, Snel, Kemper & van Mechelen, 1999). Psychological stress has also been identified as a potential trigger for acute coronary events and an exacerbating factor in various coronary symptoms (Kop, 1999; Rozanski, Bairey, Krantz, Friedman, Resser, Morell, Hilton-Chalfen, Hestrin, Beitendorf & Berman, 1988). Possible mechanisms for this effect include stress-induced increases in levels of catecholamines and cortisol (Rozanski et al., 1988).

Chronic illness has been cited as the most prevalent of all the major life stressors (Felton, 1990). Diabetes mellitus, which co-occurs with overweight and obesity in 96% of patients, is a chronic endocrine disease which significantly increases morbidity and mortality and constitutes the fourth leading cause of death due to a disease in the United States (Mokdad, Ford, Bowman, Dietz, Vinicor, Bales & Marks, 2003). Glycemic control in diabetics has been shown to be adversely affected by stress via activation of the HPA axis, and subsequent secretion of glucose counteregulatory hormones (Sulway, Tupling, Webb & Harris, 1980). Stress-induced release of growth hormone by the pituitary gland can also cause insulin resistance and sympathetic stimulation of pancreatic hormones (Surwit, Ross & Feinglos, 1991).

Tobacco and alcohol abuse are often a maladaptive attempt to cope with stressful situations, and stress can maintain the use of these substances (Best, Wainwright, Mills, & Kirkland, 1988; Feverstein, Labbe & Kuczmierczyk, 1986; Williams, Stinson, Parker, Harford, & Noble, 1987). Nicotine has been shown to

potentiate sympathetic arousal, and smoking compromises physiological systems (e.g., cardiopulmonary and immune) susceptible to stress (Trap-Jensen, 1988; McGill, 1988). Evidence also suggests that stress may be an important factor in predicting alcoholism, and individuals who become alcoholics may lack skills in stress management (Brantley & Garrett, 1993; Cotton, 1990).

Brownell has posited that the increase in obesity and stress-related disorders over the past century has resulted from sedentary lifestyles (1982). A renewed interest in exercise during the past two decades has paralleled research suggesting that physical fitness is a significant stress moderator (Brandon & Loftin, 1991; Roth & Holmes, 1985). Moreover, Everly contends that exercise, more than any other stress management strategy, prevents disease by ventilating the pathophysiological changes associated with the stress response (1989).

Stress and Weight Gain

Several investigators have associated psychological stress and weight gain, with the mechanism of action hypothesized to involve abnormalities in the neuroendocrine stress response, such that overproduction of cortisol and other stress hormones results in metabolic abnormalities. For example, glucocorticoids have been shown to produce insulin insensitivity, causing hyperglycemia, hypertryglyceridemia, hypercholestemia and hyperinsulinemia. In addition, glucocorticoids are capable of acting in concert with insulin to decrease energy expenditure and promote energy deposition (Brindley & Rolland, 1989). Other evidence has suggested that mild chronic stressors increase trough corticosteroid levels in both human and animal studies. This elevation of trough concentrations has often been accompanied by a reduction in peak values, suggesting

that stress acts to "level-out" the normally cyclical nature of hormone release, producing a more constant, rigid secretion pattern (Dallman, Akana, Bhatnagar, Bell & Strack, 2000).

Evidence for this stress-weight connection exists in several studies reporting psychosocial influences on obesity or its sequelae. For example, in a study examining predictors of weight gain in male fire fighters and paramedics, Gerace and George reported that higher levels of worry and stressful life events predicted significantly greater weight gain 7 years later (Gerace & George, 1996).

Vitaliano and colleagues, testing a model of chronic stress in primary caregivers of Alzheimer's patients, found increases in depression scores, psychological burden and daily hassles for caregivers versus control subjects over 15-18 months, as well as significantly greater weight gain, body mass index and fasting insulin and blood glucose levels (Vitaliano, Scanlan, Krenz, Schwartz & Marcovino, 1996; Vitaliano, Russo, Scanlan & Greeno, 1996). Similarly, other investigators have observed abnormal serum insulin and glucose responses following application of laboratory stressors in Pima Indian samples, a population commonly used as a genetic model of obesity (Esposito-Del Puente, Lillioja, Bogardus, McCubbin, Feinglos, Kuhn & Surwit, 1994).

Using a retrospective cross-sectional design, Ferreira et al. observed higher incidences of stressful life events and inversely correlated levels of serum prolactin and urinary cortisol in women who had gained at least 5 kg. in the previous 12 months, thus suggesting an abnormal neuroendocrine stress response in the women (Ferreira, Sobrinho, Pires, Silva, Santos & Sousa, 1995). Seematter and colleagues have similarly observed abnormal glucose and insulin responses in obese women compared to lean

controls after application of a laboratory stressor (Seematter, Guenat, Schneiter, Cayeux, Jequier & Tappy, 2000).

Stress and Body Fat Distribution

A characteristic visceral obesity has been observed in Cushing's Disease, and it is hypothesized that the excess glucocorticoid production present in this disease of primary hypercortisolism affects both energy storage and metabolism. (Peeke & Chrousos, 1995). Evidence for cortisol and catecholamine involvement in body fat changes has also been demonstrated in studies involving subjects with body fat redistribution secondary to antiretroviral treatment for HIV infection. Specifically, visceral adipose tissue was associated with significantly higher levels of 24-hour urinary cortisol and catecholamine levels than in control subjects (Renard, Fabre, Patris, Reynes & Bringer, 1999). Likewise, subjects expressing a genetic growth hormone deficiency, a hormone that normally antagonizes the effects of cortisol, have been shown to similarly exhibit central adiposity, dyslipidemia and other features of increased cardiovascular health risk (Barreto-Filho, Alcantara, Salvatori, Barreto, Sousa, Bastos, Souza, Pereira, Clayton, Gill & Aguiar-Oliveira, 2002).

Bujalska and colleagues, in an interesting study using adipose tissue cultured from subjects undergoing elective abdominal surgery, found that visceral, but not subcutaneous fat was capable of generating active cortisol from inactive cortisone via a distinct enzymatic expression. In addition, the enzyme response was enhanced in the presence of cortisol and insulin, suggesting that a constantly increasing cycle of glucocorticoid exposure may be a maintaining factor in viscerally obese subjects (Bujalska, Kumar & Stewart, 1997).

Raeikkoenen first observed that stress may differentially affect subjects based on their patterns of body fat distribution after finding that in lean men, a physical stress response was positively associated with increased waist-hip ratios, whereas for moderately obese men, moderate stress and higher depression scores were associated with WHR (Raeikkoenen, Hautenanen & Keltikangas-Jaervinen, 1994). Similarly, other investigators have found significant positive associations between self-reported stress, mood and higher BMI and WHR in samples of women with Type 2 diabetes (Bell, Summerson, Spangler & Konen, 1998).

Epel's 1999 cross-sectional study examined cortisol reactivity and psychological factors in response to lab-induced stressors in women with central versus gluteofemoral fat distribution. She found that subjects with high waist-hip ratios had higher levels of 24-hour urinary cortisol, greater cortisol reactivity, and exhibited more passive trait coping strategies and trait negative affect than the low WHR subjects (Epel, 1999). Davis et al. have observed that women classified as centrally obese exhibited an increased vascular stress response compared to peripherally obese matched controls, specifically larger stress-induced increases in diastolic blood pressure and total peripheral resistance (Davis, Twamley, Hamilton & Swan, 1999).

In an excellent study using adult MZ twin pairs discordant for visceral obesity, Mariemi and colleagues have observed higher levels of urinary cortisol, noradredaline excretion, alcohol consumption, sleep disturbance, and depressive symptoms to be present in the viscerally obese versus lean cotwins (Mariemi, Kronholm, Aunola, Toikka, Mattlar, Koskenvuo & Ronnemaa, 2002).

The "Civilization Syndrome"

Visceral obesity has been associated with the Metabolic Syndrome, a syndrome marked by multiple endocrine abnormalities, including hyperinsulinemia, insulin resistance, hypertryglyceridemia, low high-density lipoproteins and hypertension (Bouchard, Bray & Hubbard, 1990). Bjorntorp (1993) has proposed that visceral obesity develops as the result of a chronically elevated activation of the HPA axis, which occurs secondary to psychological stress. Specifically, he hypothesizes that chronic stress produces discrete, periodic elevations of cortisol secretion on a daily basis, which over time is followed by a rigid cortisol pattern characterized by low morning values, and higher "troughs" in the secretion cycle. Normal regulatory mechanisms eventually become compromised as the feedback control is diminished, and a parallel inhibition of sex steroid and growth hormones occur, which under normal conditions act to antagonize the effects of cortisol.

The physiological effects of this neuroendocrine perturbation include insulin resistance, hyperinsulinemia, hypertension, dyslipidemia and accumulation of body fat to visceral depots, all hallmark features of the Metabolic Syndrome. Bjorntorp has alternatively referred to this set of symptoms as a "Civilization Syndrome," highlighting the pressures of modern, competitive lifestyles in the generation of chronic stress (see Figure 2). He also points to the increased prevalence of high-risk health behaviors as contributing factors, such as increased tobacco and alcohol consumption, overeating and physical inactivity (Bjorntorp, Holm & Rosmond, 1999; 2000; Bjorntorp & Rosmond, 1999; 2000).

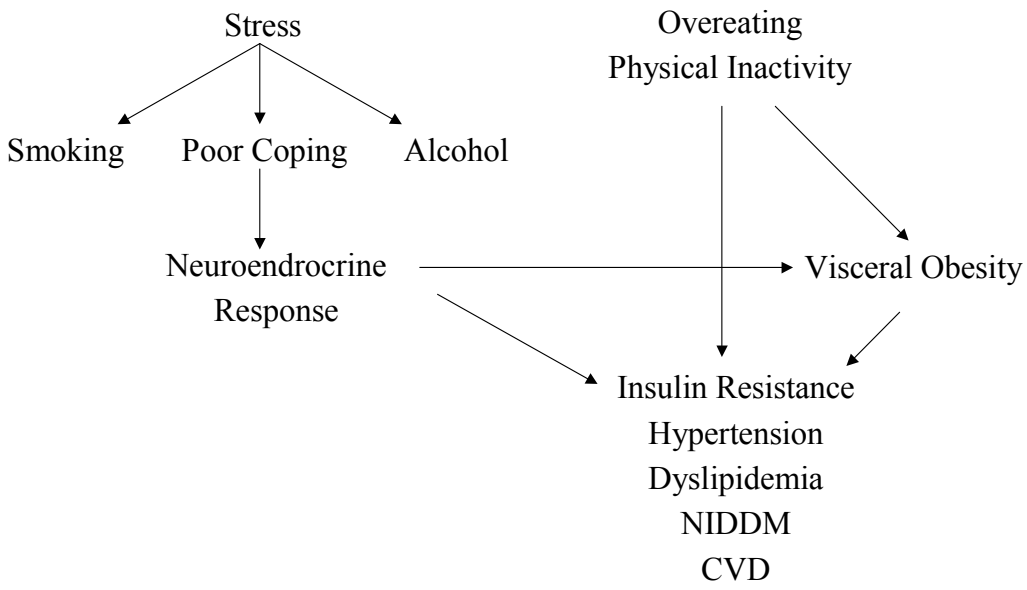

Figure 2. Civilization Syndrome[1]

Laboratory and clinical studies have shown that the Metabolic Syndrome is characterized by an increased deposition of intraabdominal, visceral adipose tissue, and that the mechanism of the fat deposition involves multiple hormonal abnormalities, specifically high levels of cortisol, adrenocorticotropin hormone and insulin, and low levels of growth hormone and sex steroid hormones (Bjorntorp, 1996b; Kissebah, Vydelingum, Murray, Evans, Hartz, Kalkhoff & Adams, 1982). Interestingly, this same pattern of hormonal abnormalities has been observed in animal studies of subjects exposed to environmental stress, suggesting that stress may act as catalyst for both visceral adiposity and the Metabolic Syndrome (Wallace, Shively, & Clarkston, 1999; Jayo, Shively, Kaplan & Manuck, 1993).

The stress reaction associated with these abnormalities has been described by Bjorntorp as a "depressive reaction" such that stressors perceived as chronic or

[1] Figure reprinted by permission of Obesity Research

overwhelming cause the organism to exhibit a defeatist or hopelessness response over time, rather than the more typical "fight or flight" response associated with acute stressors. This depressive reaction is hypothesized to result in an energy-conserving adaptation in the endocrine system, such that cortisol, adrenocorticotropin and insulin production are increased, growth and sex steroid hormone production is inhibited or halted completely, and triglyceride energy stores are increased and redistributed to areas where they can be swiftly utilized (i.e., the central visceral region) (Bjorntorp, 1993; 1996a).

This pattern is identical to that seen in the Metabolic Syndrome, and the studies reviewed previously have associated visceral obesity with several markers of increased psychological distress, such as higher levels of depression, anxiety, alcohol and tobacco consumption, as well as increased cortisol secretion in response to acute laboratory stressors. In addition, several other factors have been correlated with visceral obesity, including high rates of unemployment, difficulties with work when employed, low income, low standard of housing, lower educational levels, higher divorce rates and higher levels of alcohol consumption, all factors which may be described as *chronic minor stressors* (Bjorntorp, Holm & Rosmond, 1999; 2000; Bjorntorp & Rosmond, 1999; 2000).

Evidence for this mechanism of fat deposition exists in a series of excellent prospective studies with primates. Researchers from the Wake Forest School of Medicine have observed that both male and female cynomolgus monkeys exposed to social stress over several months developed greater intraabdominal fat depots than their non-stressed counterparts (Wallace, Shively, & Clarkston, 1999; Jayo, Shively, Kaplan

& Manuck, 1993). This group used computer-assisted tomography in order to assess intraabdominal fat in their monkeys, however, prospective studies using precise imaging techniques in humans are noticeably absent.

Evidence for the neuroendocrine aspect of the syndrome in humans is being tested in a series of studies conducted by Pasquali and colleagues, who have observed HPA axis hyperactivity in both males and females with visceral obesity. The method they developed, measuring stress hormone response to corticotropin-releasing factor (CRF) and adrenocorticotropin hormone (ACTH) stimulation during application of a laboratory stressor, has been well-documented and replicated (Pasquali, Vicennati, Calzoni, Gnudi, Gambineri, Ceroni, Cortelli, Menozzi, Sinisi & Rio, 2000; Vicennati & Pasquali, 2000; Pasquali, Anconetani, Chattat, Biscotti, Spinucci, Casimirri, Vicennati, Carcello & Labate, 1996). Similarly, subjects with higher waist-hip ratios have been observed to abnormally respond to standard dexamethasone suppression tests, a physiological challenge which typically reduces cortisol secretion in normal subjects (Ljung, Andersson, Bengtsson, Bjorntorp & Marin, 1996).

With regard to the role of mood disturbance in viscerally obese subjects, Arborelius and collegues have reported that corticotropin-releasing factor (CRF) appears to mediate endocrine responses in depression, suggesting that higher CRF levels in cerebrospinal fluid may be a state marker for depression in these subjects (Arborelius, Owens, Plotsky & Nemeroff, 1999).

In order to examine the effects of depression on both cortisol levels and body fat distribution, researchers in Germany recently measured salivary cortisol and visceral adipose tissue in 22 postmenopausal women with Major Depressive Disorder and 23

age-matched controls. Results indicated that only in the depressed women with elevated concentrations of free cortisol were visceral fat depots significantly greater. These women additionally exhibited higher oral glucose and serum insulin levels than both the control subjects and the depressed women with normal cortisol values, suggesting that the *interaction* of stress hormones and depression in these women is associated with their increased visceral adiposity (Weber-Hamann, Hentschel, Kniest, Deuschle, Colla, Lederbogen & Heuser, 2002).

Taken collectively, the studies reviewed above appear to suggest the existence of a relationship between stress, depressive symptoms, abnormal neuroendocrine responses and visceral obesity. However, studies investigating the specific sequence of events hypothesized by Bjorntorp are lacking. Conspicuously, each of the human studies reviewed previously uses cross-sectional or correlational designs, and most additionally use laboratory stress tasks and biochemical assay of stress hormones to infer the presence of naturally occurring stress.

Limitations of Previous Research

Thoits (1995) reviewed the current state of stress research and highlighted several directions for future investigations. For example, further elucidation of the relationship between stress and physical health outcomes is needed. In terms of chronic stress, issues related to chronic employment stress have been most consistently studied, while examination of *other types of chronic stress* (marital, parental, financial) are lacking. Investigations clarifying the relationships between the *sequence* of stressful life events and both physical and psychological consequences is desired, as well as assessment of multiple outcomes, in order to *identify associations between resulting*

physical and mental health effects. Finally, emerging interest in the *physiological mechanisms* underlying the stress-related outcomes has stemmed from recent studies correlating stress with various medical conditions and syndromes (Thoits, 1995).

There have also been a number of global criticisms of life event measures. Not surprisingly, the subjective nature of life-event instruments has fueled disputes regarding stress assessment. Brown (1989) cited the possibility of response biases creating, exaggerating, or attenuating associations between stress and relevant outcome variables. Additionally, others have debated whether stress measures employing subjective ratings have greater predictive power than measures with weighted ratings (Brown, 1989; Rahe, 1974). The reliability of life event scales can also be compromised when subjects are asked to recall events for longer than one year, leading some researchers to argue for the use of clinical interviews (Dohrenwend, Dohrenwend, Dodson & Shrout, 1984). However, despite these criticisms, a significant problem exists in measuring stress and Metabolic Syndrome susceptibility via long-term HPA axis activation, because of the highly variable nature of both cortisol and ACTH secretion, and the necessity of multiple daily measurements of either salivary or urinary cortisol over a period of weeks or months.

Body mass index (BMI), while it is the most commonly used metric in classifying obesity, is only a surrogate measure of body fatness. As such, BMI can be misleading in many cases, and is not recommended for use with several populations, including infants and children, minorities, aging adults, athletes and certain clinical populations (Prentice & Jebb, 2001). Similarly, the waist-hip ratio, which has been used in most of the studies published to date, is only a surrogate marker of body

composition, and is not useful in estimating the degree of abdominal obesity in lean subjects, or other populations exhibiting a relative atrophy of gluteal muscle, such as alcoholics (Bjorntorp, 1993).

A recent study using magnetic resonance imaging to measure body fat depots in 341 Caucasian men and women concluded that the combination of BMI and waist circumference independently predicted total fat mass, nonabdominal, abdominal subcutaneous and visceral fat masses. However, these assessments were less than optimal in measuring visceral adipose tissue, as they only predicted 57% of the variance in men and 60% of the variance in women (Janssen, Heymsfield, Allison, Kotler & Ross, 2002). For this reason, several authors have called for the increasing use of *direct measures* of body composition (Prentice & Jebb, 2001).

Furthermore, in most of the studies cited to date asserting support for Bjorntorp's theory, poor methodology and/or measurement of the variables of interest make inferences regarding causation impossible. The studies that Bjorntorp directly cites in support of the notion that psychosocial variables influence body composition have used cross-sectional data drawn almost exclusively from large, epidemiological studies conducted in northern Europe. These studies, while they have found significant correlations between the variables of interest, have used very broad, general measures to assess relevant outcomes. For example, the patients in these studies provided self-reported height and weight for the assessment of BMI, and self-recorded waist and hip measurements for the calculation of WHR. In addition, while stress is cited as the foundation of his theory, in these studies it was not directly measured. Rather, stress was inferred from surrogate variables obtained from demographic measures, such as

level of income and education, type of work, use of healthcare facilities, use of tobacco and alcohol, and use of drugs for anxiety or depression (Lapidus, Bengtsson, Hallstrom & Bjorntorp, 1989; Lapidus, Bengtsson, Larsson, Pennert, Rybo & Sjostrom, 1984; Larsson, Seidell, Svardsudd, Welin, Tibblin, Wilhelmsen & Bjorntorp, 1989; Larsson, Svardsudd, Wilhelmsen, Bjorntorp & Tibblin, 1984).

Several subsequent studies have improved the measurement issue by using validated measures of psychosocial variables (Epel, 1999; Bell, Summerson, Spangler & Konen, 1998; Vitaliano, Scanlan, Krenz, Schwartz & Marcovino, 1996; Vitaliano, Russo, Scanlan & Greeno, 1996; Raeikkoenen, Hautenanen & Keltikangas-Jaervinen, 1994). However, these studies have also used weight gain, body mass index or waist-hip ratio calculations, less than optimal measures of body composition.

A few studies have used both well-validated stress measures and precise measures of body composition (Mariemi, Kronholm, Aunola, Toikka, Mattlar, Koskenvuo & Ronnemaa, 2002; Weber-Hamann, Hentschel, Kniest, Deuschle, Colla, Lederbogen & Heuser, 2002; Arborelius, Owens, Plotsky & Nemeroff, 1999). However, these studies have each been cross-sectional in design, and have used predominantly laboratory stressors in order to test the effects of stress on biochemical assays.

Several of the best studies conducted to date have been molecular studies of the mechanisms of HPA axis activation or fat deposition. However, even these studies have been only cross-sectional designs, barring causal inferences (Pasquali, Vicennati, Calzoni, Gnudi, Gambineri, Ceroni, Cortelli, Menozzi, Sinisi & Rio, 2000; Vicennati &

Pasquali, 2000; Pasquali, Anconetani, Chattat, Biscotti, Spinucci, Casimirri, Vicennati, Carcello & Labate, 1996).

Excellent prospective studies by one group of researchers using primate subjects provide the strongest evidence for the causal role of stress in promoting deposition of visceral adipose tissue (Wallace, Shively, & Clarkston, 1999; Jayo, Shively, Kaplan & Manuck, 1993). However, obesity research over the past several years has been plagued by the problem of reproducing in humans similar results to those seen using comparative biology designs.

In the only prospective study in humans published to date, Nelson et al. reported cynism, anxiety and anger to be statistically significant predictors of waist-hip ratio in males, and depression to be a statistically significant predictor of WHR in females (Nelson, Palmer, Pedersen & Miles, 1999). However, the practical significance of this study is questionable, since the psychosocial predictors accounted for only 8.2% of the variance in men, and 2.0% of the variance in women. In addition, this study used only a very general measure of body composition, the waist-hip ratio. Therefore, *prospective studies using well-validated psychosocial measures and precise assessments of body fat distribution in humans are needed*.

Preliminary Studies

Preliminary studies examining these variables have also been conducted by the current investigator, using samples drawn from larger investigations. In one study, several significant associations between stress and measures of body composition were found in a sample of perimenopausal Caucasian women drawn from an ongoing study investigating the effects of menopause on cardiovascular risk. This study improved

upon existing studies by using a well-validated measure of stress (Weekly Stress Inventory), and precise measures of body composition (CT and DEXA).

Results indicated that increases in stress over 12 months were significantly correlated with visceral adiposity ($r = .25$) at 12 months. In women who gained weight (≥ .5 kg) over one year, total stress scores were significantly related to weight ($r = .45$), body fat percentage ($r = .37$), fat mass ($r = .44$) and total adiposity ($r = .43$) at 12 months. For women who were already obese at baseline (BMI ≥ 30), increases in stress over the year were significantly associated with baseline weight ($r = .56$), body fat percentage ($r = .55$), fat mass ($r = .68$) and total adiposity ($r = .64$). This pattern was similar to women who significantly increased their stress over the year (WSI-E ≥ 10; 1 SD), whose stress increase was positively correlated with baseline body fat percentage ($r = .44$), fat mass ($r = .48$), total adiposity ($r = .49$) and visceral adiposity ($r = .44$). Finally, women who reported an increase in stressful life events over one year gained significantly more weight ($t = 2.802$, $p = .01$) and had higher BMI's ($t = 2.770$, $p = .01$) than women who did not report an increase in stressful life events (Rhode, Lovejoy, Smith, Dutton & Brantley, 2001).

This study, while suggestive of a relationship between stress and body composition, also had significant limitations, the most obvious being that the results were strictly correlational, thus barring causal inferences. Less apparent, the results may be attenuated by the fact that the women followed in this study were perimenopausal, and therefore by definition did not have well-controlled levels of estrogen and progesterone. As noted previously, sex steroid hormone levels have been shown to significantly impact body composition, and women going through menopause

without hormone replacement therapy are known to gain significant amounts of both total and visceral adipose tissue (Simkin-Silverman & Wing, 2000).

The second study used a sample from a larger clinical trial on primary care office management of obesity to prospectively examine the effect of major stress, minor stress and depression on weight change in a sample of low-income African-American women. Again, this study improved upon previous investigations by using well-validated measures of stress (Life Experiences Survey and Weekly Stress Inventory) and depression (Center for Epidemiological Studies Depression Scale).

For this analysis, stress and depression scores over 12 months were standardized and averaged in order to create single composite major stress, minor stress and depression predictor variables. The variables were then entered into a regression equation which controlled for both smoking status and use of hormone replacement therapy. Results indicated that the model accounted for 26% of the variance in weight change over 12 months ($R^2 = .26$, $p < .02$). When this same model was used to predict change in weight from the end of the weight loss intervention (6-months) to 12 months, only depression and minor stress scores were found to be significant, accounting for 29% of the variance ($R^2 = .29$, $p < .004$). Finally, when the model was used to predict BMI at 12 months, only minor stress scores were found to contribute significantly, accounting for 31% of the variance ($R^2 = .31$, $p < .002$) (Rhode, Martin, Dutton & Brantley, 2003).

This study strongly suggests several hypotheses, specifically that both stress and depression influence body composition, and that perhaps those two variables interact to create their effects. It additionally suggests that minor life events exert an effect

independent of and more significant than that of major life events. Finally, because the relationship was more significant after the end of the clinical weight loss intervention (from 7-12 months), it suggests that different mechanisms may be involved in weight loss versus weight maintenance periods of treatment intervention, and therefore stress and depression may be particularly important variables in the absence of a formal weight loss intervention. However, similar to the criticisms of several previous studies, this study did not examine precise measures of body composition; rather it used the very general assessments of body weight and BMI.

Summary and Study Rationale

Taken collectively, the research reviewed above suggests that psychosocial variables, specifically stress, depression, arousal and certain demographic variables may influence the preferential deposition of body fat in general, and visceral adipose tissue specifically. Reviewers have emphasized the need for studies aimed at identifying associations between physical and mental health effects and in clarifying the physiological mechanisms underlying stress-related health outcomes. However, the studies conducted thus far in this area have been limited by several methodological concerns, such a lack of standardized measures, the use of collateral rather than direct measures of body composition, and the use of quasi-experimental and correlational research designs.

The current study was proposed to address several of these previous limitations by using a randomized, prospective design, well-validated psychosocial measures and precise assessments of body fat distribution. The goal of the current study was to

further elucidate the relationship between the deposition of body fat and the psychosocial variables hypothesized to influence body composition.

Collateral evidence on the mechanism and precise relationships between stress, depression, arousal and body fat deposition have implications for directing further research in the area of overweight and obesity, as well as potential utility in developing clinical interventions. For example, if psychological variables such as stress and depression were found to have a causal role in the growing epidemic of obesity, future research and clinical endeavors could target these variables more directly. Psychological treatments could then be developed to complement the standard physiological interventions utilized at present.

Therefore, the current study was a randomized, prospective design examining the relationship between trait arousal, mood and stress on the deposition of visceral adipose tissue over 12 months. The study used well-validated self-report measures to assess trait arousal, depressive symptomatology, major and minor stress, and dual-energy X-ray absorptionmetry and computer-assisted tomography to assess visceral adiposity. Table 1 illustrates the independent, dependent and potential control variables assessed in the current study.

Hypotheses

1. It was hypothesized that trait arousal, composite depression, major stress and minor stress scores over 12 months would each be significant predictors of increases in visceral adipose tissue between baseline and 12 months.
2. It was hypothesized that the interaction between arousal, depression and stress would predict more variance in the deposition of visceral adipose tissue than any of

the three variables singly or in pairs. Specifically, it was hypothesized that those subjects identified *a priori* as more highly arousable, and who also reported higher depression and stress scores over 12 months would also exhibit significantly larger deposits of visceral fat at 12 months than those subjects exhibiting either lower arousal or lower depression and stress scores during the same period.

3. It was hypothesized that subjects reporting high trait arousal at baseline would also report more subjective distress in response to both major and minor life events reported at baseline.

Table 1

Study Variables

Variable	Assessment Points
Control Variables (Potential Covariates)	
Gender	Baseline
Age	Baseline
Race	Baseline
Tobacco Use	Baseline, 6 and 12 months
Alcohol Intake	Baseline, 6 and 12 months
Hormone Replacement Therapy	Baseline, 6 and 12 months
Physical Activity	Baseline, 6 and 12 months
Predictor Variables (Independent Variables)	
Arousal (APS)	Baseline
Depression (CES-D)	Baseline, 6 and 12 months
Minor Stress (WSI)	Baseline, 6 and 12 months
Major Life Events (LES)	Baseline, 6 and 12 months
Criterion Variable (Dependent Variable)	
Visceral Adipose Tissue (CT)	Baseline and 12 months

METHOD

Subjects

Subjects for the study were obtained from a project conducted at the Pennington Biomedical Research Center entitled Reversal of Early Atherosclerotic Changes by Diet (REACH). The goal of this project was to examine the effects of a dietary intervention on physiological markers of atherosclerotic disease and its progression. We recognize that participants involved in changing their dietary habits would be inappropriate for a study examining body fat deposition, so for this reason subjects used in the current study were members of the control group of the larger project (N = 120; 75 female and 45 male). Participants were healthy men and post-menopausal women between the ages of 45 and 70. Conditions requiring exclusion from the REACH study included: the presence of coronary artery disease; the use of lipid-lowering or antihypertensive medications; diabetes mellitus; uncontrolled hypertension; renal, hepatic, endocrine, or gastrointestinal disease; body mass index > 35; a history of alcohol or drug abuse; a history of eating disorder; the presence of a psychotic disorder or use of antipsychotic or mood-stabilizing medications.

Measures

Demographic Information. Demographic information was obtained via chart review on all subjects, and was collected during the pre-enrollment screening period for the study. Information collected for the current project included: age, gender, marital status, educational level, smoking status and use of hormone replacement therapy.

Body Weight. Subjects were weighed twice on an electronic scale, after overnight fasting wearing a hospital gown, at all assessment points. The average of the two weights was used to determine the final body weight.

Body Mass Index (BMI). Height was obtained via stadiometer during the baseline period. BMI was calculated for each assessment period using the following formula: kg/m^2.

Dual-Energy X-Ray Absorptionmetry (DEXA). Body composition variables were assessed in overnight fasted subjects using a Hologic QDR2000 DEXA scanner (Waltham, MA) at baseline and 12 months. Variables assessed included total body mass, total fat mass, lean body mass, bone mass, trunk fat mass, right arm fat mass, right leg fat mass, left arm fat mass, and left leg fat mass. The variables used in the present analyses were total fat mass (FAT) and trunk fat mass (TRK).

Computerized Tomography (CT). Abdominal fat distribution variables were assessed in overnight fasted subjects using a GE High Speed Advantage CT scanner (GE Medical Systems, Milwaukee, WI) at baseline and 12 months. Scans were performed at the level of the interspace between the forth and fifth lumbar vertebrae (10-mm thick) for determination of total abdominal adipose tissue (TAT), visceral adipose tissue (VAT), and subcutaneous adipose tissue (SAT). Images were stored on digital tape and analyzed at the Pennington Center using the Analyze software package (CNSoftware, Rochester, MN) run on a Sun Sparc 20 workstation (Sun Microsoft, San Jose, CA). The software allows for segmentation of sequential images into adipose and nonadipose tissue pixel values measured in Houndsfield units (HU). The adipose tissue pixel values for each subject were determined using a histogram sampling technique, in order

to decrease error due to volume averaging and scanner drift over time. Total adipose tissue (TAT) was defined as the sum of adipose tissue pixels inside a line tracing of the skin. VAT was segmented by drawing a line around the interior of the peritoneal cavity and summing all adipose tissue pixels within this area. The difference between TAT and VAT represents SAT. A single reader performed all CT image analyses. The variable used in the present analyses was visceral adipose tissue (VAT).

Baecke Physical Activity Questionnaire (Baecke, Burema & Frijters, 1982). Physical activity was assessed with the Baecke physical activity questionnaire at baseline, 6 and 12 months. The measure is a 16-item self-report questionnaire providing 3 semicontinuous indices of the level of physical activity engaged in over the past 2 weeks. Items are rated on a 5-point Likert scale ranging from *Never (1)* to *Always* or *Very Often (5)*. The scale renders four scores, a Work Index (WI), Sport Index (SI), Leisure Index (LI), and a composite Physical Activity Index (PAI), which is calculated by summing the other three indices. Occupational physical activity level is defined as low *(housework, shopkeeping, clerical work, driving, teaching, studying, all occupations with a university education)*, middle *(factory work, plumbing, carpentry, farming)* or high (*sport, dock work, construction work*). Sport physical activity level is classified as low, middle, and high, depending on the average energy expenditure per hour. The sport score is calculated from the intensity of the sport, time per week spent to play sport, and the proportion of the year the sport is performed. In addition, for the two most frequently reported sports, the subject is asked to report the number of months per year and hours per week of participation. Leisure physical activity level is defined as low *(watching television*), middle *(walking, shopping*), or high *(bicycling,*

walking/biking to work). The instrument has demonstrated good test-retest reliability (*r* = .74 to .93), and concurrent validity with aerobic capacity measured by VO_2 max testing (*r* = .52) and with markers of atherosclerosis (carotid intima-medial thickness) (Richardson, Ainsworth, Wu, Jacobs & Leon, 1995; Jacobs, Ainsworth, Hartman & Leon, 1993). The index used in the present analyses was the composite Physical Activity Index (PAI).

Arousal Predisposition Scale (APS; Coren, 1988). The APS was used at baseline to assess trait arousal. The measure is a 12-item empirically derived questionnaire listing common behaviors and self-perceptions, and respondents are asked to rate which response best describes themselves and their behavior. Responses are rated and scored on a 5-point Likert scale ranging from *Never (or almost never) (1)* to *Always (or almost always) (5)*. Norms for the APS are available, and the instrument has been demonstrated to possess good split-half reliability (*r* = .83). Validity studies have demonstrated convergent validity with physiological measures of sleep disturbance (*r* = .45), the Activation-Deactivation Adjective Check List (Thayer, 1967), and both electrodermal and electromyogram measures of arousal. The APS has also been shown to differentiate between high and low arousability in subjects reporting stress-related physical symptoms (Hicks, Conti & Nellis, 1992). The following are example items from the APS: 2) I get flustered if I have several things to do at once; 6) My mood is quickly influenced by entering new places; 10) I startle easily.

Weekly Stress Inventory (WSI; Brantley, Jones, Boudreaux & Catz, 1997). The WSI was used to assess the frequency and impact of minor life events at baseline, 6, and 12 months. The measure is an 87-item questionnaire assessing the frequency and impact of

minor stressors likely to have been experienced in the past week. Items are rated on an 8-point Likert scale ranging from *Did not occur (0*) to *Extremely stressful (7*). The scale renders two scores, an event score (WSI-E), which is the total number of events endorsed, and an impact score (WSI-I), which is the sum of the subjective ratings of distress of the items endorsed. Norms for the WSI are available, and the instrument has demonstrated good internal consistency (α = .92-.97), test-retest reliability (*r* = .80-.83) and concurrent validity with the Daily Stress Inventory (*r* = .77-.84) and the Hassles Scale (*r* = .61-.69) (Kanner, Coyne, Schaefer & Lazarus, 1981). In a recent longitudinal study, psychological assessments across a 6-month period provided a stable indicator of minor stress in a sample of adults recruited from primary care medical clinics (Scarinci, Ames, & Brantley, 1999). The following are example items from the WSI: 6) Hurried to meet a deadline; 16) Ran out of pocket money; 51) Argued with a friend.

Life Experiences Survey (LES; Sarason, Johnson & Siegel, 1978). The LES was used to measure the number and impact of major life events experienced over the past 0-6 months and 7-12 months, and was assessed at baseline, 6, and 12 months. Stress is scored by counting the number of negatively rated events reported and by summing the subjective impact of those events. This 57-item scale instructs subjects to rate item desirability and degree of impact on a 7-point Likert scale ranging from *Extremely negative (- 3)* to *Extremely positive (+ 3)*, and renders three scores: positive, negative, and total. The scale has been demonstrated to possess good psychometric properties, with reliability coefficients ranging from .66 to .88, and coefficients of total change

ranging from .63 to .64. The following are example items from the LES: 1) Marriage; 3) Death of a spouse; 19) Major change in financial status.

Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977). The CES-D was used to assess depressive symptoms at baseline, 6, and 12 months. The scale is a 20-item self-report measure of depressive symptomatology experienced in the past week. Respondents rate the frequency of occurrence of each symptom on a 4-point Likert scale ranging from *Rarely or none of the time (less than one day)* to *Most of the time or all of the time (5-7 days)*. A score of 16 or greater indicates increased risk for the diagnosis of Major Depressive Disorder. Norms for the CES-D are available, and the instrument has demonstrated good internal consistency (α = .85), split-half reliability (r = .85) and concurrent validity with several other measures including the Hamilton Depression Scale and the Beck Depression Inventory (r = .61-.89). In addition, the CES-D was developed for use with community rather than psychiatric samples, and has been used extensively in primary care and community based studies (Brantley, Mehan & Thomas, 2000). The following are example items from the CES-D: 3) I felt that I could not shake the blues, even with help from my family and friends; 8) I felt hopeful about the future; 9) I thought my life had been a failure.

Procedure

Participants for the REACH study were recruited through standard media for a two-year study investigating the effects of a low-fat diet on progression of heart disease. Seven cohorts were recruited beginning in August 1998, and the final cohort completed the study in May 2003.

Informed consent was obtained (see Appendix A), and after enrollment subjects completed a 4-week pre-intervention period, during which baseline assessment data was collected. After the baseline assessment period, subjects were randomized to either a dietary intervention group, or to the no-intervention control group. Participants in the control group did not receive any formal dietary counseling, and were not encouraged to change their dietary habits.

Both the intervention and control groups completed formal data assessments at baseline, 6 and 12 months. Measures used for the current study were psychosocial questionnaires and imaging data collected at baseline, 6 and 12 months.

Statistical Analyses

1. Descriptive statistics for the sample were completed using pre-intervention baseline assessment measures, in order to obtain demographic information, means and standard deviations on all variables.
2. In order to test Hypothesis 1, that trait arousal, depression, major stress and minor stress scores over 12 months would be significant predictors of increases in visceral adipose tissue between baseline and 12 months, CES-D, LES and WSI scores at baseline, 6 and 12 months were standardized and averaged, in order to create single composite depression, major stress and minor stress variables.

b. A change score in the dependent variable, visceral adiposity, was created by subtracting baseline visceral fat from 12-month visceral fat. Negative scores on this variable represented a loss of visceral fat from baseline to 12 months, while positive scores indicated an increase in visceral fat during the same period. The change

score was created in order to control for the potential effect of total adiposity on the analyses.

c. Control variables were then identified by examining the bivariate correlations between potential demographic covariates and visceral adipose tissue. Potential covariates included age, race, gender, smoking status, alcohol intake, level of physical activity and the use of hormone replacement therapy.

d. A correlational matrix was then constructed to determine the bivariate associations between the covariates, the predictor variables (arousal, depression, major stress and minor stress) and the criterion variable (visceral adipose tissue).

e. Arousal and the composite depression, major stress and minor stress variables were then entered into a hierarchical regression analysis to predict visceral adipose tissue. The first step involved the forced entry of the identified control variables, in order to covary out the effects of any demographic predictors of visceral adiposity. The second step included arousal and the composite depression, major stress and minor stress variables.

3. In order to test Hypothesis 2, that the interaction between arousal, depression and stress would predict more variance in the deposition of visceral adipose tissue than any of the three variables singly or in pairs, the third step of the regression analysis was the entry of arousal-depression, arousal-stress, stress-depression and arousal-depression-stress interactions into the regression equation.

b. Any significant differences in visceral adiposity and in the arousal, depression and stress interactions were then evaluated using scatterplots and simple slope analyses. A t-test of the interaction was used to determine which of the simple slopes was

significantly different from zero, in order to identify under what conditions the interaction was significant. Specifically, plots were created by solving the regression equation at one standard deviation above and below the mean for each of the components of the interaction.

4. In order to test Hypothesis 3, that subjects reporting high trait arousal at baseline would also report more subjective distress in response to both major and minor life events reported at baseline, baseline arousal scores were calculated, and the sample divided by a median split into high arousal and low arousal subgroups. The high and low arousal subgroups were then compared for significant differences in baseline major and minor stress with t-tests, using the LES negative scale score and the WSI-Impact score divided by the WSI-Event score.

RESULTS

Descriptive Statistics

Data were analyzed using Statistical Package for the Social Sciences (SPSS), Version 10.1 (SPSS, Inc., 2000). Exploratory data analyses were performed on the initial sample of 120 subjects, in order to identify missing data, invalid data, and subjects lost to follow-up. These cases were then corrected, entered, or eliminated from subsequent analyses, such that subjects retained for further inclusion (n = 95) had complete data on all study variables. Outlying scores were also identified, defined as any score greater than 3 standard deviations from the mean on any of the variables of interest (Hair, Anderson, Tatum & Black, 1998; Licht, 1995; Cone & Foster, 1993). Using this metric, three cases were eliminated as outliers, resulting in a final sample of 92 subjects. One year attrition for the sample was calculated to be 23%. Validity checks were then performed on the complete data for 10% of the subjects in order to ascertain integrity of the data entry procedures. No subjects from the 10% subsample were found to have incorrect, missing or invalid data.

Descriptive statistics were then used to create a profile of the sample based on the following information: gender, race, age, marital status, and education. As reflected in Table 2, 94.6% of the sample was Caucasian, 66.6% were female, 75% were married, 77.2% were college graduates, and the mean age of the sample was 55.74 years.

T-tests were conducted to evaluate statistically significant differences between Caucasians, representing 94.6% of the sample, and African-Americans (5.4%) with regard to all study variables. No significant differences were found, so all remaining subjects were retained in subsequent analyses. In addition, subjects completing the

study were compared to subjects lost to attrition on all study variables. No significant differences were found.

Table 2

Demographic Information for the Selected Sample

Variable	%	*N*	*Mean* (± *SD*)	*Range*
Race				
Caucasian	94.6	87		
African-American	5.4	5		
Gender				
Male	33.7	31		
Female	66.3	61		
Marital Status				
Single	2.2	2		
Married	75.0	69		
Divorced	20.7	19		
Widowed	2.2	2		
Education (Years)		92	16.2 (± 3.36)	12 - 20
Age (Years)		92	55.74 (± 5.87)	45 – 69

Predictors of Visceral Adiposity

Prior to further analysis, the arousal, depression, major stress and minor stress scores were standardized and averaged, in order to create single predictors representing scores on each variable from baseline to 12 months. Similarly, a change score in the criterion variable, visceral adiposity, was created by subtracting baseline visceral fat from 12-month visceral fat. Negative scores on this variable represented a loss of visceral fat from baseline to 12 months, while positive scores indicated an increase in visceral fat during the same period. By creating the change score for visceral adiposity, the potential effect of total adiposity was directly controlled for, and therefore not included in subsequent analyses. Descriptive statistics for each of the variables of interest were then calculated, and are reflected in Table 3. An analysis of weight change in the subjects indicated that 33% of the sample lost weight over the course of

the study, whereas 67% of the sample gained an average of 2.06 kg. (4.53 lbs.) between baseline and 12 months.

Table 3

Descriptive Characteristics of the Study Variables

Variable	*N*	*Mean* (± *SD*)	*Range*
Predictor Variables			
Arousal	119	27.2 (± 6.09)	15 - 42
Depression	105	7.19 (± 6.21)	0 - 34
Minor Stress (# Events)	105	27.3 (± 12.13)	4 - 61
Minor Stress (Impact)	104	56.06 (± 37.36)	9 - 180
Major Life Events (# Events)	92	3.24 (± 2.68)	0 - 10
Criterion Variable			
Visceral Adipose Tissue (HU)	92	6.43 (± 22.48)	- 49.13 – 56.35
Weight Change (kg.)		.64 (± 2.88)	- 10.0 – 10.0
Gained Weight (kg.)	62	2.06 (± 2.00)	.10 – 10.0
Lost Weight (kg.)	30	-2.12 (± 2.26)	-.10 – -10.0

Variable distributions were then evaluated on all variables in order to detect violations of the normality assumption (Hair, Anderson, Tatum & Black, 1998; Licht, 1995; Cone & Foster, 1993). Significant violations from the normal distribution were detected by inspecting the normal probability plots of the expected normal versus observed values on all variables. Figures 3 and 4 illustrate the raw and transformed distributions for depression in the current analyses. In addition, a statistical test of normality, the Kolmogorov-Smirnov test with Lilliefors significance level, was also examined for each of the variables (Hair, Anderson, Tatum & Black, 1998; Licht, 1995).

The tests for normality indicated that the distributions of the arousal, depression and minor stress variables each deviated significantly from the normal. Inverse, square root, log 10 and natural logarithm transformations were then performed, and the normal probability plots and Kolmogorov-Smirnov statistics re-examined in order to determine

the best data transformation procedure (Hair, Anderson, Tatum & Black, 1998; Licht, 1995). For each of the distributions, a natural logarithm transformation was determined to be the most appropriate.

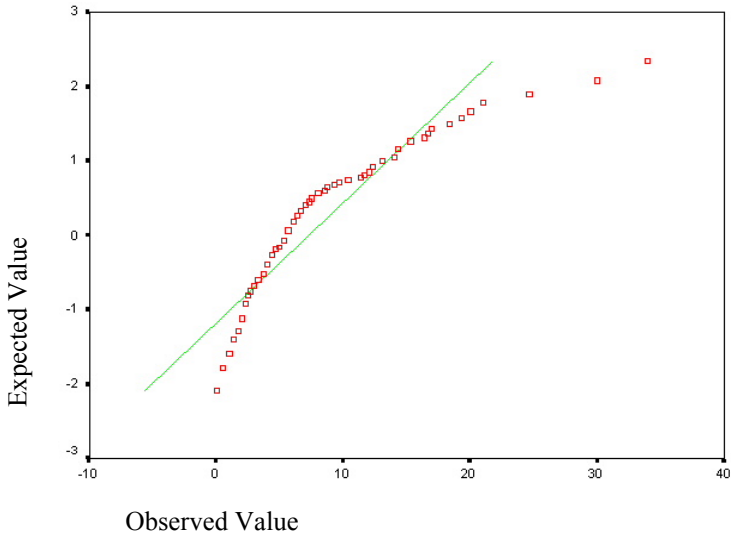

Figure 3. Normal Probability Plot of Raw Depression Scores – Skewed Distribution

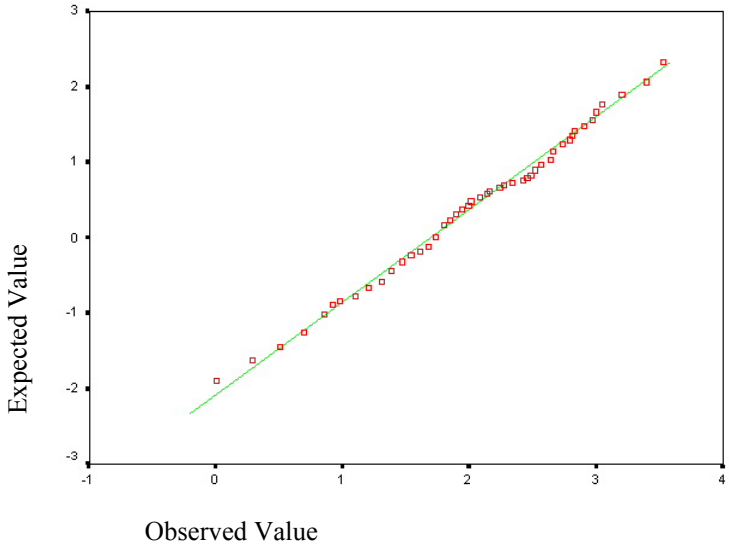

Figure 4. Normal Probability Plot of Transformed Depression Scores – Normal Distribution

Bivariate correlations were then conducted between hypothesized covariates and the criterion variable, in order to determine whether control variables would need to be included in the regression analyses. Hypothesized control variables included gender, age, race, smoking status, alcohol intake, the use of hormone replacement therapy, and level of physical activity. As reflected in Table 4, level of physical activity was the only hypothesized control variable to be significantly correlated with visceral fat (r = -.21, p < .05), suggesting that it should be controlled for in subsequent analyses.

Table 4

Correlations Between Potential Control Variables and Visceral Adipose Tissue (r)

Covariate	Visceral Fat
Gender:	-.15
Age:	.02
Race:	.03
Tobacco:	.03
Alcohol Intake:	-.06
Hormone Replacement Therapy:	-.08
Physical Activity:	-.21*

*p < .05

A correlational matrix of the predictor and criterion variables was then constructed in order to determine whether the creation of composite scores would be necessary in order to control for multicollinearity among the predictor variables. Bivariate correlations between the predictor variables and visceral adiposity are presented in Table 5.

As shown in Table 5, intercorrelations among the predictor variables ranged from r = -.02 to .41. While the correlations between the depression variable and the arousal and stress variables were statistically significant, intercorrelations below .41 are not interpreted as causing significant violations of the multicollinearity assumption, and

were therefore retained as single variables in subsequent analyses (Tabachnick & Fidell, 2001; Hair, Anderson, Tatum & Black, 1998; Licht, 1995).

Table 5

Correlational Matrix of Predictor and Criterion Variables (*r*)

Variables	PA	APS	DPX	LES	WSI	VAT
Physical Activity (PA):	--	-.15	-.15	.06	-.02	-.21*
Arousal (APS):		--	.31**	-.07	.14	-.16
Depression (DPX):			--	.23*	.41**	.05
Major Stress (LES):				--	.27*	.03
Minor Stress (WSI):					--	.16
Visceral Fat (VAT):						--
					*$p < .05$	**$p < .01$

In order to test Hypothesis 1, that trait arousal, depression, major stress and minor stress scores over 12 months would predict increases in visceral adipose tissue between baseline and 12 months, a hierarchical multiple regression analysis was performed to analyze the contribution of the predictor variables on visceral adiposity. In Step 1, physical activity was entered into the equation as a covariate, and the model was significant, accounting for 7.9% of the variance [F (1, 77) = 6.57, $p < .01$]. In Step 2, arousal, depression, major stress and minor stress were entered, and the model continued to be significant, accounting for 16.9% of the variance [F (5, 73) = 2.96, $p < .02$]. As hypothesized, the results of the analysis indicated that the primary predictors of arousal, depression, major and minor stress explained significant variance ($\Delta R^2 = .09$) beyond that accounted for by physical activity. The results of this initial regression are reflected in Table 6.

Contrary to what was hypothesized, only physical activity ($\beta = -.28$, $p < .01$) and arousal ($\beta = -.31$, $p < .01$) were significant predictors of visceral adiposity, independent

of the contributions of stress and depression. Depression and stress, while not significant independent predictors, were in the expected direction. Therefore, in order to test the possibility that a more parsimonious model would explain the results, a second regression analysis was constructed using only physical activity and arousal as predictor variables. This model was found not to be significant [F (2, 89) = 2.41, $p <$ .12], so the original model containing each of the four primary predictor variables was re-constructed and tested further. The final model for Steps 1 and 2 is illustrated graphically in Figure 5.

Table 6

Hierarchical Regression Analysis, Steps 1 and 2

			Results in Final Step	
Step and Predictor Variable (n = 78)	R^2	ΔR^2	B	β
Step 1 (Covariate)	.079**	.079**		
Physical Activity			-6.49**	-.32**
Step 2 (Predictors)	.169*	.090*		
Arousal			-10.95**	-.31**
Depression			3.33	.12
Major Stress			.51	.06
Minor Stress			4.85	.14

*$p < .05$ **$p < .01$

In order to test for violations of the statistical assumptions relating to multivariate multiple regression, the residual plots of observed versus predicted values for the dependent variable were then analyzed, as recommended by Hair et al. (1998). Examination of standardized residual plots for the entire model as well as each of the partial regression plots were null, thus indicating that the analysis met the assumptions of lack of multicollinearity, linearity of the relationship, and constant variance of the error term (homoscedasticity), both with regard to the individual predictor variables and

the variate. The standardized residual plot for the entire model is represented in Figure 6.

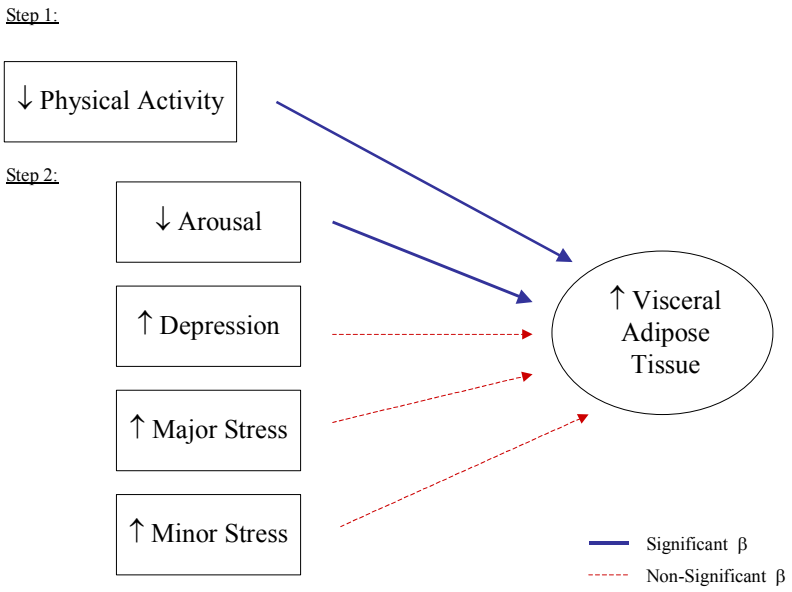

Figure 5. Hierarchical Regression Analysis, Steps 1 and 2

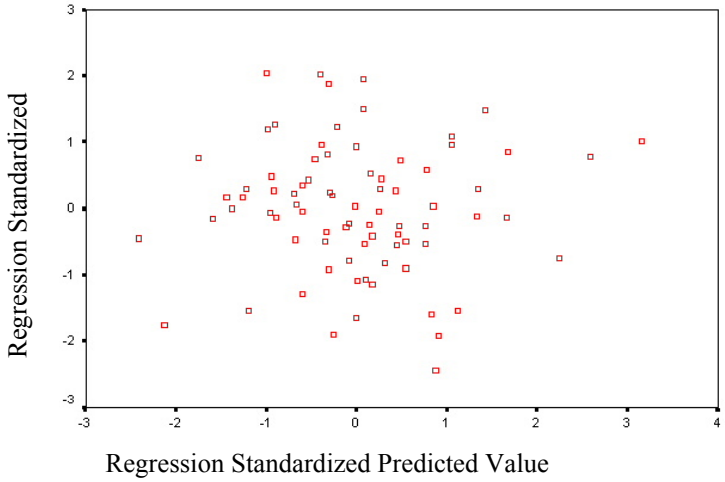

Figure 6. Standardized Residual Plot

The final statistical assumption of multivariate multiple regression, normality of the error term distribution, was then tested by an examination of the normal probability

plot of the observed versus expected values of the standardized residuals. As illustrated in Figure 7, the normal probability plot revealed that the distribution of the error terms did not deviate significantly from normal.

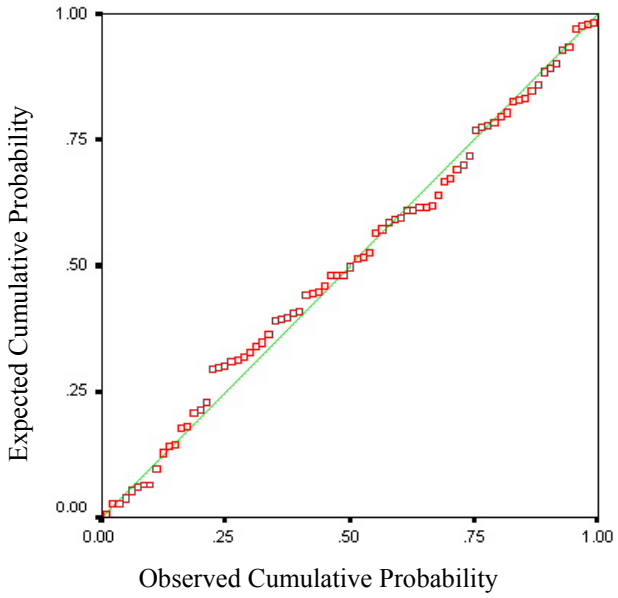

Figure 7. Normal Probability Plot of Standardized Residuals

Interactions of Predictors of Visceral Adiposity

In order to test Hypothesis 2, that the interaction between arousal, stress and depression would be predictive of more variance in the deposition of visceral adipose tissue than any of the three variables singly or in pairs, interaction terms representing the two and 3-way interactions were created. These interaction terms were created by centering the individual variables and multiplying them together to create two-way interaction terms between arousal-depression, arousal-stress and depression-stress, and a 3-way interaction term between arousal-depression-stress (Aiken & West, 1991). Centering is done in order to place each of the predictor variables on a common metric, and is performed by subtracting the mean from each individual variable score to

produce variables with a mean of zero (Aiken & West, 1991). Prior to analysis, the presence or absence of multivariate outliers was determined based on the Mahalanobis Distance, and no multivariate or univariate outliers were identified (Tabachnick & Fidell, 2001; Aiken & West, 1991). Bivariate correlations between the interaction variables and visceral adiposity are presented in Table 7.

Table 7

Correlational Matrix of Interaction and Criterion Variables (*r*)

Variables	AD	AS	DS	ADS	VAT
Arousal-Depression	--	.51**	.29**	.32***	-.14
Arousal-Stress		--	.41**	.29**	-.01
Depression-Stress			--	.16	-.12
Arousal-Depression-Stress				--	.11
Visceral Fat (VAT):					--
				*$p < .05$	**$p < .01$

The regression model was then constructed again, with the interaction terms entered as Steps 3 and 4. In Step 3, the two-way interaction terms between arousal-depression, arousal-stress, and depression-stress were entered, and the model was not significant [F (8, 70) = 1.90, $p < .07$]. In Step 4, the arousal-depression-stress interaction was entered, and the model was significant, accounting for 20.9% of the variance [F (9, 69) = 2.03, $p < .05$].

Contrary to what was hypothesized, the two-way interactions were not significant. However, the 3-way interaction between arousal-depression-stress did explain significant variance ($\Delta R^2 = .04$) beyond that accounted for by the individual predictor variables. The results of Steps 3 and 4 of the regression model are reflected in Table 8.

Table 8

Hierarchical Regression Analysis, Steps 3 and 4

Step and Predictor Variable	R^2	ΔR^2	Results in Final Step	
			B	β
(*n* = 78)				
Step 3 (2-way interactions)	.179	.010		
Arousal-Depression			-2.62	-.13
Arousal-Stress			-.26	-.02
Depression-Stress			-.95	-.07
Step 4 (3-way interaction)	.209*	.030*		
Arousal-Depression-Stress			2.17*	.22*

*$p < .05$

As reflected above, the arousal-depression-stress interaction (β = .22, $p < .05$) was a significant predictor of visceral adiposity independent of the contributions of the other single predictors and interactions. However, contrary to Hypothesis 2, this interaction did not account for more variance in the model than the individual predictor variables. Steps 3 and 4 of the model are illustrated graphically in Figure 8.

In order to test for violations of statistical assumptions relating to the new regression model, the residual and normal probability plots of the observed versus predicted values for the dependent variable were again analyzed, and no violations of statistical assumptions were identified.

The interaction between arousal-depression-stress was then examined using scatterplots and simple slope analyses. A t-test of the interaction was used to determine which of the simple slopes was significantly different from zero, in order to identify under what conditions the interaction was significant. Specifically, plots were created by solving the regression equation at one standard deviation above and below the mean for each of the components of the arousal-depression-stress interaction (Aiken & West, 1991).

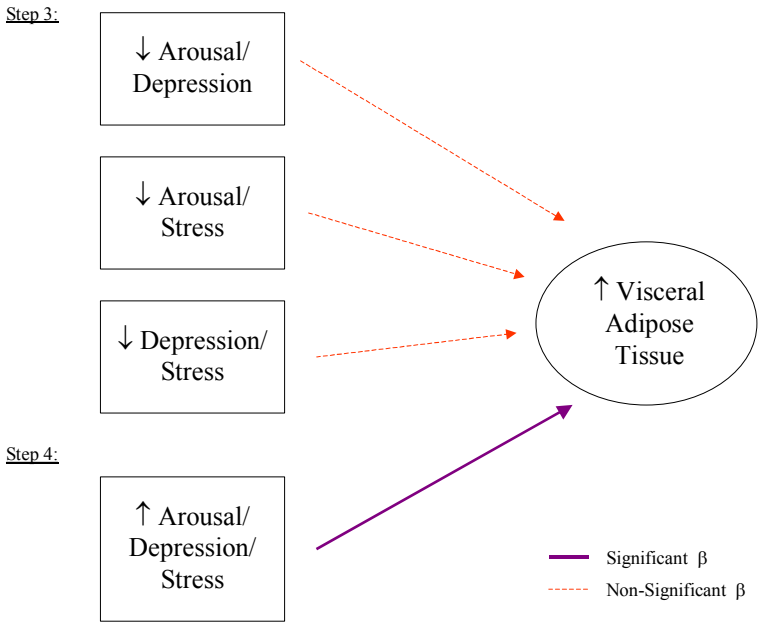

Figure 8. Hierarchical Regression Analysis, Steps 3 and 4

These analyses revealed that the interaction was significant at lower levels of arousal, and higher levels of depression and stress $M = 1.75$ ($SD = .75$) versus $M = 2.27$ ($SD = .84$), $t = -2.47$ ($p = .02$). The direction of the interaction is reflected in the bivariate correlations between each of the single predictors at high and low levels of arousal (produced by median split) (Table 9). The arousal-depression-stress interaction is represented graphically in Figure 9.

Power Analysis

A power analysis was performed in order to ensure adequate power for the primary analyses using the statistical software program, Power and Precision (Biostat, Inc., 2001), and was calculated as follows:

1. The model accounts for inclusion of 1 covariate, which yielded an R^2 of .08, in order to account for a small to medium effect size.

Table 9

Correlations Between Predictors at High and Low Levels of Arousal (*r*)

Variables	APS	DPX	STR
Low Arousal Condition			
Arousal	--	-.01	-.02
Depression		--	.65**
Stress			--
High Arousal Condition			
Arousal	--	.35	.22
Depression		--	.34
Stress			--

*$p < .01$

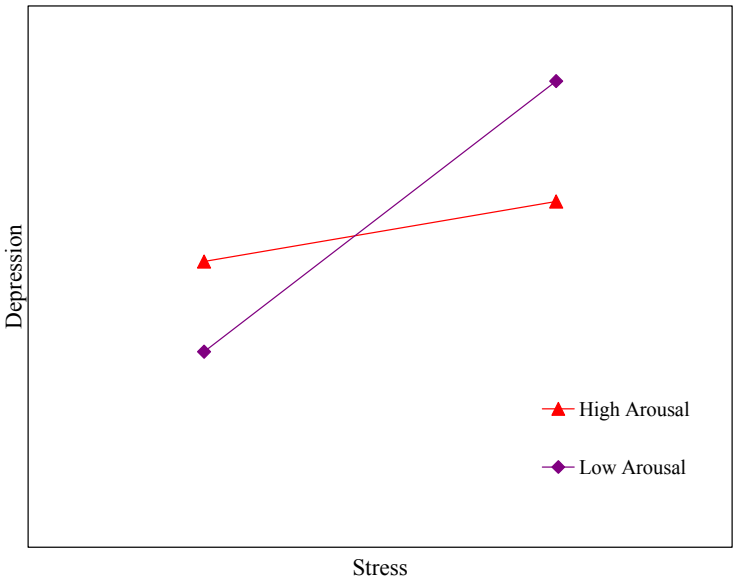

Figure 9. Interaction Between Arousal, Depression and Stress

2. The model also included 4 variables in the set of interest, which yielded an increment of .09, in order to detect a medium effect size in testing Hypothesis 1. The variables in the set of interest are the 4 primary predictor variables (arousal, depression, major stress, minor stress).

3. Finally, the model included 4 interaction variables, which yielded an increment of .04, in order to detect a small effect size in testing Hypothesis 2. The total R^2 for the 16 variables in the model was .21.
4. With the given sample size of 78 and alpha set at .05, the study had power of .86.

The test is based on Model 2 error, which means that variables entered into the regression equation subsequent to the set of interest served to reduce the error term in the significance test, and therefore were included in the power analysis.

Relationship Between Arousal and Stress

In order to test Hypothesis 3, that subjects reporting high trait arousal at baseline would also report more subjective distress in response to both major and minor life events reported at baseline, the sample was divided by median split into high and low arousal subgroups. In order to calculate an average intensity rating for minor stress, WSI-Impact scores were divided by WSI-Event scores for each subject. In order to calculate an average intensity rating for major stress, the LES Negative Impact scale score was used. The high and low arousal subgroups were then compared for significant differences in LES and WSI average intensity scores using t-tests.

As hypothesized, the results indicated that subjects reporting higher arousal scores also reported higher levels of minor stress, $M = 2.11$ ($SD = .78$) versus $M = 1.73$ ($SD = .64$), $t = 2.60$ ($p = .01$). However, contrary to prediction, while high arousal subjects tended to report higher levels of major stress, the differences between the groups was not significant, $M = 2.43$ ($SD = 3.23$) versus $M = 1.45$ ($SD = 2.80$), $t = 1.48$ ($p = .14$).

Secondary Analyses

In order to more fully examine the relationship between weight gain, arousal, depression and stress, post-hoc secondary analyses were performed on several other dependent variables of interest. It was hypothesized that if the relationships observed in the analysis of visceral fat were replicable, and not simply artifacts of this particular analysis or sample, other variables should also reflect the influence of the predictor variables on weight change. Other available variables within this sample included total body weight, body mass index, total fat mass and trunk fat mass.

All analyses previously conducted using visceral fat were repeated using the additional dependent variables of interest. Bivariate correlations between the previously hypothesized covariates and each of the new criterion variables indicated that age was significantly correlated with each of the dependent variables. Because no other hypothesized control variable was significantly correlated, age was controlled for in all subsequent analyses. Descriptive statistics for the covariate and each of the new criterion variables were then calculated, and are reflected in Table 10.

Table 10

Descriptive Characteristics - Secondary Analyses

Variable	*N*	*Mean* (± *SD*)	*Range*
Covariate			
Age (Years)	120	55.43 (± 5.99)	45 - 70
Criterion Variables			
Body Weight (kg.)	97	.64 (± 2.88)	- 10.0 - 10.0
Body Mass Index	93	.24 (± .83)	- 2.79 - 2.49
Total Fat Mass (kg.)	97	.40 (± 2.33)	- 5.56 - 7.9
Trunk Fat Mass (kg.)	95	.13 (± 1.43)	- 2.8 - 3.33

Change scores for each of the new dependent variables were then created by subtracting the baseline score from the 12-month score. Positive scores on these variables represented increases in weight, body mass index, total body fat and trunk fat from baseline to 12 months, while negative scores indicated decreases during the same period.

Variable distributions for the covariate and each of the criterion variables were then examined using normal probability plots and Kolmogorov-Smirnov statistics. Significant deviations from the normal were detected in the distributions for age, total body weight, and body mass index. Transformation procedures were then used to correct the distributions via the most appropriate method. Age was corrected by using the inverse transformation, and body weight was corrected using the log 10 transformation. No transformation procedure was able to produce a better distribution for body mass index, so this variable was not transformed, and subsequently analyzed with a slightly positively skewed distribution.

A correlational matrix of the predictor and criterion variables was then constructed, and is presented in Table 11.

Table 11

Correlational Matrix of Predictor and Criterion Variables – Secondary Analyses (*r*)

Variables	AGE	APS	DPX	LES	WSI	WT	BMI	FAT	TRK
Age (AGE):	--	-.02	.07	.23*	.23*	.38**	.29**	.21*	.22*
Arousal (APS):		--	.31**	-.07	.14	.00	-.29**	-.12	-.08
Depression (DPX):			--	.23*	.41**	.06	.10	.07	-.03
Major Stress (LES):				--	.27*	.23	.02	.05	.14
Minor Stress (WSI):					--	.19	.09	.22*	.22*
Total Body Weight (WT):						--	--	--	--
Body Mass Index (BMI):							--	--	--
Total Body Fat (FAT):								--	--
Trunk Fat (TRK):									--

*$p < .05$ **$p < .01$

Secondary Regression Analyses

Hierarchical multiple regression analyses were then performed in order to analyze the contributions of age, arousal, depression, major stress and minor stress on the additional dependent variables of interest (total body weight, body mass index, total body fat and trunk fat). For ease of interpretation, these secondary regressions will be presented individually.

To assess the contribution of the predictor variables on total body weight, a regression model was constructed and age was entered into the equation as a covariate at Step 1. The model was significant at this step, accounting for 14.6% of the variance [F (1, 57) = 9.72, $p < .003$]. In Step 2, arousal, depression, major stress and minor stress were entered, and the model continued to be significant, accounting for 19.9% of the variance [F (4, 53) = 2.64, $p < .03$]. The results of this analysis indicated that the primary predictors of arousal, depression and stress explained significant variance (ΔR^2 = .05) beyond that accounted for by age. Of these primary predictor variables, only age (β = .37, $p < .01$) was a significant predictor of body weight, independent of the contributions of arousal, depression and stress. Arousal, depression and stress, while not significant independent predictors, were each in the same direction as in the previous analyses for visceral fat.

In Step 3, arousal-depression, arousal-stress and stress-depression two-way interaction terms were entered, and the model continued to be significant, accounting for 26.6% of the variance [F (8, 50) = 2.27, $p < .04$]. In Step 4, the arousal-depression-stress 3-way interaction was entered and the model was no longer significant [F (9, 49) = 2.00, $p < .06$]. Because the model was not significant with the inclusion of the 3-way

interaction, this step was then dropped from the analysis in order to create the most parsimonious model. In addition, none of the interaction terms were significant independent contributors to the model, and therefore were not examined further. The results of the final model are presented in Table 12.

Table 12

Regression Analysis for Body Weight

			Results in Final Step	
Step and Predictor Variable (n = 58)	R^2	ΔR^2	B	β
Step 1 (Covariate)	.146**	.146**		
Age			72.82*	.37*
Step 2 (Predictors)	.199*	.054*		
Arousal			-.12	-.19
Depression			3.27	.06
Major Stress			1.95	.01
Minor Stress			.11	.18
Step 3 (2-way interactions)	.266*	.067*		
Arousal-Depression			.12	.30
Arousal-Stress			-1.76	-.08
Depression-Stress			-2.96	-.12

*$p < .05$ **$p < .01$

To assess the contribution of the predictor variables on body mass index, a regression model was constructed and age was entered into the equation as a covariate in Step 1. The model was significant at this step, accounting for 9.5% of the variance [F (1, 79) = 8.27, $p < .005$]. In Step 2, arousal, depression, major stress and minor stress were entered, and the model continued to be significant, accounting for 19.4% of the variance [F (5, 75) = 3.62, $p < .005$]. The results of this analysis indicated that the primary predictors of arousal, depression and stress explained significant variance (ΔR^2 = .10) beyond that accounted for by age. Of these primary predictor variables, both age (β = .38, $p < .001$) and arousal (β = -.37, $p < .001$) were significant predictors of body mass index, independent of the contributions of depression and stress. Depression and

stress, while not significant independent predictors, were in the same direction as in the previous analyses.

In Step 3, arousal-depression, arousal-stress and stress-depression two-way interaction terms were entered, and the model continued to be significant, accounting for 30.4% of the variance [F (8, 72) = 3.93, $p < .001$]. In Step 4, the arousal-depression-stress 3-way interaction was entered and the model continued to be significant [F (9, 71) = 2.46, $p < .001$]. However, because the inclusion of the 3-way interaction added only negligible variance to the prediction ($\Delta R^2 = .001$), this step was dropped from the analysis in order to create the most parsimonious model.

One interaction term, the depression-stress interaction, contributed significant variance to the model independent of the covariate, single predictors and other interactions ($\beta = .22$, $p < .04$). Further examination of this interaction revealed that the interaction between stress and depression was a significant independent contributor to increases in body mass index when both stress and depression were high. The results of the final model are presented in Table 13.

To assess the contribution of the predictor variables on total body fat, a regression model was constructed and age was entered into the equation as a covariate at Step 1. The model was significant at this step, accounting for 4.9% of the variance [F (1, 91) = 4.72, $p < .04$]. In Step 2, arousal, depression, major stress and minor stress were entered, and the model was not significant [F (5, 76) = 1.66, $p < .15$]. An examination of the beta weights and partial correlations revealed that major stress appeared to be acting as a suppressor variable in the analysis, so the regression was reconstructed with major stress left out of the analysis (Hair, Anderson, Tatum & Black,

1998). For this model, Step 2 was now significant, accounting for 10.3% of the variance [F (4, 88) = 2.52, $p < .05$].

Table 13

Regression Analysis for Body Mass Index

Step and Predictor Variable (n = 80)	R^2	ΔR^2	Results in Final Step B	β
Step 1 (Covariate)	.095**	.095**		
Age			150.46***	.38***
Step 2 (Predictors)	.194**	.100**		
Arousal			-.49***	-.38***
Depression			3.64	.04
Major Stress			2.84	.10
Minor Stress			3.81	.03
Step 3 (2-way interactions)	.304***	.109***		
Arousal-Depression			-.14	-.17
Arousal-Stress			-6.58	-.18
Depression-Stress			.18*	.23*

*$p < .05$ **$p < .01$ ***$p < .001$

The results of this analysis indicated that the primary predictors of arousal, depression and minor stress explained significant variance ($\Delta R^2 = .05$) beyond that accounted for by age. Of these primary predictor variables, only arousal (β = -.24, $p < .05$) was a significant predictor of total body fat, independent of the contributions of age, depression and minor stress. Depression and minor stress, while not significant independent predictors, were in the same direction as in the previous analyses.

In Step 3, arousal-depression, arousal-minor stress and depression-minor stress two-way interaction terms were entered, and the model was no longer significant [F (7, 85) = 1.70, $p < .12$]. In Step 4, the arousal-depression-minor stress 3-way interaction was entered and the model was also no longer significant [F (8, 84) = 1.83, $p < .08$]. Because the model was not significant with the inclusion of the two and 3-way

interactions, these steps were dropped from the analysis in order to create the most parsimonious model. The results of the final model are presented in Table 14.

Table 14

Regression Analysis for Total Body Fat

Step and Predictor Variable (n = 92)	R^2	ΔR^2	Results in Final Step B	β
Step 1 (Covariate)	.049*	.049*		
Age			210.92	.17
Step 2 (Predictors)	.103*	.054*		
Arousal			-.94*	-.24*
Depression			3.13	.01
Minor Stress			.40	.16

*p < .05

To assess the contribution of the predictor variables on trunk fat, a regression model was constructed and age was entered into the equation as a covariate at Step 1. The model was significant at this step, accounting for 5.4% of the variance [F (1, 92) = 5.30, p < .02]. In Step 2, arousal, depression, major stress and minor stress were entered, and the model was not significant [F (5, 74) = 1.12, p < .36]. An examination of the beta weights and partial correlations revealed that both major stress and depression appeared to be acting as suppressor variables in the analysis, so the regression was re-constructed with these predictors left out of the analysis (Hair, Anderson, Tatum & Black, 1998). For this model, Step 2 was now significant, accounting for 9.4% of the variance [F (3, 90) = 3.12, p < .03].

The results of this analysis indicated that the primary predictors of arousal and minor stress explained significant variance (ΔR^2 = .04) beyond that accounted for by age. Of these primary predictor variables, both age (β = .19, p < .06) minor stress (β = .19, p < .06) approached significance as independent predictors of trunk fat. Arousal

and minor stress, while not significant independent predictors, were in the same direction as in the previous analyses.

In Step 3, the arousal-minor stress two-way interaction term was entered, and the model continued to be significant, accounting for 9.8% of the variance [F (4, 89) = 2.43, $p < .05$]. However, because the inclusion of the two-way interaction added only negligible variance to the prediction ($\Delta R^2 = .004$), this step was dropped from the analysis in order to create the most parsimonious model. The results of the final model are presented in Table 15.

Table 15

Regression Analysis for Trunk Fat

			Results in Final Step	
Step and Predictor Variable	R^2	ΔR^2	B	β
(n = 92)				
Step 1 (Covariate)	.054*	.054*		
Age			144.58†	.19†
Step 2 (Predictors)	.094*	.094*		
Arousal			-.26	-.11
Minor Stress			.40†	.19†

*$p < .05$ †$p < .06$

In order to test for violations of statistical assumptions relating to the additional regression models, the residual and normal probability plots of the observed versus predicted values for each of the dependent variables were analyzed. No violations of statistical assumptions were identified for body weight, total body fat, or trunk fat. However, as expected, the normal probability plot for body mass index indicated that this variable had a slightly positively skewed distribution. Based on the robustness of the hierarchical multiple regression statistical test to minor violations of the normality

assumption, it was determined that the analysis for body mass index would be retained, rather than omitted (Cohen & Cohen, 1983).

DISCUSSION

The purpose of the current study was to examine the relationships between the psychological variables of arousal, depression and stress on the deposition of visceral adipose tissue, while controlling for various demographic variables. The study was conceptualized on Bjorntorp's theory of "Civilization Syndrome", and based on previous research suggesting a relationship between the psychosocial variables and adverse changes in body composition. The study was a significant contribution to the literature in that it was the first prospective study to attempt to test the specific components of Bjorntorp's theory in a human sample. The present study also substantially improved upon both the methods and measures which had been utilized in previous studies in this area, by using more sophisticated statistical procedures, valid and reliable measures of the psychological variables, and precise measures of body composition.

Specifically, this study examined the relationship between stress, depression, arousal and the deposition of visceral adipose tissue in a sample of middle-aged males and females. The results were found to be surprising, however, in that they only partially supported the original hypotheses. Specifically, the individual predictors of arousal, stress and depression did not significantly and independently predict visceral fat, apart from the other primary predictor variables. However, the entire model of arousal, stress and depression was significant in predicting visceral fat, and within this model, arousal was a significant independent predictor variable. Unexpectedly, arousal was found to negatively correlate with visceral fat, whereas the original presumption theorized that this relationship would be positive.

Similarly, the hypothesis that the two and 3-way interactions between the variables would each be significant, and would account for more variance than the individual predictors, was also not completely supported. Rather, neither the two-way interactions nor the entire model at this step were significant. However, inclusion of the 3-way interaction between arousal, stress and depression was significant, both as an independent predictor variable, and for the entire model. Examination of this interaction revealed that it significantly predicted visceral fat when arousal was low, and stress and depression were high. This finding was contrary to the *a priori* hypothesis, that the interaction would be significant when arousal, stress and depression were all high. In addition, this interaction did not account for more variance than the original predictors, as initially hypothesized.

Likewise, the third hypothesis, that subjects reporting high arousal would also report more subjective distress in response to both major and minor stress, as measured by the average intensity ratings for each, was partially supported. Specifically, the differences between the high and low arousal subjects were significant with regard to minor stress, but not for major stress.

These results, while unexpected, are not entirely unexplainable. The most plausible and likely explanation for both the magnitude and direction of the results is the restricted range that was observed for all of the psychological variables. Most notably, a severely restricted range was observed in the scores for arousal, depression, major stress and minor stress, suggesting that the subjects in this study were either underreporting their psychological symptoms, or were comprised of a biased sample of unusually healthy subjects.

Fundamentally, the mean arousal score for the present sample was 27.2, whereas the mean arousal score for the normative sample was 36.1. The arousal scores for the current sample translate to a difference of 3.4 standard deviations below the mean of the normative sample. The mean of the "high" arousal group in this study was 32.1 (1.5 standard deviations below the normative mean) and the mean of the "low" group was 22.3 (5.3 standard deviations below the normative mean). This range of scores indicates that the *a priori* hypothesis that, "highly arousable subjects who report more stress and depression over 12 months will also deposit more visceral fat" was never adequately tested in this sample. There were no "highly arousable" subjects in this sample, which in previous studies was defined as arousal scores greater than 44 (Coren & Mah, 1993; Hicks, Conti & Nellis, 1992). Therefore, the scores on the arousal scale were such that in the arousal subgroup analysis, the "high" arousal group had scores reflective of low arousal, and the "low" arousal group had scores reflective of extremely low arousal.

The depression scores show a similar restriction of range in this sample, such that the mean in the present study was 7.2, which is not quite one standard deviation below the normative mean of the scale, which is 11.3 (Radloff, 1977). Mean minor stress-impact scores in the present sample were 56.1, whereas 86.6 was the normative mean, a difference of approximately one-half a standard deviation (Brantley, Jones, Boudreaux & Catz, 1997). Likewise, the mean major stress score for the present sample was 3.2, which was once again approximately one-half a standard deviation below the normative mean of 6.6 (Sarason, Johnson & Siegel, 1978).

This restriction of range among all the psychological variables within the study strongly suggests that the hypothesized relationships between the variables were not adequately tested in the present sample. It is possible that in a sample of subjects with more normal ranges of scores on arousal, stress and depression, the relationships would have been stronger, and stress and depression would have been independent predictors, as they had been in a previous sample (Rhode et al., 2003).

One can speculate as to the reason for this severe restriction of range. However, a possible explanation is that the recruiting procedures implemented for the larger REACH study excluded the subjects with more normative scores on the psychological measures. Because the REACH project was testing the effects of a dietary intervention on pre-morbid markers of cardiovascular disease, they were interested in including subjects who were healthy. It is possible that the recruiters and screeners for this project included only subjects who were unusually healthy from a psychological standpoint. For example, conditions requiring exclusion from the REACH study included: the presence of coronary artery disease; the use of lipid-lowering or antihypertensive medications; diabetes mellitus; uncontrolled hypertension; renal, hepatic, endocrine, or gastrointestinal disease; body mass index > 35; a history of alcohol or drug abuse; a history of eating disorder; the presence of a psychotic disorder or use of antipsychotic or mood-stabilizing medications. While these exclusion criteria do not directly target depression and stress, both the medical and psychological conditions requiring exclusion are known to have high rates of co-morbidity with both depressive and stress-related disorders. Therefore, it is possible that the subjects who were excluded from the REACH study for the above reasons would also have been

those who had more normative levels of stress and depression, and who, in retrospect, would have been more appropriate and interesting to examine for the present study.

The restriction of range on the arousal variable may also explain why lower arousal, rather than higher arousal, was associated with both higher levels of stress and depression, and was predictive of gains in visceral adipose tissue. It could be possible that people who reported high arousal in this study, but who in absolute terms actually had low levels of arousal, were accurately reporting relatively modest levels of stress and depression. Since these subjects were below the normative mean on all three variables, it would follow that their stress and depression scores were not high enough to produce the fat deposition effect hypothesized to occur in subjects with higher levels of stress and depression.

Similarly, those subjects who reported low arousal in this study, but who in absolute terms actually had extremely low levels of arousal, may have had higher levels of stress, depression and subsequent fat deposition because they represent a subgroup of people whose normal physiological state is so deactivated that they do not as easily cope with normal psychological distress, such as stress and depression. This idea is echoed in Hans Eysenck's three-factor theory of personality, and is hypothesized to occur in individuals who demonstrate very low levels of "emotionality", the term he used to describe people who have a predisposition to react bodily, or physiologically, to stressful events (Eysenck, 1967). In the current study, the concept of "arousability" assessed by the Arousal Prediposition Scale is very similar to the construct of "emotionality" described by Eysenck. Specifically, Eysenck's theory posits that emotionality can serve as a drive which motivates behavior, and that people who are

either very low or very high on this trait may possess less-than-optimal emotional and behavioral repertoires in dealing with stressors (Eysenck, 1967).

Applied to the current study, the subjects extremely low in arousal may be avoidant, or vegetative, to a degree that they actually experience *higher* levels of stress and depression than subjects with both more normal levels of arousal and more typical emotional and behavioral repertoires. For example, it is well documented that optimal state arousal has a bell-shaped curve, such that both too little arousal and too much arousal hinders task performance (Yerkes & Dodson, 1908). Perhaps it is possible that trait arousal also has a bell-shaped curve, and that mid, rather than high or low, levels of arousal produce optimal responsiveness to psychological distress. That would explain why in this study, low arousal was independently predictive of fat gain, and why the subjects with the lowest levels of arousal also had the highest levels of stress and depression.

Such a relationship would also explain the results of Hypothesis 3, that the subjects reporting "high" arousal reported higher average intensity ratings in response to minor stressors, but yet the subjects reporting "low" arousal reported higher absolute numbers of stressors. This relationship suggests that the subjects in the "high" arousal group reported more congruence between the number of minor stressors experienced and their impact, whereas the subjects in the "low" arousal group reported more stressful events, but a lower impact. The implication is that the subjects in the more normative "high" arousal subgroup more accurately assessed the relationship between stressors and their impact than the subjects in the "low" arousal subgroup.

The secondary analyses performed on the additional dependent variables of change in body weight, body mass index, total fat mass and trunk fat mass were very similar to the findings from the analyses using the primary outcome measure of visceral fat. Most interestingly, the relationships between stress and depression in these analyses were in the same directions, and were of similar magnitudes as in previous samples (Rhode et al., 2003).

While the results of the secondary analyses provide convergent evidence that the relationships between the predictor and criterion variables in the current sample represent true relationships, the same questions regarding the direction of the results apply. In addition, while it is true that the magnitude of the results of the body weight and body mass index analyses were more congruent with findings from previous samples, it is possible that this may be an artifact of the imprecise nature of both body weight and body mass index as indicators of body composition. This is feasible because the magnitude of the results of the total fat mass and trunk fat mass analyses, which both used DEXA imaging to assess the dependent variables, were more similar in terms of effect size to the findings from the analyses of visceral fat, which used CT data. This suggests that both the current and previous results reported using more imprecise measures of body composition, such as body weight and body mass index, may reflect larger effect sizes due to unaccounted for measurement error.

In addition, the finding that major stress appeared to *suppress* the relationship between arousal, depression and minor stress in the total fat mass analysis, and that both major stress and depression were suppressor variables in the trunk fat analysis, is inconsistent with the results of the analyses for visceral fat. These findings suggest that

the relationships between the predictor and criterion variables may be different at varying levels of stress, depression and arousal, or different for varying measures or types of body composition. For example, arousal and minor stress alone may be salient in predicting more global measures of body fat such as total fat and trunk fat, but both depression and major stress may be needed when predicting more specialized areas of fat deposition, such as the central visceral area. Therefore, it is possible that the results of these analyses could be influenced by imprecisely classified predictor variables or unaccounted for measurement error.

Overall, the results of both the primary and secondary analyses are somewhat surprising, in light of the previous research in this area. As emphasized in the literature review, the majority of previous studies in this area have not directly examined the variables hypothesized by Bjorntorp to comprise the "Civilization Syndrome" as a mechanism for visceral fat deposition. They have also failed to use longitudinal research designs, which is needed in order to infer causality. Therefore, in these respects, the current study has made both a substantial improvement upon, and contribution to, the existing literature. However, because it is the first endeavor into this research area using both precise measures and a prospective research design, the results of this study should be considered provisional.

The previous research most strongly supports a relationship between the neuroendocrine stress response, as measured in the laboratory, and adverse patterns of body composition, as measured by body weight, body mass index or waist-hip ratio. As previously noted, this relationship has been observed in many populations with known body composition abnormalities, such as individuals with Cushing's Disease (Peeke &

Chrousos, 1995), those being treated with HIV antiretroviral therapy (Renard et al., 1999), subjects with genetic growth hormone abnormalities (Barreto-Filho et al., 2002) Pima Indians (Esposito-Del Puente et al., 1994) and MZ twins discordant for visceral obesity (Mariema et al., 2002).

The evidence for a stress-body composition relationship is less strong in studies using more generalizable life-event measures of stress, and additional psychological indices, such as depression, in samples drawn from normal populations. Whereas several of these studies have reported significant associations between body weight, body mass index or waist-hip ratio and the psychosocial variables examined, the results reported were modest, in that they typically employed only cross-sectional research designs and simple correlational or equality-of-means analyses (Vitaliano et al., 1996; Bell et al., 1998; Epel, 1999; Rhode et al., 2001).

The present study appears to fall between the latter two groups of studies, in that it used more generalizable and valid measures of stress and depression, but a prospective research design, and therefore produced results suggestive of a medium effect size for the role of psychosocial variables in the deposition of body fat. However, the current study is a substantial improvement from previously published reports in many respects. Methodological rigor may be posited as a strength of the current study, in that many of the limitations identified in previous studies were addressed in the present analyses. This study also appears to be unique in its use of a more sophisticated research design and statistical analyses, and in the use of precise measures of body composition.

Nevertheless, the current study has several limitations, most notably, the reliance on exclusively self-report measures of the psychological variables. As such, the definitions of each construct were limited to the domains assessed by each of the corresponding self-report scales. However, its limitations notwithstanding, the use of self-report methodology for assessing psychological constructs remains the most frequently used assessment tool for research purposes. This method can be more objective, reliable and efficient than other forms of assessment; however, utilization of this methodology requires that the instruments used have sound psychometric properties. In fact, for clinical use, the practical difficulties in using other assessment methods can be considerable, and few viable alternatives to the self-report methodology are currently available outside of a research laboratory setting.

Despite the fact that the current study used psychometrically valid and reliable measures of self-reported stress, depression and arousal, the study would have been strengthened with the use of collateral measures of these variables, such as blood or urinary assays of corticosteroid or catecholamine levels, the employment of a dexamethasone response test, or the use of electrodermal or electromyogram measures of arousal. Future studies employing both self-report and direct physiological measures of the variables of interest would improve validity of the results reported, and allow for more direct comparisons between the clinical and basic research studies in this area.

The present study would also have been strengthened by additional observations of the study variables, allowing for a follow-up period beyond 12 months. While the study was the first to investigate these relationships prospectively over one year, it can be argued that 12 months is a comparatively short period of time for the hypothesized

relationships between the psychological variables and physiological outcomes to emerge. It would be plausible, therefore, that future studies examining these relationships over two, three or four years would provide more substantive evidence of the theorized relationships between the variables of interest.

In addition, the magnitude of the effect sizes observed in the current study may reflect limitations in the measures chosen for the study, the use of an inappropriate sample, problems with the theory on which the study was based, or some combination of these factors. For example, the measures used in the current study were not specifically developed based on Bjorntorp's theory. Rather, they were chosen based on their perceived conceptual similarity to the components of the Civilization Syndrome. As such, it was probably unrealistic to expect significant results in all analyses, and particularly across methods.

While the current study supports the notion that the psychological variables of stress, depression and arousal are associated with adverse changes in body composition, the erudition of the mechanism of this effect still remains to be established. Hierarchical regression analysis, while a stronger test of causality than many other statistical tests, particularly when used in a longitudinal design as in the current study, can only infer, rather than unequivocally prove, causality. Future studies employing prospective designs and more sophisticated path analysis statistical procedures will be required for both causality, and Bjorntorp's theory, to be confirmed.

Despite the fact that the results of the present study were somewhat unexpected, they did support the premise that the psychological variables of arousal, stress and depression adversely impact the deposition of visceral adipose tissue. However,

because of the limitations of the current investigation, most notably the restricted range of scores observed for each of the psychological variables, the results of the current study should be considered tentative. Future studies using samples drawn from more normative populations will need to be conducted in order to clarify the interaction between arousal, stress and depression, and to further examine the relationship between these variables and adverse changes in body fat deposition.

In addition, by providing collateral evidence on the mechanism and precise relationships between stress, depression, arousal and body fat deposition, this study has implications for directing further research in the area of overweight and obesity, as well as potential utility in developing clinical interventions. The importance of obesity research has been underscored by the World Health Organization, the National Institutes of Health, and the Centers for Disease Control and Prevention, who have each labeled obesity as a health crisis of epidemic proportions. Therefore, studies targeting both the clinical and basic science aspects of the disorder have become a national research priority. Finally, the importance of further investigations of the biological, behavioral, social and environmental sequelae of obesity and obesity-related disorders can be summed up by the Director of the National Institute of Diabetes, Digestive and Kidney Diseases Allen M. Spiegel, in his April 2003 testimony to the United States Senate,

> "Obesity and its associated diseases result from complex interactions of biologic and environmental factors. The environmental factors include social, demographic and economic changes that encourage people to eat more food than necessary to meet their energy requirements, and discourage physical activity that would increase their energy expenditure. […] We much approach obesity, not as a cosmetic or moral problem, but rather as a health problem. To address this problem, research is vital…"
>
> (NIDDK, 2003)

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APPENDIX A

INFORMED CONSENT FORM

CONSENT TO PARTICIPATE IN A RESEARCH STUDY

Title of Study:

Reversal of Early Atherosclerotic Changes by Diet

What you should know about a research study:

- We give you this consent so that you may read about the purpose, risks and benefits of this research study.
- The main goal of research studies is to gain knowledge that may help future patients.
- You have the right to refuse to take part, or agree to take part now and change your mind later on.
- Please review this consent form carefully and ask any questions before you make a decision.
- Your participation is voluntary.

1. Who is doing the study?

Investigator Information:

Principal Investigator: Michael Lefevre, Ph.D.
(504) 763-2569

Medical Investigator: Steve Smith, M.D.
Day Phone: (504) 763-3028
24-hr. Emergency Phone Nos.:
763-2672 (Weekdays 8:00a.m.-5:00 p.m.)
765-4644 (After 5:00 p.m. and Weekends)

Co-Investigators: Catherine Champagne, Ph.D.
Richard Tulley, Ph.D.
Michael Welsch, Ph.D.

Dr. Michael Lefevre directs this study, which is under the medical supervision of Dr. Steve Smith. This study is being conducted at the Pennington Biomedical Research Center. We expect 500 people will be in this study. The study will take place over a period of 4 years. Your expected time in this study will be 25 months.

2. *Where is the study being conducted?*

This study takes place in the Metabolic Unit at the Pennington Center.

3. *What is the purpose of this study?*

Current recommendations are for everyone above the age of 2 years to consume diets containing 30% or less of calories from total fat and 8 to 10% of calories from saturated fat. However, there are those who advocate that the American Public consume even lower levels of total and saturated fat. While low fat diets have, on average, been shown to slow the progression and development of atherosclerotic plaques in populations with definite coronary artery disease, little if any information is available concerning the effectiveness of these diets in slowing or reversing the progression of atherosclerotic disease in the general population. This study will directly examine the effects of dietary reductions in total fat, saturated fat, and cholesterol on markers of early atherosclerotic disease and on biomarkers of atherosclerotic disease progression.

4. *Who is eligible to participate in the study? Who is ineligible?*

You are eligible for this study if you: 1) are between the ages of 45 and 70, 2) (for females) are at least six months post-menopausal; 3) have normal to moderately elevated blood cholesterol levels; 4) have moderately thickened carotid arteries; and 5) are otherwise in good health.

You are ineligible if you: 1) are not between the ages of 45 and 70; 2) have very low or very high blood cholesterol levels; 3) (for females) have had a menstrual cycle within the last six months; 4) have documented coronary artery disease; 5) have clinically significant carotid artery disease; 6) are on lipid lowering medication; 7) have uncontrolled hypertension; 8) have diabetes; 9) have any other kidney, liver, endocrine, gastrointestinal or other systemic disease which would interfere with your ability to safely participate; and 10) have a recent history of alcohol or drug abuse.

5. *What will happen to you if you take part in the study?*

If selected for this study, you will be assigned to either a control group or to a diet intervention group.

Control Group: If you are assigned to the control group, you will not receive any formal diet instructions other than how to properly record your food intake. However, you will be free to make changes in your dietary habits on your own or on the advice of your physician.

Diet Intervention Group: If you are assigned to the diet intervention group, you will receive intensive dietary counseling consisting of both group and individual sessions aimed at substantially reducing your intake of total fat,

saturated fat and cholesterol, while increasing your intake of complex carbohydrates. The nutritional goals will be to reduce total fat intake to 15% of calories, saturated fat intake to 4% of calories, and cholesterol intake to less than 60 mg/1000 Kcal. Group sessions will occur weekly for two months, and every other week for months 3 and 4. Sessions will be monthly for the remainder of year one and every other month during year two.

Food Intake Records, Food Frequency Questionnaires and 24-hr Diet Recalls: Throughout the course of this study, you will be asked to provide information regarding the types and amounts of food that you consume. This information will be provided in the form of 4-day complete food intake records and food frequency questionnaires. These assessments will occur at baseline and at months 1, 3, 6, 12, 18 and 24. Additionally, you will be called at home unannounced by a study dietitian and asked to provide a listing of the types and amounts of food that you have consumed in the previous twenty-four hours.

Cardiovascular disease risk factor assessments: Throughout the course of this study, you will be asked to provide samples of blood for determination of cardiovascular disease risk factors. These assessments will occur twice at baseline; once at months 1 and 3; and twice at months 6, 12, 18 and 24. The total amount of blood taken on any day will not exceed 60 mls (approximately 2 fluid ounces).

DEXA scan: You will be asked to undergo two DEXA scans to measure your percent body fat. These will occur during baseline and at the end of the study.

Carotid artery ultrasound: Throughout the course of this study, measurements will be taken of the thickness of the lining of your carotid arteries. This will be done non-invasively using an ultrasound machine with the ultrasound probe applied to the side of your neck. This assessments will occur twice at baseline; once at months 6, 12, and 18; and twice at month 24.

Vascular reactivity: Throughout the course of this study, measurements will be taken of how well your arteries respond to changes in blood flow. This test will be conducted on your arm. Using ultrasound, measurements will be taken of the width of your brachial artery in your upper arm before and after five minute occlusion of forearm blood flood with a blood pressure cuff. This assessments will occur twice at baseline; once at months 3, 6, 12, and 18; and twice at month 24.

6. What are the possible risks and discomforts?

Blood sampling: Bruising, bleeding and/or infection at site of needle insertion; possible fainting.

DEXA scan: There is minimal risk associated with exposure to x-ray radiation during the DEXA scan. The amount of radiation exposure is roughly equivalent to eight hours exposure to sunlight.

Carotid artery ultrasound: Possibility of fainting and/or temporary slowing of the heart rate. Remote possibility of carotid plaque destabilization with resulting stroke.

Vascular reactivity: Discomfort in forearm and hand. Temporary numbness and tingling in hand similar in sensation to having your hand "fall asleep".

In addition to the risks listed above, you may experience a previously unknown risk or side effect.

7. What are the possible benefits?

We cannot promise any direct benefits from your being in the study. The knowledge gained during these studies may help individuals in the future through the formulation of dietary recommendation which may reduce overall risk of developing cardiovascular disease. There are no medical benefits to you from your taking part in this study. If you are assigned to the diet intervention group, you will receive information about how to lower dietary fat intake. If you are assigned to the control group, you will not receive any formal diet instructions. All study volunteers will receive information about your blood cholesterol levels and be screened for significant atherosclerosis in your carotid arteries.

8. If you do not want to take part in the study, are there other choices?

There are no alternative procedures available that would involve less risk. However, you have the choice not to participate in this research study.

9. If you have any questions or problems, whom can you call?

If you have any questions about your rights as a research volunteer, you should call the Institutional Review Board Office at (504) 763-2693 or Dr. Claude Bouchard, Executive Director of PBRC at (504) 763-2513. If you have any questions about the research study, contact Dr. Michael Lefevre at (504) 763-2569. If you think you have a research-related injury or medical illness, you should call Dr. Steve Smith at (504) 763-3028 during regular working hours. After working hours and on weekends you should call the answering service at (504) 765-4644. An on-call physician will respond to your call.

10. What information will be kept private?

Every effort will be made to maintain the confidentiality of your study records. However, someone from the Food and Drug Administration, the Pennington Biomedical Research Center, and the National Dairy Council (

the sponsor), may inspect and/or copy the medical records related to the study. Results of the study may be published; however, we will keep your name and other identifying information private. Other than as set forth above, your identity will remain confidential unless disclosure is required by law.

11. Can your taking part in the study end early?

Dr. Michael Lefevre or the study sponsor can withdraw you from the study for any reason or for no reason. Possible reasons for withdrawal include: 1) not coming to the scheduled diet counseling sessions; 2) not coming to the scheduled endpoint assessments; 3) inability to make significant changes in dietary fat intake; 4) illness; and 5) use of medications not allowed on the study. Also, the sponsor of the study may end the study early.

You may withdraw from the study at any time without penalty.

12. What if information becomes available that might affect your decision to stay in the study?

During the course of this study there may be new findings from this or other research which may affect your willingness to continue participation. Information concerning any such new findings will be provided to you.

13. What charges will you have to pay?

There will be no charges that you or your insurance company will have to pay. All instructions and medical tests associated with this study will be provided to you free of cost.

14. What payment will you receive?

You will be compensated for the inconvenience associated with the endpoint assessments. This includes $5 for every 4-day food intake record and $10 for each blood draw, ultrasound visit; and DEXA measurement. If you complete all assessments, the total amount of money you will receive is $255. If you are or have been an employee of LSU within the current calendar year, the normal employee payroll deductions will be withheld.

15. Will you be compensated for a study-related injury or medical illness?

The Pennington Center is a research facility and does not provide medical care. In the event of injury or medical illness resulting from the research procedures in which you participate, you will be referred to a treatment facility. No form of compensation for medical treatment is available. Medical treatment may be provided at your expense or at the expense of your health care insurer (e.g., Medicare, Medicaid, Blue Cross-Blue Shield, etc.) which may or may not provide coverage.

16. Signatures

The study has been discussed with me and all my questions have been answered. I understand that additional questions regarding the study should be directed to the study investigators. I agree with the terms above and acknowledge that I have been given a copy of the consent form.

_____	_____
Signature of Volunteer	Date
_____	
Social Security No. of Volunteer	
_____	_____
Signature of Witness	Date
_____	_____
Investigator *(Michael Lefevre, Ph.D.)*	Date
_____	_____
Medical Investigator (*Steve Smith, M.D.)*	Date

APPENDIX B

PERMISSIONS FOR USE OF FIGURE AND MEASURES

>>> "Michele Taussig" <michelet@naaso.org> 07/29/03 11:05 AM >>>

Paula:

Permission is granted to use figure 2, page 217 "The Civilization Syndrome" from the publication listed below:

Journal: Obesity Research
Issue: Volume 1, No. 3, May 1993
Article: Visceral Obesity: A "Civilization Syndrome"
Author(s): Per Bjorntorp

As I understand, this figure will be reproduced in your dissertation.

Please ensure that the appropriate source reference is provided. Any future use or adaptation will require separate permission. Copyright is retained by the North American Association for the Study of Obesity.

Best of luck to you in your future endeavors.

Regards,
Michele

-----Original Message-----
From: Paula Rhode [mailto:rhodepc@pbrc.edu]
Sent: Tuesday, July 29, 2003 11:32 AM
To: michelet@naaso.org
Subject: Reproduction Request

Ms. Taussig,

Pursuant to our telephone conversation this morning, I am writing to request permission to reproduce the attached figure for my dissertation manuscript.

The citation for the source article is:
Bjorntorp, P. (1993). Visceral obesity: A "Civilization Syndrome". Obesity Research, 1, 206-222.

>>> "NIMH Information Center" <NIMHINFO@circlesolutions.com> 07/29/03 11:47 AM >>>

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* Clinical Trials Information http://clinicaltrials.gov
  Provides information on federally funded and other clinical trials

Department of Health and Human Services
National Institutes of Health

>>> Stan Coren <scoren@psych.ubc.ca> 07/29/03 13:30 PM >>>

Dear Paula Rhode

Please let this note serve as permission to use and reproduce the APS scale for research purposes.

Sincerely

Prof. Stanley Coren, Ph.D., F.R.S.C.
Department of Psychology
University of British Columbia
2136 West Mall
Vancouver, Canada V6T 1Z4

Phone (604) 822-6458
Fax (604) 822-6923
E-mail: scoren@psych.ubc.ca
Website: http://www.stanleycoren.com

>>> Irwin Sarason <isarason@u.washington.edu> 07/29/03 13:22 PM >>>

You have my permission.
Irwin Sarason

Irwin Sarason
Department of Psychology
Box 351525
University of Washington
Seattle, Washington 98195
Phone:206 543-6542
FAX:206 685-3157

On Tue, 29 Jul 2003, Paula Rhode wrote:

Dr. Sarason,

I am currently a doctoral student finishing up my dissertation at Louisiana State University.

I am requesting permission to use your questionnaire, the Life Experiences Survey, as a measure in my dissertation, which will examine psychosocial predictors of visceral adiposity.

I do not intend to reproduce the scale in my manuscript, and I will, of course, properly cite it throughout my document.

However my committee has requested that I gain your permission for use of the scale for research purposes only.

Thank you for your cooperation and assistance in this matter,

Very truly yours,

Paula Rhode
Doctoral Candidate
Pennington Biomedical Research Center
Louisiana State University

>>> Phillip Brantley 07/29/03 14:30 PM >>>

Yes, you have my permission to use the WSI.

Phillip J. Brantley, PhD
Professor and Director
Division of Educational Programs
Chief, Behavioral Medicine
Pennington Biomedical Research Center
Louisiana State University
6400 Perkins Road
Baton Rouge, LA 70808-4124
voice: (225) 763-3046
fax: (225) 763-3045
email: BrantlPJ@pbrc.edu

>>> Paula Rhode 07/29/03 01:36PM >>>

Dr. Brantley,

I am currently a doctoral student finishing up my dissertation at Louisiana State University.

I am requesting permission to use your questionnaire, the Weekly Stress Inventory, as a measure in my dissertation, which will examine psychosocial predictors of visceral adiposity.

I do not intend to reproduce the scale in my manuscript, and I will, of course, properly cite it throughout my document.

However my committee has requested that I gain your permission for use of the scale for research purposes only.

Thank you for your cooperation and assistance in this matter,

Very truly yours,

Paula Rhode
Doctoral Candidate
Pennington Biomedical Research Center
Louisiana State University

>>> "Burema, Jan" <Jan.Burema@wur.nl> 08/11/03 08:26 AM >>>

Dear Paula Rhode,

I herewith give permission to you to use the short questionnaire for the measurement of habitual physical activity in epidemiological studies (Baecke et al, Am J Clin Nutr 1982;36:936-942) for research purposes.

On behalf of JAH Baecke,
Jan Burema

Division of Human Nutrition
Dep. Agrotechnology and Food Sciences
Wageningen University
PO box 8129
6700 EV Wageningen
The Netherlands

VITA

Paula C. Rhode was born on November 12, 1967 in Twinsburg, Ohio to William F. and Charlotte M. Rhode. She attended high school in West Liberty, Ohio and received her baccalaureate degree in history and political science from the University of Tampa in Tampa, Florida. In December 2000, she received a Master of Arts degree in clinical psychology from Louisiana State University in Baton Rouge, Louisiana. She completed her clinical residency at the University of Mississippi Medical Center/Jackson Veteran's Affairs Medical Center Consortium in 2002-2003. The preceding work represents the culmination of her studies at Louisiana State University for the degree of Doctor of Philosophy in clinical psychology. She currently holds the position of Assistant Professor in the Department of Preventive Medicine at the University of Kansas School of Medicine in Kansas City, Kansas.