2012

The Effects of Psychological Stress, Depressive Symptoms, and Cortisol on Body Mass and Central Adiposity in 10-to12-Year-Old Children

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THE EFFECTS OF PSYCHOLOGICAL STRESS, DEPRESSIVE SYMPTOMS,
AND CORTISOL ON BODY MASS AND CENTRAL ADIPOSITY
IN 10- to 12-YEAR-OLD CHILDREN

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A DISSERTATION

Submitted to the graduate faculty of The University of Alabama at Birmingham,
in partial fulfillment of the requirements for the degree of
Doctor of Philosophy

BIRMINGHAM, ALABAMA

2012

THE EFFECTS OF PSYCHOLOGICAL STRESS, DEPRESSIVE SYMPTOMS,
AND CORTISOL ON BODY MASS AND CENTRAL ADIPOSITY IN
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ABSTRACT

The percentage of children with elevated body mass and central adiposity has tripled in the U.S. over the past thirty years. While physical factors, such as decreased physical activity and poor nutrition, have been noted to influence elevated body mass and central adiposity in school-aged children, less is known about psychological factors, such as psychological stress and depressive symptoms, especially in 10-to12-year-old children. Further, it has been suggested that cortisol, a biomarker of psychological stress, plays a role in the underlying mechanism that links psychological stress and depressive symptoms to increased body mass and central adiposity, but this has not been addressed.

The purpose of this study was to examine the influence of psychological stress and depressive symptoms on body mass and central adiposity in 10-, 11-, and 12-year-old children and included controlling for gender, ethnicity, puberty, and SES. In addition, the principal researcher sought to determine the mediating role of cortisol in the relationships among psychological stress, depressive symptoms, body mass, and central adiposity.

A convenience sample of 147 (84 girls, 63 boys; 67.3% White, 11.6% African American, 17.7% Hispanic, 2.0% Asian, and 1.4% More than one race) 10-, 11-, and 12-year-old children were enrolled from a rural southeastern school district. More than half

(51.7%) of the participants were overweight or obese. A majority of the participants had cortisol levels within the normal range of 1.69 to12.81 nmol/l, while 8.3% had cortisol levels above the range. Approximately, 15.6 % of boys had WC measures at or above the 90th percentile, while 19% of girls had WC measures at or above the 90th percentile.

After controlling for gender, puberty, ethnicity, and SES, the predictor variable depressive symptoms explained a significant amount of the variance in body mass and central adiposity. The predictor variable psychological stress explained a significant amount of the variance in both body mass and central adiposity when the independent variable depressive symptoms was not included in the model. A lack of statistically significant relationships between psychological stress and cortisol, and between depressive symptoms and cortisol precluded the need for additional testing of cortisol for mediation.

Key Words: depressive symptoms, psychological stress, body mass, central adiposity, school-aged children

DEDICATION

I dedicate this dissertation to my loving mother, Hong Ngoc Thi Phan. She taught me the

value of hard work, commitment, courage, strength, love of family, and the importance

of having dreams and aspirations. Mom, I am grateful for your unconditional love and

support.

ACKNOWLEDGMENTS

and Andrea Craig, who helped me during the research study. Their organization, energy, and ability to work with children were extremely valuable. Thank you to Barbara Hull, who is one of the best editors that I have ever met.

I want to extend my deepest gratitude to Superintendent, Dr. Barry Carroll for his unyielding support. Thank you to Anthony Hilliard; I would not have been able to access the school system without his belief in this project. Thank you to all of the wonderful principals, teachers, and school children who showed interest and incredible kindness.

I wish to thank my wonderful Leadership and Education in Child Health Nursing peers, Betsy Gulledge, Jeannie Rodriguez, Luz Huntington-Moskos, Ann Johnson, Chrissy Feeley, Azita Amiri, and Susan Williams. You are always supportive of me and give me the kindest assistance. I am so fortunate to have experienced the PhD program with you. I am honored to know each and every one of you.

I want to thank my husband, Tom. I know how much you have done to support me and help me complete the dissertation. Thanks for being my personal chef and making sure that I had enough study breaks during those countless days and nights. Your patience and love for me is priceless.

Finally, thank you to my mother for her unconditional love and support. Without you, I would not be here to achieve my dreams. Your encouragement always gives me strength and courage. I am proud of being your daughter.

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CHAPTER 1

INTRODUCTION

Both elevated body mass and central adiposity have increased dramatically in

children, particularly in developed countries such as the United States (U.S.), and these

increases are associated with children being classified as overweight and obese (Li, Ford,

Mokdad, & Cook, 2006; World Health Organization [WHO], 2010). Body mass is

defined as the two compartments of the human body consisting of fat mass and fat-free

mass (Vestbo et al., 2006). Fat mass, in principle, is the metabolic inactive energy store,

while fat-free mass encompasses the metabolic active organs and the skeletal muscle

(Vestbo et al., 2006). Body mass can be assessed by using body mass index (BMI),

defined as weight in kilograms divided by the square of the height in meters (Centers for

Disease Control and Prevention [CDC], 2011).

While often associated with body mass, central adiposity is defined as excessive

accumulation of both central subcutaneous and visceral fat embedded in the abdominal

area encasing the visceral organs (Donoho, Weigensberg, Emken, Hsu, & Spruijt-Metz,

2010; Li, Ford, Mokdad, & Cook, 2006) and can be measured by waist circumference.

Based on the National Health and Nutrition Examination Surveys (NHANES) data, the

percentage of central adiposity among U.S. children increased 65.4% in boys and 69.4%

for girls from 1988 to 2004 (Li et al., 2006). Data from 1999 to 2004 revealed that the

percentage of 6- to 11-year-old children with high BMI scores rose about 25%; however,

the increase in central adiposity of the same group was more than a 35% increase over the same period (Li et al., 2006).

These elevations in body mass and/or central adiposity have economic implications. In 2004, the U.S. spent an estimated $98 to $129 billion for associated obesity health care costs in adults alone (Koplan, Liverman, & Kraak, 2005). The national cost of childhood obesity is estimated at approximately $11 billion for children with private insurance and at $3 billion for those with Medicaid (Marder & Chang, 2005).

Generally, overweight and obesity in adults and children are defined in terms of BMI. However, the terms *overweight* and *obesity* in children have been defined in several ways and used inconsistently across studies in children (Flegal, Tabak, & Ogden, 2006). According to Barlow and the Expert Committee (2007), children with a BMI in the 85th to 94th percentile are classified as overweight, whereas children at or above the 95th percentile are classified as obese, based on age- and gender-specific growth charts.

Within the last 30 years, the prevalence of BMI for age at or above the 95th percentile has tripled from 6.5% to 19.6% in children aged 6 to 11 years (NHANES, 2010). Geographical differences in child BMI trends have emerged in the U.S. with the southeastern states having the highest prevalence of elevated BMI (Singh, Kogan, & van Dyck, 2010). Children in some of the southern states have obesity rates that exceed 20%, which is twice the rate in other regions in the nation (Singh et al., 2010).

Elevations in body mass and central adiposity in childhood have been associated with overweight and obesity in adults and co-morbidities for children and later adulthood (Magarey, Daniels, Boulton, & Cockington, 2003; Must & Strauss, 1999; Serdula et al., 1993). In one study of children aged 8 to 12 years, researchers reported that 43% of

obese children remained obese as adults, and that another 29% were overweight as adults (Maffeis et al., 2002). Children at the highest levels of BMI are generally at the greatest risk of adverse health outcomes. Elevated blood pressure and insulin resistance were found to be twice as common in children with BMIs above the 97th percentile as in children within the 95th to 97th percentile (Freedman, Dietz, Srinivasan, & Berenson, 1999). Once seen mostly in adults, these long-term medical complications are now witnessed in children with an elevated BMI (Daniels, 2006). The prevalence of hypertension in children is estimated to be 2-5% (Sorof, Lai, Turner, Poffenbarger, & Portman, 2004). The combined prevalence of prehypertension and hypertension in adolescents who are obese is greater than 30% in boys and is 23-30% in girls (McNiece et al., 2007). One study reported a concordant increase in body mass and systolic blood pressure in middle school students aged 10 to 14 years (Luepker, Jacobs, Prineas, & Sinaiko, 1999).

In addition to health risks associated with elevated BMIs, high levels of plasma lipids and lipoprotein levels have been correlated with a greater waist circumference measurement in adults (Flodmark, Sveger, & Nilsson, 1994; Freedman, Serdula, Srinivasan, & Berenson, 1999). This measurement is regarded as representing one of the early criteria for cardiometabolic syndrome, and independently of BMI, has been recognized to be an important indicator for cardiovascular risks in the adult population (Bacha, Saad, Gungar, & Arslanian, 2006; Janiszewski, Janssen, & Ross, 2007; Klein et al., 2007). Studies with children have noted that in comparison with overall adiposity, a greater deposition of central fat is correlated with cardiovascular disease risk factors (Flodmark, Sveger, & Nilsson, 1994; Freedman, Serdula, Srinivasan, & Berenson, 1999;

Lurbe, Alvarez, & Redon, 2001; Savva et al., 2000). Together, waist circumference measurement and BMI predict cardiovascular risks such as hypertension, better than either measure used alone (Janssen, Katzmarzyk, & Ross, 2004; Messiah, Arheart, Lipshultz, & Miller, 2008; Ng et al., 2007; Zhu et al., 2004). Thus, tracking body mass through BMI and central adiposity through waist circumference measurements in childhood is especially valuable.

Having an elevated body mass and increased central adiposity involves multifactorial influences including genetic, behavioral, environmental, and cultural factors (Dishman, 2008; Dunton, Berrigan, Ballard-Barbash, Graubard, & Atienza, 2009; Giles-Corti, Timperio, Bull, & Pikora, 2005; Marti, A., Moreno-Aliagra, Hebrebrand, & Martinez, 2004; Mustein, Silventoinen, Pietiläinen, Rissanen,& Kaprio, 2009). Research aimed at understanding the etiology of obesity has focused on two conventional areas, poor nutrition and low physical activity. Physical factors such as poor nutrition and low physical activity influence body mass and central adiposity (Fox, Dodd, Wilson, & Gleason, 2009; Floriani & Kennedy, 2007; Hills, King, & Armstrong, 2007). Other factors that have been noted to influence body mass and central adiposity are gender (Alleyne & LaPoint, 2004; Goldfield et al., 2007; Hammer et al., 1991; Morrison et al., 1994), ethnicity (Biro et al., 2010; Herman-Giddens et al., 1997; Kaplowitz, Slora, Wasserman, Pedlow, & Herman-Giddens, 2001) socioeconomic status (Fernald & Gunnar, 2009; Lupien, King, Meaney, & McEwen, 2001), and puberty (Kiess et al., 1995; Knutsson et al., 1997; Netherton, Goodyer, Tamplin, & Herbert, 2004; Ribeiro, Santos, Duarte, & Mota, 2006). However, less is known about psychological factors, particularly psychological stress and depressive symptoms, which, in turn, may

contribute to behavioral factors such as decreased physical activity and poor nutritional habits that lead to elevated BMI and increased waist circumference.

The prevalence of depressive disorders ranges from 5.2% to 6.3% in a nonclinical population (Poznanski & Mokros, 1994), and approximately 18% of fifth and sixth graders experience mild to severe depressive symptoms (Chang, Zauszniewski, Heinzer, Musil, & Tsai, 2007). During this period of developmental transition, children often experience psychological stress, especially associated with the school context (Brobeck, Marklund, Haraldsson, & Berntsson, 2007); this experience of psychological stress further complicates depressive symptoms (Antonovsky, 1981; Cornell & Furman, 1984; Washington, 2009). Psychological stress, defined as the particular relationship between the person and environment that is appraised by the person as taxing or exceeding his or her resources and endangering his or her well being (Lazarus & Folkman, 1984), may also contribute to an increased body mass and central adiposity via underlying physiological mechanisms. Such mechanisms have been proposed whereby stress activates the hypothalamic-pituitary-adrenal (HPA) axis, and this activation could promote visceral fat deposits and overall adiposity (Adam & Epel, 2007). Björntorp (2001) reported that the elevation of cortisol, an end product of HPA activation, contributed to peripheral fat and visceral fat accumulation.

Although psychological stress is a universal concept that generally affects all individuals, different populations such as children experience unique events or circumstances thought to be especially stressful for them (Lewis, Siegel, & Lewis, 1984). According to Antonovsky (1981), individuals tend to become more vulnerable during

periods of biological, social, and psychological transition. In middle and late childhood,

children undergo transitions that can cause psychological stress (Washington, 2009).

The stages of passing from childhood to adolescence and from adolescence to

adulthood have been viewed as developmental transitions (Cornell & Furman, 1984).

Psychological stress typically arises from normative developmental transitions such as

entrance into middle school. School transitions, often characterized by many social and

academic stressors, adversely affect academic motivation, performance, and school

engagement, as well as emotional well-being (Cornell & Furman, 1984). Children aged

10 to 12 years are in a transitional stage from childhood to adolescence; this period is a

turning point in the lives of children and involves psychologically stressful challenges

that may result in depressive symptoms (Herman-Stahl & Peterson, 1996). Specific to

children, Kovacs (1985) defined depressive symptoms as encompassing negative mood,

interpersonal problems, ineffectiveness, anhedonia, and negative self-esteem. Negative

mood reflects feeling sad, feeling like crying, worrying about "bad" things, being

bothered or upset by things, and being indecisive (Kovacs, 1992). Interpersonal

problems indicate difficulties in interactions with people, including trouble getting along

with people, social avoidance, and social isolation (Kovacs, 1992). Ineffectiveness

reflects a negative evaluation of one's ability and school performance (Kovacs, 1992).

Anhedonia is characterized by impaired ability to experience pleasure (Kovacs, 1992).

Negative self-esteem includes low self-esteem, self-dislike, and feelings of being unloved

(Kovacs, 1992).

During the middle school-aged child's period of transition, children are more

vulnerable to chronic stress, which is persistent stress and a state of unrelenting demands

over resources (Thoresen & Eagleston, 1983). Rudolph and colleagues (2001) confirmed that the experience of school-related stress (such as poor academic performance, negative feedback from parents and teachers about schoolwork, and daily turmoil in the school environment) is associated with depressive symptoms in the context of a transition into middle school.

Research has shown that chronic stressors are strongly correlated with depressive symptoms in adults (Phelan, Bromet, & Dew, 1991). In comparison with acute events, chronic stress seems to have a greater impact on depressive symptoms (McGonagle & Kessler, 1990). Hammen (1991) reported the bidirectional relationship between stress and depression or depressive symptoms; other researchers noted the association between depressive symptoms and occurrence of stressful life events in an individual's lifetime (Cohen & Wills, 1985; Kessler & Magee, 1994). Results of several studies supported a strong relationship between psychological stress and depressive symptoms in the adult population (Hammen, 2005; Kessler & Magee, 1994; Phelan, Bromet, & Dew, 1991). Research conducted within the past few decades also found an association between stressful life events and depressive symptoms in children and adolescents; however researchers did not target school-aged children but rather combined school-aged children and adolescents within their studies, thus making it difficult to address this association in school-aged children alone (Birmaher et al., 1996; Compas, Slavin, Wagner, & Vannatt, 1986; Sund, Larsson, & Wichstrom, 2003; Williamson, Birmaher, Dahl, & Ryan, 2005).

Both psychological stress and depressive symptoms have been linked to increased body mass and increased central adiposity in children, but the mechanisms for these linkages remain unclear (Bahreinian et al., 2011; Pine, Goldstein, Wolk, & Weissman,

2001; Roemmich, Smith, Epstein, & Lambiase, 2007). One of the primary mechanisms

linking psychological stress and depressive symptoms may be the elevation of cortisol, a

glucocorticoid hormone and end product of the HPA axis; cortisol is produced in

response to stress in an individual's environment (Cohen, Janicki-Deverts, & Miller,

2007; Hanrahan, McCarthy, Kleiber, & Lutgendorf, & Tsalikian, 2006). Elevated levels

of this hormone contribute to accumulation of visceral fat, the body adipose tissue located

within the abdominal cavity and surrounding the visceral organs; this accumulation may

lead to abdominal obesity (Björntorp, 2001).

 Although psychological stress is associated with a general increase in body mass

and central adiposity, only a limited number of studies have examined the associations

between stress and weight in children and adolescents (Ter Bogt et al., 2006; Goldbacher,

Matthews, & Salomon, 2005). Most researchers have specifically studied work stress

and associations with weight in adults, with differing outcomes found between men and

women (Kivimäki et al., 2006; Kouvonen, Kivimäki, Cox, Cox, & Vahtera, 2005). This

difference may exist because BMI was primarily investigated as an outcome. Stress has

been linked with elevated central adiposity, in which case waist circumference would be

a sensitive indicator (Björntorp, 2001). Van Jaarsveld and associates (2009) examined

perceived stress or psychological stress and changes in weight, waist circumference, and

BMI in youths aged 11 to 16 years; the researchers concluded that across time, waist

circumference and BMI increased more in higher stress groups than in lower stress

groups.

 Although little is known about the relationship between depressive symptoms and

elevated body mass (McElroy et al., 2004), research results suggest that children and

adolescents with depressive symptoms may be at increased risk of developing elevated body mass. Findings from a number of studies have shown that depressive symptoms influence body mass; however, most of these studies included primarily adolescent and adult populations (Goodman & Whitaker, 2002; Matziou, Perdikaris, & Mellou, 2010; Pine, Goldstein, Wolk, &Weissman, 2001; Wardle, Williamson, Johnson, & Edwards, 2006; Xie et al., 2005). In a recent study, adults with depressive symptoms were found to have greater increases in BMI scores and waist circumference (Needham, Epel, Adler, & Kiefe, 2010). Depressive symptoms in adolescents were associated with an increased BMI in adulthood, even when participants with elevated BMI were excluded at baseline (Pine et al., 2001). In some studies, researchers found positive relationships between depressive symptoms and body mass in children and adolescents; however, these relationships existed in girls only (Erikson, Robinson, Haydel, & Killen, 2000; Xie et al., 2005). Few studies involved examining depressive symptoms and development of an elevated body mass over time in both children and adolescents (Goodman & Whitaker, 2002; Pine, Goldstein, Wolk, & Weissman, 1997; Pine et al., 2001; Richardson et al., 2003). In one study, researchers confirmed that cortisol mediated the association between depressive symptoms and BMI; however, significance was found only in girls (Dockray, Susman, & Dorn, 2009). Findings from another study suggested that elevated cortisol levels underlie the association between depressive symptoms and high BMI in children (Endocrine Society, 2009).

The influence of depressive symptoms on central adiposity has also been examined, but this research has been done primarily in adults (Everson-Rose et al., 2009; Hach, Ruhl, Klotsche, Klose, & Jacobi, 2006; Lee et al., 2005). Vozelzangs and

colleagues (2008) found an association between depressive symptoms and elevated central adiposity in a large population of older adults over time. Depressive symptoms were shown to have a positive association with visceral fat distribution in women (Lee et al., 2005). To date, little research has been done with children and adolescents.

Strong associations have been shown in the relationships among elevated cortisol, depressive symptoms, and central adiposity primarily in adults (Brown, Varghese, & McEwen, 2004). In a 1997 research study, Rosmond reported significant associations between abdominal fat and depressive symptoms in middle-aged men. A later study by Rivenes, Harvey, and Mykleturn (2009) found that central adiposity was associated with depressive symptoms in both men and women. The investigators provided an explanation for their findings that support metabolic pathways or disturbances involving the HPA axis; thus, cortisol plays a role in the development of central adiposity. Most studies have examined relationships in adults. However, no studies to date have examined these relationships in children.

When studying the factors that contribute to increased body mass and central adiposity, researchers must control for variables that have been shown to influence these factors including gender (Hammer et al., 1991; Morrison et al., 1994), ethnicity (Biro et al., 2010; Herman-Giddens et al., 1997; Kaplowitz, Slora, Wasserman, Pedlow, & Herman-Giddens, 2001), socioeconomic status, (SES) (Fernald & Gunnar, 2009; Lupien, King, Meaney, & McEwen, 2001), and puberty (Biro et al., 2010; Herman-Giddens et al., 1997; Kaplowitz et al., 2001), in studies involving school-aged children (10-, 11-, and 12-year-olds). Such variables can exert an independent influence on cortisol levels, body mass, and central adiposity. Pubertal stage may independently activate the HPA axis,

with subsequent cortisol production (Kiess et al., 1995; Knutsson et al., 1997; Netherton, Goodyer, Tamplin, & Herbert, 2004). Additionally, increased body mass and adiposity in children may influence various aspects of pubertal development (Solorzano & McCartney, 2010). Pubertal changes have been associated with increased body mass, central adiposity, and cortisol (Bratberg, Nilsen, Holmen, & Vatten, 2007; Kaplowitz et al., 2001; Knuttson et al., 1997; Netherton, Goodyer, Tamplin, & Herbert, 2004; Ribeiro et al., 2006).

In addition to puberty, low socioeconomic status has been linked to HPA stimulation resulting in higher cortisol levels in children (Fernald & Gunnar, 2009; Lupien, King, Meaney, & McEwen, 2001). Children from families with low SES report greater exposure to stressful life events, and these children can experience subsequent increases in cortisol secretion (Lupien et al., 2001). Gender can also confound results because it uniquely influences the relationships among elevated BMI, socioeconomic status, ethnicity, and psychological factors (Alleyne & LaPoint, 2004; Goldfield et al., 2007). In addition to gender, ethnicity has played a role in early puberty. African American girls in the United States were shown to experience puberty at an earlier age than other ethnic groups (Biro et al., 2010; Herman-Giddens et al., 1997; Kaplowitz et al., 2001). On the other hand, the effects of excess adiposity in boys remain unclear; however, some researchers report a delay of pubertal development in boys (Solorzano & McCartney, 2010). Because of the potential effects of gender, ethnicity, socioeconomic status, and puberty, these variables were controlled.

Conceptual Framework

The conceptual framework used to guide this study is based on an expansive biobehavioral interaction model that incorporates theoretical underpinnings from three models (Kang, Rice, Park, Turner-Henson, & Downs, 2010). This comprehensive model is based on Selye's stress theory, McEwen's theory of allostasis and allostatic load, and Lazarus and Folkman's transactional model of stress, appraisal, and coping. This integrated model combines the strengths of all three models and provides a more complete representation of a biobehavioral framework. Within this model, psychosocial, behavioral, individual, and environmental factors that can individually and/or interactively influence biological responses that impact health outcomes are included (Kang et al., 2010). Biological responses generally serve as a mediator for the influence of various psychosocial, behavioral, individual, and environmental factors on health outcomes (Kang et al., 2010). Psychosocial factors such as stress reflect the factors processed through individual appraisals and also reflect cumulative effects (both acute and chronic) on biological responses (Kang et al., 2010). Acute cumulative effects may develop when multiple factors occur simultaneously during a short time, whereas chronic cumulative effects may arise when a selected number of psychosocial, behavioral, individual, and environmental factors persist over a long period to change biological responses (Kang et al., 2010). Additionally, this model allows for substantial flexibility because it addresses potential bidirectionality between various factors and health outcomes.

One of the theories that undergirds the biobehavioral model is Selye's stress theory. Selye popularized the concept of stress, and many researchers trace the origin of

its study and definition to Selye (Ice & James, 2007). The work of Selye described stress as a nonspecific response of the body to noxious stimuli and focused on the physiological response to physical stimuli or environmental stressors (Selye, 1946). The physiological response is always the same regardless of the stimulus. Selye (1946) expanded the physiological response process known as the general adaptation syndrome (GAS). The stress response (GAS), a defensive response, does not depend on the nature of the stressor (Selye, 1946). This syndrome consists of three stages: a) alarm reaction, b) stage of resistance, and c) stage of exhaustion (Selye, 1946). In the alarm stage, the body reacts to a stimulus by activating the HPA axis. The resistance stage signals successful adaptation to the stimulus (Selye, 1946). If the stress is not adequately resolved, then the body's stores of glucocorticoids, such as cortisol, which are the output of the HPA axis becomes depleted (Selye, 1946). Exhaustion occurs when exposure to stimuli is prolonged; in this stage, disease states could eventually occur.

Selye's basic theoretical tenet that stress was a physiological phenomenon did not include consideration of the concept of psychological stress. Stress research focused on major life events, and researchers further expanded events that might be considered stressful (Ice & James, 2007). Although Selye's stress theory detailed physiological factors such as activation of the sympathetic nervous system (SNS) and of the HPA axis, the theory did not address cognitive-perceptual factors (Lyon, 2000). As the stress research continued to expand to focus on major life events, it became clear to researchers that there were individual differences in the response to events considered stressful (Ice & James, 2007). Lazarus and Folkman (1984) viewed the stress process as a transaction between person and environment. Stress is experienced when the demands of a situation

exceed a person's resources and when some type of harm or loss is anticipated (Lazarus & Folkman, 1984).   The interactional approach emphasizes the importance of processes within the person that determine both whether a stress response will occur and the nature of the response (Lazarus & Folkman, 1984). Lazarus and Folkman (1984) defined the stress process as a "particular relationship between the person and the environment that is appraised by the person as taxing or exceeding his or her resources and endangering his or her well-being" (p. 19).

The perception of threat and the ability to adequately cope with the demand play a key role in determining the activation, intensity, and duration of the stress responsive system (Lazarus & Folkman 1984). Although Selye originally conceptualized the stress response as nonspecific in nature, Mason (1968) demonstrated that physical stressors such as cold or heat did not activate the HPA axis and the stress response unless a perception of uncertainty and a sense of emotional distress were present. Unlike the physiological stress investigated by Selye, the stress experienced by humans almost always results from a cognitive mediation (Lazarus, 1966). Selye did not address or specify the mechanisms that may explain the cognitive transformation of "objective" noxious agents into an individual's subjective experience of being distressed (Gallagher, Nelson, & Weiner, 2003)

Lazarus and Folkman (1984) emphasized that individual appraisal constitutes the most important source of information in the stress response. Because they are developing causal thinking, school-aged children can understand and appraise events and experiences. School-aged children are also able to describe their thoughts and no longer need to rely on actions to explain themselves (Board, 2005).

While Lazarus and Folkman (1984) described the cognitive appraisal of stress, more information has been revealed about cognitive processes, physiological responses, and health-related outcomes associated with psychological stress (Gallagher, Nelson, & Weiner, 2003). McEwen introduced the long-term effects of stress on humans and proposed a model of allostasis and allostatic load to explain the relationship between stress and disease (McEwen & Lasley, 2003). The concepts of allostasis and allostatic load are described within the biobehavioral interaction model developed by Kang, Rice, Park, Turner-Henson, and Downs (2010). The allostatic load model provides explanations of the human body's adaptation processes in response to stress and of the results of failed adaptation over time (McEwen, 2003).

McEwen and Wingfield (2003) studied the allostatic load and the physiological effects of the sustained activation of the HPA axis and the SNS and proposed that repeated stimulation of glucocorticoids such as cortisol may lead to allostatic load, which is characterized by chronically elevated cortisol levels, as well as by changes in other physiological systems. Their model, known as the allostatic load model, is based on the concept of allostasis, which represents the process of adaptation of complex physiological systems to physical, psychosocial, and environmental challenges (McEwen & Wingfield, 2003). Although allostasis is critical to adaptation and survival, allostatic load is defined as "the price the body pays over long periods of time for adapting to challenges" (McEwen, 2001, p. 44).

Frequent overactivation of the stress response can overwhelm the ability of the body to properly manage stress (McEwen & Lasley, 2003). Individuals who have experienced excessive stress in their lives have been linked with more depressive

symptoms and have shown decline in physical and mental functioning (McEwen & Lasley, 2003).

Repeated allostasis such as changes in cortisol and catecholamine secretion in response to stress and depressive symptoms affects allostatic load. Activation of the HPA axis with subsequent sustained overactivation of the HPA can increase allostatic load (See Figure 1). Cortisol, a glucocorticoid hormone, is the end product of HPA axis activation in humans (Hanrahen, McCarthy, Kleiber, Lutgendorf, & Tsalikan, 2006).

An example of an alteration in allostatic load includes the accumulation of abdominal fat (McEwen, 2002). Results of population studies have shown that adrenal stress hormones, such as cortisol, are strongly associated with the deposition of body fat around the waist and with overall adiposity (Björntorp, 2001). Additionally, glucocorticoids influence the brain to increase appetite for food and to increase food seeking behavior (Leibowitz & Hoebel, 1997). According to Björntorp (2001), the ultimate result of continued glucocorticoid exposure might be stress eating with subsequent increase in body mass and/or central adiposity.

The experience of psychological stress and depressive symptoms activate the SNS and the HPA axis. Through these activations, cortisol levels could increase, with subsequent glucose circulated in the system. Chronically elevated glucocorticoids can hinder the action of insulin to promote glucose uptake (McEwen, 2002). As a result, insulin and glucocorticoid elevation may promote the deposition of body fat (McEwen, 2002). Psychological stress and depressive symptoms may likely influence body mass and central adiposity through the mediation of cortisol. Little research has addressed the

potential effects of psychological stress, depressive symptoms, and cortisol on body mass

and central adiposity in school-aged children.
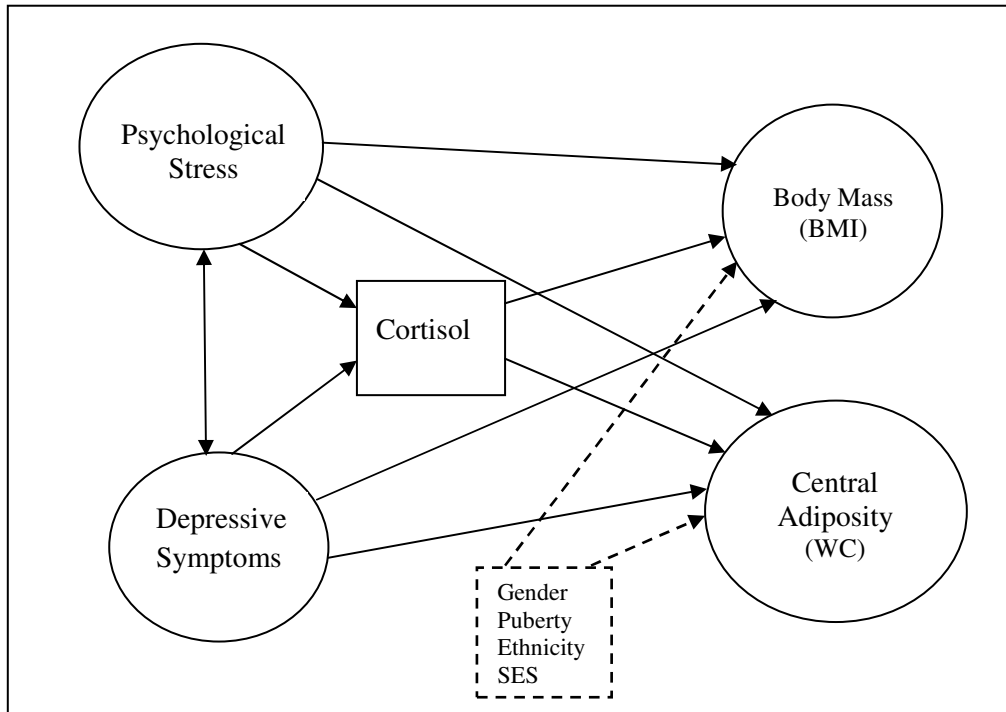
Figure 1. Diagram of conceptual framework

Purpose

The overall purpose of this study was to determine the effects of psychological

stress and depressive symptoms on body mass and central adiposity in 10-, 11-, and 12-

year-old children. The investigator also sought to determine the mediating role of

cortisol in the relationships among psychological stress, depressive symptoms, body

mass, and central adiposity in this population. Specific research questions and

hypotheses were as follows:

Research Questions and Hypotheses

Research Question 1: What is the relationship between psychological stress and depressive symptoms in 10-, 11-, and 12-year-old children?

Hypothesis 1: There is a positive relationship between psychological stress and depressive symptoms in 10-, 11-, and 12-year-old children.

Research Question 2: How much of the variance in body mass and central adiposity is explained by psychological stress and depressive symptoms in 10-, 11-, and 12-year-old children after controlling for gender, ethnicity, puberty, and SES?

Hypothesis 2: Psychological stress and depressive symptoms will explain a significant amount of the variance in body mass and central adiposity in 10-, 11-, and 12-year-old children after controlling for gender, ethnicity, puberty, and SES.

Research Question 3: Does cortisol mediate the relationship between psychological stress and body mass and/or between depressive symptoms and body mass in 10-, 11-, and 12-year-old children after controlling for gender, ethnicity, puberty, and SES?

Hypothesis 3: Cortisol will mediate the relationship between psychological stress and body mass and/or between depressive symptoms and body mass in 10-, 11-, and 12-year-old children after controlling for gender, ethnicity, puberty, and SES.

Research Question 4: Does cortisol mediate the relationship between psychological stress and central adiposity and/or between depressive symptoms and central adiposity in 10-, 11-, and 12-year-old children after controlling for gender, ethnicity, puberty, and SES?

Hypothesis 4: Cortisol will mediate the relationship between psychological stress and central adiposity and/or between depressive symptoms and central adiposity in 10-, 11-, and 12-year-old children after controlling for gender, ethnicity, puberty, and SES.

Significance of Study

This study could provide insight into other factors that could influence elevated body mass and central adiposity in children aged 10-, 11-, and 12-years. In light of the findings from the literature, consideration should be given to the possibility that psychological stress and depressive symptoms are more prevalent in school-aged children, and therefore, prevention and intervention programs should include the assessment and monitoring of psychological stress and depressive symptoms early in the school-aged years to prevent elevated body mass and central adiposity. Nurses and other healthcare providers, especially those in primary care, school-based, and community health settings have the potential to play an important role in helping children and parents to recognize and properly manage children's experiences with psychological stress and depressive symptoms.

Psychological factors, such as psychological stress and depressive symptoms, can impact children's development of elevated body mass and central adiposity. Children who have elevated BMI and increased waist circumference commonly become obese adults with consequent morbidity and mortality risks such as increased rates of hypertension, diabetes, coronary artery disease, arthrosclerosis, and certain cancers (Biro & Wien, 2010). Additionally, children today are experiencing normally adult-onset co-morbidities, such as dyslipidemia, Type 2 diabetes, chronic inflammation, sleep apnea,

and orthopedic complications (Janssen, Katzmarzyk, & Ross, 2004; Jolliffe & Janssen, 2006; Must & Anderson, 2003). It is important to understand how psychological factors can impact body mass and central adiposity in school-aged children; this understanding could provide valuable information about the mechanisms involved and the knowledge base necessary to incorporate preventative and intervention strategies in programs to effectively combat elevated body mass and development of central adiposity in this population.

Assumptions

Several assumptions were made for this study of 10-, 11-, and 12-year-old children and their health. For the purposes of this study, the following assumptions were made:

1. Psychological stress can be harmful to one's health.

2. Depressive symptoms can lead to negative health outcomes.

3. Psychological stress and depressive symptoms can be measured and self-reported by school-aged children 10-, 11-, and 12-years-old.

4. Stress is an occurrence in life and school-aged children are able to appraise a situation as stressful or not stressful.

5. Cortisol levels can be measured in children.

Summary

Elevated body mass and central adiposity have been identified as important risk factors for a number of adverse health outcomes such as Type 2 diabetes, hypertension,

and cardiovascular disease. From the research published to date, it is suggested that measures of general and central adiposity should be used together in order to best identify individuals at increased risk of ill health, especially in children. Psychological stress, depressive symptoms, and cortisol may influence increases in body mass and central adiposity in school-aged children. Knowledge about the role these factors play could be used to decrease the incidence of overweight and obesity in school-aged children.

CHAPTER 2

LITERATURE REVIEW

In this chapter, research was reviewed that addresses children's psychological stress; depressive symptoms; and body mass, central adiposity, and cortisol. Each variable was discussed in the context of the available research. Additionally, research related to gender, ethnicity, puberty, and SES as confounding variables was presented. To identify published work of relevance to this study, a search was conducted using databases such as CINAHL, PubMed, PsycINFO, and those of the CDC and NHANES. In addition, bibliographies of published articles were used to identify additional articles relevant to the study.

Introduction

Although psychological stress and depressive symptoms are pervasive in modern society, most studies (Hammen, 1991; Kirschbaum, Pirke, & Hellhammer, 1995; Williamson et al., 1998; Wong, Leung, Ping, & Woo, 2011) have focused more on adults and adolescents and less on younger children. Results of several studies indicated that not only do depressive symptoms exist in childhood, but symptoms are similar to those of adult depression (Carlson & Cantwell, 1982; Puig-Antich et al., 1979). At any given time, it is estimated that 15% of children and adolescents experience some depressive symptoms (Bhatia & Bhatia, 2007). The prevalence of depressive disorders ranged from 5.2% to 6.3% in a nonclinical child population (Poznanksi & Mokros, 1994), and

approximately 18% of fifth and sixth graders had mild to severe depressive symptoms (Chang, Zauszniewski, Heinzer, Musil, & Tsai, 2007). During this developmental period, children often undergo transitions and experience psychological stress, which further complicate depressive symptoms (Antonovsky, 1981; Cornell & Furman, 1984; Washington, 2009).

One of the challenges in reviewing the literature is the inconsistent use of terminology across studies, particularly surrounding the concept of depressive symptoms. The terms *depression*, *depressive disorder*, *depressive syndrome*, and *depressive symptoms* have been used interchangeably; however, each concept is distinctive and should be used according to the context of the study (Cytryn & McKnew, 1996; Kovacs, 1992). In this study, depressive symptoms were examined because they are more commonly experienced than the clinical syndrome (Cytryn & McKnew, 1996). Furthermore, depressive symptoms are often insidious and may go undetected in children (Avenevoli, Knight, Kessler, & Merikangas, 2008). Over time, children who persistently experience depressive symptoms are more likely to develop depression that may be undiagnosed (Friedman, Katz-Levey, Manderschied, & Sondheimer, 1996).

More recently, there has been increased interest in the relationships between psychological factors (psychological stress and depressive symptoms) and their influence on body mass and on central adiposity (Anderson et al., 2011; Brunner, Chandola, & Marmot, 2007; Everson-Rose et al., 2009; Roemmich, Smith, Epstein, & Lambaise, 2007). Although the body of knowledge related to psychological stress and depressive symptoms as contributing factors to elevated body mass and increased central adiposity has been growing, the research involving children remains sparse. Therefore, this

literature review of related research includes the adult and adolescent population. The purposes of this study consisted of: (a) determining the effects of psychological stress and depressive symptoms on body mass and central adiposity in 10-, 11-, and 12-year-old children, and (b) investigating the mediating role of cortisol in the effect of psychological stress and depressive symptoms on body mass and on central adiposity in this population.

This chapter is organized into several sections. These sections consist of review of research on: (a) psychological stress and depressive symptoms; (b) psychological stress and cortisol, body mass, and central adiposity; (c) depressive symptoms and cortisol, body mass, and central adiposity; (d) cortisol as a mediator of the relationships between psychological stress and body mass and between psychological stress and central adiposity; (e) cortisol as a mediator of the relationships between depressive symptoms and body mass and between depressive symptoms and central adiposity; and (f) the potential confounding effects of gender, ethnicity, puberty, and socioeconomic status.

Psychological Stress and Depressive Symptoms

Results of several previous studies supported a strong association between psychological stress and depressive symptoms in the adult population (Hammen, 2005; Kessler, 1997; Kessler & Magee, 1994; Mazure, 1998; Monroe & Hadiyannakis, 2002; Phelan, Bromet, & Dew, 1991). Investigations of this relationship in children remain less common. Several groups of researchers examined the association between psychological stress and depressive symptoms, with a focus on stressful life events (Birmaher et al., 1996; McGonagle & Kessler, 1990; Williamson et al., 2005). Research conducted during

the past few decades revealed a relationship between stressful life events and the onset of depressive symptoms in children and adolescents (Compas, Slavin, Wagner, & Vannatt, 1986; Compas, Grant, & Sydney, 1994; Goodyer, 1994; Williamson et al., 1995). Furthermore, other studies focused on events that individuals perceive as stressful that occur in the person's environment (Kessler, 1997; Silberg et al., 1999; Williamson et al., 1995); such events may relate to depressive symptoms (Cohen & Wills, 1985; Kessler & Magee, 1994).

In their study, Csorba and colleagues (2001) examined the total number of life events among preadolescents (mean age 12.73 years) and found that childhood life events (recent and past life stressors) increased during this developmental time. Findings from other investigations revealed a significant connection between stressful life events and depressive symptoms in clinical and community samples of children and adolescents (Birmaher et al., 1996; Sund, Larsson, & Wichstrom, 2003; Williamson et al., 1998). Exclusively in the case of girls, researchers found a connection between depressive symptoms and total stress events in Rudolph and Hammen's (1999) cross-sectional and Ge and colleagues' (1994) prospective studies. According to results of the prospective research of Silberg and associates (1999), stressful life events predicted severe depressive symptoms (clinical depression) more frequently in the case of girls than in the case of boys. In their 4-year longitudinal study, Ge and colleagues (1994) found that, after the age of 13, girls experienced more psychological stress than boys were found to do and that only in case of girls were stressful events associated with a higher risk of depressive symptoms. In a later study, Williamson and associates (2005) examined psychological stress (the occurrence of stressful life events) in children with depressive symptoms. In

their study, 6-to 12-year-old children with an anxiety disorder ($n = 20$), with depression ($n = 45$), and controls lacking either symptom ($n = 11$) were assessed by using the Life Events Record, a questionnaire containing life events that are both clearly negative and stressful. The authors found that in comparison with both anxious and control children, children with depressive symptoms experienced significantly more stressful life events. Williamson and associates' (2005) finding that stressful life events occurred significantly more frequently among the depressed group than among controls is consistent with earlier reports of the correlation between stressful life events and symptoms of depression (Compas et al., 1986; Johnson & McCutcheon, 1980; Mullins, Siegel, & Hodges, 1985; Swearingen & Cohen, 1985).

In summary, a positive association has been found between psychological stress and depressive symptoms; however, this relationship has been seen primarily in studies conducted with adults. Examination of this relationship has been studied in children, but to a lesser extent in school-aged children. Findings from several investigations showed a significant association between psychological stress (stressful life events) and depressive symptoms in both clinical and community samples of children and adolescents.

Psychological Stress and Cortisol

In this study, psychological stress was conceptualized as the particular relationship that exists between a person and the environment and that is appraised by the person as taxing or exceeding his or her resources and as endangering his or her well-being (Lazarus & Folkman, 1984). Lazarus and Folkman (1984) purported that psychological stress consists of the interaction that takes place between the individual and

a specific situation, whereby the individual perceives the situation as potentially

threatening and also doubts the adequacy of the available resources. According to

Tennes and Mason (1982), psychological stress or stimuli trigger cortisol elevation by

producing negative emotional states, and elevated levels of cortisol therefore serve as

indices of negative emotions. Research has shown that the HPA system is responsive to

psychological stress (De Kloet, 1991; Kudielka et al, 2004). For example, the activity of

the HPA system can be estimated by measuring cortisol, a hormonal end-product of the

system. Cortisol, the most potent glucocorticoid produced by the human adrenal system

in response to stress (Dickerson & Kemeny, 2004; Kudielka et al., 2004), can be assayed

from samples of free-flowing saliva (Gunnar, Bruce, & Hickman, 2001). Salivary

cortisol reflects the unbound, biologically active fraction of the hormone and correlates

highly with plasma concentrations of cortisol (Kirschbaum & Hellhammer, 1994).

In her pioneering work, Megan Gunnar investigated psychological stress and

cortisol; her early studies involved examining stress reactions in neonates and infants

(Gunnar, 1992; Gunnar, Hertsgaard, Larson, & Rigatuso, 1991; Gunnar, Porter, Wolf,

Rigatuso, & Larson, 1995). Gunnar (1986) found that, in response to physical and

psychological stressors, newborns were highly reactive. In some studies, research groups

examined the adrenocortical response to a classic psychological stressor, such as

separation from the mother or primary attachment figure (Gunnar, Mangelsdorf, Larson,

& Hertsgaard, 1989; Tennes, Downey, & Vernadakis, 1977). Gunnar and her students

(1989) studied the salivary cortisol response to separation among 9-month-old infants in a

laboratory setting. The investigators assessed a long separation of 40 min and a short

separation of 9 min and found a statistically significant cortisol response in infants during

the long separation. In their study of relationships between parents' psychological stress and children's salivary cortisol levels, Koch and coworkers (2009) assessed parenting stress and serious life events at birth and at 1, 2, 5, and 8 years. Eighty-two paired saliva samples were collected from the parents' children. A significant relationship was found between higher parenting stress at child age 1 and at child age 8 and elevated cortisol levels.

Several groups of researchers have investigated the HPA axis responses to psychological stress in adults (Dickerson & Kenemy, 2004; Kirschbaum, Pirke, & Hellhammer, 1995; Kudielka, Buske-Kirschbaum, Hellhammer, & Kirschbaum, 2004; Rosmond, Holm, & Björntorp, 2000). In children, because of the limited number of studies, findings have been inconclusive with regard to psychological stress and cortisol. Additionally, researchers studied acute stress events rather than chronic stressors. In one of the few studies of school-aged children, Yim and colleagues (2010) modified a laboratory stressor for children aged 9 to 12 years to directly compare children's and adults' stress responses. Salivary cortisol levels significantly ($p < .001$) correlated with results from the Trier Social Stress Test (TSST). Additionally, children exhibited a significantly greater number of behaviors indicative of distress.

In summary, psychological stress has been associated with cortisol; however this relationship has been studied primarily in adults (De Kloet, 1991; Dickerson & Kenemy, 2004; Kudlieka, Buske-Kirschbaum, Hellhammer, & Kirschbaum, 2004; Rosmond et al., 2000). The relationship between psychological stress and cortisol has been studied more widely in young children, specifically with neonates and infants (Gunnar, 1992; Gunnar, Porter, Wolf, Rigatuso, & Larson, 1995; Tennes, Downey, & Vernadakis, 1977). Few

studies (Koch et al., 2009; Yim et al., 2010) have been conducted with children, especially school-aged children, to support any conclusions in this population.

Psychological Stress and Body Mass

The relationship between psychological stress and body mass in children has not been widely studied. More studies have been done with adults and adolescents (Brunner, Chandola, & Marmot, 2007; Goldbacher, Matthews, & Salomon, 2005; Jern, Bergbrant, Björntorp, & Hansson, 1992; Ter Bogt et al., 2006); in those studies, most researchers focused on work stress and associations with weight in adults, with differing outcomes found between men and women (Kivimäki et al., 2006; Kouvonen, Kivimäki, Cox, Cox, & Vahtera, 2005). Kivimäki and coauthors (2006) used the Whitehall study, a prospective cohort of British civil servants, to analyze the prospective association between work stress (psychological stress) and weight change over 5 years; the authors found that greater job strain and lesser job control were associated ($p = .05$ and $p = .02$, respectively) with increased BMI among men who were overweight or obese at baseline. However, a similar association was not observed among women. Roemmich and associates (2007) determined the association between psychological stress (reactivity) and total adiposity as measured by BMI and body fat percentage; the investigators analyzed data from two independent samples of children used to study the influence of an interpersonal stressor on either eating or physical activity. Participants included a sample of sixty-three 8- to 12-year-old boys and girls. In the study, Roemmich and coworkers (2007) measured perceived or psychological stress by using a visual analog scale

depicting very relaxed and very worried and found an association between greater

psychological stress reactivity and greater total adiposity as measured by BMI.

The relationship between psychological stress and body mass has been studied in

adults; yet, the findings have been equivocal (Kivimäki et al., 2006; Kouvonen,

Kivimäki, Cox, Cox, & Vahtera, 2005). Researchers used different instruments to

measure psychological stress; furthermore, some researchers defined psychological stress

as work stress or perceived stress. In addition, too little research has been done in

children and adolescents to support any conclusions.

Psychological Stress and Central Adiposity

Results of previous studies (Björntorp, 2001; Georges, Wear, & Mueller, 1992)

revealed a strong link between psychological stress and central adiposity or abdominal fat

accumulation; however, findings from other studies (Kuo et al., 2008; La Fleur, Akana,

Manalo, & Dallman, 2004; Shively, Register, & Clarkson, 2009) have been mixed and

not as clear   Most studies consisted of examining the relationship between psychological

stress and central adiposity in adults (Björntorp, 2001; Brunner, Chandola, & Marmot,

2007; Georges, Wear, & Mueller, 1992). In a large prospective study of adults, Brunner,

Chandola, and Marmot (2007) found chronic stress to be associated with increases in

central adiposity over a 19-year period; the authors examined 6,895 men and 3,413

women enrolled in the Whitehall II study and observed a dose-response relationship

between work stress (psychological stress) and both general and central adiposity in both

men and women. This finding supports conclusions from two previous cross-sectional

studies that showed an association between psychological stress related to work stress

and higher central adiposity in two samples of men (Georges, Wear, & Mueller, 1992; Jönsson et al., 1999).

Some researchers have examined psychological stress and central adiposity in adolescents (Goldbacher, Matthews, & Salomon, 2005; Van Jaarsveld et al., 2009). Goldbacher, Matthews, and Salomon (2005) studied the association between central adiposity (measured by waist circumference) and cardiovascular reactivity to psychological stress among 211 adolescents between 14 to 16 years of age; approximately 50% of the sample consisted of girls. The authors found that, in boys only, participants with a greater waist circumference exhibited greater physiological responses (greater systolic blood pressure reactivity and greater diastolic blood pressure reactivity) to psychological stress; their study results are consistent with those of earlier studies that involved investigating central adiposity and reactivity to stress in adults (Björntorp, 2001; Davis et al., 1999; Jern et al., 1992; Waldstein et al., 1999). In a later study, Van Jaarsveld and associates (2009) found that perceived stress or psychological stress in adolescents aged 11 to 15 years was associated with increased waist circumference in cross-sectional analyses but was not significant in longitudinal analyses.

The results of research on the relationship between psychological stress and central adiposity have been equivocal, possibly because of differences in measuring psychological stress. The measurement of stress in human studies uses subjective measures of stress. Life events represent more objective measures of stress because they measure events generally considered stressful, whereas perceived stress measures the subjective evaluation of an event as stressful (Cohen, Kessler, & Underwood, 1995). The relationship between psychological stress and central adiposity has not been widely

examined in children and particularly not in school-aged children. More studies have

been conducted with adults and adolescents (Rosmond, 2005).

Depressive Symptoms and Body Mass

In addition to examining the relationship between psychological stress and body

mass, groups of researchers explored the relationships between depressive symptoms and

body mass but have done so primarily in adults and adolescents (Heo, Pietrobelli, &

Fontaine, 2006; Matziou, Perdikaris, & Mellou, 2010; McElroy et al, 2004; Xie et al.,

2005). The relationship between depressive symptoms and body mass was studied to a

greater extent in adults than in adolescents, and fewer investigators enrolled children in

their studies (Anderson et al., 2011; Mather, Cox, Enns, & Sareen, 2009; Pine, Goldstein,

Wolk, & Weissman, 2001). Although the association between body mass and depressive

symptoms remains unclear because of mixed results in the literature (Dong, Sanchez, &

Price, 2004; Faith & Jorge, 2002; Mather, Cox, Enns, & Sareen, 2009; Sullivan et al.

1993), these inconsistent results led researchers to further examine the links between

these two concepts. Some investigators found correlations between depressive symptoms

and elevated body mass in both younger (Becker, Margraf, Turke, Soeder, & Neumer,

2001; Mather, Cox, Enns, & Sareen, 2009) and older adults (Hasler et al., 2004; Heo,

Pietrobelli, & Fontaine, 2006; Istvan, Zavela, & Weidner, 1992; Roberts, Deleger,

Strawbridge, & Kaplan, 2003). One previous national study consisted of examining the

association between BMI and depressive symptoms as measured by the Center for

Epidemiological Studies Depression Scale (CED-S), and results revealed a significant

positive finding in women but not in men (Istvan, Zavela, & Weidner, 1992). Similarly,

in a later study, Heo and colleagues (2006) found that, in comparison with non-overweight women, women with elevated BMI were significantly more likely to have sustained depressive symptoms.

Results of several studies showed a positive association between depressive symptoms and elevated body mass in adolescents (Anderson et al., 2011; Cortese et al., 2009; Goodman & Whitaker, 2002; Matziou, Perdikaris, & Mellou, 2010; Pine, Goldstein, Wolk, & Weissman, 2001; Wardle, Williamson, Johnson, & Edwards, 2006; Xie et al., 2005). However, data from the literature on the link between body mass and depressive symptoms remain inconclusive (Elgar, Roberts, Tudor-Smith, & Moore, 2005). In a study of Greek adolescents (72 males and 128 females), Matziou and colleagues (2010) examined the association between weight status and depressive symptoms among 18-year-olds, and reported that weight status (assessed by BMI) was negatively related to depressive symptoms. In a recent study with adults, Needham and colleagues (2010) found that participants with depressive symptoms had greater increases in BMI values and waist circumference.

In an earlier study, Needham and colleagues (2005) extended previous research on the association between elevated body mass and depressive symptoms among adolescents and young adults after analyzing data from the National Longitudinal Study of Adolescent Health, which contained a sample of 18, 294 adolescents aged 11 to 21 years. The investigators reported a stronger positive association between elevated BMI and depressive symptoms existed among adolescents in lower grades. With the use of standardized psychiatric evaluations, Pine and associates (2001) followed for 10 to 15 years two age- and gender-matched groups of children 6 to 17 years old with either

elevated depressive symptoms ($n = 87$) or no psychiatric symptoms ($n = 90$) and determined that a significant positive association existed between childhood depression and increased BMI in adulthood. In a later cohort study of 9,374 adolescents, Goodman and Whitaker (2002) investigated whether depressive symptoms predicted the development of elevated BMI at one-year follow-up; the authors reported that having increased depressive symptoms at baseline independently predicted elevated BMI at follow-up.

Cortese and colleagues (2009) examined the relationship between body mass and depressive symptoms in 678 young adolescents aged 11 to 14 years. In the total sample, the investigators found a significant association between BMI z-scores and Children's Depression Inventory scores. The lowest depressive symptom scores were evident for BMI z-scores around -1, indicating that a moderately underweight status is associated with the lowest severity of depressive symptoms.

Hasler and colleagues (2005) tested the hypothesis that depressive symptoms during childhood were associated with weight gain and obesity during young adulthood. Their study included 591 young adults followed for 20 years. The findings revealed that, among women, depressive symptoms before age 17 years were associated with increased weight gain. In a few studies, researchers identified positive relationships between depressive symptoms and body mass in children and adolescents; however, these relationships existed in girls only (Erikson, Robinson, Haydel, & Killen, 2000; Xie et al., 2005). Few studies involved examining depressive symptoms and development of an elevated body mass over time in both children and adolescents (Goodman & Whitaker, 2002; Pine et al., 1997; Pine et al., 2001; Richardson et al., 2003). Most studies grouped

school-aged children with adolescents instead of specifically focusing on middle school-aged children (10 to 12 years of age).

Depressive Symptoms and Central Adiposity

Earlier examinations of the relationship between depressive symptoms and central adiposity focused primarily on adults (Everson-Rose et al., 2009; Hach, Ruhl, Klotsche, Klose, & Jacobi, 2006; Kahl et al., 2005; Lee et al., 2005; Thakore et al., 1997; Weber-Hamann et al, 2002), and only a limited number of studies have been conducted with school-aged children (Bahreinian, Ball, Colman, Becker, & Kozyrskyi, 2011). Because of the dearth of studies in children, this section contains a review of studies in the adult population.

Researchers examined the association between depressive symptoms and central adiposity in exclusive groups based on gender (Everson-Rose et al., 2009; Lee et al., 2005; Lapidus, Bengtsson, Hallstrom, Björntorp, 1989). For example, Lee and colleagues (2005) found a significant association ($p < .001$) between depressive symptoms and increased waist circumference in a sample of 101 overweight premenopausal women. Similarly, in a later study, Everson-Rose and associates (2009) determined a strong association ($r = .77$, $p < .001$) between depressive symptoms and increased waist circumference in a sample of 409 overweight or obese middle-aged women. Lapidus and associates (1989), in their study of community dwelling women in Sweden, found that increased depressive symptoms were more strongly associated with central adiposity than with total body adiposity. Those authors compared 1,743 women with elevated BMI with 89 women classified as normoweight. In a prospective study of

2,088 participants, Vogelzangs and colleagues (2008) showed in a large population of adults that the presence of depressive symptoms at baseline was positively associated (β = .050, p = .009) with an increase in central adiposity independent of overall obesity; in addition, significant associations existed in White men and women but not in African American women. However, conflicting findings from a recent study by Wong and colleagues (2011) indicated that depressive symptoms resulted in a decrease in abdominal obesity or central adiposity in a sample of 3,998 Chinese elderly men and women. Needham and associates (2010) examined data from a sample of 4,643 participants from Years 5, 10, 15, and 20 of the Coronary Artery Risk Development in Young Adults (CARDIA) study and found a relationship between higher levels of depressive symptoms at baseline and increases in BMI in Caucasians. They also noted a significant positive association between depressive symptoms and increases in waist circumference in both African American and Caucasian groups.

As shown, earlier studies of the relationship between depressive symptoms and central adiposity yielded equivocal results for adults. To date, limited studies have been done with children and adolescents; in one, Bahreinian and associates (2011) found in 431 children aged 11 to 14 years a 10-cm increase in waist circumference of girls who reported higher depressive symptoms; in boys, increased waist circumference was not associated with depressive symptoms. The paucity of studies with adolescents and children prevents the support of any conclusions.

Depressive Symptoms and Cortisol

The literature examining the relationship between depressive symptoms and cortisol has been equivocal, with some studies showing no significant association (Van Eck et al., 1996). However, some studies showed positive associations between depressive symptoms and cortisol, primarily those involving adults (Deuschle et al., 1997; Otte et al., 2004; Young et al., 2001). To date, limited studies have examined the relationship between cortisol and depressive symptoms in children and especially in school-aged children (Forbes et al., 2006; Goodyer, Herbert, Moor, & Altham, 1991; Goodyer, Park, & Herbert, 2001; Puig-Antich et al., 1989).

In an experimental study, Puig-Antich and associates (1989) examined plasma cortisol concentration in prepubertal children with major depressive disorder (MDD). Children aged 6 to 12 years were studied for cortisol hypersecretion during an episode of elevation in depressive symptoms. The authors measured plasma cortisol concentrations at 20-minute intervals over a 24-hr time period and identified hypersecretion of cortisol in only four children with depressive symptoms (Puig-Antich et al., 1989). In a prospective longitudinal study, Goodyer and colleagues (1991) determined the secretory pattern of cortisol in children and adolescents aged 8 to 16 years with major depressive disorder. Salivary cortisol samples were collected at 4-hr intervals over 24 hr both when the participants experienced high levels of depressive symptoms and when they recovered from symptoms. Cortisol levels were significantly higher in the older group of participants. Rao and colleagues (2008) studied 55 adolescents (30 with major depressive disorder and 25 healthy participants) for variability in HPA functioning attributed to experiential factors. Before and after measuring the Trier Social Stress Test

(TSST) in both groups, cortisol concentrations were measured via saliva samples. Children with increased depressive symptoms exhibited elevated and prolonged cortisol secretion in response to the TSST.

Studies of the relationship between depressive symptoms and cortisol used a variety of cortisol sampling techniques; as a result, findings prove inconsistent. These techniques include urinary, salivary, and serum samples measured at different times. Because of the variations in measuring cortisol levels, comparison of findings presents a challenge.

Mediating Effect of Cortisol on Relationships Between Psychological Stress and Body Mass and Between Psychological Stress and Central Adiposity

Although several groups of investigators have examined the direct relationships between psychological stress and body mass (Block, He, Zaslavsky, Ding, & Ayanian, 2009; Kivimäki et al., 2006; Kouvonen, Kivimäki, Cox, Cox, & Vahtera, 2005; Vicennati, Pasqui, Cavazza, Pagotto, & Pasquali, 2009) and between psychological stress and central adiposity (Brunner, Chandola, & Marmot, 2007), the few studies of biological pathways that may link psychological stress, cortisol, and body mass or central adiposity in humans (Brown, Varghese, & McEwen, 2004) have been limited to adults. In their experimental laboratory study, Epel and colleagues (2000) subjected 59 healthy postmenopausal women (30 with a high waist circumference and 29 with a low waist circumference) to a stress-inducing protocol. Results noted that, independently of BMI, women with a high waist circumference had significantly higher cortisol levels associated with stress and felt more threatened under stress; controlling for this high threat appraisal

partially attenuated the association between waist circumference and cortisol levels. In a later study of 36 women, Vicennati and associates (2009) investigated the dynamics of weight gain and HPA activity in women who developed weight gain after a stressful event; the authors found that women who had an elevated BMI at the onset of weight gain after an important stressful event experienced rapid onset of obesity (as indicated by high BMI scores and waist circumference measures) and over-activated HPA function. Additionally, women with elevated BMI had significantly ($p < .001$) higher than normal urinary free cortisol levels compared to a nonstress-related obesity group and normal weight group.

Associations have been found between cortisol and body mass and between cortisol and central adiposity; however, it is unclear whether cortisol mediates the relationships between psychological stress and body mass and between psychological stress and central adiposity (Björntorp, 2001; Talbott, 2007). Pathological activation of the HPA axis, such as occurs in Cushing's syndrome, is associated with increased central adiposity (Kumari, Chandola, Brunner, & Kivimäki, 2010). However, in nonclinical populations, the reported associations of salivary cortisol levels and anthropometric measures such as BMI and waist circumference remain equivocal. For example, Wallerius and colleagues (2003) described a positive association of morning cortisol levels with waist circumference in 28 healthy men, and Steptoe and associates (2004) reported an association between cortisol awakening response (CAR) and waist circumference in 58 healthy White men but no association in healthy White women or in cortisol measures throughout the day. Similarly, Therrien and colleagues (2007) described a positive association of the CAR in 51 men but found no association in 31

women. In a recent study, Baltrus and colleagues (2010) examined the relationships among socioeconomic status, psychological stress, cortisol, and abdominal fat deposition in a sample of 1,850 African American and White American women (921 in the urban area and 929 in the rural area). Among White women, those less educated had a waist circumference larger ($p < .05$) than that of more highly educated women. African American women tended to have a greater BMI and waist circumference and lower cortisol levels than White women were found to have.

In one of the few studies of the relationship among psychological stress, cortisol, and central adiposity in children, Donoho and associates (2011) examined the independent and interactive effects of stress and cortisol in predicting central adiposity in a sample of Hispanic female participants aged 8 to 11 years. The authors found that chronic stress such as school-related stress in children was associated with high cortisol awakening response (CAR) but not in children with low CAR. Similar to job stress in adults, school-related stress in children may contribute to central adiposity, especially for girls with high CAR.

To date, no studies have involved examining cortisol as a mediator in the relationships between psychological stress and body mass, as well as in the relationships between psychological stress and central adiposity. Thus, whether cortisol plays a mediating role in the relationships among these variables remains unclear.

Mediating Effect of Cortisol on Relationships Between Depressive Symptoms and Body

Mass and Between Depressive Symptoms and Central Adiposity

In studies of the direct relationships between depressive symptoms and body mass and between depressive symptoms and central adiposity, investigators have noted positive associations (Everson-Rose et al., 2009; Hach, Ruhl, Klotsche, Klose, & Jacobi, 2006; Lee et al, 2005; Needham et al., 2010). Some researchers proposed that high cortisol levels may serve as the link between depressive symptoms and central adiposity (Björntorp, 2001; Brown, Varghese, & McEwen, 2004). Study samples consisted of primarily adults; fewer included children. In an earlier study, Rosmond (1997) reported significant associations between central adiposity and depressive symptoms in middle-aged men. Thakore and associates (1997) examined normal-weight women with elevated depressive symptoms and concluded that these women had more than twice the cortisol level and intraabdominal fat of those without depressive symptoms. In a cross-sectional study, Muhtz and colleagues (2009) examined the association of current depressive symptoms, circadian cortisol levels, and variables of the metabolic syndrome in 215 men ($n = 107$) and women ($n = 108$). Post hoc ANOVA analysis revealed that in comparison with women without depressive symptoms, women with such symptoms had larger waist circumferences and higher salivary cortisol levels. Similarly, results of a later study by Rivenes and associates (2009) showed an association between central adiposity and depressive symptoms in both men and women; the researchers provided an explanation for their findings that support the hypothesis that cortisol and metabolic pathways involving the HPA axis play roles in the relationship between depressive symptoms and central adiposity. One study consisted of examining cortisol as a mediator

between depressive symptoms and body mass and/or central adiposity in children (Dockray, Susman, & Dorn, 2009). Dockray and coauthors (2009) examined the relationships among depressive symptoms, physical activity, BMI, and cortisol as a mediator among these variables in their sample of 111 children and adolescents aged 8 to 13 years. The authors assessed symptoms of depression by using the Child Behavior Checklist and measured cortisol reactivity by the Trier Social Stress Test. Findings revealed that depressive symptoms were positively associated with BMI in both girls and boys. In girls but not in boys, cortisol reactivity mediated the association between depressive symptoms and BMI (Dockray et al., 2009). Pervanidou and colleagues (2009) found a positive correlation between cortisol levels and depressive symptoms and confirmed the link between elevated BMI and depressive symptoms in children. In their study, salivary cortisol levels were measured five times a day in 50 obese children and obese adolescents. The 20 boys and 30 girls aged 8 to 15 years completed the Children's Depression Inventory to assess self-reported depressive symptoms. The findings indicated that elevated BMI and depressive symptoms may have an abnormal hormonal link that underlies the relationship between the two variables.

Potential Confounding Variables

Gender, ethnicity, puberty, and SES may exert an independent influence on body mass, central adiposity, and cortisol. Although not the focus of this study, these variables must be considered as possible confounding variables. This section contains a review of the research related to these variables and their outcomes.

Puberty

One potential confounding variable that may independently influence the outcomes of this study of 10- to 12-year-old children is puberty, a multifactorial process that encompasses a series of changes affecting biological, physiological, and emotional development in children (Hayward, 2003). The onset of puberty proves challenging to determine with some children initiating puberty as young as 6 years of age but with an average being 8 years (Hayward, 2003). This period is typically associated with the development of secondary sex characteristics (Hayward, 2003). In earlier studies, puberty has been assessed by Tanner staging (Baker, Birch, Trost, & Davison, 2007; De Ridder et al., 1992; Ribeiro, Santos, Duarte, & Mota, 2006), physical examination (Dockray, Susman, & Dorn, 2009; Kaplowitz et al., 2001), or self-assessment (Bonat et al., 2002; Brooks-Gunn, Warren, Rosso, & Garguilo, 1987; Carskadon & Acebo, 1993). Pubertal changes have been associated with increased body mass and cortisol (Kaplowitz et al., 2001; Knutsson et al., 1997; Netherton, Goodyer, Tamplin, & Herbert, 2004; Ribeiro et al., 2006).

Increased body mass and adiposity in children may influence various aspects of pubertal development (Solorzano & McCartney, 2010). Children with elevated body mass and central adiposity may experience an earlier onset of puberty (Biro & Wien, 2010; De Ridder et al., 1992; Herman-Giddens et al., 1997; Kaplowitz et al., 2001; Riberio, Santos, Duarte, & Mota, 2006). Kaplowitz and colleagues (2001) examined data from 17,077 girls aged 3 to 12 years and noted an earlier onset of puberty in young girls with elevated BMI. In their study, the authors measured girls for pubertal status by using physical examination. Age at pubertal onset is decreasing; although girls are more

extensively studied in this respect, reports also exist of trends in early pubertal onset in boys (Parent et al., 2003). In their study, Van Lenthe and coinvestigators (1996) examined the role of pubertal timing for adult fat phenotype in 177 in girls and boys. Results showed a higher BMI for children who matured early. Beunen and colleagues (1994) reported similar results from the Leuven Growth Study, which included 173 male participants; the investigators used age at peak height velocity (PHV) as an assessment of pubertal timing and demonstrated higher BMI in early maturers. In a later study of 819 children, Ribeiro and associates (2006) found a relationship between early sexual maturity and overweight in both girls and boys. Early sexual maturity or pubertal status was assessed with the use of physical examination and Tanner staging in the Ribeiro and coworkers (2006) study.

Puberty may independently activate the HPA axis and subsequently impact cortisol production (Kiess et al, 1995; Knutsson et al. 1997; Netherton, Goodyer, Tamplin, & Herbert, 2004). Netherton and colleagues (2004) studied the basal levels of cortisol in relation to pubertal development in 129 children and adolescents aged 8 to 16 years; the authors found cortisol levels to be highest in the postpubertal stage of development, more notably evident in girls. In a similar study, Adam (2006) found that, of 52 adolescents, participants at a higher pubertal stage of development produced higher levels of cortisol. Cortisol values increased by 23% with every point of increase on a 5-point pubertal-development scale. However, Rosmalen and associates (2005) did not find an association between puberty and cortisol levels for either boys or girls in 1,768 participants aged 10 to 12 years.

In summary, evidence suggests that puberty may independently influence cortisol, body mass, and central adiposity. Thus, puberty was controlled in order to examine the effects of psychological stress and depressive symptoms on body mass and central adiposity beyond the influence of puberty. Although puberty is a factor that has been noted to be associated with elevated body mass and central adiposity, much of the variability in body mass and central adiposity is not explained by puberty; thus, other factors which influence body mass and central adiposity should be examined.

Socioeconomic Status

Researchers have linked low socioeconomic status (SES) to adverse health outcomes (cardiovascular disease, diabetes, and psychological disorders) in adults (Cohen, Janicki-Deverts, & Miller, 2007; Melchior et al., 2011; Nicklett, 2011; Pollitt, Rose, & Kaufman, 2005; Vonneilich et al., 2011). Evidence exists for a link between low SES and HPA stimulation with resultant higher cortisol levels in children (Fernald & Gunnar, 2009; Lupien, King, Meaney, & McEwen, 2001). Children from families with low SES reported greater exposure to stressful life events, and these children can experience subsequent increases in cortisol secretion (Lupien, King, Meaney, & McEwen, 2001). In several studies, researchers linked elevated cortisol levels to underlying mechanisms that resulted in adverse health outcomes such as cognitive decline, cardiovascular disease, immunosuppression, and insulin resistance (Lupien et al., 1998; Rosmond, 2005; Rosmond & Bjorntorp, 2000). In a study of 217 school children aged 6 to 10 years, Lupien and associates (2001) found that lower family income was associated with higher morning cortisol levels. Children in families with lower SES had significantly higher levels of salivary cortisol; however, the relationship did not exist and,

in fact, appeared reversed in children aged 12 to 16 years (Lupien et al., 2001). Evans and Kim (2007) reported a relationship between lower family income and increased levels of cortisol in a sample of 287 children aged 8-10 years. In a later study of cortisol and SES, Evans and Kim (2007) found a significant relationship between duration of poverty and higher cortisol levels, after controlling for wave 1 cortisol values. Finally, in a study of 50 children aged 9- to 18- years-old, Chen and colleagues (2010) found, over a 2-year span, cortisol levels in low-SES children nearly doubled those of high-SES children; moreover, the researchers found associations between SES and cortisol trajectories most pronounced in postpubertal children.

Results of several studies showed a relationship between low SES and abdominal fat deposition in adults (Baltrus et al., 2010; Chen & Tunstall-Pedoe, 2005; Cohen, Doyle, & Baum, 2006). Reports also indicated profound effects of low SES on myriad mental and physical health outcomes in children (Baltrus, Everson-Rose, Lynch, Raghunathan, & Kaplan, 2007). For instance, higher BMI appeared more prevalent in the lower SES segments of the United States (Rutt & Coleman, 2005; Sallis, Zakarian, Hovell, & Hofstetter, 1996; Wang, 2001). Results also indicated a clear inverse relationship between SES and BMI in children and adolescents (Booth, Macaskill, Lazarus, & Baur, 1999; Moore, Howell, & Treiber, 2002). In their study, Moore and coinvestigators (2002) examined the effects of race, gender, and SES on changes in youth overweight over a 7-year period in a longitudinal study of cardiovascular risk factors. Participants at the beginning of the study had a mean age of 8.8 ± 2.0 years and were followed for an average of 7.2 ± 0.5 years. Over the span of the study, BMI percentile increased significantly in both males and females; neither gender nor race predicted the

increase in BMI, except when the authors considered SES as a variable (Moore et al.,

2002). In one of the few studies of the relationship of SES and depressive symptoms to

increased BMI, Goodman and associates (2003) evaluated the public health impact of

SES on adolescent depression and obesity in a nationally representative sample of 15,112

adolescents; research results indicated large population attributable risks (PAR) for

depression and obesity for both income and education. For increased BMI, the adjusted

PAR for income was 32% and for education was 39%. The authors concluded that an

association existed between low SES and a significant disease burden in children

(Goodman et al., 2003).

Gender

Gender can confound results because it uniquely influences the relationships

among elevated body mass, SES, ethnicity, and psychological factors such as depressive

symptoms (Alleyne & LaPoint, 2004; Goldfield et al., 2007; Pesa, Syre, & Jones, 2000).

Thus, gender must be studied as a confounding variable. In their comprehensive review,

Wang and Beydoun (2007) provided a report of trends relative to gender, age, SES,

ethnicity, and geographic regions in the United States on the basis of national data. Their

analyses revealed that between 1971-1974 and 1999-2002, children and adolescents in

the nation had a BMI that increased an average of 1.4 points and 2.0 points among

adolescent boys and girls, respectively. A study by Martin and Ferris (2007) consisted of

examining relationships among adult obesity, childhood overweight, and food insecurity

in a convenience sample of 200 parents and their 212 children, aged 2 to 12 years. The

authors found that being female and having an obese parent doubled the likelihood of a

child's being overweight (OR = 2.56, *p* = .01; OR = 2.32, *p* = .02).

Researchers also noted a link between differences in cortisol levels and gender. Netherton and colleagues (2004) found in their study of 129 children aged 8 to 16 years that salivary cortisol levels were higher in girls than boys. Furthermore, Rosmalen and associates (2005) measured cortisol levels five times a day in 1,768 children aged 10 to 12 years. The authors found that, in comparison with males, females had greater cortisol levels at all five times; also, significant differences were noted in three of the five measurements (Rosmalen et al., 2005).

Ethnicity

Among the factors frequently associated with body mass and central adiposity is ethnicity. Individuals of different ethnicities have significant differences in body mass and central adiposity so that, the influence of this confounding variable must be considered (Freedman et al., 2005; Gordon-Larsen, Adair, & Popkin, 2003; Lutfiyya et al., 2008).

A recent study by Saab and coworkers (2011) consisted of examining trends in prevalence and the odds of elevated body mass index (BMI) and obesity among ethnically diverse adolescents. The authors studied data from countywide (Miami-Dade) health screenings from 1999 to 2005 in 77,050 adolescents (51.0% girls, 9.4% White non-Hispanic, 59.2% White Hispanic, 16.4% African American, 7.0% Black Hispanic, and 8.0% Black Caribbean). Overall, the analyses revealed that, in comparison with White non-Hispanic youths, youths from minority backgrounds had greater odds of obesity and that differential rates occurred among minority groups (Saab et al., 2011).

In a secondary data analysis of the National Survey of Children's Health (NCSH), Lutfiyya and colleagues (2008) noted significant differences in overweight and obesity

(elevated body mass) among African American, Hispanic, and White children. Analyses

revealed that, in comparison with their White peers, African American and Hispanic

children had higher BMI values. This finding partially supports previous work by

Dennison and coworkers (2002), who found mean television/video viewing time higher

for African American and Hispanic children than for White children and who concluded

that this increased the risk for greater body mass in the first two populations. Several

studies showed that menarche occurred earlier in African American than in White girls

and that, a widening of this difference developed over the past few decades (Freedman et

al., 2002; Styne, 2004). Investigators involved in the Bogalusa Heart Study reported a

spread of 2 months in the 1970s, which grew to 4 months in the 1990s (Freedman, 2002).

Such changes might result from a greater increase in average weight for African

American girls than for White girls, possibly because of nutritional-supplementation

programs. Moreover, Russell and coworkers (2001) reported a greater advancement in

bone age for African American girls than for White girls. In a recent study, Anderson and

colleagues (2011) investigated data collected from 918 adolescent girls from the sixth and

eighth grades in the Trial of Activity for Adolescent Girls (TAAG). The authors

examined the bidirectional associations between elevated BMI and depressive symptoms

in girls and determined whether associations differed by racial/ethnic groups (Black,

Hispanic, and White girls in middle schools). Racial/ethnic group proved a statistically

significant effect modifier in both directions of association ($p < .02$). The strongest

associations between depressed mood and obesity were seen for White girls (Anderson et

al., 2011).

Although some research suggests that elevated BMI in boys and girls may be becoming stable, a study by Madsen and her colleagues (2010) yielded findings that certain racial and ethnic groups may have increasing rates of obesity. Based on ethnicity, the investigators found that, in comparison with White girls, Black, Hispanic, and American Indian girls face two to three times higher odds of having a high BMI. A disparity was noted in the highest BMI category, which encompassed 1.3% of White girls, 4.6% of Black girls, and 4.9% of American Indian girls (Madsen, Weed, & Crawford, 2010). Although elevated body mass and central adiposity are evident across all ethnicities, there are differences among ethnic groups with some ethnicities affected more significantly than others. Thus, ethnicity could confound the influences seen in body mass and central adiposity from psychological stress, depressive symptoms, and cortisol.

<div align="center">Summary</div>

The literature relevant to this study yielded many reports noting the association between psychological stress and depressive symptoms. However, this relationship has been studied more in the adult population, with less focus placed on school-aged children. Studies of children often involve broad age groups that reach into the adolescent years.

Relationships among psychological stress, cortisol, body mass, and central adiposity have been reported; however, such associations have been noted primarily in adults and adolescents. Although the relationships among depressive symptoms, cortisol, body mass, and central adiposity have also been examined, much about these

relationships in children remains unknown. Instrumentation to measure psychological stress and depressive symptoms varied across studies, thus comparing findings across studies is difficult. In addition, some studies involved small samples calling into question the power to test the hypotheses.

Last, few studies consisted of evaluating cortisol as a mediator between psychological stress and body mass and between psychological stress and central adiposity. Investigators noted direct relationships between psychological stress and cortisol, between psychological stress and body mass, and between psychological stress and central adiposity. In addition, direct relationships were reported between depressive symptoms and cortisol, depressive symptoms and body mass, and depressive symptoms and central adiposity. Although researchers reported associations among these relationships, the results have been equivocal and have involved primarily adult studies. Thus, conclusions cannot be determined in studies involving children.

Therefore, this study involved examining the effects of psychological stress and depressive symptoms on cortisol, body mass, and central adiposity in 10-to 12-year-old children. Also investigated were the mediating effects of cortisol on the relationships between psychological stress and body mass, between psychological stress and central adiposity, between depressive symptoms and body mass, and between depressive symptoms and central adiposity. Additionally, because puberty, SES, gender, and ethnicity independently exert influence on the variables of study, they were controlled in the study.

CHAPTER 3

METHODOLOGY

The purposes of this study were to examine the influence of psychological stress and depressive symptoms on body mass and central adiposity in 10-, 11-, and 12-year-old children after controlling for gender, ethnicity, puberty, and SES. In addition, the principal investigator also sought to determine the mediating role of cortisol in the relationships among psychological stress, depressive symptoms, body mass, and central adiposity in this population. In this chapter, the research design, sample and setting, methods of data collection, instrumentation, protection of vulnerable subjects, data management, and data analysis are discussed.

Design

A descriptive, cross-sectional, predictive design was used to address the research questions. The research questions were as follows: (1) What is the relationship between psychological stress and depressive symptoms in 10-, 11-, and 12-year-old children?; (2) How much of the variance in body mass and central adiposity is explained by psychological stress and depressive symptoms in 10-, 11-, and 12-year-old children after controlling for gender, ethnicity, puberty, and SES?; (3) Does cortisol mediate the relationship between psychological stress and body mass (BMI), and/or between depressive symptoms and body mass (BMI) in 10-, 11-, and 12-year-old children after controlling for the confounding variables gender, ethnicity, puberty, and SES?; and

(4) Does cortisol mediate the relationship between psychological stress and central adiposity (waist circumference), and/or between depressive symptoms and central adiposity (waist circumference) in 10-, 11-, and 12-year-old children after controlling for the confounding variables gender, ethnicity, socioeconomic status, and puberty?

Few studies have examined relationships among the variables of psychological stress, depressive symptoms, and cortisol on body mass and central adiposity in school-aged children. Because children should not be randomized in groups based on psychological stress and depressive symptoms, a non-experimental, predictive design was appropriate for this study. Predictive designs are used to predict the value of one variable on the basis of values obtained from another variable or variables (Burns & Groves, 2005). According to Polit and Beck (2008), descriptive studies describe the relationships among the variables by directly examining samples of the population. In this study, data were collected by administration of three self-report questionnaires, collection of a saliva sample, and measurement of body mass index and waist circumference.

Setting and Sample

Characteristics of the Sample

The convenience sample for this study included 10-, 11-, and 12-year-old school-aged students recruited from three elementary schools and one middle school in a rural southeastern state. This age group (10-, 11-, and 12-year-old) is important to study because children undergo a transitional stage of development from childhood to adolescence (Ball & Bindler, 2008). In addition, children are in Erikson's stage of industry, which is a crucial period when they learn to develop a sense of self-worth (Ball

& Bindler, 2008). The schools selected represented various racial, ethnic, cultural, and socioeconomic characteristics. The sample was inclusive of fifth and sixth grade students who were able to assent to participate in the study and meet the following eligibility requirements: (1) current fifth or sixth grader and 10, 11, or 12 years of age, (2) mentally capable of signing assent and participation in the study, (3) capable of understanding, speaking, and responding in English, (4) cognitively capable of responding to instruments and following instructions, and (5) having parental consent. Children needed to be able to understand, speak, and respond in English because the psychological instruments were written in English, and all instructions for this study were delivered in this language. Exclusion criteria included: (1) non-English speaking children, and (2) children cognitively unable to complete instruments.

Characteristics of the Setting

The setting of this study was public elementary and middle schools that accommodated students in fifth and sixth grades. Arrangements were made with school principals and teachers for data collection periods prior to distribution of information packets. Efforts were made to collect data at times other than instructional time for required classes. Students were assembled for data collection during non-academic elective courses, such as art, band, orchestra, and home economics. Data collection also occurred during physical education class.

Sample Size and Justification of Sample Size

Multiple regression analysis was used to create a linear model for body mass and central adiposity in terms of the predictor variables (psychological stress and depressive symptoms), mediation variable (cortisol), and the confounding variables (gender,

ethnicity, puberty, and SES). In multiple regression analysis, the relationship between the criterion variable and the linear combination of explanatory variables is summarized by the coefficient of determination R^2 (Tabachnick & Fidell, 2007). This coefficient, ranging from 0 to 1, estimates the proportion of variability in the dependent variable explained by the linear combination of the explanatory variables (Tabachnick & Fidell, 2007). Larger R^2 values indicate that the linear model has accounted for greater amounts of the variance of the dependent variable. For testing a value of R^2, the following rule for the required sample size is: $N \geq 50 + 8m$, where N is the number of subjects and m is the number of predictors (Tabachnick & Fidell, 2007). Following this rule, this study required 122 = (50 + 8(9)) participants to detect a medium-size relationship between the dependent variable and the set of explanatory variables, with an estimated significance level of 0.05 and at 80% power. A medium effect size was deemed feasible based on a previous study with similar variables of interest such as depressive symptoms, cortisol levels, and BMI in a group of children aged 8- to 13-years (Dockray, Susman, & Dorn, 2009). A more accurate estimate of the detectable effect size based on multiple regression techniques can be calculated using PASS (Power Analysis and Sample Size) software (Hintze, 2008). According to this calculation, a sample size of 136 achieves 80% power to detect an R^2 of 0.11 (|R| ≈0.31, a medium effect size) attributed to nine independent variables using an F-test with a significance level of 0.05 (Hintze, 2008). Based on the calculation using PASS software, 136 participants will be enrolled for the study. Figure 2 illustrates detectable values of R^2 with sample sizes ranging from 100 to 150 with nine predictors.
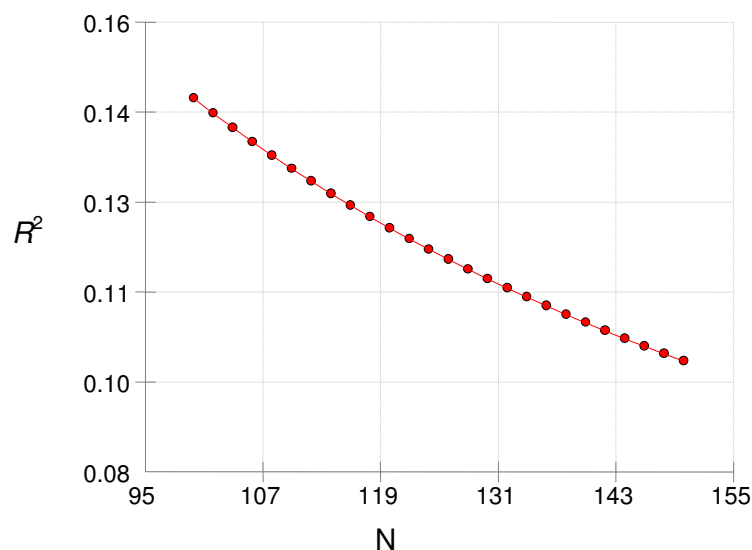
Figure 2. Power Analysis: Detectable values of R^2 attributed to 9 predictors, for sample sizes ranging from 100 to 150, at alpha=0.05 and with 80% power. Calculations were conducted in PASS 2008 (Hintze, 2008) software.

Protection of Vulnerable Subjects

Approval of the proposal for protection of human subjects was obtained from the Institutional Review Board at the University of Alabama at Birmingham, prior to the initiation of this study. Additionally, the study was reviewed and approved by the Superintendent of the school system.

The psychological risks associated with participation in the study included possible emotional upset while responding to the depressive symptoms and psychological stress questionnaires. Children were informed that there were no penalties or consequences if they chose not to answer a question that might cause them to feel uncomfortable. The risks associated with the physiological procedures were described in the parent consent form. Also described in the consent form, participants could have experienced some psychological discomfort or potential embarrassment when height,

weight, and waist circumference were measured. Efforts were made to ensure privacy when physical measurements were obtained. All physical measurements were collected behind privacy screens. Privacy screens were used to deter children from observing each other and to protect access to information by other children. The risks were discussed with the children prior to their signing of the assent form. Children were given an opportunity to ask questions and to resign from the study at any time, without repercussions. Only children who had parental consent were allowed to provide assent. The assent form was signed in the presence of the principal researcher. If either parental consent or child assent was not granted, the child was not enrolled in the study.

Data were collected in a designated area (classroom or conference room) to ensure privacy. During completion of questionnaires, children were seated in separate desks and given cover sheets to prevent children from viewing each other's responses. The school counselor and school nurse were informed about data collection procedures and requested to assist if children became upset as a result of responding to survey items about depressive symptoms and psychological stress.

Confidentiality was maintained by the use of a numerical coding system in the database. The names of participants were known only to the principal researcher. Neither the participant's name nor any identifying information appeared on any data collection sheet or within any database. The data was accessed by the principal researcher who has password access to the computer. Raw data related to the research was secured in a locked cabinet when not in use. The data were analyzed and reported collectively as a group, without identification of schools or teachers

Pilot

A pilot study was conducted during spring 2011 to determine reliability and validity of instrumentation. Additionally, the pilot study helped to address potential issues with participant recruitment and the data collection process. The sample was comprised of 30 10-12 year-old students recruited from an elementary school in a rural southeastern state. From a pool of 76 eligible participants, 43 students responded; however, 13 parent consent forms were incomplete. Thus, 30 students participated in the study. Of the 30 children recruited, 12 (40%) were male and 18 (60%) were female. The number of participants in each ethnicity group was as follows: 20 (66.7%) Caucasian; 9 (30%) Hispanic; and 1 (3.3%) Native American.

The pilot study included the following instrumentation: physiological and physical measurement (cortisol, height, weight, and waist circumference), the Children's Depression Inventory (CDI), The Feel Bad Scale (FBS), and the Self-Administered Rating Scale for Pubertal Development. Descriptive statistics of physiological and physical data were as follows: Body mass index mean was 22.8 (range of 15.0 to 38.1); waist circumference mean was 32.58 inches (range of 21.438 to 42.188). Cortisol concentration level mean was .128 (range of .002 to .314). A Pearson correlation coefficient was calculated to determine the relationship between participants' depressive symptoms (CDI scores) and psychological stress (FBS scores). A positive correlation was found ($r = .475$, $p < .01$), indicating a significant linear relationship between the two variables. Children, who reported higher levels of depressive symptoms, also reported increased psychological stress.

Using Cronbach's alpha to determine reliability, the CDI had a level of reliability at .822 and is considered to be reliable for this age group and thus, was used in the full scale study. Additionally, The Feel Bad Scale had a Cronbach's alpha of .896 and was considered to be highly reliable for the full scale study. The Rating Scale for Pubertal Development had an acceptable reliability level of .749; however, the children had difficulty with certain terminology within the questionnaire. For example, children had questions about the term "underway" on the pubertal development questionnaire. For the full scale study, additional instructions about the completion of questionnaires were provided to children. Also, research assistants were available to assist children who had questions about the instruments during the larger study.

<div align="center">Instrumentation</div>

The variables in this research study included: body mass, central adiposity, cortisol, psychological stress, and depressive symptoms. Data were also collected on gender, ethnicity, SES, and pubertal status; information about these variables is important to study because of their potential influence on body mass and central adiposity. Instruments in this study were selected based on the conceptual framework, reliability and validity, and feasibility of instrumentation.

Demographic Information

In addition to its use as a screening device, the demographics questionnaire was used to assess participants' age, grade level, ethnicity, gender, and parents' occupation and level of education. Information on the demographic questionnaire was completed by parents and returned with the consent form. Parents marked a box on the form that

corresponded to the child's age in number of years. Also, parents marked a box to indicate the child's grade level and gender. For ethnicity, there were five different ethnic groups (African American, Asian, Caucasian, Hispanic, and Native American), from which the parent chose the group that reflected the child's ethnicity. In addition, there was a blank space to write in the child's ethnicity if the ethnic group was not listed on the questionnaire.

To assess SES, the Nam-Powers-Boyd Occupational Status Scale was used. The Nam-Powers-Boyd Occupational Status Scale scores provide equal weight to education and earnings (Nam & Boyd, 2004). Moreover, the Nam-Powers-Boyd occupational status scores range from 0 to 100 and have a specific and clear interpretation; they can be regarded as the percent of individuals in the civilian labor force who are in occupations that have composite levels of education and earnings below the reference occupation (Nam & Boyd, 2004). For example, managerial and professional occupations tend to have the highest scores, while service and production occupations tend to have the lowest scores. The highest score of 100 is found for dentists, physicians, and surgeons; the lowest score is assigned to counter attendants in cafeterias and food concessions. Parents were asked to report their occupation; a blank space was provided on the questionnaire for parents to record occupation. Parental report of occupation is noted to be a reliable source of information to determine SES (Lien, Friestad, & Klepp, 2001). According to Hauser and Warren (1996), respondents are more willing and able to describe occupations, which can also be reliably coded. Based on the occupation reported by the parent on the demographic questionnaire, a score was provided for the occupation listed

on the Nam-Powers-Boyd Occupational Status Scale. Demographic data were used to describe the sample and to control for confounding variables.

Cortisol

Cortisol was measured by salivary specimens. Collection of cortisol from saliva followed protocol established by Parameter Cortisol Assay (2012) and the literature (Hanneman, Cox, Green, & Kang, 2011; Hanrahan, McCarthy, Kleiber, Lutgendorf, & Tsalikan, 2006) describing the collection of saliva sample to measure cortisol. Participants' salivary samples were collected within a one-hour time frame due to effects of circadian rhythmic fluctuations. Sample collection was avoided within 60 minutes after participants' consumption of a meal. The participants were instructed to rinse their mouth thoroughly with water 10 minutes before the sample was collected; this procedure minimized potential pH variability and bacterial contamination. As a group, the children were given 3 minutes to provide the specimen. Children were asked to direct their saliva accumulations from their mouth into a short straw that drained into a polypropylene vial. After three minutes, the vials were collected, secured in a container, and placed in a Styrofoam cooler with dry ice for transportation to a freezer with a temperature set at -20°C or lower for storage and later batch processing. Samples were placed in a freezer within four hours after collection.

Cortisol levels from saliva specimens were analyzed by high-sensitivity enzyme immunoassay with the use of commercially prepared kits (Parameter Cortisol Immunoassay Six-Pack; item number SKGE008) from R & D Systems (2012). A research assistant at the University of Alabama at Birmingham-Department of Physiology and Biophysics performed the cortisol assays. The research assistant had over twenty-one

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years of laboratory and experimental analyses experience and fifteen years of ELISA assays experience. The sensitivity of the cortisol assay was reported, and the minimum detectable dose of cortisol ranged from 0.030-0.111 ng/mL (Parameter, 2012). The assay kit measures the bound cortisol peroxidase to the goat anti-mouse antibody coated on a microtitre plate. Cortisol levels were obtained and actual ranges from participants were assessed by comparing them with expected morning range of cortisol (1.69 to 12.81 nmol/l) in children (Safarzadeh, Mostafavi, & Haghi, 2005).

All reagents and the microtitre plate were brought to room temperature. The microtitre plate included 96 available wells. The first 10 wells were reserved for the standards, zero standard, and non-specific binding (NSB) wells that were packaged with the kit. Each plate was coated with monoclonal antibodies to cortisol. Cortisol in the standards and unknown wells competed with the cortisol linked to the horseradish peroxidase for the antibody-binding sites. The standards were reconstituted with 1.0 mL of distilled water that produced a stock solution of 100 ng/mL. Calibrator Diluent RD5-43 (900 µL) was pipetted into the 10 ng/mL tube, and 500 µL was pipetted into the remaining tubes. The 100 ng/mL standard stock was used to produce a dilution series that resulted in cortisol concentrations of 10 ng/mL, 5 ng/mL, 2.5 ng/mL, 1.25 ng/mL, 0.625 ng/mL, 0.312 ng/mL, and 0.156 ng/mL. After the number of assays was determined, 200 µL of Calibrator Diluent RD5-43 was added to the NSB wells and to the zero standard wells. Next, 100 µL of standards and samples were placed in the appropriate wells. With a multichannel pipette, 50 µL of cortisol conjugate was added to each well. After the wells were filled with cortisol conjugate, 50 µL of primary antibody solution was added to each well except the NSB wells. After the wells were filled, the

plates were set on a microplate rotator set at 500 rpm to incubate for two hours. After the incubation was complete, the plates were washed for four times with a 1X wash buffer and blotted dry on paper towels after each wash. Next, 200 µL of substrate solution was added to each well with a multichannel pipette; the plates were then incubated in the dark at room temperature for an additional 30 minutes. Next, 50 µL of stop solution were added to each well with a multichannel pipette. Within 30 minutes, the plates were placed on a microplate reader at 450 nm. Cortisol level concentrations were then evaluated for all samples.

Height

The participant's height was measured with a portable stadiometer according to established recommendations (Heyward & Wagner, 2004). Participants were informed of how height would be measured and that the procedure would be obtained behind a privacy screen. Participants were asked to take off shoes and then stand at a right angle to the vertical rod of the stadiometer. The participant was asked to stand up straight with back against the vertical backboard and eyes focused straight ahead. Body weight was evenly distributed between both feet and arms. The arms hung freely by the sides of the body with palms facing the thighs. Heels were together with toes pointing outward at a 60 degree angle. When possible, the participant was instructed to have the back of the head, scapula, and buttocks touch the vertical backboard. As the horizontal board of the stadiometer was lowered to the superior point on the head compressing the hair, the participant was asked to inhale deeply while maintaining the head erect and eyes focused straight ahead. The measurement was recorded to the nearest ¼ inch.

Weight

Weight was measured by use of a freestanding weight beam scale behind a privacy screen. The scale was calibrated to zero prior to the participant stepping on the scale. The participant was asked to remove shoes and step on the platform and stand facing the balance beam. Body weight was evenly distributed between both feet. The scale weights were adjusted until the balance beams hung freely. The measurement was recorded to the nearest ¼ of a pound.

Body Mass Index

Body mass index (BMI), an indicator of body mass, was calculated during data analysis. Although, BMI is not a direct measure of adiposity, its use has been reported to be a valid predictor of adiposity in children (CDC, 2011; Pietrobelli et al., 1998). Despite the ease of use and popularity of BMI as an anthropometric tool, it has certain limitations. For example, BMI does not take into account the wide variation in distribution of body fat relative to body weight (Zhu et al., 2002). Hence, BMI measurement does not provide information with regard to where body fat is stored (Zhu et al., 2002), which is important in determining whether an individual has central adiposity; thus, waist circumference was also measured.

BMI was calculated based on the following formula: weight in kilograms (kg) divided by the square of height in meters (m^2) (CDC, 2011). After BMI scores were determined from height and weight, BMI z-scores, measures of relative weight adjusted for child's age and sex, were computed using an algorithm described within the CDC website (CDC, 2012). BMI z-scores were used in the interpretation of data analysis.

Waist circumference

Waist circumference, an indicator of central adiposity, was measured according to protocol by Hayward and Wagner (2004). The participant was measured behind a privacy screen. A flexible, non-stretchable tape measure was used to determine the participant's waist circumference. The principal researcher stood to the right of the participant and palpated the upper hip bone to locate the right iliac crest. Just above the uppermost lateral border of the right iliac crest, a horizontal mark was drawn, and then crossed with a vertical mark on the midaxillary line. The tape measure was placed in the horizontal plane around the abdomen at the level of the marked point on the right side of the trunk. The plane of the tape remained parallel to the floor at all points and was snug without compressing the skin. The participant was asked to breathe normally, and the measurement was recorded to the nearest 1/16 inch. Waist circumference was measured twice, and an average reading from the two measurements was calculated during analysis.

Rating Scale for Pubertal Development

Participants were measured for pubertal development with the use of a self-report rating scale, A Self-Administered Rating Scale for Pubertal Development (Carskadon & Acebo, 1993). The rating scale was developed individually for females and males based on five indices of pubertal growth. All participants were asked about growth, body hair, and skin changes (especially pimples). Males were asked about changes to voice and growth of facial hair, and females were asked about breast development. Additionally, females were asked about onset and age of menstruation. Each item was followed by five possible choices, of which the child selected by circling the response that most closely represented his or her physical growth. The five response options for each item are (1) "I have not yet begun", (2) "I have barely started", (3) "The change is definitely underway,

(4) "The change is complete", and (5) "I don't know" (Carskadon & Acebo, 1993). Each response has an assigned numerical value. Scores range from 0 to 20 with higher scores indicating a greater level of pubertal maturation. For females, the following point values are assigned as follows: Pre-pubertal = 3; Early Puberty = 3 without menses; Mid-pubertal = 4 without menses; Late Puberty = ≤ 7 with onset of menses; Post-pubertal = 8 with onset of menses (Carskadon & Acebo, 1993). For males, the point values are assigned as follows: Pre-pubertal = 3; Early Puberty = 4 or 5; Mid-pubertal = 6-8; Late pubertal = 9-11; Post-pubertal = 12. Children are considered pubertal if their scores are within mid-pubertal scores or greater.

To validate the instrument, the authors (Carskadon & Acebo, 1993) had physicians rate physical development based on Tanner stages. The Tanner stages represent physical development characterized by external primary and secondary sex characteristics (Tanner, 1962). Ratings on Tanner stages were correlated with scores on the self-ratings of the Self-Administered Rating Scale for Pubertal Development. The Spearman correlation coefficient between participants' self-ratings and pediatricians' ratings on physical development based on Tanner staging was high ($r = 0.868-0.841$, $p < 0.0001$) (Carskadon & Acebo, 1993). The correlations between the students' and pediatricians' ratings were strong with one stage of developmental difference between the comparisons. Significant correlations were also found between students and parents for measures on the scales. The internal consistency of the student version has been assessed by Cronbach's coefficient alpha with ranges from 0.67 to 0.70 (Carskadon & Acebo, 1993. As reported earlier, the Self-Administered Rating Scale for Pubertal Development

had a Cronbach's alpha coefficient of .749 in the pilot study that was conducted by the

principal researcher. Permission to use the scale was obtained from the author.

Feel Bad Scale (FBS)

The Feel Bad Scale (FBS) consists of two sets of 20 questions that measure

intensity and frequency of children's self-reported stress using a Likert-type scale (Lewis,

Siegal, & Lewis, 1984). The FBS has 20 items that originated from focus groups on fifth

and sixth graders' interviews about situations that "made them feel bad" (Lewis, Siegal,

& Lewis, 1984). The feel bad items include such statements as "being overweight or

bigger than others" and "feeling left out of a group". Participants were asked to report

how they would feel if the situation described happened to them and rate it on a Likert-

type scale. Participants were asked first to rate the intensity of the item by using a 5-

point scale with 1 representing *not bad* to 5 representing *terrible*. Next, children were

asked to indicate how often, if ever, the situation had occurred on a 5-point scale with 1

for *never* to 5 for *always*. The FBS scores are sums of values for each of the subscales

(i.e., the Intensity Scale and the Frequency Scale) and the total score is the sum of

subscale scores. Higher total scores indicate greater levels of stress.

Lewis and colleagues (1984) reported the ratings of internal consistency using the

coefficient alpha at .82 in a sample of 2,480 ethnically diverse group of children, and

factor analysis supported construct validity of the FBS (Lewis, Siegal, & Lewis, 1984).

In secondary data analysis of a study with 1,026 fourth-, fifth-, and sixth grade children,

an alpha of .85 was reported by Jenkins and Rew (2005).   Cronbach's alpha coefficients

were determined in a recent study by Rew and associates (2010) with a sample of

children in the fourth to sixth grades. The Cronbach's alpha value for the total score was

.84 (Rew, Horner, & Fouladi, 2010). In a recent pilot study conducted by the principal

researcher, a Cronbach's alpha coefficient was determined at .89 in a group of fifth and

sixth graders. Factor analysis supported construct validity of the scale (Lewis et al.,

1984). Factor analysis revealed three dimensions, comprised of items related to: 1)

anxieties surrounding conflict with parents; 2) self-image and peer relationships; and 3)

geographic mobility. The FBS is available for use without charge to the public domain;

however, permission to use the instrument was obtained from the author.

Children's Depression Inventory (CDI)

The Children's Depression Inventory (CDI) is a 27-item self-report questionnaire

designed to measure depressive symptoms in children aged 7-17 years-old (Kovacs,

1992). According to Hammen and Gotlib (1992), the CDI is the most widely used self-

report measure of depressive symptoms in children. The instrument encompasses

cognitive, affective, and behavior functioning of depression. Each of the items describes

depressive symptoms such as disturbance in mood, sleep, appetite, or interpersonal

relationships. In the present study, one modification was made to the original CDI, which

was the removal of the item that assesses suicidal ideation. This item was removed

because the question was of concern to some of the principals and this is in line with

other previous studies (Abela, Aydin, & Auerbach, 2007; Cole, Martin, Peek,

Seroczynski, & Hoffman, 1998; Jordan & Cole, 1996). Total scores range from 0 to 52.

A cutoff score of 13 has been suggested to indicate mild depressive symptoms and 19 to

indicate severe depressive symptoms (Kovacs, 1981). Higher scores indicate increasing

depressive symptoms severity. The inventory took 10 to 15 minutes to complete and was

administered to participants individually. Children were asked to select one of three

statements that best describe how they have felt over the past two weeks. The items are

scored 0 (absence of symptoms), 1 (mild symptoms), or 2 (definite symptoms). The CDI is available for purchase through Pearson Assessments at www.pearsonassessments.com. Permission to use the CDI is granted by the author once the instrument is purchased through the publisher.

The reliability of the CDI has been examined in terms of internal consistency, test-retest reliability, and standard error (Kovacs, 1992). The normative sample used for scoring the CDI included 1,266 public school students with a relatively even split of boys and girls divided into two groups (aged 7-11 and 12-17). The internal consistency coefficients ranged from .71 to .80, and the test-retest coefficients ranged from .74 to .83 spanning over a two-week time interval (Kovacs, 1992). Cronbach's alpha for the CDI in a normative sample was .86, indicating good internal consistency reliability (Kovacs, 1985). Kovacs (1985) found the Total Score coefficient alpha to be .86 with a heterogeneous, psychiatric referred sample of children, .71 with a pediatric-medical outpatient group, and .87 with a large sample of public school students. Abela (2001) reported moderate to high internal consistency in third and seventh graders; alpha coefficients ranged from .39 to .84 and .89 to .94, respectively.

Although less research is available in terms of validity, the CDI still has acceptable validity. The CDI has demonstrated strong correlations with measures of related constructs, specifically anxiety and self-esteem (Kovacs, 1992). The Coopersmith's Self-Esteem Inventory has been shown to be negatively correlated with the CDI (for girls, $r = -.72$, $p < .0001$, for boys, $r = -.67$, $p < .0001$) (Green, 1980). Intuitively, it makes sense that children who endorse higher depressive symptoms would have lower self-esteem. Saylor, Finch, Spirito, and Bennett (1984) determined the

concurrent validity of the CDI by administering the CDI along with the Piers-Harris Self

Concept Scale. The participants included 28 children (19 males, 9 females) in an

inpatient psychiatric setting. The correlation results with the Piers-Harris scores was

significant ($r = -.64$, $p < 0.05$) indicating that negative self-concept was highly related to

increased depressive symptoms as measured by the CDI. Self-report of low self-concept

corresponded consistently with higher CDI scores (Saylor, Finch, Spirito, & Bennett,

1984).

Romano and Nelson (1988) compared the CDI child form (CDI: C) and parent

form (CDI: P) to the Acenbach Child Behavior Checklist (CBCL) for discriminant and

concurrent validity. The authors found that all three tests in combination could

discriminate between children with depressive symptoms, children with other

psychopathologies, and normal children between 8- and 11-years-old. The groups

differed significantly, Wilks's lambda = .22, which is equivalent to $F(6, 32) = 6.10$, $p <$

.0002). A one-way analysis of variance (ANOVA) revealed a significant difference

among the three groups on the CDI-C, $F(2, 18) = 22.10$, $p < .0001$, supporting both the

initial diagnostic groupings and the concept that children can report on their depressive

symptoms.

Procedure

Prior to the study, approval from the Institutional Review Board at the University

of Alabama at Birmingham was granted. Approval of the study was also granted by the

Superintendent of the school system. An information packet was sent home to parents of

each student attending the fifth and sixth grades, ages 10, 11, and 12 years. The fifth and

sixth grade teachers of each school distributed this information packet to students in their respective grades as part of the parent-school communication process. The information packet included a letter explaining the purpose of the study and when data would be collected, a demographic questionnaire, and two parental consent forms (one for parents to return and one to keep for their records). Three days after the information packets were sent home, the principal researcher sent an email to teachers of the fifth and sixth grades. This email included a request for teachers to remind students to return signed parent consent forms and the demographic questionnaire. Returned forms were collected by the teachers on the sixth day after distribution. The teachers placed collected forms in a sealed box designated for the study that was located in the school's main office or principal's office.

On the day(s) of data collection, only those students with parental consent, child assent, and who met inclusion criteria participated in the study. Children were asked to provide assent to the study on the day of data collection. Participants were escorted to a designated area (empty classroom or conference room) within the school to ensure privacy. The assent process took place during homeroom, physical education period, or elective periods. Students were instructed that they could decline to participate in the study without consequences.

Physiological and physical measurements were collected first prior to psychological measurements because responding to questions about stress and feelings could potentially influence cortisol levels. The principal researcher and two adults were present during the data collection process; the two adults were available to help maintain order and to escort children to their class. Saliva samples were collected from children as

a group. The collection of salivary samples was explained to the children before the specimen was obtained. Data were collected at mid-morning (9:30 AM-11:00 AM) in order to standardize the time of collection and to potentially control for factors that may interfere with measurement (Hanneman, Cox, Green, & Kang, 2011; Hanrahan, McCarthy, Kleiber, Lutgendorf, & Tsalikian, 2006). Additionally, this time frame did not interfere with scheduled lunch time for children. Children did not consume anything for at least one hour before the sampling procedure. Then, children were asked to rinse their mouth and wait 3 to 5 minutes to re-establish a natural oral environment and pH level prior to sampling (Hanrahan et al., 2006). Each participant was given a pre-labeled tube with a straw inside and rubber band attached to the tube. As a group, children were instructed to direct their saliva into the specimen tube. The children were then instructed to chew on the rubber band or suck in the sides of their mouth if they had difficulty producing saliva.

After saliva samples were collected, each child's height, weight, and waist circumference measurement were obtained based on protocols described earlier. The Rating Scale for Pubertal Development was completed privately by each child behind the privacy screen. Each participant was given individual questionnaires to complete. Instructions and examples were provided prior to completion of the questionnaires. Psychological instruments, The Feel Bad Scale and Children's Depression Inventory, were completed by children individually at their desk and then collected by the principal researcher. After completion of data collection, children were given a choice of incentive items such as scented pencils, erasers, or notebooks.

Data Management and Analysis

Data Management

Data were collected from four schools on separate dates over a three month time span. To ensure integrity, data were reviewed for unintentional omissions and checked for errors in documentation by the principal researcher and research assistant at the data collection sites. Data were initially entered into a database using Microsoft Excel software. The data analysis package SPSS/PASW (Statistical Package for Social Sciences, 2010) 18.0 for Windows was used for all statistical analyses. All data were entered twice, into two separate files, and then compared for any discrepancies. A code book was created based on each author's instructions and guidelines for scoring of instruments. Raw data were maintained in a locked file cabinet and accessed only by the principal researcher.

Data Analysis

To ensure data quality, the data were examined to identify missing or invalid values and errors in data entry. In addition, each study variable was examined for normal distribution and outliers. Distributions for each variable were examined using descriptive statistics (frequencies, percentages, means, standard deviations, and range). Additionally, Cronbach's alpha coefficients were calculated to test for instrument reliability.

Each regression statistical model was analyzed for adherence to assumptions, such as existence, independence, linearity, normality, homoscedasticity, and multicollinearity. Several analyses were performed to address the research questions and hypotheses. The following is a description of the type of statistical analyses that addressed each research question and hypothesis.

Research Question 1: What is the relationship between psychological stress and depressive symptoms in 10-, 11-, and 12-year-old children?

Hypothesis 1: There is a positive relationship between psychological stress and depressive symptoms in 10-, 11-, and 12-year-old children.

For research question 1, bivariate correlation analysis was used to evaluate the relationship between the two variables in research question 1. The Pearson Product Moment Correlation Coefficient (r) was used to assess the relationship between psychological stress and depressive symptoms.

Research Question 2: How much of the variance in body mass and central adiposity is explained by psychological stress and depressive symptoms in 10-, 11-, and 12-year-old children after controlling for gender, ethnicity, puberty, and SES?

Hypothesis 2: Psychological stress and depressive symptoms will explain a significant amount of the variance in body mass and central adiposity in 10-, 11-, and 12-year-old children after controlling for gender, ethnicity, puberty, and SES.

For research question 2, hierarchical multiple regression techniques (R^2), controlling for gender, ethnicity, puberty, and SES, were conducted to determine the amount of variance explained by the variables psychological stress and depressive symptoms on body mass and central adiposity. In order to determine the amount of variance accounted for by the set of predictor variables, hierarchical regression techniques were used, entering the confounding variables gender, ethnicity, puberty, and SES on block 1 and the set of predictor variables on block 2.

Research Question 3: Does cortisol mediate the relationship between psychological stress and body mass (BMI), and/or between depressive symptoms and body mass (BMI)

in 10-, 11-, and 12-year-old children after controlling for the confounding variables gender, ethnicity, puberty, and SES?

Hypothesis 3: Cortisol will mediate the relationship between psychological stress and body mass (BMI) and/or between depressive symptoms and body mass (BMI) in 10-, 11-, and 12-year-old children after controlling for gender, ethnicity, puberty, and SES.

Research Question 4: Does cortisol mediate the relationship between psychological stress and central adiposity (WC), and/or between depressive symptoms and central adiposity (WC) in 10-, 11-, and 12-year-old children after controlling for the confounding variables gender, ethnicity, socioeconomic status, and puberty?

Hypothesis 4: Cortisol will mediate the relationship between psychological stress and central adiposity (WC), and/or between depressive symptoms and central adiposity (WC) in 10-, 11-, and 12-year-old children after controlling for gender, ethnicity, puberty, and SES.

For research questions 3 and 4, univariate regression models were used to determine the mediation of cortisol between the relationships described. A mediation model is a statistical process that helps to identify and explain the mechanism that underlies the observed relationship between an independent variable and a dependent variable through the inclusion of a third variable, known as a mediator (MacKinnon, 2008). The mediator variable serves to clarify the nature of the relationship between the independent and dependent variables (MacKinnon, 2008).

Baron and Kenny (1986) described the simple mediation model, which has been described widely for testing the direct effects of a mediator. According to Baron and

Kenny (1986), cortisol is deemed to function as a mediator (a) if there is a significant

relationship between the independent variables and the hypothesized mediator

Psychological Stress → Cortisol

Depressive Symptoms → Cortisol;

(b) if there is a significant relationship between the independent variables and the

dependent variables

Psychological Stress → Body Mass

Depressive Symptoms → Body Mass

Psychological Stress → Central Adiposity

Depressive Symptoms → Central Adiposity;

(c) if there is a significant relationship between the hypothesized mediator and the

dependent variables

Cortisol → Body Mass

Cortisol → Central Adiposity;

and (d) if the coefficient relating the independent variables (psychological stress and

depressive symptoms) to the dependent variables (body mass and central adiposity) is

reduced or becomes nonsignificant when the mediator (cortisol) is entered into the model.

Cortisol → Body Mass, after Psychological Stress is statistically controlled

Cortisol → Body Mass, after Depressive Symptoms is statistically controlled

Cortisol → Central Adiposity, after Psychological Stress is statistically controlled

Cortisol → Central Adiposity, after Depressive Symptoms is statistically controlled

The strongest representation of mediation will be present if the relationship of

psychological stress or depressive symptoms (independent variables) to body mass or

central adiposity (dependent variables) is zero when the mediating variable (cortisol) is included in the model (Baron & Kenny, 1986). Baron and Kenny (1986) purport that in psychological analyses, most phenomena are explained as multifactorial. Therefore, a mediating relationship of zero may be improbable, and any reduction in significance will reveal some support of mediation.

Building on the work of Baron and Kenny (1986), Sobel (1990) and MacKinnon (1994) presented statistical techniques that provided a more precise picture of the mediation path. MacKinnon and Dwyer (1993) further presented the test of the mediation path with introduction of the Sobel test. The Sobel test, a statistical test, is performed to determine the significance of the indirect effect of the mediator by testing the hypothesis of no difference between the total effect and the direct effect. The total effect is the sum of the direct and the indirect effects. The indirect effect of the mediator is the product of path ab as in Figure 3. Sobel's test provides an approximate significance test for the indirect effect of the independent variable on the dependent variable via the mediator. This statistical method allows for the amount of explained variance accounted for by the mediation to be estimated (Dudley, Benuzillo, & Carrico, 2004).
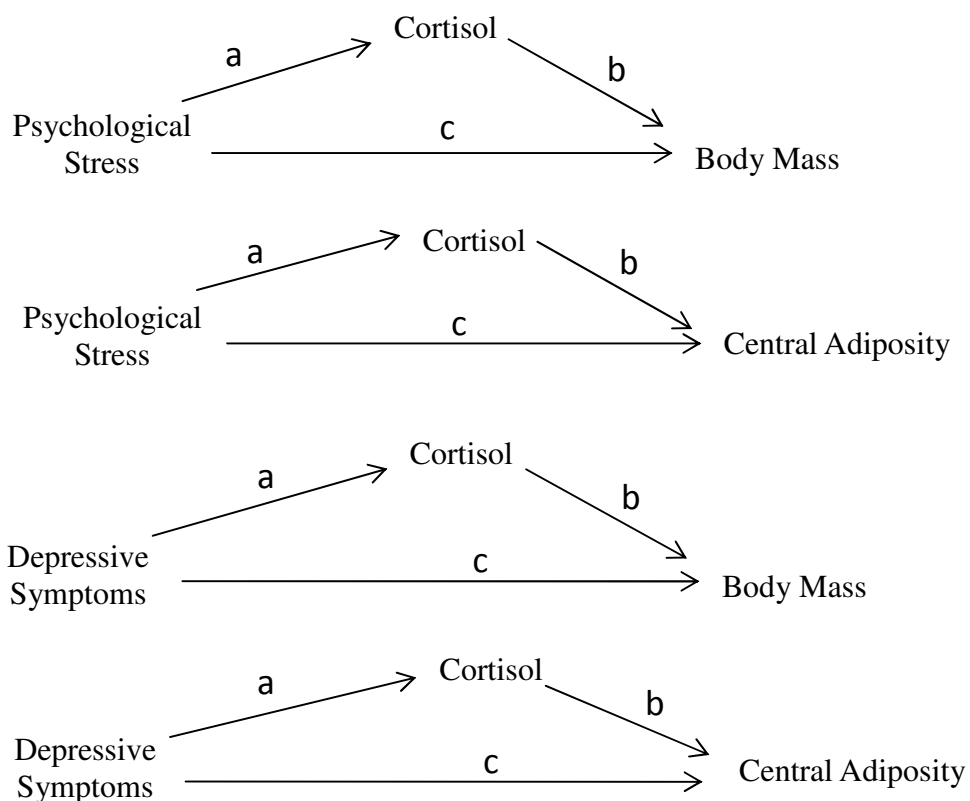
Figure 3. Conceptual model of cortisol as a mediator.

Summary

This chapter described the methodology for assessing the influence of

psychological stress, depressive symptoms, and cortisol on body mass and central

adiposity in 10-, 11-, and 12-year-old school-aged children. The rationales for the

research design, sample, setting, data collection methods, and instrumentation were

addressed. The plan for protection of vulnerable subjects was outlined. In addition, the

plan for data management and analysis was described.

CHAPTER 4

FINDINGS

The purpose of this study was to examine the influence of psychological stress, depressive symptoms, and cortisol on body mass and central adiposity in 10-, 11-, and 12-year-old children after controlling for gender, ethnicity, pubertal status, and SES. Additionally, cortisol was examined as a mediator in the relationships between psychological stress and body mass, between psychological stress and central adiposity, between depressive symptoms and body mass, and between depressive symptoms and central adiposity. The findings from the study are addressed in this chapter. First, the characteristics and demographic information of the participants, including age, gender, race/ethnicity, and pubertal status are described by using frequencies and descriptive statistics (mean, range, and standard deviation). Then, descriptive information about the study variables is included using descriptive statistics. Reliability information for the instruments for this study is discussed. The study variables and the relationships among the variables are presented. Last, a summary of the findings of each specific research question and hypothesis is presented. A lack of statistically significant relationships between psychological stress and cortisol and between depressive symptoms and cortisol precluded the need for additional testing of cortisol for mediation among the study variables.

Sample and Setting Characteristics

A convenience sample of 147 children was recruited from a southeastern city school system in the United States. Letters and consent forms were sent via the schools' communication protocol to all of the parents of 524 children enrolled in the fifth and sixth grades of three elementary schools and one middle school. The parents of 150 of the children consented to their child's participation in the study. Of those 150 children, every child met the eligibility requirements and was enrolled in the study. At the time of data collection, 147 children were present and assented to participate. The final sample was composed of 147 (84 female and 63 male) children, which represented a participation rate of 28.6% of the 524 children enrolled in the fifth and sixth grades (See Figure 3).
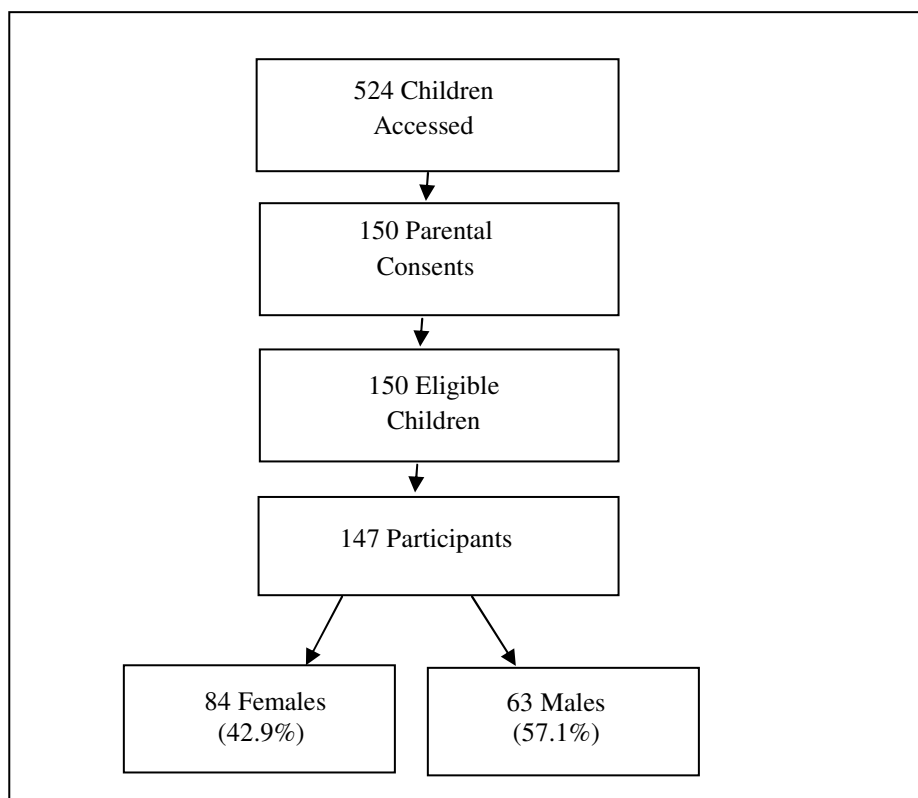
Figure 3. Participant Response.

Demographic data, including age, gender, race/ethnicity, grade level, pubertal status, and socioeconomic status are presented in Table 1.   The sample included 60 ten-year-olds, 64 eleven-year-olds, and 23 twelve-year-olds; 85 were in the fifth grade, and 62 were in the sixth grade. Sixty-seven percent ($n = 99$) of the participants were White, 17.7% ($n = 26$) of the sample were Hispanic, 11.6% ($n = 17$) were African American, 2% ($n = 3$) were Asian, and 1.4% ($n = 2$) described themselves as more than one race. In the accessed schools, 12.8% of the students were African American, 78.2% were White, 7.1% were Hispanic, and 1.8% were other races/ethnicity (Alabama Department of Education, 2012; D. Owens, personal communication, March 5, 2012). Participants in this study closely represented the schools from which they were drawn. The percentage of students enrolled in free and reduced lunch for School A was 77%, for School B was 27%, for School C was 69%, and for School D was 24%.

Socioeconomic status was determined with the use of the Nam-Powers-Boyd occupational status tool (Nam & Boyd, 2004). Parents self-reported occupation on the demographic questionnaire. The occupations were assigned a score based on the occupation listed on the Nam-Powers-Boyd Scale and then arranged into low, middle, and high classifications. Of the 147 participants, 38 (25.8%) were classified as low socioeconomic status, 98 (66.7%) middle socioeconomic status, and 11 (7.5%) high socioeconomic status. Approximately 47% ($n = 70$) of the children reported that they were prepubescent and 48.3% ($n = 71$) of the children indicated that they were in puberty; four percent ($n = 6$) of children were unsure or marked "I don't know" on the pubertal status questionnaire.

Table 1. *Demographic Characteristics of Sample (N = 147)*

Variable	*M (SD)*	*N (%)*
Age (range 10-12 years)	10.75 (.71)	
10 years		60 (40.8)
11 years		64 (43.5)
12 years		23 (15.6)
Gender		
Female		84 (57.1)
Male		63 (42.9)
Grade		
5th		85 (57.8)
6th		62 (42.2)
Race/Ethnicity		
African American		17 (11.6)
Asian		3 (2.0)
White		99 (67.3)
Hispanic		26 (17.7)
More than one race		2 (1.4)
Puberty		
Prepubescent		70 (47.6)
Females		24 (34.3)
Males		46 (65.7)
Pubertal		71 (48.3)
Females		56 (78.9)
Males		15 (21.1)
Unknown		6 (4.0)
SES		
Low		38 (25.9)
Middle		98 (66.7)
High		11 (7.5)

Note. Because of rounding, not all percentages total 100.

Descriptive Statistics of Study Variables

Descriptive statistics for the study variables are presented in Table 2. The

descriptive statistics of the physical data of the sample are as follows: BMI mean for

participants was 21.93 (range of 13.42 to 36.54); standard deviation was 5.006. Of the

147 participants, 2.0% were determined as underweight (BMI under the 5th percentile),

46.3% were determined as healthy weight (between 5th percentile and 84th percentile),

and 51.7% were determined as either overweight (BMI between the 85th to less than the 95th percentile) or obese (BMI equal to or greater than the 95th percentile). Of the 51.7% participants classified as overweight or obese, 26 participants (17.7%) were classified as overweight, and 50 participants (34%) were classified as obese. In the overweight category, 73.1% ($n = 19$) were White, 19.2% ($n = 5$) were Hispanic, and 7.7% ($n = 2$) were African American. In the obese category, 62% ($n = 31$) were White, 22% ($n = 11$) were Hispanic, 14% ($n = 7$) were African American, and 2.0% ($n = 1$) were Asian. Of the 71 participants who were pubertal, 45% ($n = 32$) of children had BMIs at the 85th percentile and above, while 54.3% ($n = 38$) of prepubertal children had BMIs at the 85th percentile and above.

Waist circumference (WC) mean for the group was 30.178 inches (range of 20.125 to 45.0); $SD = 5.282$. WC mean value for males was 30.29; for girls, the mean value was 30.09. Based on WC references established by Fernandez and colleagues (2004), approximately 15.6 % of boys had WC measures at or above the 90th percentile, while 19% of girls had WC measures at or above the 90th percentile.

Of the 147 participants, three children did not provide adequate saliva samples for testing of cortisol. Thus, 144 total samples were included in the analysis of cortisol. The mean value of cortisol concentration for the group was 6.752 nmol/l (range of 1.971 to 46.085 nmol/l); standard deviation was 6.753. Cortisol values were within the normal limits of 1.69 to 12.81 (Safarzadeh, Mostafavi, & Haghi, 2005) for approximately 91.7% ($n = 132$) of the group; however, 8.3% ($n = 12$) of the children had values that were greater than 12.81 nmol/l.

Depressive symptoms in this group of 10-, 11-, and 12-year-olds, as measured by

the Children's Depression Inventory (CDI), ranged from 0 to 37 out of a possible range of 0 to 52. A cut-off score of 13 denotes mild depressive symptoms, and a score of 19 indicates severe levels of depressive symptoms (Kovacs, 1981; Kovacs, 1992). In the group, 16.3% ($n = 24$) of children reported mild to moderate depressive symptoms, while 23.1% ($n = 34$) endorsed severe levels of depressive symptoms. For the group, the mean score was 11.14; standard deviation was 8.978. The mean score for boys was 11.05 (range of 0 to 36); standard deviation was 8.876. The mean score for girls was 11.20 (range of 0 to 37); standard deviation was 9.105. Similarly, Kovacs (1992) reported the CDI mean score of 11.1 ($SD = 6.6$) in a normative sample of children. Of children who reported mild to moderate depressive symptoms, 58.3% ($n = 14$) of children had elevated BMI values, and approximately 86% ($n = 12$) had BMI's at or above the 95th percentile. For children who reported severe depressive symptoms, 82.4% ($n = 28$) of children had BMI's at or above the 95th percentile. More children had elevated WC values with depressive symptoms than with BMI. Of children who experienced mild to moderate depressive symptoms, 33.3% ($n = 8$) children had WC values at or above the 75th percentile, while 37.5% ($n = 9$) of children had WC values at or above the 90th percentile. Approximately, 29.1% *($n = 7$)* of children with WC values between the 10th – 50th percentile reported mild to moderate depressive symptoms, and 11.7% *($n = 4$)* of children within the same percentile reported severe depressive symptoms. For children who reported severe depressive symptoms, 17.6% ($n = 6$) of children had WC values at or above the 75th percentile, while 67.6% ($n = 23$) had WC values at or above the 90th percentile.

Psychological stress was measured by the Feel Bad Scale (FBS). On the FBS, scores can range from 20 to 500. According to Lewis, Siegel, and Lewis (1984), there is no established cut-off score that delineates between low levels and high levels of stress in children; thus, higher scores indicate greater distress on the FBS. For the group, children had actual scores that ranged from 40 to 290. The mean score was 124.44 and standard deviation was 54.902. In an earlier study conducted by the authors of the instrument, the mean score was 135.1 (range of 20 to 500) with a standard deviation of 46.7 (Lewis, Siegal, & Lewis, 1984). For the group, the mean score for boys was 117.5 and standard deviation was 57.5; girls had a mean score of 129.64 and standard deviation was 52.5. Of the 147 children, 46.3% ($n = 68$) had FBS scores higher than the mean value of 124.44 for the group. Of the 68 children, 4.4% ($n = 3$) had BMI values between the 85th and 94th percentile; however, 58.8% ($n = 40$) had BMI values at or above the 95th percentile. The median score for the FBS was 120; seventy-two children reported scores above the median. Of those 72 children, 63.9% ($n = 46$) of girls had FBS scores above the median, while 36.1% ($n = 26$) of boys had FBS scores above the median. Approximately, 27.7 % ($n = 20$) of children with WC values between the 10th -50th percentile reported FBS scores above the median. Of children who had FBS scores above the median, 18% ($n = 13$) of children had WC values at or above the 75th percentile, while 54.2% ($n = 39$) of children had WC values at or above the 90th percentile.

Table 2. *Descriptive Statistics for Study Variables (N=147)*

Study Variable	Actual Range	Mean (*SD*)	Median
Body Mass (BMI)	13.42-36.54	21.93 (5.006)	20.76
Central Adiposity (WC)	20.125-45.0	30.178 (5.282)	29.5
Cortisol	1.971-46.085	6.752 (6.753)	4.686
Psychological Stress	40-290	124.44 (54.902)	120
Depressive Symptoms	0-37	11.14 (8.978)	9

Note. Psychological stress corresponds to the FBS = Feel Bad Scale; depressive symptoms corresponds to the CDI = Children's Depression Inventory

Instrument Reliability

The instruments used in the data collection for this study have been found to have adequate reliability in prior studies for children who are 10-, 11-, and 12-years-old. The Feel Bad Scale (FBS), Children's Depression Inventory (CDI), and the Self-Administered Rating Scale for Pubertal Development were used in this study. Internal consistency for the Feel Bad Scale, Children's Depression Inventory, and the Self-Administered Rating Scale for Pubertal Development were evaluated using Cronbach's alpha coefficients. Reliability estimates for the FBS and CDI were in the good to excellent range (Table 2) and consistent with the previously conducted pilot study and earlier studies (Abela, 2001; Kovacs, 1985; Lewis, Siegel, & Lewis, 1984; Rew, Horner, & Fouladi, 2010; Siegel & Brown, 1988). In an earlier study, the CDI was evaluated in a normative sample of children aged 7-11 years; good internal consistency was determined with an alpha coefficient at .83 (Kovacs, 1992). For the FBS, a Cronbach's alpha coefficient was reported as .84 in a study by Rew and associates (2010). The Self-Administered Rating

Scale for Pubertal Development used in this study was noted to have a Cronbach's alpha

coefficient of .605 for the group as a whole; however, Cronbach's alpha was .656 and

.521 for males and females, respectively. These coefficients were lower than the

coefficient range of .67 and .70 as reported in the study by Carskadon and Acebo (1993).

Table 3. *Psychological Instrument Reliability*

Instrument	No. of items	Possible score	Cronbach's α coefficient
Feel Bad Scale	40	20-500	.888
Children's Depression Inventory	26	0-52	.904

Correlations among Study Variables

Bivariate correlations using Pearson Product-Moment Correlation were calculated

to describe the relationship between the study variables (psychological stress, depressive

symptoms, body mass, and central adiposity) and the confounding variables (gender,

ethnicity, puberty, SES). Cortisol data were log transformed to correct their skewed

distribution and were used in calculating correlation coefficients. A significant

relationship was found between gender (female) and pubertal status ($r = .450.$ $p < .001$).

No significant relationships were found between ethnicity and the confounding and/or

study variables. In addition, socioeconomic status demonstrated no statistically

significant correlations with any of the confounding and study variables. Body mass

showed a significantly positive association to central adiposity ($r = .858$, $p < .001$).

Cortisol showed no significant correlations except with the log transformed cortisol,

which was expected. Likewise, the log transformed cortisol did not reveal any significant

associations. Psychological stress demonstrated significant correlations with body mass

($r = .233$, $p < .01$), central adiposity ($r = .385$, $p < .001$), and depressive symptoms

($r = .559$, $p < .001$). Similarly, depressive symptoms was significantly associated with

body mass ($r = .396$, $p < .001$) and central adiposity ($r = .507$, $p < .001$).

Table 4. Bivariate Correlations among Study Variables

	Female	Puberty	White	SES	BMIz	WC	Cortisol	Cortisol[a]	FBS	CDI
Female	1									
Puberty	.450***	1								
White	-.046	.020	1							
SES	.036	.239	.137	1						
BMIz	-.121	-.017	.012	-.076	1					
WC	-.019	-.009	-.012	-.139	.858***	1				
Cortisol	.074	.037	-.053	.053	.133	.175	1			
Cortisol[a]	.035	-.011	-.039	.084	.142	.189	.893***	1		
FBS	.110	.057	-.011	-.007	.233**	.385***	.172	.169	1	
CDI	.009	-.014	.062	-.135	.396***	.507***	.135	.107	.559***	1

Note. ** $p < .01$, *** $p < .001$
[a] Natural log transformed data were used.
FBS= Feel Bad Scale; CDI = Children's Depression Inventory

Analysis for Research Question 1

Research Question 1: What is the relationship between psychological stress and

depressive symptoms in 10-, 11-, and 12-year-old children?

Table 5. *Bivariate Correlation between Psychological Stress and Depressive Symptoms*

	FBS (Psychological Stress)
CDI (Depressive Symptoms) Pearson Correlation	.559
Sig (2-tailed)	.000

The Pearson Product-Moment Correlation was used to determine if there was a bivariate relationship between the predictor variables. A significant bivariate relationship between the independent variables (psychological stress and depressive symptoms) was identified. Hypothesis 1 was supported indicating a positive relationship between psychological stress and depressive symptoms in 10-, 11-, and 12-year-old children.

Analyses for Research Question 2

Research Question 2: How much of the variance in body mass (measured by BMI) and central adiposity (measured by WC) is explained by psychological stress and depressive symptoms in 10-, 11-, and 12-year-old children after controlling for gender, ethnicity, puberty, and SES?

For this specific research question, the interpretation of diagnostic tests is dependent on the examination of model assumptions that include normal distribution, linearity among independent and dependent variables, and homoscedasticity. Scatterplots and histograms of multiple regression data were examined and met assumptions of distribution, linearity, and homoscedasticity. Statistical output was examined for extreme outliers, tolerance, Variance Inflation Factor (VIF), normal distribution, and multicollinearity. One extreme outlier was noted with one child having a BMI z-score of -2.28. After further examination of raw data, a BMI value of 13.4 is plausible for a child

who is ten-years-old; thus, the extreme outlier was included in analyses. Collinearity

diagnostics included the examination of tolerance values and VIF statistics for the model.

The model did not reveal any multicollinearity among the variables.

Hierarchical multiple regression techniques were performed in order to determine

the amount of variance in body mass and central adiposity, in turn, accounted for by the

predictor variables. In this study, goodness of fit and statistical significance of the

regression model were evaluated using the R-squared value and an F-test. Confounding

variables of gender, ethnicity, puberty, and SES with the dependent variable body mass

(BMI z-scores) were entered in block 1 and revealed that the set of confounding variables

did not provide a significant contribution, $F(5, 141) = 1.121$, $p = .352$ and accounted for

3.8% of the variance in body mass (Table 6). Independent variables of psychological

stress and depressive symptoms were then entered in block 2.

Table 6. *Hierarchical Regression Model with Confounding Variables and Body Mass (BMI)*

Model	R	R^2	Adjusted R^2	R^2 Change	F Change	df1	df2	Sig. F Change
1	.196	.038	.004	.038	1.121	5	141	.352

Predictors: (Constant), Gender, Ethnicity, Puberty, SES

The full model including confounding variables, depressive symptoms, and

psychological stress revealed a significant contribution to the variance in body mass

scores $F(7, 146) = 4.490$, $p < .001$, R^2 .184. The full model explained 18.4% of the

variance in body mass. The ΔR^2 value of .146 indicated a 14.6% change in the amount of

variance explained in body mass when the predictor variables (depressive symptoms and

psychological stress) were added to the model. Specifically, depressive symptoms

revealed a significant t-test (3.907, $p < .001$); thus, this predictor variable provided a

significant contribution. Regression analysis showed the contribution of the variables

(Table 7-8).

Table 7. *Regression Analysis of Confounding Variables, Psychological Stress, and Depressive Symptoms on Body Mass (BMI)*

ANOVA[b]					
Model	Sum of Squares	df	Mean Square	F	Sig. (p.05)
2 Regression	31.996	7	4.571	4.490	.000[a]
Residual	141.494	139	1.018		
Total	173.490	146			

a. Predictors: (Constant), Gender, Ethnicity, Puberty, SES, Psychological Stress, Depressive Symptoms
R Squared =.184 (Adjusted R Squared = .143)
b. Dependent Variable: Body Mass (BMI)

Table 8. *Regression Coefficients of Confounding Variables, Psychological Stress, and Depressive Symptoms on Body Mass (BMI)*

Coefficients[a]							
Model		Unstandardized Coefficients B	Std. Error	Standardized Coefficients Beta	t	Sig (p.05)	Tol.
2	Constant	.442	.416		1.061	.291	
	Male	.354	.189	.161	1.870	.064	.788
	Prepubertal	-.184	.190	-.085	-.969	.334	.764
	SES (low)	-.004	.362	-.002	-.010	.992	.276
	SES (mid)	-.174	.331	-.076	-.527	.599	.285
	White	-.045	.181	-.019	-.246	.806	964
	CDI	.045	.011	.369	3.907	.000	.656
	FBS	.001	.002	.034	.365	.715	.669

a. Dependent Variable: Body Mass (BMI)
CDI = Children's Depression Inventory; FBS = Feel Bad Scale

In a separate model using hierarchical multiple regression, the study variable

psychological stress was solely included for testing as the independent variable. When

confounding variables of gender, ethnicity, puberty, and SES with the outcome variable

body mass were entered in block 1, the confounding variables did not reveal a significant

contribution, $F(5, 141) = 1.121$, $p = .352$, $R^2 .038$ and accounted for 3.8% of the variance

in body mass. When psychological stress was entered in the model, a significant

contribution was revealed $F(6, 146)$, $p = .028$, $R^2 .095$, and the full model accounted for

9.5% of the variance explained in body mass (Table 9-10). In this model, psychological

stress revealed a significant t-test (2.960, $p = .004$). The ΔR^2 value of .057 represented a

5.7% change in the amount of variance explained in body mass when the predictor

variable psychological stress was added to the model.

Table 9. *Regression Analysis of Confounding Variables and Psychological Stress on Body Mass (BMI)*

ANOVA[b]					
Model	Sum of Squares	df	Mean Square	F	Sig. ($p.05$)
2 Regression	16.459	6	2.446	2.446	.028[a]
Residual	157.031	140	1.122		
Total	173.490	146			

a. Predictors: (Constant), Gender, Ethnicity, Puberty, SES, Psychological Stress
R Squared = .095 (Adjusted R Squared = .057)
b. Dependent Variable: Body Mass

Table 10. *Regression Coefficients of Confounding Variables and Psychological Stress on Body Mass (BMI)*

Coefficients[a]							
Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig (*p*.05)	Tol.
		B	Std. Error	Beta			
2	Constant	.320	.436		.733	.465	
	Male	.380	.199	.173	1.913	.058	.789
	Prepubertal	-.168	.200	-.077	-.840	.402	.764
	SES (low)	.145	.378	.059	.384	.701	.279
	SES (mid)	-.164	.347	-.071	-.471.	.638	.285
	White	.025	.189	.011	.132	.895	.973
	FBS	.005	.002	.240	2.960	.004	.980

a. Dependent Variable: Body Mass (BMI)
FBS = Feel Bad Scale

Confounding variables of gender, ethnicity, puberty, and SES with the outcome variable WC were entered in block 1 and revealed that the set of confounding variables did not provide a significant contribution, $F(5, 141) = 1.009$, $p = .415$, $R^2 .035$ and accounted for 3.5% of the variance in central adiposity (Table 11).

Table 11. *Hierarchical Regression Model with Confounding Variables and Central Adiposity (WC)*

Model	R	R^2	Adjusted R^2	R^2 Change	F Change	df1	df2	Sig. F Change
1	.186	.035	.000	.035	1.009	5	141	.415

Predictors: (Constant), Gender, Ethnicity, Puberty, SES

After controlling for confounding variables, the study variables, psychological stress and depressive symptoms were added to the model in the second block. The full model including the confounding variables, depressive symptoms, and psychological

stress, revealed a significant contribution to the amount of variance explained in central

adiposity, F (7, 146) = 7.925, $p < .001$, R^2 .285 and accounted for 28.5% of the variance

in central adiposity. The ΔR^2 value of .249 revealed a 24.9% change in the amount of

variance explained in central adiposity when the predictor variables depressive symptoms

and psychological stress were added to the model. Depressive symptoms were found to

significantly contribute to central adiposity, while psychological stress showed a

nonsignificant contribution in the model. The predictor variable depressive symptoms

revealed a significant t-test (4.538, $p < .001$); regression coefficients for each variable are

presented (Table 12-13).

Table 12. *Regression Analysis of Confounding Variables, Psychological Stress, and Depressive Symptoms on Central Adiposity (WC)*

ANOVA[b]					
Model	Sum of Squares	df	Mean Square	F	Sig. (p.05)
2 Regression	1161.973	7	165.996	7.925	.000[a]
Residual	2911.653	139	20.947		
Total	4073.627	146			

a. Predictors: (Constant), Gender, Ethnicity, Puberty, SES, Psychological Stress, Depressive Symptoms
R Squared = .285 (Adjusted *R* Squared = .249)
b. Dependent Variable: Central Adiposity

Table 13. *Regression Coefficients of Confounding Variables, Psychological Stress, and Depressive Symptoms on Central Adiposity (WC)*

Coefficients[a]							
Model		Unstandardized Coefficients B	Std. Error	Standardized Coefficients Beta	t	Sig (*p*.05)	Tol.
2	Constant	25.345	1.888		13.421	000	
	Male	.653	.859	.061	.760	448	.788
	Prepubertal	-.638	.864	-.061	-.739	.461	.764
	SES (low)	1.298	1.642	.108	.790	.431	.276
	SES (mid)	.058	1.501	.005	.039	.969	.285
	White	-.068	.820	-.006	-.083	.934	.964
	CDI	.236	.052	.402	4.538	.000	.656
	FBS	.015	.008	.160	1.827	.070	.669

a. Dependent Variable: Central Adiposity

CDI = Children's Depression Inventory; FBS = Feel Bad Scale

Psychological stress was tested as a sole independent variable in a separate model using regression techniques. Confounding variables of gender, ethnicity, puberty, and SES with the outcome variable central adiposity were entered in block 1; the confounding variables did not significantly explain the amount of variance in central adiposity $F (5, 141) = 1.009$, $p = .415$, $R^2.035$ and accounted for 3.5% of the variance in central adiposity. When psychological stress was entered in the model, a significant contribution was revealed $F (6, 146)$, $p <.001$, $R^2 .179$, and the full model accounted for 17.9% of the variance explained in central adiposity (Table 14). The ΔR^2 value of .144 represented a 14.4% change in the amount of variance explained in central adiposity when the predictor variable psychological stress was added to the model. Regression coefficients are presented in Table 15.

Table 14. *Regression Analysis of Confounding Variables and Psychological Stress on Central Adiposity (WC)*

ANOVA[b]					
Model	Sum of Squares	df	Mean Square	*F*	Sig. (*p*.05)
2 Regression	730.544	6	121.757	5.099	.000[a]
Residual	3343.083	140	23.879		
Total	4073.627	146			

a. Predictors: (Constant), Gender, Ethnicity, Puberty, SES, Psychological Stress
R Squared = .179 (Adjusted *R* Squared = .144)
b. Dependent Variable: Central Adiposity

Table 15. *Regression Coefficients of Confounding Variables and Psychological Stress on Central Adiposity (WC)*

Coefficients[a]							
Model	Unstandardized Coefficients B	Std. Error	Standardized Coefficients Beta	t	Sig (*p*.05)	Tol.
2 Constant	24.702	2.011		12.286	.000	
Male	.790	.917	.074	.861	.390	.789
Prepubertal	-.551	.922	-.052	-.597	.551	.764
SES (low)	2.083	1.743	.173	1.195	.234	.279
SES (mid)	.116	1.603	.010	.072	.943	.285
White	.298	.871	.027	.342	.733	.973
FBS	.037	.007	.384	4.970	.000	.980

a. Dependent Variable: Body Mass (BMI)
FBS = Feel Bad Scale

Analyses of Research Question 3

Research Question 3: Does cortisol mediate the relationship between psychological stress and body mass (BMI), and/or between depressive symptoms and body mass (BMI)

in 10-, 11-, and 12-year-old-children after controlling for the confounding variables gender, ethnicity, puberty, and SES?

Univariate regression models were planned to be used to determine whether, after gender, ethnicity, puberty, and SES were controlled, cortisol mediated the relationships between psychological stress and body mass and/or between depressive symptoms and body mass. The first regression technique performed was to test if the independent variable (psychological stress) was a significant predictor of the mediator (cortisol). In the model, psychological stress did not significantly predict cortisol (Table 16). Because this condition was not met, further testing for cortisol as a mediator ceased.

Table 16. *Univariate Regression Model with Psychological Stress and Cortisol*

Model	R	R^2	Adjusted R^2	F	df	Sig.
1	.163	.027	.020	3.893	1	.065

Predictor: (Constant), Psychological Stress

Next, a regression analysis was performed to test if depressive symptoms significantly predicted cortisol. The regression equation with depressive symptoms as a predictor was not significant (Table 17); thus, further steps to test for mediation were not continued.

Table 17. *Univariate Regression Model with Depressive Symptoms and Cortisol*

Model	R	R^2	Adjusted R^2	F	df	Sig.
1	.129	.017	.010	2.402	1	.123

Predictor: (Constant), Depressive Symptoms

Analyses of Research Question 4

Research Question 4: Does cortisol mediate the relationship between psychological stress and central adiposity (WC), and/or between depressive symptoms and central adiposity (WC) in 10-, 11-, and 12-year-old children after controlling for the confounding variables gender, ethnicity, puberty, and SES?

As described earlier in Analyses of Research Question 3, the univariate regression models to test whether cortisol mediated the relationships between psychological stress and body mass and between depressive symptoms and body mass were nonsignificant (Table 10-11). Thus, further testing for mediation did not continue.

Summary

Statistical analyses of variables revealed a significant bivariate relationship between psychological stress and depressive symptoms. After controlling for gender, puberty, ethnicity, and SES, the predictor variable depressive symptoms explained a significant amount of the variance in body mass and central adiposity. The predictor variable psychological stress significantly contributed to explaining the amount of variance in both body mass and central adiposity when the independent variable depressive symptoms was not included in the model. A lack of statistically significant relationships between psychological stress and cortisol, and between depressive symptoms and cortisol precluded the need for additional testing of cortisol for mediation among the study variables.

CHAPTER 5

DISCUSSION

The purpose of this study was to examine the influence of psychological stress and depressive symptoms on body mass and central adiposity in 10-, 11-, and 12-year-old children and included controlling for gender, ethnicity, puberty, and SES. In addition, the principal researcher sought to determine the mediating role of cortisol in the relationships among psychological stress, depressive symptoms, body mass, and central adiposity. This chapter includes a discussion of the findings related to descriptive statistics of the sample and research questions. In addition, a discussion of the study limitations and implications for nursing practice, education, and future research are included.

Findings Related to the Sample

A majority of participants enrolled in this study were overweight or obese. This finding is in contrast to those of Ogden and colleagues (2010), who reported percentages for BMIs at the 85th to 95th percentile in children 6- to 11- years-old to be 35.5% and for BMIs at the 95th percentile and greater to be 19.6%. In the southeastern states, the prevalence of overweight and obesity in this age group is estimated to be 50% (Singh, Kogan, & van Dyck, 2010). Results in this study of 10-, 11-, and 12-year-old children in the obese category exceed that of the NHANES (National Health and Nutrition Examination Surveys) study (Ogden et al., 2010) but are similar to the data estimated in the southern states (Singh, Kogan, & van Dyck, 2010) and are not unexpected. Waist circumference mean in this group of children was 30.2 inches for the group. This finding

differs from Li and colleagues' report from national data that found children aged 6-11 years had an average waist circumference of 24.4 inches (Li, Ford, Mokdad, & Cook, 2006). Given the number of children with elevated body mass in the southern states, it is not surprising to see central adiposity in this sample (Akil & Ahmad, 2011).

Although not the focus of this study, puberty was considered as a confounding variable because it may independently influence body mass and central adiposity. A child as young as 6 years of age with an average age of 8 can be in the beginning stages of puberty (Hayward, 2003). Thus, children ages 10-, 11-, and 12-year-old typically are considered as approaching the onset of puberty. The numbers of children who were pubertal and prepubertal status were nearly equivalent in this study; however, more girls reported themselves as pubertal. Six children self-reported that they were unsure or marked "I don't know" on questions about their pubertal development. Although the children completed the questionnaire behind a privacy screen, and the principal researcher was available to answer questions, children may have been hesitant to respond to sensitive questions about development. Furthermore, some children who are overweight or obese may have difficulty differentiating adiposity from secondary sex changes. Children, as young as those in this sample, may have some difficulty distinguishing the appearance of breast tissue or other indications of puberty from adiposity. Because discrimination of breast (glandular) tissue from fat may be difficult, especially in overweight or obese girls with excessive subcutaneous fat, the risk of erroneous classification of breast development is problematic. Children were encouraged to ask the principal researcher if they had questions while completing the instrument. In the pilot study and full-scale study, some children asked the researcher to explain some

terminology (e.g., "underway" and "menstruation") stated within the questionnaire; other children may have been unwilling to ask the researcher for clarification.

As a result of the possible confusion about puberty, the internal consistency of the Self-Administered Rating Scale for Pubertal Development was low, and thus, would influence the reliability of the findings. While it would be helpful to use another instrument to measure puberty, there are few self-report measures of pubertal status available.

Elevated body mass and central adiposity did not seem to be related to the onset of puberty. Of the pubertal children, 45% of children were overweight or obese, whereas 54.3% of prepubertal children were overweight or obese. These findings are in contrast to those of other researchers who found that early pubertal timing was linked to overweight status in children (Biro et al., 2010; Kaplowitz, et al, 2001; Ribeiro et al., 2006). Thus, other factors, such as low physical activity and/or poor nutritional habits need to be considered as influencing the development of overweight or obesity in this group of children. For example, physical activity has been noted to decrease in this age group as children approach adolescence (Maximova et al., 2009).

More girls than boys reported that they were pubertal; this finding is consistent with that of Biro and colleagues (2010), who reported that girls had started puberty earlier. Some researchers noted a pattern of early pubertal timing in girls who experienced environmental stressors and challenges (e.g., parental conflict) (Deardorff et al., 2011; Tither & Ellis, 2008), and thus, the relationship between girls' experience of stress and pubertal timing is interesting and may explain girls' earlier onset of puberty.

Future analysis of gender comparison related to pubertal status may prove beneficial and needs to be considered in studies with children.

Based on race, a greater percentage (61.5%) of Hispanic children were overweight or obese. This finding coincides with the report that Hispanic children and African American children have higher rates of obesity (Ogden et al., 2010). In their study of the NHANES sample, Ogden and associates (2010) reported the prevalence of high BMI in U.S. children by sex, age, and race/ethnicity. They indicated that in 6- to 11-year-old children who had BMIs at the 85th to 95th percentile: 34.5 % were Non-Hispanic White children; 37.6 % were Non-Hispanic Black children; and 42.6 % were Hispanic children.

Approximately, 44.8% of children in this sample had waist circumference values above the group mean. On average, compared to boys in this study, girls had higher WC values. On the basis of the WC references established by Fernandez and colleagues (2004), 15.6 % of boys had WC measures at or above the 90th percentile, while 19% of girls had WC measures at this percentile. Higher WC measures were seen in prepubertal girls. These findings corroborate the results of other studies that found girls to have higher WC measurements than boys (Aeberli, Gut-Knabenhans, Kusche-Ammann, Molinari, & Zimmerman, 2011; Wang et al., 2007). In this study, more White children had WC measures at or above the 90th percentile, which conflicts with findings from data reported by Messiah and associates (2011), who found that Hispanic children had higher WC measurements than those of non-Hispanic Black and non-Hispanic White children. Because the study was conducted in a rural school district, consideration needs to be given to geographical factors that could explain the dissimilarities in findings. Results

from other studies note geographic differences of eating behaviors and consumption of energy-dense and nutrient-poor foods, particularly in rural areas of the U.S. (Dean & Sharkey, 2011; Lufiyya, Lipsky, Wisdom-Behounek, & Inpanbutr-Martinkus, 2007; Nicklas, Meyers, Reger, Beech, & Berenson, 1998; Sharkey, Johnson, Dean, 2011). Perhaps these geographic factors were apparent in this sample and could result in elevated central adiposity. It may be helpful in future research to consider the geographical location and nutrition of participants.

In addition to weight, height, and waist circumference, cortisol levels from saliva samples were measured. A majority of children had cortisol values within the normal range; however, 8.3% of children had cortisol values above the norm. Of the children categorized as obese, approximately16.0% of those children had cortisol values greater than the reported norm, while 83.3% of healthy weight children had cortisol values within the norm. In this study, the cortisol values were not related to elevated body mass or central adiposity. This finding contrasts with that of Dimitrou and associates (2003), who found a relationship between an increase in cortisol levels and body fat in children. Other researchers have noted that central adiposity is associated with increased cortisol secretion; however, these studies were done with adults (Björntorp, 1991; Marin et al., 1992; Pasquali et al., 1993). Although some researchers reported positive associations between cortisol levels and body mass and between cortisol levels and central adiposity in adolescents (Sen, Aygun, Yilmaz, & Ayar, 2008; Weigensberg, Toledo-Corral, & Goran, 2008), other investigators reported an association between elevated body mass in children and a decrease in cortisol levels that results from a blunting effect of chronic HPA stimulation (Chalew, Nagel, Burt & Edwards, 1997). Similarly, Stewart and

colleagues (1999) found a similar relationship between a dysregulation in the HPA axis

that resulted in blunted cortisol levels and the development of central adiposity; however,

the results were noted in adults. Because few studies exist with children, it is difficult to

draw any conclusions with regard to the relationships between cortisol and body mass

and between cortisol and central adiposity. However, given that no relationships

involving cortisol levels were noted in this study, it may be that the sample size was too

small to detect a moderate effect size with this biomarker. Generally, researchers in the

social sciences and related field use power ranges of .20 to .50 for detection of small to

medium effects (Dockray, Susman, & Dorn, 2009).

On the basis of SES, a majority of children (66.7%) were categorized as having

medium SES, while approximately 26% of children were categorized as having low SES.

In addition, over half (57.8%) of overweight or obese children were determined to have

medium SES, while approximately 34% of overweight or obese children were determined

to have low SES. Of children who were overweight or obese, 8% were considered to

have high SES. This finding differs from results of other research that found that low

SES was linked to elevated body mass or central adiposity in children (Booth, Macaskill,

Lazarus, & Bauer, 1999; Moore, Howell, & Trieber, 2002; Samani-Radia & McCarthy,

2008). Interestingly, when the SES data were examined according to separate subgroups

of SES, 68.4% of children within the low SES category were overweight and obesity,

followed by children in the high SES group with 54.4% with overweight and obesity.

Less than half of children in the medium SES were found to be overweight or obese. In

addition, more than half of children (58.8%) who had WC above the 90th percentile were

in the medium SES category. Although, it is generally accepted that having a low SES

places an individual at greater risk of overweight and obesity (Sobal & Stunkard, 1989; Wang, 2001), the relationship between SES and overweight and obesity in this group of children seems quite different. Given that over half (54.5%) of the children in the high SES group were overweight or obese, factors other than SES may influence body mass and central adiposity. As suggested earlier, the geographic location and the associated nutritional habits (e.g., increased consumption of energy-dense foods and sweetened beverages) could play a factor in the risk of developing overweight and obesity.

In addition to physical and physiological measurements, psychological stress and depressive symptoms were measured by use of self-report instruments. Approximately, 16.3% of children reported mild to moderate depressive symptoms, and 23.1% of children reported severe levels of depressive symptoms. The percentages are slightly higher than those of other researchers (Bhatia & Bhatia, 2007; Chang, Zauszniewski, Heinzer, Musil, & Tsai, 2007) in studies with school-aged children. The percentage of children with severe levels of depressive symptoms also corroborates the finding from a recent study that found that 23% of children had elevated levels of depressive symptoms (McCabe, Ricciardelli, & Banfield, 2011). The mean CDI scores reported by both girls and boys were equivalent, and the mean score of the entire group was consistent with that found in Kovacs' (1992) study in a normative sample of children. A higher percentage (58.6%) of children in the middle SES group reported having mild, moderate, and severe levels of depressive symptoms, compared with 34.5% of children in the low SES group who also reported depressive symptoms.

Children with elevated BMIs had higher levels of depressive symptoms. This relationship was also true of WC and levels of depressive symptoms; of children who

reported severe levels of depressive symptoms, a majority of those children had elevated WC. However, children with normal BMIs and WC also reported depressive symptoms. So, it cannot be construed that elevated BMI and WC cause depressive symptoms or even precede the occurrence of depressive symptoms over time.

In addition to reporting depressive symptoms, children also reported their experience of psychological stress. Nearly half of the children reported FBS scores higher than the mean score for the group. However, the mean score of 124.4 was lower than that found by Lewis, Siegal, and Lewis (1984) with fifth and sixth grade children. In the current study, the median score was 120. Of those who reported scores above the median, more than half 64% (*n* = 46) were girls, while 36.1% (*n* = 26) of boys had FBS scores above the median. This finding supports the results of Christiansen and colleagues (2008), who noted significant gender differences among young rural adolescents, with girls reporting higher levels of perceived or psychological stress. In future studies, gender may need to be examined in self-reports of psychological stress.

Findings Related to the Research Questions

The relationship between psychological stress and depressive symptoms was examined through bivariate correlation analysis, which revealed a positive relationship between psychological stress and depressive symptoms for the entire group of children; this association supported findings from previous studies with adolescents (Braet, Vlierberghe, Vandevivere, Theuwis, & Bosmans, 2012; Sund et al., 2005). Few studies have found positive associations between depressive symptoms and psychological stress in children. Most studies grouped children into broad age groups that included

adolescents (Ge, Lorenz, Conger, Edler, & Simons, 1994; Williamson et al., 2005). This study was one of few with children aged 10 to 12 years. Consistently, it seems that children with psychological stress had higher levels of depressive symptoms. When considering gender, several studies yielded results indicating that the relationship between psychological stress and depressive symptoms in boys differed markedly from that found in girls (Ge et al., 1994; Silberg et al., 1999; Williamson et al., 2005). In the case of girls, researchers found that psychological stress was significantly associated with a higher risk of depressive symptoms (Ge et al., 1994; Silberg et al., 1999; Rudolph & Hammen, 1999; Williamson et al., 2005). However, in this study, a significant relationship existed between psychological stress and depressive symptoms in both girls and boys. Perhaps, in this group of children, boys were more willing to disclose their feelings of psychological stress and depressive symptoms. Furthermore, researchers reported that gender differences related to depressive symptoms typically emerge between the ages of 13 and 15 years. Prior to this age, depressive symptoms appear to be similar for young girls and boys (Hankin & Abramson, 1999; Saluja et al., 2004; Sweeting & West, 2003); the finding from the current study supports their contention.

　　　None of the confounding variables (gender, ethnicity, puberty, and SES) explained a significant amount of the variance in body mass and central adiposity. This finding is inconsistent with results from previous studies that found that gender, puberty, ethnicity, and SES had significant effects on body mass and/or central adiposity (Biro et al., 2010; Booth, Macaskill, Lazarus, & Baur, 1999; Kaplowitz et al., 2001; Madsen, Weed, & Crawford, 2010; Martin & Ferris, 2007; Moore, Howell, & Treiber, 2002). The lack of effects is surprising, considering the presented literature. However, this finding

may indicate other factors that involve biological, environmental, and/or psychological aspects yield a greater impact on body mass and central adiposity than the included variables. As discussed earlier, researchers have noted a relationship between elevated body mass and central adiposity and earlier onset of puberty. However, Dockray and colleagues (2009) did not find that pubertal status contributed to the variance in BMI. Due to mixed results in the literature, it is difficult to draw conclusions about the importance of puberty in the consideration of body mass and/or central adiposity.

Gender did not significantly explain the variance in body mass and central adiposity in this study; male and female participants had the same rate (50%) of overweight and obesity. However, more female participants had larger WC values, with 3.4% (at or above the 90th percentile) having higher values than boys. This finding is inconsistent with results from previous research that gender influenced body mass and central adiposity, especially in girls (Martin & Ferris, 2007; Wang & Beydoun, 2007). Although not specific to WC elevations, it has been noted that increased body mass is not gender-based. For example, Ogden and colleagues (2010) reported no significant difference in obesity prevalence in the 6- to 11-year-old group based on gender.

In addition to gender and puberty, ethnicity did not significantly explain the variance found in body mass and central adiposity, which are findings that are inconsistent with those of previous research (Dennison et al., 2002; Lutfiyya et al., 2008; Saab et al., 2011). In one report, Ogden and associates (2010) noted significant differences in prevalence of high BMI among children on the basis of racial and ethnic groups; for instance, in comparison with non-Hispanic White boys, Hispanics had significantly higher odds of having high BMI at all three BMI cutoffs. The findings

reported by Ogden and colleagues (2010) are consistent with results of research by

Madsen and associates (2010), who determined that certain racial and ethnic groups had

increasing rates of obesity, and that, when compared with White girls, Black, Hispanic,

and American Indian girls faced 2 to 3 times higher odds of having a high BMI (Madsen,

Weed, & Crawford, 2010). The contrary finding in the current study may have been due

to the limited number of minority participants. In addition, factors other than ethnicity

may explain the variance in body mass and central adiposity. For example, results of

research have shown that children participate less in physical activities and engage in

more sedentary activity on a regular basis (Kendall & Serrano, 2006). Given the

percentage of children who reported depressive symptoms, it is possible that children

with low energy would engage less in physical activities. A decrease in energy levels and

feelings of fatigue are symptoms commonly experienced with depressive symptoms. In

the current study, 46.9% of children reported that they felt "tired" on a survey item about

feelings of tiredness on the Children's Depression Inventory. Furthermore, nearly half of

youths aged 12 to 21 years-old are not vigorously active (Kendall & Serrano, 2006),

compounding concerns with depressive symptoms. Considering this, it is conceivable

that the increase of depressive symptoms and decrease in physical activity may impact

body mass and central adiposity in children.

Last, SES did not significantly explain the variance in body mass and central

adiposity. Children with elevated BMI and WC were fairly evenly distributed among low

and medium SES. These findings differ from those of other studies that have noted

relationships between SES and elevated BMIs and between SES and WC in children and

adolescents (Booth, Macaskill, Lazarus, & Baur, 1999; Moore, Howell, & Treiber, 2002;

Samani-Radia & McCarthy, 2008). Results of several studies noted a relationship

between low SES and abdominal fat deposition in adults (Baltrus et al., 2010; Chen &

Tunstall-Pedoe, 2005; Cohen, Doyle, & Baum, 2006). Few studies involved examining

the association between SES and central adiposity in children (Samani-Radia &

McCarthy, 2008), so that it is difficult to draw any conclusions. Associations have been

found between SES, ethnicity, and elevations in central adiposity in children aged 6 to 11

years (Okuson, Boltri, Eriksen, & Hepburn, 2006), so it may be that there are factors

other than SES that affect the association. Research on the mechanisms between low

SES and increased risk of elevated BMI has failed to produce a clear conclusion;

however, some investigators suggested that access to food, leisure-time physical

activities, and level of behavioral skills about weight maintenance are related to SES

(Lobstein & Jackson-Leach, 2007; Sarlio-Lähteenkorva, 2007). Other factors such as

parental obesity or genetic factors may influence both SES and elevated BMI (Sarlio-

Lähteenkorva, 2007) and, thus, need to be considered in research with children.

Furthermore, prevalence of overweight and obesity has been reported to be higher in rural

than in urban areas in the U.S.; therefore, because this study was conducted in a rural

school district, findings related to the effects on BMI and WC in children may need to be

considered in light of rural or urban setting.

Although the confounding variables (gender, ethnicity, puberty, and SES) did not

significantly explain the variance in body mass and central adiposity, depressive

symptoms were shown to be associated with body mass and central adiposity in this

sample. These findings coincide with those of previous studies conducted with

adolescents (Needham & Crosnoe; 2005; Pine et al., 2001). Cortese and associates

(2009) examined the relationship between depressive symptoms and body mass and found a significant association between BMI z-scores and CDI scores. The lowest depressive symptoms were evident for BMI z-scores around -1, suggesting that a moderately underweight status is associated with the lowest severity of depressive symptoms (Cortese et al., 2009). The findings of the current study were similar to those of Cortese and colleagues (2009), whereby the lowest depressive symptoms were evident for a BMI z-score around -1.5, indicating depressive symptoms were seen in a child with a low, normal weight. Most studies that examined depressive symptoms and body mass grouped school-aged children with adolescents, instead of specifically focusing on children aged 10 to 12 years (Goodman & Whitaker, 2002; Needham & Crosnoe, 2005; Pine et al., 2001). Few studies have been done solely in school-aged children, which make drawing conclusions difficult.

Researchers have identified positive relationships based on gender between depressive symptoms and body mass in children and adolescents; these relationships existed in girls only (Erikson, Robinson, Haydel, & Killen, 2000; Xie et al., 2005). In this study, gender-based relationships between depressive symptoms and body mass were not evident. Male and female participants reported nearly the same levels of depressive symptoms. Boys may have been more willing to report their depressive symptoms. Additionally, based on other studies, boys and girls experience the same degree of depressive symptoms during a certain developmental stage, and then later in adolescent years, experience differences in levels of depressive symptoms between genders (Hankin & Abramson, 1999; Saluja et al., 2004; Sweeting & West, 2003).

Depressive symptoms were also shown to be associated with central adiposity in this group of children. This finding is consistent with previous studies in adults that revealed that depressive symptoms were associated with central adiposity (Everson-Rose et al., 2009; Lee et al., 2005; Rosmond et al., 1996; Raikkonen, Matthews, & Kuller, 1999). Few investigators have examined the relationship between depressive symptoms and central adiposity in children (Bahreinian, Ball, Colman, Becker, & Kozyrskyi, 2011), so it is difficult to compare results across studies.

As depicted in the conceptual diagram (Fig. 1), psychological stress was hypothesized to influence body mass; however, psychological stress did not significantly explain the variance in body mass in the presence of depressive symptoms as a covariate. This finding differs from results of other research (Brunner, Chandola, & Marmot, 2007; Jern, Bergbrant, Björntorp & Hanssen, 1992) that noted positive associations between psychological stress and body mass in adults. Fewer investigations have been done with children, specifically school-aged children (Roemmich et al., 2007). The paucity of studies in children makes comparing results across studies difficult.

Psychological stress in the model also did not significantly explain the variance in central adiposity in this group of children in the presence of depressive symptoms as a covariate. This finding is contrary to findings that psychological stress has a positive association with central adiposity both in adults (Brunner, Chandola, & Marmot, 2007; Georges, Wear, & Mueller, 1992; Koch, Sepa, & Ludvigsson, 2008) and in adolescents (Goldbacher, Matthews, & Salomon, 2005; Van Jaarsveld et al., 2009). Because there is a lack of research with children, comparison of findings is problematic.

Interestingly, in the full model for this study, psychological stress did not significantly explain the variance in body mass and central adiposity. However, when depressive symptoms were removed from the model and psychological stress was examined separately, a significant effect was shown for both body mass and central adiposity. Thus, an alternative model could be considered because psychological stress was shown as a contributing factor to the outcome variables. Conceivably, psychological stress could impact depressive symptoms; in turn, depressive symptoms could have an effect on body mass and central adiposity. Hence, psychological stress could be considered as the predictor variable directly influencing depressive symptoms (mediator variable). Measures of multicollinearity (Tolerance and Variance Inflation Factor) indicated no issues with multicollinearity; thus, the two variables are not measuring the same concept, so each is considered as an important predictor of body mass and central adiposity. There are some studies that have examined stress as a predictor of depression in adults (Hammen, 1991; Kessler, 1997; McGonagle & Kessler, 1990), but few researchers have studied this relationship in children (Cole et al., 2006; Goodyer, Herbert, Tamplin, & Altham, 2000). Although no studies were found to support the model with the variables used in this study, it is possible that an alternative model may explain the relationships among the study variables.

In this study, no significant relationships were found between psychological stress and cortisol and between depressive symptoms and cortisol. This finding is inconsistent with results from studies indicating a positive association between elevated cortisol levels and depressive symptoms in children (Dockray, Susman, & Dorn, 2009; Puig-Antich et al., 1989; Rao et al., 2008). For instance, in an earlier study, Dockray, Susman, and Dorn

(2009), concluded that depressive symptoms were associated with BMI in both girls and boys, and, in girls, cortisol mediated the relationship between depressive symptoms and BMI.

Although previous researchers have noted significant relationships between depressive symptoms and cortisol in children (Dockray, Susman, & Dorn, 2009; Feder et al., 2004; Goodyer, Herbert, Moor, & Altham, 1991; Forbes et al., 2006; Rao et al. 2008), those findings were not replicated in this study. Therefore, without the existence of a significant relationship between depressive symptoms and cortisol, mediation could not be tested. However, these findings are in concert with previous research by Peeters and associates (2003), who found no differences between the cortisol levels of adult participants who had severe depressive symptoms and the levels of those who did not report having such symptoms. It may be that cortisol responses are blunted in individuals with chronic depressive symptoms. Burke and colleagues (2005) examined the association between depressive symptoms and salivary cortisol responses to stress in low-income women and found that women with high levels of depressive symptoms exhibited blunted cortisol responses to a naturalistic psychological stressor. Because studies primarily included adults (Burke, Fernald, Gertler, & Adler, 2005; Peeters et al., 2003), comparing findings across studies with children proves difficult. In other studies with children, depressive symptoms were associated with both increased (Dockray, Susman, & Dorn, 2009; Rao, Hammen, Ortiz, & Chen, 2008) and reduced (Luby et al., 2003) cortisol responses to psychological challenges.

In the diagrammed conceptual model (Fig. 1), depressive symptoms and psychological stress were hypothesized to elicit a response resulting in the stimulation of

the HPA axis; this stimulation is followed by an increase in cortisol levels. For this group

of children, the events depicted in the conceptual model did not occur. However, this

absence of a rise in cortisol levels may have resulted in habituation or some form of

dysregulation of the HPA axis (Buske-Kirschbaum et al., 2003; Chalew et al., 1997;

Luby et al., 2003; Otte et al., 2005; Wambodlt et al., 2003). Although not expected based

on physiologic responses to stress, cortisol secretion may be blunted. In one review, Cyr

and Romero (2009) provided explanations that could cause a diminished stress response

to a repeated stressor. One explanation is that an individual may perceive a stressor

differently at different periods or stages in one's life; thus, a stress response may show

variability during a lifetime (Cyr & Romero, 2009). Another possible explanation is

habituation, or the familiarization of a repeated stressor, which may result in a diminished

stress response (Cyr & Romero, 2009). A third possible explanation for a diminished

stress response is the desensitization of the stress response. Desensitization without

habituation can occur if an individual cannot adapt to a stressor after prolonged exposure;

thus, the individual perceives that stressor as noxious (Cyr & Romero, 2009). Last, the

authors explained that a diminished stress response could occur because of physiological

exhaustion; an individual is too fatigued to maintain the HPA axis, and thus unable to

effectively elicit a stress response (Cyr & Romero, 2009). Tyrka and colleagues (2010)

noted that lower or attenuated cortisol levels were associated with emotional problems in

a community sample of 8- to 11-year-old boys. In a study with adolescents, Harkness

and colleagues (2011) found that adolescents with depressive symptoms and a history of

maltreatment had cortisol responses to psychological stress that were lower than those of

adolescents who experienced minimal depressive symptoms or were healthy participants.

It is unclear whether cortisol acts as a mediator in the relationships among psychological stress, depressive symptoms, and body mass. However, in this study, the hypotheses that cortisol plays a mediating role in the relationships between psychological stress and body mass and central adiposity and between depressive symptoms and body mass and central adiposity were not supported. Given that cortisol levels were not affected in this study, other biomarkers need to be considered. It is documented that chronic stress produces immune suppression and increased production of glucocorticoids, such as cortisone, and catecholamines (Janusek, Cooper, & Mathews, 2012). Glucocorticoid excess has been linked to clinical observations of glucose intolerance, insulin intolerance, hypertension, and central adiposity (Wang, 2005). Perhaps, incorporating another biomarker, such as cortisone could provide valuable information about which underlying mechanisms are operating in the relationships between psychological stress and body mass and central adiposity and between depressive symptoms and body mass and central adiposity.

To date, no studies have examined cortisol as a mediator in the relationships between depressive symptoms and central adiposity and between psychological stress and central adiposity. Thus, whether cortisol plays a mediating role in the relationships among these variables remains tentative, and drawing conclusions based on the present research is not possible.

Last, as discussed above, a blunting effect of cortisol may be present. In this study, the majority of children had normal cortisol levels. Some researchers noted that severe depressive symptoms were associated with blunted cortisol responses in adults (Gold et al., 1996; Otte et al., 2005; Pfohl, Sherman, Schlecthte, & Stone, 1985).

Because of those findings, whether cortisol acts as a mediator between psychological

stress and central adiposity and between depressive symptoms and central adiposity

remains unclear.

Study Limitations

Several limitations must be considered with this study, including low participation

rate, self-report instrumentation, one-time data collection, and only a single geographic

region. The participation rate of 28.5% for the study is low. The study was conducted

within the public school system during the students' regular physical education class or

elective class. Children who did not want to miss physical education class or their

elective class may have decided not to participate in the study. Children who were

overweight or obese may have been unwilling or embarrassed to participate in a study

that included body composition measurements (height, weight, and waist circumference).

Furthermore, their parents may have also been hesitant to allow their children to

participate in the study. However, of those enrolled, 51.7% of the children in the study

were classified as overweight or obese in this study, so it does not seem like this was a

major consideration. In the future, strategies designed to optimally communicate with

parents and community members about the research study should be employed; for

example, having a parent representative and school principal distribute letters or flyers

describing the study in general terms may prove helpful. Although the principal

researcher scheduled a time to be available to meet with parents about the study,

investigators conducting school-based research might increase participation by also

attending and presenting the study at parent-teacher organization meetings, introducing

themselves at "Back to School Night" gatherings, and scheduling available time each week at the school to answer questions or concerns (Blom-Hoffman et al., 2009).

Although, the Children's Depression Inventory and Feel Bad Scale were considered to have good to excellent reliability, the reliability coefficients of the Self-Administered Rating Scale for Pubertal Development were less than optimal for the group as a whole, as well as for both boys and for girls as separate subgroups. While some may recommend completion of instruments by parents, this approach may not produce reliable data because parents may not be knowledgeable about bodily changes and the pubertal status of their children. It would be beneficial to compare the Self-Administered Rating Scale for Pubertal Development with the Tanner Stages. The Tanner Stages screening is based upon a scale of discrete stages of secondary sexual characteristics that permit health care professionals to determine the degree of pubertal maturation in children and adolescents, regardless of chronological age (Tanner, 1962). This usually requires actual visualization and physical examination of the children. Self-assessment of pubertal maturation using Tanner Stages has been conducted in studies with children and adolescents (Boas, Falsetti, Murphy, & Orenstein, 1995; Leone & Comtois, 2007; Williams et al., 1988). Although, the use of Tanner Stages or physical examination may prove beneficial, it may not be feasible to include such screening methods in the school setting.

The Nam-Powers-Boyd Occupation Status Scale was used to determine the socioeconomic status of the participants (based on parents' self-report of occupation). The scale is based on a composite of education and income of certain occupations from the 2000 U.S. Census. The scale has no corresponding score for individuals who self-

reported that they were disabled or unemployed; therefore, the SES was inferred for some of the participants. Although the Demographic Questionnaire (enclosed in the information packet to parents) included a question about the length of employment of a position/career, the Nam-Powers-Boyd Scale would not be applicable to those who are not employed or are disabled. Therefore, more suitable measures of SES need to be considered. For example, some researchers reported the use of free and/or reduced school meal eligibility as a measure of parental income (Harvell & LeBeau, 2010; Sirin, 2005). This proxy measure of SES could be beneficial and included on the Demographic Questionnaire.

Another limitation with regard to self-report instrumentation relates to the phenomenon of participants providing common responses to test items on study questionnaires. In essence, children may be biased or more likely to respond to items that are similar. For example, on the Feel Bad Scale, the child is asked to respond to a statement on how bad it would make him/her feel if he/she was "left out of a group"; the Children's Depression Inventory includes an item that asks the child to rate feelings of "being alone", which may be similar to the one item noted above on the Feel Bad Scale. It can be problematic if several items are measuring the same underlying concept; therefore, it is important for researchers to identify *a priori* if test items are measuring the same concept or constructs. In the current study, measures of multicollinearity revealed no issues with multicollinearity and indicated that psychological stress and depressive symptoms were separate concepts.

This study was cross-sectional, and data collected from participants occurred at one time. A prospective, longitudinal study would provide further insight into children's

cortisol levels, psychological stress, and depressive symptoms over time. Although some of the findings of this study corroborate those of similarly conducted studies with children (Bahreinian, Ball, Colman, Becker, & Kozyrskyi, 2011; Cortese et al., 2009; Laessle and Lindel, 2010), the study did not support the hypothesis that cortisol acts as a mediator of the effects of psychological stress and depressive symptoms on body mass and central adiposity. The relationships would need to be studied over time, and this would help to determine whether blunted cortisol effects or dysregulation of the HPA actually precede, accompany, or follow depressive symptoms. Therefore, a prospective, longitudinal study of psychological stress and depressive symptoms could adequately address this concern.

Because the design of this study was cross-sectional, no causal relationships among the study variables can be concluded. Therefore, it is possible that the directions of the relationships of psychological stress and depressive symptoms with body mass and central adiposity are reversed (Blaine, 2008). It is important to note that children with normal BMIs were included in this sample, and relationships with depressive symptoms and psychological stress were also found. So, the requirement that elevation in body mass and/or central adiposity precede depressive symptoms or psychological stress would not be met.

Last, the study involved recruiting participants from one school system in one geographical region. A distinct geographic location limits generalizability to other areas of the country and may not be reflective of fifth- and sixth- grade students in other regions. The study would need replicating in other parts of the country and in urban settings to determine whether results are similar. Additionally, because of the low

response rate of participants in this study, it is difficult to generalize the data to this rural region. Children who participated may have been different from children who did not participate.

Implications for Nursing Practice, Education, and Research

Although elevated BMI and waist circumference were not criteria for inclusion in this study, a majority of participants had elevations and would be classified as overweight or obese based on BMI. Therefore, it is important to, at the least, assess the BMI of children to identify children with elevated levels at well-child or primary health care visits and intervene before co-morbidities develop. In this study, a strong correlation was found between BMI and WC; thus, further evaluation of WC as a screening measure in children needs to be explored.

The percentage of children aged 6-11 years in the United States who were obese increased from 7% in 1980 to nearly 20% in 2008 (CDC, 2011). Children with elevated body mass and central adiposity are at risk for adult health problems such as heart disease, Type 2 diabetes, stroke, osteoarthritis, and several types of cancer (USDHHS, 2010). During 2002-2005, among U.S. youth ages 10 years and older, the rate of new cases for Type 1 diabetes was 18.6 per 100,000 and 8.5 per 100,000 for Type 2 diabetes (USDHHS, 2011). Some researchers also noted a prevalence of 14.5% for the cardiometabolic syndrome in obese children aged 8-11 years (Messiah et al., 2008; Pan & Pratt, 2008). Health concerns about the increase in childhood obesity exists within the health care field, and the number of children with elevated BMI and WC should be of

concern to all nurses and other health care professionals (Nauta, Byrne, & Wesley, 2009; Story et al., 2002).

Further, the Healthy People 2020 objectives support measurement of BMI on a regular basis (USDHHS, 2010). As stated earlier, at the least, measurement of BMI should constitute part of the required record for school-aged children. WC measurement may also prove beneficial in screening of overweight and obesity in children.

In addition to BMI and WC, children should be assessed for psychological stress and depressive symptoms. Approximately 16% of children reported mild to moderate depressive symptoms, while 23% reported severe levels of depressive symptoms. Of children who reported severe depressive symptoms, 82.5% of those children had elevated BMIs (at or above the 95th percentile). Psychological stress was also evident in this group, with 46.3% of children reporting stress scores above the mean; a greater percentage (58.8%) of children in the obese category reported higher levels of psychological stress. In this study, children with elevated BMIs showed higher levels of depressive symptoms and psychological stress. In addition, children with normal weight also reported depressive symptoms and psychological stress; this finding underscores the need for nurses and other health care professionals to incorporate into the health programs for children the assessment and monitoring of levels of depressive symptoms and psychological stress.

In addition, nurses can teach families that regular screening for depressive symptoms and psychological stress in children should form a part of primary prevention assessments by health care providers. Given that children reported psychological stress and depressive symptoms regardless of elevations in BMI and WC, it would be important

to assess for psychological concerns in children. It is evident that children experience psychological stress and depressive symptoms, and often times; this may be unobserved during well-child visits. Nurses play an essential role in educating and counseling children and families about regular BMI screening and WC measurements. Some parents may be reluctant to discuss their child's weight, and some may not even recognize a weight problem. BMI and WC reporting to parents/guardians can open a dialogue with parents about the growth and health of their child. In addition, taking body composition measurements regularly, such as BMI and WC makes it possible to monitor individual children over time and provides an opportunity for early intervention in overweight and obesity prevention.

Nurse educators need to enhance their students' understanding of the contributing risk factors of elevated body mass and central adiposity to the associated physical problems documented in acute care settings. Various factors, that include environmental, genetic, behavioral, and psychological factors, need to be incorporated in a teaching program to prevent childhood obesity. Nurse educators also need to stress to students the importance of understanding the prevalence and significance of normally adult-onset chronic illnesses now seen in children and health care costs associated with elevated body mass and central adiposity. Specifically, nurse educators should detail the many potential health complications evident in overweight and obese children; these concerns can be addressed through traditional didactic teaching methods, in pediatrics clinical experiences, and simulation techniques. Nursing students should also be educated about depressive symptoms and psychological stress in children. Depressive symptoms can lead to depression, which can ultimately lead to serious outcomes such as suicidal

ideations if not appropriately treated. Additionally, nursing students need to understand the importance of regular screening of body mass and central adiposity, and the inclusion of screening for depressive symptoms and psychological stress in children.

Expanding on this study, the hypotheses could be retested by using a larger sample size. Although the participants in this study closely represented the schools from which they were drawn, the researcher may need to access sites with greater minority enrollments, particularly with African American children. A greater diversity of racial and ethnic groups would help determine whether findings would be consistent. Additionally, expanding the study to other regions that include higher percentages of racial and ethnic groups would increase representation of specific groups. Low participation rates can be remedied in future research by incorporating strategies to promote studies in the community. For instance, researchers can generate interest about the study by meeting regularly with principals, teachers, parents, and students; also, researchers can increase participation in studies by communicating information about the study through scheduled visits at schools and attendance at Parent-Teacher Association meetings.

In future research, it may be beneficial to consider other biomarkers that may be indicative of underlying physiological mechanisms that may be occurring among the relationships of psychological stress, depressive symptoms, body mass, and central adiposity. Glucocorticoids, specifically cortisone, have been recognized as a key factor in producing metabolic abnormalities or insulin resistance in the body. Elevated glucocorticoids, together with insulin, can result in increased intraabdominal fat stores. Excessive glucocorticoids (cortisone) and subsequent insulin resistance have been

associated with hypertension, central adiposity, and dyslipidemia (Qi & Rodrigues,

2007). Elevated glucocorticoids are reported to have damaging effects, particularly

associated with inflammation, which could lead to adverse health conditions, such as

diabetes, cardiovascular disease, and the cardiometabolic syndrome (Dallman et al.,

2004).

Continued research aimed at understanding, identifying, and treating depressive

symptoms and psychological stress are needed to ensure that childhood disorders receive

attention. Replication of this research with other ages and ethnicities to determine

consistent findings needs to be conducted. Childhood obesity is a pressing issue

necessitating continued research, especially with increasing numbers of children with

elevated body mass and central adiposity. This study was one of the few studies that

examined the relationships among psychological factors (psychological stress and

depressive symptoms), body mass, and central adiposity in children. This study was the

first study to examine cortisol as a mediating variable between psychological stress and

body mass; between psychological stress and central adiposity; between depressive

symptoms and body mass; and between depressive symptoms and central adiposity in

children. Given the paucity of studies in children that included the variables of study, it

is difficult to draw any conclusions. Thus, more studies are needed in order to make

meaningful and comparative evaluations across studies. Because the confounding

variables gender, ethnicity, puberty, and SES did not reveal a significant contribution to

the variance in body mass and central adiposity, other factors need to be considered.

Understanding which variables do not contribute to body mass and central adiposity

allows researchers to focus on other possible explanations for elevated body mass and

central adiposity. Nurses and other health care professionals need to consider psychological factors in their research of childhood obesity, given the finding that depressive symptoms and psychological stress influence body mass and central adiposity in school-aged children. Furthermore, in this study, healthy weight children were found to also experience levels of psychological stress and depressive symptoms; this finding has implications for the mental and physical health of today's children. A greater knowledge of these concerns will help ensure suitable preventive measures and interventions that foster healthier, more positive and happier children.

Summary

In this study, the principal researcher examined the relationships among psychological stress, cortisol, body mass and central adiposity and among depressive symptoms, cortisol, body mass, and central adiposity in 147 (84 girls, 63 boys) 10-, 11-, and 12-year-old children in the school setting. In this group of children, the mean scores for depressive symptoms and psychological stress were similar to those found in previous studies. Children with higher scores on the depressive symptoms and psychological stress instruments had elevated body mass and central adiposity, although children with normal BMIs and waist circumference values noted depressive symptoms and psychological stress. The majority of children were either overweight or obese despite recruitment of essentially well children. Cortisol levels for the majority of the group fell within the normal range of 1.69 to12.81 nmol/l.

For the group, a significant bivariate relationship was seen between psychological stress and depressive symptoms. This finding was noted in the few previous studies of

children in this age group. In addition, no issues with multicollinearity were evident between depressive symptoms and psychological stress; thus, these two variables were determined to be two separate factors.

Depressive symptoms explained a significant amount of the variance in body mass and central adiposity for the entire group when confounding variables gender, puberty, ethnicity, and SES were controlled in the model. This has not been demonstrated in previous studies. Psychological stress did not significantly explain the variance in body mass and central adiposity in the full model with depressive symptoms as a covariate; however, when depressive symptoms was removed, psychological stress accounted for a significant portion of the variance in body mass and central adiposity. Because no significant relationships existed between depressive symptoms and cortisol, or between psychological stress and cortisol, the testing for mediation did not continue. Last, although relationships have been previously reported among psychological stress, cortisol, body mass, and central adiposity, and among depressive symptoms, cortisol, body mass, and central adiposity in adults, only some of these relationships were supported in the study. Depressive symptoms had direct effects on body mass and central adiposity in these children but were not mediated by cortisol levels.

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APPENDIX A

PARENTAL LETTER

Dear Parents of 5th and 6th Grade Children,

My name is Thuy Lam, and I am a doctoral student at the University of Alabama at Birmingham School of Nursing. I am conducting a research study as part of my doctoral program.

I am inviting your child to participate in the study. This research study involves looking at how your child's feelings affect their health. This research study will examine the effects of psychological stress, depressive symptoms, and cortisol on body weight and size in children ages 10-12 years. This study will enroll approximately 136 participants from the Limestone County Schools and will come from the fifth and sixth grade class.

The study procedures are described in the attached parent/guardian consent document. We will also ask your child to sign an assent form, to make sure your child wants to participate. Parental consent and child assent are required if your child is to participate. If you agree for your child to be in the study, please sign the enclosed parent/guardian consent document and the enclosed Demographic Questionnaire and return them to the school in the enclosed envelope. The second copy of the consent document is for you to keep.

Your child may choose not to be in the study or you or your child may stop participation in the study at any time. This will not affect your child's class standing or grades at ____[Name]_____ School. There will be no cost to you for your child taking part in the study. Your child will receive a choice of scented pencils, erasers, or notebooks at the completion of the study.

If you have questions about the study, I will be available at the school on ____[Date]_____. I will be more than happy to answer your questions. Please return the consent document and Demographic Questionnaire by ___[Date]_____.

I want to thank you in advance for your consideration in this matter. Please feel free to contact me with any questions or concerns at (256) 479-0240 or by email at lamt@uab.edu

Thank you for your time and consideration,

Thuy Lam, MSN, RN

Please remember to return the parent/guardian consent document and the Demographic Questionnaire in the enclosed envelope by ___[Date]_____.

APPENDIX B

DEMOGRAPHIC QUESTIONNAIRE

Code Number_____

DEMOGRAPHIC QUESTIONNAIRE

Directions: Please fill in the blanks or check the box next to the correct response.

1. CHILD'S AGE:

- ☐ 10
- ☐ 11
- ☐ 12

2.   SEX:

- ☐ Male
- ☐ Female

3. GRADE LEVEL:
- ☐ 5th Grade
- ☐ 6th Grade

4. RACE/ETHNICITY:

- ☐ African American
- ☐ Asian
- ☐ Caucasian
- ☐ Hispanic
- ☐ Native American
- ☐ Other, please indicate_____

5. PARENTS' OCCUPATION: Please be specific as possible and list the parent's job title.   For example, BANKER is too general. BANK TELLER is specific.

Father _____

  How long have you held this job title? _____ (in months or years)

Mother _____

How long have you held this job title? _____ (in months or years)
6. What is mother's highest level of education? Please select one.

□   Less than high school degree
□   GED
□   High school degree
□   Some college/community college
□   Bachelor's degree
□   Graduate/professional degree
□   Other, please specify: _____

7. What is father's highest level of education? Please select one.

□   Less than high school degree
□   GED
□   High school degree
□   Some college/community college
□   Bachelor's degree
□   Graduate/professional degree
□   Other, please specify: _____

APPENDIX C

PARENTAL CONSENT

Parent/Guardian Consent Document

TITLE OF RESEARCH: The Effects of Psychological Stress, Depressive Symptoms, and Cortisol on Body Mass and Abdominal Obesity in 10 to 12-Year-Old Children

IRB PROTOCOL: X101011007

INVESTIGATOR: Thuy Lam, MSN, RN; Marti Rice, PhD, RN (faculty advisor)

SPONSOR: The University of Alabama at Birmingham School of Nursing

For Children/Minors (persons under 19 years of age) participating in this study, the term *You* addresses both the participant ("you") and the parent or legally authorized representative ("your child").

Explanation of Procedures

Your child is being asked to participate in a study that will examine how children's feelings affect their health. This research study is designed to examine the effects of psychological stress, depressive symptoms, and cortisol (a hormone related to stress) on body weight and size in children ages 10 to 12 years. This study will enroll approximately 136 participants from Limestone County Schools who are: in the 5th and 6th grade, 10, 11, or 12 years of age, and capable of completing the surveys. The first 136 participants who are eligible will be enrolled in this study.

You will be asked to provide demographics information about your child.

Your child will be asked to provide samples of saliva (spit). The saliva (spit) will be used to measure your child's stress hormones. Your child's height, weight, and waist circumference will be measured. These measurements will be conducted in a private room or behind a screen and written on an information sheet. Your child will complete a questionnaire that asks about his/her physical growth and pubertal development.

Your child will also be asked to complete questionnaires about his/her feelings and how he/she normally acts. Your child will be asked to complete a set of questionnaires about stress and depressive symptoms that should take about 35 minutes.

All data collection with your child will occur at the school during school time. All information from your child will be collected during your child's physical education or elective class on one day. It will take approximately 1 hour to get all of this information. By participating, your child will not miss class information or school work.

UAB IRB

09/28/2011

Date of Approval 10-12-11

Not Valid On 10-12-12

Initials of Parent/Legal Guardian: _____

Page 1 of 3

Risks and Discomforts

Your child may experience some psychological discomfort or potential embarrassment related to being weighed, measured for height and waist circumference, or during the completion of the pubertal development, stress, and depressive symptoms questionnaires; however, the risk involved is minimal. Efforts will be made to limit the potential for other students' knowledge of your child's weight, height, or waist circumference through the use of privacy screens. A school counselor will be available should your child experience any emotional upset during measurement. If your child becomes upset during completion of any of the questionnaires, they may stop at any time. If your child's responses to the questionnaires indicate a high level of stress or depressive symptoms, then a school counselor will be notified for recommendations based on the school's policy.

Benefits

Your child may not benefit directly from participation in this research. However, findings from this study can guide us and other health professionals in developing better programs for helping children.

Alternatives

The alternative is for your child not to participate in the study.

Confidentiality

Information obtained for this will be kept private to the extent allowed by law. The research results may be published for scientific purposes; however, your child's identity will not be revealed in any way by name. Research information that identifies your child may be shared for ensuring compliance with the University of Alabama at Birmingham (UAB) Institutional Review Board (IRB) and others who are responsible for ensuring compliance with laws and regulations related to research, including the Office for Human Research Protections (OHRP). In order to minimize the risk of a breach in confidentiality, all questionnaires will be coded with randomly assigned numbers. The computer database used to store the data will be password-protected and will be accessible only to the principal investigator of this study. All research data will be stored in a locked cabinet with access limited to the principal investigator.

Refusal or Withdrawal without Penalty

Your child is taking part in this study by choice. There will be no penalty if you or your child decides not to take part in the study. You and your child may choose not to be in the study, or you may withdraw (stop) the study at any time before it is over. If you do, you should tell the investigator or tell the child's teacher. This will not affect your child's class standing or your class grades. If you or your child would like to withdraw, tell Ms. Thuy Lam.

Cost of Participation

There will be no cost to you for your child's participation in this study.

Payment for Participation in Research

Your child will receive a small incentive for participating in the study. This incentive will be the choice of scented pencils, erasers, or notebooks. The incentive is valued at approximately $2.00.

Questions

If you have any questions, concerns, or complaints about the research, please contact: Ms. Thuy Lam, RN, MSN. She will be glad to answer any of your questions. Ms. Lam's number is (256) 479-0240. If you have questions about your rights as a research participant, or concerns or complaints about the research, you may contact the Office of the Institutional Review Board for Human Use (OIRB) at (205) 934-3789 or 1-800-822-8816. If calling the toll-free number, press the option for "all other calls" or for an operator/attendant and ask for extension 4-3789. Regular hours for the OIRB are 8:00 a.m. to 5:00 p.m. CT, Monday through Friday. You may also call this number in the event the research staff cannot be reached or you wish to talk to someone else.

Legal Rights

You are not waiving any of your or your child's legal rights by signing this informed consent document.

Signatures

You are making a decision whether or not to have your child participate in this study. Your signature indicates that you have read (or been read) the information provided above and decided to allow your child to participate. You have been provided a copy of this consent form.

_____   _____
Signature of Participant or Legally Authorized Representative   Date

_____   _____
Child's Name   Date

_____   _____
Signature of Principal Investigator   Date

09/28/2011

APPENDIX D

CHILD ASSENT FORM

Child Assent Form

Title: The Effects of Psychological Stress, Depressive Symptoms, and Cortisol on Body Mass and Abdominal Obesity in 10 to 12-Year-Old Children

IRB Protocol No.:   X101011007

Sponsor: The University of Alabama School of Nursing

Investigators: Thuy Lam, MSN, RN; /Marti Rice, PhD, RN (faculty advisor)

The investigators named above are doing a research study.

These are some things we want you to know about research studies:
We are asking you to be in a research study. Research is a way to test new ideas. Research helps us learn new things.

Whether or not to be in this research is your choice. You can say Yes or No. Whatever you decide is OK.

Why am I being asked to be in this research study?
You are being asked to be in the study because researchers want to know how your feelings affect your health.

What is the study about?
The study is about how feelings affect children's health. The study will be checking your saliva (spit) to see how it changes according to how you feel. This research is important because it can help researchers understand the health of children.

What will happen during this study?
If you agree to be in this study, you will

- Have your weight, height, and waist circumference (around your waist) measured.
- Provide a saliva (spit) specimen.
- Answer questions about your feelings and physical growth.
- Get a choice of scented pencils, erasers, or notebooks for participating.

Will the study hurt?
Taking part in these tests will not hurt. If you not want to do any of the tests, you do not have to.

Participant's Initials _____

UAB IRB

Date of Approval 10-12-11

Not Valid On 10-12-12

What else should I know about the study?

You do not have to answer any questions that are asked of you. There may be risk of embarrassment if other students are told your weight. However, researchers will provide for your privacy by using a screen and weighing one student at a time.

What are the good things that might happen?

People may have good things happen to them because they are in a research study. These are called "benefits". Researchers may learn important facts about children that are not already known.

What if I don't want to be in this study?

You do not have to be in the study if you do not want to. You will still be able to participate in your class even if you decide that you do not want to be in the study.

Who should I ask if I have any questions?

If you have any questions about this study, you or your parents can call Ms. Thuy Lam, RN at (256) 479-0240.

Do I have to be in the study?

No, you do not have to be in the study. Even if you say yes now, you can change your mind later. It is up to you. No one will be mad at you if you don't want to do this.

Now that I have asked my questions and think I know about the study and what it means, here is what I decided:

_____ OK. I'll be in the study.   ___   No, I do not want to be in the study.

The researchers have told me about the research. I had a chance to ask questions. I know I can ask questions at any time. I want to be in the research.

If you sign your name below, it means that you agree to take part in this research study.

_____ _____ _____
Your Name (Printed)   Age   Date

_____ _____   _____
Your Signature   Date

_____   _____
Signature of Person Obtaining Consent   Date

APPENDIX E

SCHOOL LETTER OF SUPPORT

LIMESTONE COUNTY BOARD OF EDUCATION

300 South Jefferson Street
Athens, Alabama 35611
(256) 232-5353
Fax: (256) 233-6461
Website: www.lcsk12.org

April 4, 2011

Thuy Lam, MSN, RN
UAB Doctoral Student
102 Old Ivy Circle
Madison, AL 35756

Dear Ms. Lam:

This letter is in support of your research project involving the study of psychological stress, depressive symptoms, and cortisol on body mass and central adiposity in middle school-aged children that is being submitted for review to the Institutional Review Board (IRB) at the University of Alabama at Birmingham.

I am granting you access to the Limestone County Schools starting September 1, 2011 and ending on May 30, 2012 in order to recruit participants and collect data for your project. The participants will include fifth and sixth graders from elementary and middle schools within the school district.

This research project will help explain the contributing factors and underlying mechanisms of increased body mass and central adiposity in middle school-aged children, laying the groundwork for interventions to promote healthy minds and bodies at one of the most critical times in this group's physical and psychological growth. Therefore, I fully support research that can help our students in improving their overall health and well-being.
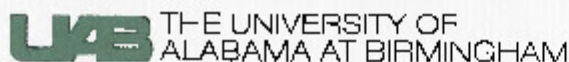
Sincerely,

Barry L. Carroll, Ed.D.
Superintendent of Education

Commitment To Children

APPENDIX F

INSTITUTIONAL REVIEW BOARD FOR HUMAN USE APPROVAL

UAB THE UNIVERSITY OF
ALABAMA AT BIRMINGHAM

Institutional Review Board for Human Use

Form 4: IRB Approval Form
Identification and Certification of Research
Projects Involving Human Subjects

UAB's Institutional Review Boards for Human Use (IRBs) have an approved Federalwide Assurance with the Office for Human Research Protections (OHRP). The Assurance number is FWA00005960 and it expires on August 29, 2016. The UAB IRBs are also in compliance with 21 CFR Parts 50 and 56.

Principal Investigator: LAM, HONG-THUY D

Co-Investigator(s):

Protocol Number: **X101011007**

Protocol Title: *The Effects of Psychological Stress, Depressive Symptoms, and Cortisol on Body Mass and Abdominal Obesity in 10–12 Year-Old Children*

The IRB reviewed and approved the above named project on ___10-12-11___. The review was conducted in accordance with UAB's Assurance of Compliance approved by the Department of Health and Human Services. This Project will be subject to Annual continuing review as provided in that Assurance.

This project received EXPEDITED review.

IRB Approval Date: ___10-12-11___

Date IRB Approval Issued: ___10-12-11___

Marilyn Doss

Marilyn Doss, M.A.
Vice Chair of the Institutional Review
Board for Human Use (IRB)

Investigators please note:

The IRB approved consent form used in the study must contain the IRB approval date and expiration date.

IRB approval is given for one year unless otherwise noted. For projects subject to annual review research activities may not continue past the one year anniversary of the IRB approval date.

Any modifications in the study methodology, protocol and/or consent form must be submitted for review and approval to the IRB prior to implementation

Adverse Events and/or unanticipated risks to subjects or others at UAB or other participating institutions must be reported promptly to the IRB.

470 Administration Building | The University of
701 20th Street South | Alabama at Birmingham
205.934.3789 | Mailing Address:
Fax 205.934.1301 | AB 470
irb@uab.edu | 1530 3RD AVE S
BIRMINGHAM AL 35294-0104

APPENDIX G

A SELF-ADMINISTERED RATING SCALE FOR PUBERTAL DEVELOPMENT

A Self-Administered Rating Scale for Pubertal Development

Introduction: These questions are about changes that may be happening to your body. These changes normally happen to different young people at different ages. Since these changes have something to do with your stress hormones, do your best to answer carefully. If you do not understand a question or do not know the answer, just mark "I don't know." <u>Please circle the letter of the response to each item that is most like you.</u>

1. What would you say about your growth in height?

    a. I have not yet begun to spurt (grow).
    b. I have barely started.
    c. My growth in height is definitely underway.
    d. My growth seems completed.
    e. I don't know.

2. What would you say about the growth of your body hair ("Body hair" means hair any place other than your head, such as under your arm)?

    a. My body hair has not yet begun to grow.
    b. My body hair has barely started to grow.
    c. The growth of my body hair is definitely underway.
    d. The growth of my body hair seems completed.
    e. I don't know.

3. Have you noticed any skin changes, especially pimples?

    a. Skin has not yet started changing.
    b. Skin has barely started changing.
    c. Skin changes are definitely underway.
    d. Skin changes seem complete.
    e. I don't know.

4. Have you noticed that your breasts have begun to grow?

    a. My breasts have not yet started growing.
    b. My breasts have barely started growing.
    c. My breast growth is definitely underway.
    d. My breast growth seems complete.
    e. I don't know.

5a. Have you begun to menstruate (started to have your period)?

    a. Yes (if yes, please answer 5b).
    b. No.

5b. If yes, how old were you when started to menstruate?

    _____ years- old

Author: Carskadon, M.

A Self-Administered Rating Scale for Pubertal Development

Introduction: These questions are about changes that may be happening to your body. These changes normally happen to different young people at different ages. Since these changes have something to do with your stress hormones, do your best to answer carefully. If you do not understand a question or do not know the answer, just mark "I don't know." <u>Please circle the letter of the response to each item that is most like you.</u>

5. What would you say about your growth in height?

    f.   I have not yet begun to spurt (grow).
    g.   I have barely started.
    h.   My growth in height is definitely underway.
    i.   My growth seems completed.
    j.   I don't know.

6. What would you say about the growth of your body hair ("Body hair" means hair any place other than your head, such as under your arm)?

    f.   My body hair has not yet begun to grow.
    g.   My body hair has barely started to grow.
    h.   The growth of my body hair is definitely underway.
    i.   The growth of my body hair seems completed.
    j.   I don't know.

7. Have you noticed any skin changes, especially pimples?

    f.   Skin has not yet started changing.
    g.   Skin has barely started changing.
    h.   Skin changes are definitely underway.
    i.   Skin changes seem complete.
    j.   I don't know.

8. Have you noticed a deepening of your voice?

    f. Voice has not yet started changing.
    g. Voice has barely started changing.
    h. Voice changes are definitely underway.
    i. Voice changes seem complete.
    j. I don't know.

9. Have you begun to grow hair on your face?

    a. Facial hair has not yet started growing.
    b. Facial hair has barely started growing.
    c. Facial hair growth has definitely started.
    d. Facial hair growth seems complete.
    e. I don't know.

PROTOCOL FOR ADMINISTRATION OF THE "SELF-ADMINISTERED SCALE
FOR PUBERTAL DEVELOPMENT"

Prior to Data Collection have:

- A second data collector
- Pencils and clipboard (if necessary)

1. Distribute instruments and pencils to each student to complete with researcher. The data collector determines that the student is ready to begin the Self-Administered Scale for Pubertal Development and asks them to turn to the page that has at the top "Self-Administered Scale for Pubertal Development." Students should be reminded not to put their names anywhere on the instrument and that extra sharp pencils are available, if needed.

2. Ask students to look at the instrument as you read the directions. These questions are about changes that may be happening to your body. These changes normally occur to young people at different ages. Since these changes have something to do with your stress hormones, do your best to answer carefully. If you do not understand or do not know the answer, just mark "I don't know." Please circle the letter of the response to each item that is most like you.

3. The data collector should allow time for students to mark their answer to questions allowing time for each child to answer before moving on. Remind students when to turn to the next page before reading the next question.

4. Ask the students to take a few minutes to look over their questionnaire to be sure they have answered all questions, and have not accidentally left questions unanswered.

5. A data collector should review each participant's questionnaire as it is turned in, to be certain that every question is marked, and no portions of the questionnaire have been accidently left blank. Students may refuse to answer certain questions.

Adapted from the PASS program, with permission from Dr. Marti Rice, PI, The University of Alabama at Birmingham.

Thuy Lam

From: Carskadon, Mary [mary_carskadon@brown.edu]
Sent: Tuesday, September 07, 2010 2:14 PM
To: Thuy Lam
Subject: Re: Rating Scale for Pubertal Development

Dear Thuy Lam. Please feel free to use the rating scale. It is not the best way to assess puberty, but if you have no other, I suppose it will do. Good luck with your research. MAC

On Tue, Sep 7, 2010 at 10:02 AM, Thuy Lam <Thuy.Lam@drakestate.edu> wrote:

Dear Dr. Carskadon,

My name is Thuy Lam and I am a nursing student in the PhD program at the University of Alabama at Birmingham. My dissertation topic involves examining how psychological stress, depressive symptoms, and cortisol influence body mass and abdominal obesity in 10 to 12-year-old children. I will be controlling for pubertal status, along with other variables as part of my research. I would like to use the Rating Scale for Pubertal Development, and I am seeking your permission to use the scale with participants (both males and females) within the study.

Please let me know if you have any questions or concerns. I will be more than happy to answer them.

Kind regards,

Thuy Lam

Thuy Lam, MSN, RN

UAB PhD Student and LECHN Fellow

Phone: (256) 479-0240

Email: Thuy.Lam@drakestate.edu or lamt@uab.edu

APPENDIX H

FEEL BAD SCALE

Feel Bad Scale

Instructions: The following is a list of things that some kids say makes them feel bad, nervous, or make them worry. For each item, mark an "X" in the box next to the best phrase showing how you would feel *if this happened to you*, or if this has happened to you, how you felt. There is no wrong or right answer.

1. **Having parents separate** — Not bad ☐ A Little Bad ☐ Pretty Bad ☐ Real Bad ☐ Terrible ☐

2. **Being pressured to try something new** — Not bad ☐ A Little Bad ☐ Pretty Bad ☐ Real Bad ☐ Terrible ☐

3. **Having your parents argue in front of you** — Not bad ☐ A Little Bad ☐ Pretty Bad ☐ Real Bad ☐ Terrible ☐

4. **Not spending enough time with your mom or dad** — Not bad ☐ A Little Bad ☐ Pretty Bad ☐ Real Bad ☐ Terrible ☐

5. **Feeling sick** — Not bad   A Little Bad ☐ Pretty Bad ☐ Real Bad   Terrible ☐

6. **Fighting with parents about house rules** — Not bad ☐ A Little Bad ☐ Pretty Bad ☐ Real Bad ☐ Terrible ☐

7. **Not having homework done on time** — Not bad ☐ A Little Bad ☐ Pretty Bad ☐ Real Bad ☐ Terrible ☐

8. **Moving from one place to another** — Not bad ☐ A Little Bad ☐ Pretty Bad ☐ Real Bad ☐ Terrible ☐

9. **Not getting along with your teacher** — Not bad ☐ A Little Bad ☐ Pretty Bad ☐ Real Bad ☐ Terrible ☐

10. **Being overweight or bigger than others your age** — Not bad ☐ A Little Bad ☐ Pretty Bad ☐ Real Bad ☐ Terrible ☐

11.	**Changing schools**	Not bad ☐	A Little Bad ☐	Pretty Bad ☐	Real Bad ☐	Terrible ☐
12.	**Not having enough money to spend**	Not bad ☐	A Little Bad ☐	Pretty Bad ☐	Real Bad ☐	Terrible ☐
13.	**Not being able to dress the way you want to**	Not bad ☐	A Little Bad ☐	Pretty Bad ☐	Real Bad ☐	Terrible ☐
14.	**Feeling left out of a group**	Not bad ☐	A Little Bad ☐	Pretty Bad ☐	Real Bad ☐	Terrible ☐
15.	**Having nothing to do**	Not bad ☐	A Little Bad ☐	Pretty Bad ☐	Real Bad ☐	Terrible ☐
16.	**Pressured to get good grades**	Not bad ☐	A Little Bad ☐	Pretty Bad ☐	Real Bad ☐	Terrible ☐
17.	**Not being good enough at sports**	Not bad ☐	A Little Bad ☐	Pretty Bad ☐	Real Bad ☐	Terrible ☐
18.	**Being late for school**	Not bad ☐	A Little Bad ☐	Pretty Bad ☐	Real Bad ☐	Terrible ☐
19.	**Feeling like your body is changing**	Not bad ☐	A Little Bad ☐	Pretty Bad ☐	Real Bad ☐	Terrible ☐
20.	**Being smaller than others your age**	Not bad ☐	A Little Bad ☐	Pretty Bad ☐	Real Bad ☐	Terrible ☐

Instructions: Now please indicate if any of these things has happened to you in the past year, and if so, how often. There is no wrong or right answer.

---

1. **Having parents separate**   Never ☐   Once or Twice ☐   Sometimes ☐   Often ☐   All the time ☐

2. **Being pressured to try something new**   Never ☐   Once or Twice ☐   Sometimes ☐   Often ☐   All the time ☐

3. **Having your parents argue in front of you**   Never ☐   Once or Twice ☐   Sometimes ☐   Often ☐   All the time ☐

4. **Not spending enough time with your mom or dad**   Never ☐   Once or Twice ☐   Sometimes ☐   Often ☐   All the time ☐

5. **Feeling sick**   Never ☐   Once or Twice ☐   Sometimes ☐   Often ☐   All the time ☐

6. **Fighting with parents about house rules**   Never ☐   Once or Twice ☐   Sometimes ☐   Often ☐   All the time ☐

7. **Not having homework done on time**   Never ☐   Once or Twice ☐   Sometimes ☐   Often ☐   All the time ☐

8. **Moving from one place to another**   Never ☐   Once or Twice ☐   Sometimes ☐   Often ☐   All the time ☐

9. **Not getting along with your teacher**   Never ☐   Once or Twice ☐   Sometimes ☐   Often ☐   All the time ☐

10. **Being overweight or bigger than others your age**   Never ☐   Once or Twice ☐   Sometimes ☐   Often ☐   All the time ☐

11.	Changing schools	Never ☐	Once or Twice ☐	Sometimes ☐	Often ☐	All the time ☐
12.	Not having enough money to spend	Never ☐	Once or Twice ☐	Sometimes ☐	Often ☐	All the time ☐
13.	Not being able to dress the way you want to	Never ☐	Once or Twice ☐	Sometimes ☐	Often ☐	All the time ☐
14.	Feeling left out of a group	Never ☐	Once or Twice ☐	Sometimes ☐	Often ☐	All the time ☐
15.	Having nothing to do	Never ☐	Once or Twice ☐	Sometimes ☐	Often ☐	All the time ☐
16.	Pressured to get good grades	Never ☐	Once or Twice ☐	Sometimes ☐	Often ☐	All the time ☐
17.	Not being good enough at sports	Never ☐	Once or Twice ☐	Sometimes ☐	Often ☐	All the time ☐
18.	Being late for school	Never ☐	Once or Twice ☐	Sometimes ☐	Often ☐	All the time ☐
19.	Feeling like your body is changing	Never ☐	Once or Twice ☐	Sometimes ☐	Often ☐	All the time ☐
20.	Being smaller than others your age	Never ☐	Once or Twice ☐	Sometimes ☐	Often ☐	All the time ☐

PROTOCOL FOR ADMINISTRATION OF THE "FEEL BAD SCALE"

Prior to Data Collection have:
- A second data collector
- Coded instruments
- Pencils and clipboards (if necessary)

1. Distribute coded instruments and pencils to students. Students should be reminded not to put their names anywhere on the instrument, and that extra pencils are available if needed.

2. Students are informed that they should raise their hand if they have a question, and the second data collector, who is circulating around the room, will help them.

3. Ask students to look at the instrument as you read the directions: "The following is a list of things that some kids say makes them feel bad, nervous, or make them worry. For each item, mark an "X" in the box next to the phrase showing how you would feel if this happened to you, or if this has happened to you, how you felt. There is no wrong or right answer. Now please indicate if any of these things has happened to you in the past year, and if so, how often. There is no wrong or right answer." Suggest that the class begin by reading the first question as an example and talking about it together as in #4 below.

4. The data collector reads the example of the first item on the questionnaire, "*Having parents separate-* you would place a mark in the box next (on the right) to the phrase that best shows how you feel if this happened to you, or how you felt when this happened to you." The data collector explains, "If you do not feel bad, then place a mark next to the box of "not bad". If you feel a little bad, pretty bad, real bad, or terrible, place a mark next to the box that best describes your feelings. Remember, choose the phrase which best describes how you feel."

5. The data collector should allow time for students to mark their answers. The data collector asks the students to begin with question one until they complete every question. Remind students to raise their hands if they have questions. Ask students to take a few minutes to look over their questionnaire to be sure they have answered all questions, and have not accidently left questions unanswered.

6. A second data collector should review each participant's questionnaire as it is turned in, to be certain that every question is marked, and no portions of the questionnaire have been accidently left blank. Students may refuse to answer questions.

Thuy Lam

From:	Judith Siegel [jmsiegel@ucla.edu]
Sent:	Wednesday, March 17, 2010 4:20 PM
To:	Thuy Lam
Subject:	Re: children questionnaire
Attachments:	Feel Bad AJPH 1984.pdf

Hello Thuy,

You may use the instrument without charge. I don't know if you have the AJPH article, but I've attached it in case you don't. It has all of the info you need to prepare a copy of the scale for your use. Good luck on your project.

At 10:12 AM 3/17/2010, you wrote:

Greetings Dr. Siegel,

My name is Thuy Lam and I am a PhD student at the University of Alabama in Birmingham. I am hoping that you could assist me with information regarding the Feel Bad Scale. I am unable to contact Dr. Lewis as he is retired from UCLA. I am interested in using the FBS in my dissertation work with children (stress and depressive symptoms influence on childhood obesity). My question- Is the instrument available for use and where may I purchase it? Would you kindly assist me? I would greatly appreciate it!

Kind regards,
Thuy Lam

Judith M. Siegel, PhD, MSHyg
Professor, UCLA School of Public Health

--
This message has been scanned for viruses and dangerous content by **MailScanner**, and is believed to be clean.

APPENDIX I

CHILDREN'S DEPRESSION INVENTORY PERMISSON

RE: research study-Children's Depression Inventory

marika kovacs [kovacs@pitt.edu]

Sent: Saturday, September 11, 2010 7:06 PM
To: Thuy Lam
Cc: Hazel Wheldon [Hazel.Wheldon@MHS.com]

Dear Thuy Lam:

Thank you for your interest in the CDI. I am pleased to hear that you plan to use for your dissertation.

May I suggest that you contact directly the publisher of the CDI, Multi-health Systems Inc (www.mhs.com) for the CDI and be sure to tell them that you are doing your dissertation research.

We are in the process of publishing the revised version of the CDI—so if you experience any delay in getting it, please feel free to contact Ms. Wheldon (publisher at MHS—she is being copied) she can assure that you receive the best possible service.

with regards.
M. Kovacs

From: Thuy Lam [mailto:Thuy.Lam@DrakeState.edu]
Sent: Saturday, September 11, 2010 1:20 PM
To: kovacs@pitt.edu
Cc: Thuy Lam
Subject: research study-Children's Depression Inventory

Dear Dr. Kovacs,

My name is Thuy Lam and I am a nursing student in the PhD program at the University of Alabama at Birmingham. My dissertation topic involves examining how psychological stress, depressive symptoms, and cortisol influence body mass and abdominal obesity in 10 to 12-year-old children. My plan is to use the CDI to assess children's depressive symptoms. I am requesting your permission to use the instrument for my research study. I understand that I may purchase the CDI through the Pearson Vue website.

Please let me know if you have any questions or concerns. I will be more than happy to answer them.

Kind regards,

Thuy Lam, MSN, RN
UAB PhD student and LECHN Fellow

(256) 479-0240
Email: Thuy.Lam@drakestate.edu or lamt@uab.edu

—

This message has been scanned for viruses and dangerous content by MailScanner, and is believed to be clean.

PROTOCOL FOR ADMINISTRATION OF THE "CHILDREN'S DEPRESSION INVENTORY"

Prior to Data Collection have:
- A second data collector
- Coded instruments
- Pencils and clipboards (if necessary)

7. Distribute coded instruments and pencils to students. Students should be reminded not to put their names anywhere on the instrument, and that extra pencils are available if needed.

8. Students are informed that they should raise their hand if they have a question, and the second data collector, who is circulating around the room, will help them.

9. Ask students to look at the instrument as you read the directions: "Kids sometimes have different feelings and ideas. This form lists the feelings and ideas in groups. From each group of three sentences, pick one sentence that describes you *best* for the past two weeks. After you pick a sentence from the first group, go on to the next group. There is no right or wrong answer. Just pick the sentence that best describes the way you have been recently. Put a mark like this X next to your answer. Put the mark in the box next to the sentence that you pick." Suggest that the class begin by reading an example of how this form works.

10. The data collector reads the example, "I read books all the time. I read books once in a while. I never read books. Please put a mark next to the sentence that describes you best."

11. The data collector should allow time for students to mark their answers. The data collector asks the students to begin with question one until they complete every question. Remind students to raise their hands if they have questions. Ask students to take a few minutes to look over their questionnaire to be sure they have answered all questions, and have not accidently left questions unanswered.

12. A second data collector should review each participant's questionnaire as it is turned in, to be certain that every question is marked, and no portions of the questionnaire have been accidently left blank. Students may refuse to answer questions.

APPENDIX J

PHYSICAL AND PHYSIOLOGICAL MEASUREMENT PROTOCOLS

Protocol for Measurement of Height

Equipment:

Stadiometer
Data collection sheet

1. Tell the child that you are going to measure his/her height.

2. Request that the child take off his shoes.

3. The child should stand at a right angle to the vertical rod of the stadiometer.

4. The child should stand with his/her weight evenly distributed between both feet, and the arms hanging by the sides with the palms facing the thighs. The heels are together, touching the vertical board of the stadiometer. The feet are spread at a 60 degree angle to each other.

5. When possible, tell the child to have his/her head, scapula, and buttocks touching the vertical board.

6. Request that the head be erect with eyes focused straight ahead.

7. Tell the child to take a deep breath and then lower the horizontal board of the stadiometer to the most superior point on the head, compressing the hair.

8. Measure the height to the nearest 1/4 inch.

9. Record the height in the space on the data collection form.

Heyward, V., & Wagner, D. (2004). *Applied composition assessment* (2nd). Champaign, IL: Human Kinetics

Adapted from the PASS program, Dr. Marti Rice, PI, The University of Alabama at Birmingham.

Protocol for Measurement of Weight

Equipment:

Weight beam scales
Data collection sheet

1. Be sure that the scales are calibrated to zero before the parent/child stands on the scale.

2. Ask one child at a time to come behind the privacy screen or the private area.

3. Ask the child to remove his/her shoes.

4. Ask the child to step up on the platform and stand facing the balance beam.

5. Ask the parent child to stand so that the body weight is evenly distributed between the feet.

6. Adjust the scale weights until the balance beam hangs free.

7. Record weight to the nearest 1/4 of a lb. on the data collection sheet.

Heyward, V., & Wagner, D. (2004). *Applied composition assessment* (2nd). Champaign, IL: Human Kinetics

Adapted from the PASS program, Dr. Marti Rice, PI, The University of Alabama at Birmingham.

Protocol for Measurement of Waist Circumference

Equipment:

Tape measure
Marking pen
Data collection sheet
Privacy Screen

1. Tell the child that you are going to measure his/her waist.

2. Be sure that the waist is measured in a private area so that only the participant and data collector (s) are present.

3. Have the child raise his/her shirt or blouse.

4. In order to determine the level at which the waist circumference is measured, the data collector must stand to the right of the participant and palpates the upper hip bone to locate the right iliac crest.

5. Just above the uppermost lateral border of the right iliac crest, a horizontal mark is drawn, then crossed with a vertical mark on the midaxillary line.

6. The measuring tape is placed in the horizontal plane around the abdomen at the level of this marked point on the right side of the trunk.

7. The plane of the tape is parallel to the floor and the tape is snug, but does not compress the skin.

8. Tell the child to breathe normally and measure the circumference to the nearest 1/16 inch.

9. Record the measurement in the appropriate boxes on the data collection sheet.

National Heart, Lung, and Blood Institute (1998). Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: The Evidence report, NIH Publication No. 98-4083, September, 1998. Retrieved from on June 18, 2011 http://www.nhlbi.nih.gov/guidelines/obesity/prctgd_c.pdf

Adapted from the PASS program, Dr. Marti Rice, PI, The University of Alabama at Birmingham.

Protocol for Collection of Saliva

1. When the participant comes into the data collection room, have a small cup of water available for each participant to rinse mouth. The water may be swallowed or spit into a garbage-bag-lined trash can.

2. For each participant, pass out the pre-labeled tubes with the straw inside and the rubber band wrapped around the outside of the tube. Give each child a tissue to use since saliva can be ropey and it can be hard to finish with a string of saliva coming from the mouth.

3. Explain that the participant needs to remove the rubber band and the label with his/her name off the tube. Then, instruct the participants to take the cap off the tube and put it down in front of them. Explain that the straw provided needs to remain inside the tube but may be pulled out slightly. Explain that saliva is what is produced in the mouth and does not come from clearing the throat or coughing up sputum.

4. Have the participants spit into the straw in the saliva specimen tube. Continue to spit until the timer goes off after three minutes.

5. Each child will have 3 minutes to provide as much saliva as they can into the tube. Tell the children you will set the timer and when it goes off they must stop spitting into the tube.

6. If they cannot spit, ask them to chew the rubber band or ask them to suck in the sides of their mouth. This will make the saliva flow. Do not give them gum, koolaid, or sugar because it can change the values of the measures. If they are unable to provide a specimen, you may give them an additional 2 minutes but do not exceed a total spitting time of 5 minutes. Be sure to note which, if any, participants need additional time so this can be recorded on the lab papers once the specimens arrive at UAB.

7. Once the timer goes off, have the child take the straw out of the tube and put the cap back on tube. The data collector will collect each tube and put it in the specimen box. The data collector will **need to push down firmly on the cap so the cap does not come off causing leakage**.

8. Once all the tubes have been collected, label the box and put the box into the cooler and put the chemical ice packs around the box.

9. Once back at UAB, take the specimen box to the lab and put on the assigned shelf in the freezer. Store specimens in -80° freezer until samples are thawed for assay.

Parameter (2012). *Cortisol assay*. Retrieved on February 28, 2012 from
http://www.rndsystems.com/pdf/kge008.pdf

Adapted from the PASS program, Dr. Marti Rice, PI, The University of Alabama at Birmingham.