Physical activity, stress, and the metabolic syndrome in 8-18 yr old boys

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Physical activity, stress, and the metabolic syndrome in 8-18 yr old boys

by

Megan E. Holmes

A thesis submitted to the graduate faculty
in partial fulfillment of the requirements for the degree of

MASTER OF SCIENCE

Major: Exercise and Sport Science
(Biological Basis of Physical Activity)

Program of Study Committee:
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This is to certify that the Masters thesis of

Megan E. Holmes

has met the thesis requirements of Iowa State University.

Signatures have been redacted for privacy.
I am among those who think that science has great beauty. A scientist in his laboratory is not only a technician: he is also a child placed before natural phenomena which impress him like a fairy tale.

~Marie Curie (1867-1934)
TABLE OF CONTENTS

CHAPTER 1. INTRODUCTION AND OVERVIEW
- Background: 2
- Purpose: 2
- References: 3

CHAPTER 2. REVIEW OF LITERATURE: PHYSICAL ACTIVITY, STRESS AND THE METABOLIC SYNDROME
- Introduction: 6
- Epidemiology of Pediatric Obesity and the Metabolic Syndrome: 7
- Physical activity and cardiovascular disease risk factors / Components of the metabolic syndrome: 9
- Role of stress in obesity and the metabolic syndrome: 11
- Relationships between physical activity, stress and the metabolic syndrome: 14
- Summary and Conclusions: 15
- References: 16

CHAPTER 3. MANUSCRIPT: PHYSICAL ACTIVITY, STRESS AND THE METABOLIC SYNDROME IN 8-18 YR OLD BOYS
- Introduction: 23
- Methods: 25
- Results: 30
- Discussion: 32
- References: 36
- Table 1: 41
- Table 2: 43
- Table 3: 44
- Figure 1: 45

APPENDIX 1. ASSOCIATIONS BETWEEN METABOLIC SYNDROME COMPONENTS AND THE METABOLIC SYNDROME SCORE: 46
APPENDIX 2. ASSOCIATIONS BETWEEN SELF-REPORT MEASURES OF STRESS AND WAKING SALIVARY CORTISOL: 47
APPENDIX 3. ASSOCIATIONS BETWEEN SEDENTARY BEHAVIORS AND THE METABOLIC SYNDROME SCORE: 48

ACKNOWLEDGEMENTS: 49
CHAPTER 1. INTRODUCTION AND OVERVIEW

Background

An estimated 16% of United States (US) children and adolescents, 6 to 19 years of age, are considered obese and an additional 30% are overweight, which represents a three-fold increase over the past few decades (1). Childhood obesity is associated with traditional cardiovascular disease (CVD) risk factors, such as hypertension and dyslipidemia (2, 3), as well as insulin resistance (4), and haemostatic risk factors for CVD (5). In addition to the immediate consequences, childhood obesity often tracks into adulthood (6) and has been linked to CVD morbidities in adulthood, including coronary artery calcification (7), dyslipidemia and hypertension (8, 9), carotid artery intima-media thickness (10), and all-cause and CVD mortality (11).

Obesity, and more specifically abdominal or visceral obesity, combined with traditional CVD risk factors is a condition that has been coined the metabolic syndrome (12). According to the most recent National Health and Nutrition Examination Survey (NHANES, 1999-2000), the prevalence of the metabolic syndrome is 6.4% among US adolescents (13), which has increased from 4.2% in the previous NHANES III (1988-1994) data (13). Population estimates suggest that there are approximately 2 million US adolescents who can be classified as having the metabolic syndrome phenotype (13). Even more troubling, an estimated 2/3 of US adolescents possess at least one characteristic of the metabolic syndrome (14, 15) which in turn is associated with an increased risk of CVD and all-cause mortality (16) compared to adults who did not have these risks as children.

With the increased prevalence of pediatric obesity and the metabolic syndrome, considerable interest in prevention is focused on diet and physical activity. However, based
on the results of a recent review by Eisenmann (17) there has been little to no change in energy intake or expenditure in the last few decades, based on self-reported indices. Thus, there is reason to consider other possible causes contributing to the increased prevalence rates of obesity and the metabolic syndrome among youths. One intriguing hypothesis is related to the chronic activation of the hypothalamic-pituitary-adrenal (HPA) axis due to various emotional, environmental, and physical stressors that can create a state of hypercortisolaemia (18-21). Chronically elevated levels of cortisol cause an up-regulation of lipoprotein lipase and subsequent storage of fat, specifically in the viscera (20). Additionally, cortisol is associated with decreased leptin sensitivity (20). Consequently, after eating, circulating leptin does not discourage additional food consumption and energy balance becomes perturbed. Previous research also suggests that increased cortisol levels negatively affect insulin sensitivity (19), a key component in the metabolic syndrome.

To date, few studies have examined the inter-relationships between physical activity, stress, and the metabolic syndrome; however, the available data suggest physical activity during adolescence may improve the risk profile for the metabolic syndrome (22) and perhaps buffer the effects of chronic stress on adiposity (23). Unfortunately, little work has been done toward understanding the relationships among physical activity, stress, and the metabolic syndrome in adolescents.

**Purpose**

The purpose of this thesis is to examine the inter-relationships between physical activity, stress and the metabolic syndrome. More specifically, this study proposes to
examine the moderating effects physical activity may have on the relationship between stress and the components of the metabolic syndrome in adolescent males.

It is hypothesized that there will be a significant relationship between waking cortisol levels as well as other stress-related measures and metabolic syndrome risk factors. Furthermore, it is hypothesized that physical activity will modify the relationship between waking cortisol levels and metabolic syndrome risk factors.

References


CHAPTER 2. REVIEW OF LITERATURE: PHYSICAL ACTIVITY, STRESS AND THE METABOLIC SYNDROME

Introduction

Secular trends in variables associated with the metabolic syndrome suggest there has been no change in total daily caloric intake or physical activity in children and adolescents over the past few decades (1). However, the prevalence of obesity and the metabolic syndrome continue to increase in the pediatric population (2). Logically, it begs to reason that other factors may augment the traditional concept of energy imbalance in the etiology and pathophysiology of obesity and the metabolic syndrome, which in turn, may further the understanding of this complex, multi-factorial phenotype. One intriguing hypothesis that requires further investigation is the role of an up-regulated hypothalamic-pituitary-adrenal (HPA) axis, which potentially links the physiology of the stress response, specifically elevated cortisol, and components of the metabolic syndrome (3). Furthermore, the potential role that physical activity may play in modulating the relationship between stress and the development of the metabolic syndrome during adolescence remains to be investigated.

Although several authors have examined the relationship between physical activity and the components of the metabolic syndrome in children and adolescents (for review, see Eisenamann (4)), few studies have examined the relationship between stress and components of the metabolic syndrome (5) or physical activity-stress interactions and components of the metabolic syndrome (6). The purpose of this review is to provide the reader with background information pertaining to physical activity, psycho-social stress, and the metabolic syndrome in the pediatric population. Topics that will be discussed in this review are: 1) the
epidemiology of pediatric obesity and the metabolic syndrome, 2) the role of stress in obesity and the metabolic syndrome, and 3) independent relationships between physical activity, stress/cortisol, and components of the metabolic syndrome.

**Epidemiology of Pediatric Obesity and the Metabolic Syndrome**

Currently, there are varying definitions to classify what is essentially the same problem. The Centers for Disease Control use the terms “at risk for overweight” and “overweight” to identify weight status in children and adolescents (≥ 85th percentile and ≥95th percentile, respectively). These classifications are statistically derived from national growth data that have been divided into age and sex-specific percentiles (7). Contrastingly, the International Task Force on Childhood Obesity (8) have developed classifications of childhood overweight and obesity using adult cut-points as a reference group. This approach used the LMS statistical method to back-extrapolate from adult overweight (BMI = 25 kg/m²) and obesity (BMI = 30 kg/m²) cut points to determine age- and sex-specific cut-points for children. For the purposes of this paper, the terms overweight and obese will be used. According to the most recent NHANES 1999-2000 data, the prevalence of overweight and obesity among US children and adolescents (ages 6-19) is approximately 31.5% (2) using the CDC cut-points.

**Immediate consequences of childhood obesity.** Childhood obesity has been associated with several adverse physiological states including insulin resistance (9), haemostatic risk factors for coronary heart disease (CHD) (10), as well as the traditional cardiovascular disease (CVD) risk factors during childhood (11-13). Traditional CVD risk factors include
dyslipidemia, hypertension, obesity, and diabetes, which are all components of the metabolic syndrome. It is the subsequent clustering of these risk factors as children grow older that creates such exponential health risks. The constellation of abdominal obesity, insulin resistance, elevated triglycerides, and low high-density lipoprotein cholesterol (HDL-C) constitute a condition that has been coined the metabolic syndrome (14). The prevalence of the metabolic syndrome is 6.4% among US adolescents (15), which is a 45% increase from the previous NHANES III data where the prevalence was 4.2% (15). An estimated 2/3 of US adolescents possess at least one characteristic of the metabolic syndrome (16).

Childhood obesity is associated with increased emotional distress (17) and decreased quality of life (QOL) during adolescence (18). QOL is a multidimensional construct which examines physical, emotional, social and school functioning (19). Schwimmer et al. (18) showed a significantly lower QOL in obese children and adolescents compared to healthy children, and furthermore, the QOL in obese subjects was comparable to that of children and adolescents who had been diagnosed with cancer.

Long-term effects of childhood obesity. While the immediate effects of childhood obesity are significant, the tracking of these effects into adulthood pose the most serious threat to health status. It is the subsequent clustering of CVD risk factors that increases the likelihood of CVD and all-cause mortality (20).

Obesity during childhood increases the likelihood of numerous CVD risk factors to manifest in adulthood. In a review examining childhood and adolescent adiposity and long-term health outcomes, high levels of fatness during childhood and adolescence markedly increased the risk of all-cause mortality (21). Likewise, several longitudinal studies have
shown that high levels of fatness during childhood are related to the development of many CVD risk factors such as hypertension (22, 23) coronary artery calcification (24), dyslipidemia (11-13, 22, 23, 25) carotid artery intima-media thickness (13, 26), hypertension, atherosclerosis (27), hyperinsulinemia (22), obesity (11, 12, 23, 25, 28) and all-cause mortality (12, 29). Maffeis (30) et al. found childhood BMI and insulin resistance to be independent predictors of adulthood BMI. Likewise, results from a follow-up of the Harvard Growth Study of 1922 to 1935, showed males who were classified as overweight during adolescence experienced more all-cause and coronary heart disease mortality than their lean counterparts (31).

**Physical activity and cardiovascular disease risk factors / Components of the metabolic syndrome**

Several excellent reviews have already extensively examined the association between physical activity and CVD risk factors (4, 32-40). This review will only provide a brief overview of these findings. It is important to note that the methodology used to measure physical activity may hinder our understanding of this topic (41).

**Physical activity and adiposity.** Currently there is not a clear association between physical activity or inactivity and adiposity in youth (42). Some population-based research has suggested that physical activity is inversely related to body mass index and overweight status in children and adolescents (43, 44); however, studies examining physical inactivity, via television viewing and other sedentary activities, and adiposity often provide stronger more statistically significant associations (43-47).
Cross-sectional associations between physical activity and lipids. When subjects are grouped by physical activity levels, children in the upper categories generally exhibit a more favorable lipid profile with higher HDL-C and lower LDL-C and TG are lower in more physically active children. However, training studies may decrease blood pressure in hypertensive adolescents (49).

1 blood pressure. Few cross-sectional studies have reported a relationship between physical activity and blood pressure (32). Physical activity has been shown to be ineffective in lowering BP in normotensive children. However, training studies may decrease blood pressure in hypertensive adolescents (49).

Physical activity and blood glucose and serum insulin. Limited research is available that examines physical activity and blood glucose or insulin in children and adolescents. Results from the Young Finns study showed lower fasting insulin levels in males, but not females, who were more active (50). Additionally, children who participated in an eight-week exercise program and saw improvements in aerobic fitness had a greater reduction in insulin levels than children who did not see improvements in aerobic fitness (50).

Physical activity and the metabolic syndrome. Physical activity, objectively measured by accelerometer, has been associated ($\beta = -0.02$, $p = 0.008$) with the metabolic syndrome in cohort of Danish children (51). This study used a metabolic syndrome composite score to examine the association between the metabolic syndrome and physical activity. Even after adjustment for physical fitness, the relationship between physical activity and metabolic syndrome remained ($\beta = -0.078$, $p = 0.004$).
Role of stress in obesity and the metabolic syndrome

Hans Selye first described stress as a syndrome produced by nocuous agents (52). Selye used this concept to describe the response of rats to various treatments such as exposure to cold and surgical injury. This concept was further developed into the General Adaptation Syndrome which suggested that regardless of the stressor, the response is composed of three basic stages: 1) Alarm Reaction, which consists of the shock and countershock phases, 2) Stage of Resistance, and 3) Stage of Exhaustion (53). More than fifteen years later, the idea of non-specificity was refuted by John Mason (54, 55), who suggested that the term “stress” was ill-defined. Furthermore, Mason suggested the concept of non-specificity was deficient for a number of reasons including Selye’s acute focus on the pituitary-adrenal-cortical system and the contradiction of homeostasis. Because homeostatic response is directed by the need of the organism, the idea of a generalized adaptability doesn’t follow logic (55). Work by Lundberg and Frankenhaeuser (56) and Henry (57) furthered the notion of a specified response by suggesting that perception of the stressor influenced response, whereby if the stressor is perceived as displeasurable, there is an increased physiological response (i.e. increased secretion of cortisol, epinephrine, etc.) and if the stressor maintains a positive connotation, this physiological response is lessened.

More recently, Chrousos and Gold (58) have summarized stress as a state of disharmony or of threatened homeostasis, evoking adaptive responses when the threat to homeostasis exceeds the threshold. The authors suggest that corticotrophin releasing hormone (CRH)-neurons and the locus coeruleus-norepinephrine/sympathetic system are chiefly responsible for mediating the stress response. Up- or down-regulation of this system
results in irregular levels of cortisol, norepinephrine, and epinephrine, and plays a role in several disorders including depression, anxiety, and hyperthyroidism.

Because homeostatic systems change frequently across a large range in adaptation to various environmental influences, the term *allostasis* was coined to describe the body’s ability to increase or decrease vital functions in adaptation (59). Unfortunately, the idea of allostasis doesn’t correspond with the long-term outcomes of a dysregulated homeostatic system. The end result of this chronic activation is a general shift to levels that may predispose individuals to disease. As a result, the term *allostatic load* was introduced to describe the effect of chronic dysregulation of the allostatic systems (60).

Elevated levels of chronic stress (i.e. work stress, depression, etc.) have been associated with the development of CVD (61). Increased cortisol secretion has been observed in various stressful conditions (62-64) and it is thought that chronic hypersecretion may lead to impaired feedback and resistance. Because the HPA axis is perturbed in states of obesity and insulin resistance, Björntorp (65) suggests that these conditions are driven by increased stress activation. To date, very few studies have examined the association between stress and subsequent cortisol secretion, adiposity, and components of the metabolic syndrome in children and adolescents. Available research suggests that children with higher levels of cortisol have higher measures of adiposity (66, 67). Likewise, high levels of cortisol in adults are associated with abdominal adiposity and the metabolic syndrome. Rosmond et al. (68) found that high levels of circulating stress-related cortisol were associated with high BMI and waist-to-hip ratio (WHR) in men. Stress related cortisol was determined via questionnaire which accompanied a saliva sample at eight time point during a normal working day. In a similar study, Epel et al., (69) found men to secrete more cortisol
subsequent to a stressor than women and those men with the highest concentrations also had higher BMI and WHR (69).

Although epidemiological studies show an association between high concentrations of cortisol and higher levels of adiposity, the mechanism that links elevated cortisol to adiposity is not fully understood. One possible explanation is the hormonal interactions between cortisol and insulin. In a study examining the associations between the glucocorticoid receptor gene markers, abdominal adiposity and dysregulation of the HPA axis, Rosmond et al. (70) found significant correlations between both BMI and WHR with circulating insulin and glucose ($r = 0.40 - 0.60$) in individuals with an HPA axis dysregulated to a greater extent. The extent of dysregulation was determined by dexamethasone secretion and total cortisol secretion. This suggests that a dysregulated HPA axis (and elevated cortisol) is associated with decreased insulin sensitivity. Kramer et al. (71) also showed relationships between various questionnaire-based measurements of stress and glycosylated hemoglobin (HbA1c), an indicator of three-month blood glucose. A higher percentage of HbA1c indicates chronically elevated blood glucose levels and is indicative of poor insulin sensitivity. To our knowledge, there are presently no data in adolescents that link high levels of cortisol to HbA1c or other blood glucose or insulin markers. Furthermore, Bjorntorp et al. (72) found similar results with regards to insulin sensitivity in Swedish men and women. Consistent adverse relationships were found between dysregulated cortisol secretion and all variables associated with the metabolic syndrome. Positive correlations are characteristically associated between dysregulated cortisol secretion and metabolic syndrome risk factors (68, 73). Based on the available data, there is a clear association between high levels of cortisol
and biomarkers associated with the metabolic syndrome in adults. However, this association has yet to be firmly established in the adolescent population.

Relationships between physical activity, stress and the metabolic syndrome

The independent associations between physical activity and the metabolic syndrome and between stress and the metabolic syndrome have been examined already in this review. Because these variables affect each other, the associations between physical activity and the stress- metabolic interaction should also be examined.

Physical activity has been shown to improve endocrinological as well as metabolic risk factors associated with the metabolic syndrome (74). Furthermore, low cortisol has also been associated with psychological constructs such as self-efficacy, which may also facilitate the improved response among the physically active (75, 76). Ratings of Perceived Exertion (RPE) were positively associated with post exercise cortisol levels, supporting the idea that cortisol is related to exercise-affective states (77). Perhaps the means by which physical activity may buffer the effects of stress is partially through positive affect.

Although less conclusive (78), metabolic syndrome risk factors appear to be improved with physical activity. Unfortunately, given the variability in methodology and confounding factors which surround this population such as normal growth and maturation the extent of the benefits of physical activity are difficult to quantify (78).

The attenuating effects of physical activity on adiposity have only recently begun to be examined in adolescents. In a recent study (6), physical activity appeared to buffer the effects of chronic stress on adiposity. Chronic stress was defined as personal stress and was assessed using the Adolescent Resource Challenge Scale. Physical activity was assessed via
self-report of how many days in or outside of school, during which physical activity was sufficient to “work up a sweat”. The results show that the stress-physical activity interaction significantly predicted sum of skinfolds and waist circumference in a hierarchical regression model. However, a limitation of this study is that both stress and physical activity were assessed by self-report. While the results of this study are promising, the true significance of the attenuating effect of physical activity on chronic stress will not be fully appreciated without the use of more objective measures of physical activity and chronic stress, via assessment of specific bio-markers of stress such as cortisol.

Summary and Conclusions

Unfortunately, the benefits of physical activity during childhood and adolescence cannot be stored and used to compensate a sedentary lifestyle in adulthood (79); however, the acute benefits are an indication of the significance of physical activity throughout life. Not only does physical activity influence the components of the metabolic syndrome, but it may also modulate the effects of chronic stress and HPA axis perturbation. This is particularly significant given that obesity has been shown to decrease the quality of life (18)(80), independent of the other co-morbidities connected with the metabolic syndrome. In the same cyclic fashion that stress, obesity, and other co-morbidities affect each other, physical activity may also affect each of these variables. Further research is warranted in the area of physical activity, stress, and the metabolic syndrome to fully understand the genesis of the metabolic syndrome during adolescence.
References


CHAPTER 3. MANUSCRIPT: PHYSICAL ACTIVITY, STRESS AND THE METABOLIC SYNDROME IN 8-18 YR OLD BOYS

Introduction

An estimated 16.5% of United States (US) children and adolescents, 6 to 19 years of age, are considered obese and an additional 15% are overweight, which represents a three-fold increase over the past few decades (1). Childhood obesity is adversely associated with traditional cardiovascular disease (CVD) risk factors, such as hypertension and dyslipidimia (2, 3), as well as insulin resistance (4), and haemostatic risk factors for CVD (5). In addition to the immediate consequences, childhood obesity often tracks into adulthood (6) and has been linked to CVD morbidities in adulthood, including coronary artery calcification (7), dyslipidimia and hypertension (8, 9), carotid artery intima-media thickness (10), and all-cause CVD mortality (11).

As previously mentioned, obesity, and more specifically abdominal or visceral obesity is often associated with elevated blood pressure, an adverse blood lipid profile, and insulin resistance. The co-occurrence of these traits has been coined the metabolic syndrome (12). According to the most recent National Health and Nutrition Examination Survey (NHANES, 1999-2000), the prevalence of the metabolic syndrome is 25% among US adults (13) and 6.4% among US adolescents (14). Furthermore, about 43% of US adolescents possess at least one characteristic of the metabolic syndrome and 17% have two or more characteristics (15, 16).

Given the increased prevalence of pediatric obesity and the metabolic syndrome, there has been considerable interest in preventing these conditions. Most prevention and treatment strategies focus on diet and physical activity (17). Since prevalence rates of
childhood obesity and metabolic syndrome continue to rise and epidemiological studies indicate the physical activity and diet only explain a modest amount of the variance (18), there is reason to consider other possible causes contributing to the increased prevalence rates of obesity and the metabolic syndrome among youths.

One intriguing hypothesis is related to the chronic activation of the hypothalamic-pituitary-adrenal (HPA) axis due to various emotional, environmental, and physical stressors that can create a state of hypercortisolaemia (19-22). Chronically elevated levels of cortisol cause an up-regulation of lipoprotein lipase and subsequent storage of fat, specifically in the viscera (21). Additionally, increased cortisol levels negatively affect insulin sensitivity (20), a key component in the metabolic syndrome. Relationships have consistently been demonstrated between stress-related cortisol secretion and markers of the metabolic syndrome in adults (23-26). Glucocorticoid excretion rates have also been associated with fatness and body mass index (BMI) in children (27).

To date, few studies have examined the inter-relationships between physical activity, stress, and markers of the metabolic syndrome; however, a recent study shows that physical activity buffers the associations between chronic stress and adiposity (28). In this study, both stress and physical activity was assessed by self-report. Additionally, this study only examines the stress-physical activity interaction with respect to adiposity. This relationship has yet to be established with the metabolic syndrome, a more comprehensive depiction of overall metabolic health.

This study examined the moderating effects physical activity may have on the relationship between measures of stress (via waking salivary cortisol and various self-report measures) and the components of the metabolic syndrome in adolescent males.
Methods

Subjects. Thirty-eight boys, ages 8-18 years, participated in the current investigation. All subjects signed assent forms and parental consent was obtained prior to data collection. This study was approved by the Iowa State University Institutional Review Board.

General Procedures. Procedures were reviewed upon arrival to the lab. The test session included explanation of waking salivary cortisol collection procedures and physical activity assessment as well as assessment of CVD risk factors. Additionally, the subject received questionnaires aimed at assessing perceived stress, anxiety, depression, self-esteem, weight and general appearance-related teasing, and television (TV) viewing time. A description of each of these measures is provided below.

Salivary cortisol. A saliva sample of 0.5 mL was collected in a sterile collection tube (Salivette) immediately after waking on a typical weekday morning. Subjects were asked to provide the sample after waking, but before eating, drinking, or brushing their teeth. Subjects returned the sample via mail and the sample was then stored at —20° C until time of assay. At that time, the sample was centrifuged at 3000 RPM for 15 minutes. Salivary cortisol concentrations were measured using a commercially available high-sensitivity ELISA kit (Salimetrics, College Park, PA).

Self-report measures of stress variables.

Physical Appearance Related Teasing Scale (PARTS). PARTS was used to determine weight and size-related and general appearance-related teasing by peers (29). The PARTS questionnaire consists of two scales which total 18 questions. The questions were changed from past to present tense to make the questionnaire age-appropriate. Examples of questions include, “Do you ever feel as though your peers are staring at you because you are
over-weight?” and “Do kids ever call you funny looking?” Subjects responded on a scale from never (1) to frequently (5). The internal consistency coefficient for the weight and size-related scale is 0.91 and the test-retest reliability is 0.86. The internal consistency coefficient for the general appearance-related scale is 0.71 and the test-retest reliability is 0.87 (29).

*Perceived Stress Scale (PSS).* The PSS(30) is a global measure of stress and was used to determine the subjects’ perception of stress in their lives over the last month. An example of a question is, “In the last month, how often have you been upset because of something that happened unexpectedly?” The coefficient alpha reliability ranged from 0.84 to 0.86 in three separate examinations (30).

*Children’s Depression Inventory (CDI).* Depressive characteristics were examined using the CDI (31). This instrument consists of 27 items assessing affective, cognitive, and behavioral symptoms of depression. Subjects were asked to pick the sentence that best describes them for the past two weeks (ex. “I am sad once in a while.” “I am sad many times.” “I am sad all of the time.”). Reliability coefficients for this instrument range from 0.71 to 0.89, indicating good to excellent internal consistency (31).

*State-Trait Anxiety Inventory for Children (STAI-C).* The STAI-C (32) was used to assess symptoms of trait anxiety. This measure consists of 20 statements such as, “I worry about making mistakes…” which subjects may respond to by choosing “hardly-ever”, “sometimes”, or “often”. The coefficient alpha reliability for the STAI-C for males is 0.78 (32).

*Self-Esteem Questionnaire (SEQ).* The SEQ (33) is composed of six subscales with a total of 42 statements. The SEQ was used to determine subjects’ global feeling of self-worth, as well as perceptions of influential factors (peers, school, and family).
Examples of questions include “I am as popular with kids my own age as I want to be.” and “I am happy about the way I look.” Subject could respond by choosing “strongly disagree”, “disagree”, “agree”, and “strongly agree”. Coefficient alphas for each of the sub-scales range from 0.81-0.91 and 0.81-0.92 in two separate examinations of internal reliability (33).

**Physical activity and television viewing time:** The Manufacturing Technology Inc. (MTI) uniaxial accelerometer (Shalimar, FL) was used in the present study. The MTI is a small, lightweight unit with a time-sampling mechanism that is designed to detect acceleration ranging in magnitude from 0.05 to 2.00 G with frequency response from 0.25 to 2.50 Hz. The filtered acceleration signal was digitized and the magnitude was summed over a user-specified epoch interval at the end of each epoch, the summed value is stored in memory and the integrator is reset. One-minute epochs were used for this study. The unit was attached to a belt worn at the mid-axillary line at the hip. The instrument was explained to the subject and then taken home and worn for 4-days during the subsequent week. The 4-day period included 1 weekend day. The accelerometers were returned via mail along with waking saliva sample. Time spent in moderate and vigorous activity was calculated using an age- and sex-specific equation. Moderate and vigorous activity are approximately equivalent to 3 and 6 METs (metabolic equivalent) (34). Moderate to vigorous physical activity (MVPA) was calculated as the total amount of time each day spent in moderate and vigorous activity.

TV viewing time was assessed via questionnaire. The questionnaire asked participants to quantify average daily time spent watching TV, during the week and on weekend days. Hours on weekdays were multiplied by 5 and hours on weekend days were multiplied by 2. These values were added together to generate hours per week.
Assessment of CVD risk factors. Subject demographics and anthropometric data were assessed following explanation of the procedures. The subject was then seated for 5-10 minutes prior to the measurement of resting BP, and blood lipids and glycosylated hemoglobin (HbA1c).

Body size. Stature and body mass were measured according to standard procedures (35). Stature was measured with a wall-mounted, fixed stadiometer (Holtain Limited, United Kingdom) with the subject standing erect, without shoes, and with weight distributed evenly between both feet, heels together, arms relaxed at the sides, and the head in the Frankfort horizontal plane. Body mass was measured without shoes and excess clothing on a balance beam scale (Seca 770, Hamburg, Germany). Stature and body mass were used to calculate body mass index (BMI, kg/m^2). Because abdominal obesity is a key feature in the metabolic syndrome, waist circumference was assessed as a measure of central adiposity. Waist circumference was measured immediately above the iliac crest (National Institutes of Health recommendation) using a Gullick tape to the nearest 0.1 cm.

Maturity offset. Since the age range of the subjects spans the period of puberty and numerous body size and physiological functions and capacities vary by pubertal status (36), an indicator of biological maturity status was assessed via the maturity offset method. The maturity offset technique is a non-invasive method of indicating biological maturity and was calculated as outlined by Mirwald et al. (37). Anthropometric variables are used to create a value that is aligned to the estimated age of peak height velocity (e.g., -1.5 yrs, etc.). This value was used as a covariate in the statistical analysis.

Resting Blood pressure. Resting BP was measured using an automated monitor (Critikon Dinamap) in accordance with standard recommendations (38). Appropriate cuff
size was determined by measuring the circumference of the right upper arm at its largest point. Three measurements were taken at 1-minute intervals, and the mean of the three values was used for data analysis.

Blood cholesterol. A blood sample was obtained by finger prick and collected in a 35 micro-liter capillary tube. Upon collection, samples were analyzed for total cholesterol (TC) and high density lipoprotein cholesterol (HDL-C) by a portable cholesterol analyzer according to the protocol of the manufacturer (Cholestech LDX System, Hayward, CA). Because subjects were in a non-fasted state, triglycerides (TG) were not be assessed. Blood sampling by finger prick was chosen for reasons of compliance and avoidance of undue stress. Intra-class reliability statistics yielded coefficients of variation ≤ 0.03 for TC and HDL-C when testing high and low standards.

HbA1c. A second finger stick was taken to determine HbA1c. The concentration of HbA1c reflects blood glucose levels over the previous 2-3 months. The sample was collected in a 10 micro-liter pipette and analyzed by a desktop analyzer (Cholestech GDX, Hayward, CA) according to the protocol of the manufacturer. Previous studies have shown that the accuracy of the Cholestech GDX falls within the limits of the National Glycohemoglobin Standardization Program (39).

Derivation of the metabolic syndrome score. A composite risk factor, or metabolic syndrome score was derived by summing the age-standardized residuals (Z-scores) for HbA1c, MAP, HDL-C, and WC. These variables were chosen because they represent the same variables used in the adult clinical criteria and this variable has been used in recent work from our laboratory (40). Because the metabolic syndrome typically does not manifest
until later in life and is a dichotomous variable, the use of a composite score allows each subject to have a value. A lower score is indicative of a better metabolic risk factor profile.

Statistical analysis. Descriptive statistics were calculated for all variables for high and low physical activity groups and the total sample. The associations between waking cortisol, self-report stress measures, physical activity and the metabolic syndrome score were examined by partial correlation, controlling for chronological age and maturity status. The moderating effects of physical activity were examined by separating the sample into low (<77 minutes of MVPA) and high (≥ 77 minutes of MVPA) physical activity groups via a median split. The difference in the magnitude of the correlations between the two physical activity groups was tested after a Z-transformation (Z = 0.5 * [ln (1 + r) − ln (1 − r)]). All statistical analyses were conducted using SPSS version 12.0.

Results

Table 1 provides the descriptive statistics for the study sample. In the total sample, mean height, weight, and BMI were all in the 50th percentile when plotted on the growth chart (data not shown). Approximately 27% and 16% of the participants were overweight or obese, respectively. The low physical activity group was taller and participated in more MVPA and vigorous physical activity when compared to the high physical activity group.

Table 2 shows correlations between physical activity and the metabolic syndrome composite score after adjustment for age and maturity offset. Correlations were low (r < -0.13), but in the expected direction (e.g., inverse) (Table 2) after adjustment for age and maturity offset. Television viewing was significantly related to the metabolic syndrome composite score (r = 0.39). Waking salivary cortisol was not associated with the metabolic
syndrome score (Table 2). Several stress-related variables were not significantly related to
the metabolic syndrome score. School-related self-esteem was inversely associated with the
metabolic syndrome score ($r = -0.46$). General appearance related teasing was negatively
associated with metabolic syndrome ($r = -0.36$). The association between sports-related self-
esteeem and trait-anxiety and the metabolic syndrome score approached significance ($P = 0.08$
and 0.09, respectively) and, therefore, were used in subsequent analysis.

Table 3 shows the correlations between stress-related variables and metabolic
syndrome score for low ($n = 18; < 77$ minutes of MVPA/day) and high ($n = 19; \geq 77$ minutes
or more MVPA/day) physical activity groups after adjustment for age and maturity offset.
Both school- and sports-related self-esteem were inversely associated with the metabolic
syndrome score ($r = -0.64$ and $-0.53$, respectively) in the low physical activity group.
Additionally, trait-anxiety was also significantly associated with metabolic syndrome score
in the low physical activity group ($r = 0.53$). Associations between stress-related variables
and metabolic syndrome score in the high physical activity group were null. Additionally,
the difference in magnitude of the correlations did not reach statistical significance. Figure 1
provides a pictorial representation of the relationship between trait-anxiety and metabolic
syndrome score in high and low physical activity groups.

**Discussion**

Previous research has examined the relationship between physical activity and the
metabolic syndrome (41) and the relationship between physical activity and stress (28)
independently, however, limited research has examined if physical activity modifies the
relationship between stress and the metabolic syndrome. The results from the present study
suggest that physical activity modifies the relationship between stress and the metabolic syndrome in 8 to 18 year old boys. Specifically, it appears that low physical activity enables various stress-related variables to significantly effect metabolic health; likewise, physical activity may buffer the effect of stress on the metabolic syndrome.

One of the main objectives of this study was to examine the relationship between physical activity, waking salivary cortisol (as an objective marker of chronic stress) and the metabolic syndrome. However, the utility of waking salivary cortisol did not prove effective in this study. In a study to determine the relationship between waking samples and area under the curve (i.e. a greater area under the curve is indicative of a more perturbed HPA axis) (42), samples were taken at 0700, 0730, and 2000. These values were represented independently and together to create area under the curve with respect to increase (AUCI), which is an indicator of one quarter of the increase between awakening levels and levels 30 minutes later, and area under the curve with respect to the ground (AUCG), which correlates ($r = 0.99$) with the sum of the three cortisol measurements. Waking cortisol was negatively associated with AUCI ($r = -0.43$), whereas cortisol sampled 30 minutes after waking was positively associated with AUCI ($r = 0.76$), which clearly demonstrates the difference in sampling times. In the current investigation, subjects were asked to provide a saliva sample after waking, but prior to eating, drinking, or brushing their teeth. In retrospect, a limitation of this method occurs because there is no impartial control of the time between waking and sampling. One possible explanation why waking salivary cortisol was not significantly associated with any variables in the present study may be due to inconsistent sampling times. Likewise, additional sampling times, which would allow for area under the curve analysis,
may have allowed a more conclusive investigation into the relationships between physical activity, cortisol (HPA function), and the metabolic syndrome.

Correlations between physical activity and metabolic syndrome score were low ($r < -0.13$), but were in the expected direction (i.e. negative). This finding is consistent with previous research examining the associations between physical activity and individual components of the metabolic syndrome (43). TV viewing was significantly related with the metabolic syndrome ($r = 0.39$). Previous research examining television viewing and other sedentary activities and adiposity provide convincing evidence to the role of physical activity in youth (44-48). In a recent study by Heelan and Eisenmann (In Press), time spent watching television and total media use were the strongest correlates ($r = 0.51$) of adiposity in girls, whereas computer usage was the strongest correlate in boys ($r = 0.31$). The present investigation furthers the current body of literature by establishing a relationship between sedentary behavior and the metabolic syndrome in youth.

The association between stress and health status in children has recently gained interest. Most research focuses on the relationship between stress and weight or BMI. Sjörberg and colleagues (49) found BMI to be associated with depression and those adolescents who belonged to the obese group suffered more often from major depression. An inverse relationship between quality of life (QOL) and obesity in children and adolescents has also been observed (50). Furthermore, the QOL in obese subjects was comparable to that of children and adolescents who had been diagnosed with cancer. QOL is a multidimensional construct which examines physical, emotional, social and school functioning (51). Each of these variables has the potential to perturb HPA axis functioning and possibly affect markers of health in addition to weight status. In the current
investigation, a variety of self-report measures were used to examine the association between stress and the metabolic syndrome. School-related self-esteem and teasing were significantly associated with the metabolic syndrome score, suggesting that the stress associated with poor self-esteem and teasing may be related to adverse health status in young people. Björntorp (21) suggests that because the HPA axis is perturbed in states of obesity and insulin resistance, poor metabolic health is driven by increased stress activation. In this example, increased stress due to poor self-esteem and teasing may result in chronic hypersecretion of cortisol and subsequent metabolic syndrome. Further research should examine this hypothesis.

Although a significant relationship between stress and the metabolic syndrome score exists, the variance suggests that other factors contribute to the metabolic syndrome. It was hypothesized that physical activity may serve as a buffer between stress and metabolic syndrome score. The main finding in the current investigation provides evidence for this hypothesis. School- and sports-related self-esteem and anxiety were significantly associated with the metabolic syndrome score in subjects who participated in low MVPA. No significant relationships were observed between any of the stress variables and the metabolic syndrome score in subjects who participated in high MVPA. This finding is of clinical relevance given that current recommendations for physical activity suggest the accumulation of 60 minutes (52). Conversely, the high physical activity group did not yield any significant associations between stress variables and the metabolic syndrome score. Although significant correlations do not equate causal inference, these results suggest that adequate MVPA is necessary for stressed individuals with poor self-esteem and high anxiety, in order to maintain metabolic health. These results are consistent with recent research on physical
activity, stress and adiposity in youth (28). In the study by Yin et al. (28), the stress-physical activity interaction significantly predicted sum of skinfolds and waist circumference in a hierarchical regression model; however, both stress and physical activity were assessed by self-report. A major strength of the present study is that it allowed examination of the associations between physical activity, stress-related variables, and the metabolic syndrome rather than merely examining weight status or individual CVD risk factors. Because the metabolic syndrome typically does not manifest until later in life, the prevalence is relatively low and does not accurately express the severity of the problem. The use of a composite score allows each subject to have a value relative to healthy or disease status. Several papers show metabolic syndrome tracks into adulthood (1, 9, 53). An additional strength of this study is the measurement of physical activity by the use of accelerometer. Previous research has been limited by questionnaire. Accelerometry provides a more objective means of quantifying physical activity and allows the partitioning of intensity levels. Lastly, this study is distinct in its conceptual design. This is the first study to examine physical activity, stress-related measures, and the metabolic syndrome. Further research is needed to examine if this relationship will remain significant in a larger sample.

In conclusion, the results show some evidence for considering stress in addition to physical activity and diet for prevention and treatment strategies for childhood obesity and metabolic syndrome. The relationship between stress and the metabolic syndrome is unique in that stress can influence all factors associated with the metabolic syndrome (21), including obesity, and, in turn, obesity has the propensity to influence stress levels via teasing and self-esteem (50, 54). Because of the two-way causal path that exists between stress and metabolic disease it’s illogical to consider these variables in a compartmentalized fashion.
Ultimately, with a greater understanding of the intimate relationships that exist between physical activity, stress, and the metabolic syndrome, the manner by which we address treatment and prevention of obesity and related diseases can be optimized.

References


Table 1. Physical characteristics of the sample. Values are mean (SD) and minimum-maximum values for normal, overweight, and total sample for anthropometric, metabolic and physical activity variables. Stress variables are represented as mean (SD) for normal and overweight groups and mean (SD) and possible range for the total sample.

<table>
<thead>
<tr>
<th>Anthropometric Variables</th>
<th>Low PA (n=18)</th>
<th>High PA (n=19)</th>
<th>Total (n=37)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>15.5 (2.1)</td>
<td>12.5 (2.4)</td>
<td>13.9 (2.7)</td>
</tr>
<tr>
<td>Ht (cm)</td>
<td>173.1 (8.1)*</td>
<td>157.9 (16.2)</td>
<td>165.3 (14.9)</td>
</tr>
<tr>
<td>APHV (yrs)</td>
<td>14.7 (0.8)</td>
<td>14.3 (0.6)</td>
<td>14.5 (0.7)</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>73.2 (14.3)</td>
<td>54.1 (21.7)</td>
<td>63.4 (20.7)</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>24.4 (4.5)</td>
<td>21.0 (5.1)</td>
<td>22.7 (5.1)</td>
</tr>
<tr>
<td>WC (cm)</td>
<td>83.8 (13.6)</td>
<td>72.8 (16.0)</td>
<td>78.1 (15.7)</td>
</tr>
<tr>
<td>Overweight</td>
<td>55.6%</td>
<td>31.6%</td>
<td>43%</td>
</tr>
<tr>
<td>Metabolic Variables</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>126.0 (10.0)</td>
<td>116.3 (10.3)</td>
<td>121.0 (11.1)</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>67.9 (3.8)</td>
<td>65.9 (3.7)</td>
<td>66.9 (3.8)</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>89.0 (4.1)</td>
<td>86.0 (4.1)</td>
<td>87.4 (4.3)</td>
</tr>
<tr>
<td>HbA1c (%)</td>
<td>5.4 (0.3)</td>
<td>5.5 (0.5)</td>
<td>5.5 (0.44)</td>
</tr>
<tr>
<td>HDL-C (mg/dL)</td>
<td>39.0 (10.8)</td>
<td>49.5 (12.4)</td>
<td>44.4 (12.6)</td>
</tr>
<tr>
<td>Metabolic Syndrome Score</td>
<td>0.18 (2.7)</td>
<td>-0.11 (2.3)</td>
<td>0.03 (2.5)</td>
</tr>
<tr>
<td>AM Cortisol (ug/dl)</td>
<td>1.0 (1.0)</td>
<td>0.87 (0.5)</td>
<td>0.95 (0.78)</td>
</tr>
</tbody>
</table>

* indicates a significant difference between groups.
<table>
<thead>
<tr>
<th>Physical Activity</th>
<th>Group 1</th>
<th>Group 2</th>
<th>Group 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>MVPA (min/day)</td>
<td>46.2 (15.9)*</td>
<td>109.7 (32.7)</td>
<td>78.8 (41.1)</td>
</tr>
<tr>
<td>Vigorous PA (min/day)</td>
<td>5.1 (7.1)*</td>
<td>18.0 (17.1)</td>
<td>11.7 (14.6)</td>
</tr>
<tr>
<td>Moderate PA (min/day)</td>
<td>41.2 (12.9)</td>
<td>91.7 (20.2)</td>
<td>67.1 (30.6)</td>
</tr>
<tr>
<td>Average counts/min</td>
<td>374.7 (130.5)</td>
<td>629.7 (241.1)</td>
<td>505.7 (232.0)</td>
</tr>
<tr>
<td>Television (hrs/wk)</td>
<td>19.9 (12.7)</td>
<td>27.4 (19.7)</td>
<td>23.8 (16.9)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Selected Stress Variables</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>SE-school</td>
<td>22.9 (6.2)</td>
<td>26.3 (3.9)</td>
<td>24.6 (5.4)</td>
</tr>
<tr>
<td>SE-sports</td>
<td>17.2 (3.6)</td>
<td>18.8 (3.7)</td>
<td>18.0 (3.7)</td>
</tr>
<tr>
<td>Anxiety</td>
<td>31.2 (5.2)</td>
<td>31.7 (5.1)</td>
<td>31.4 (5.1)</td>
</tr>
<tr>
<td>PARTS-GA</td>
<td>32.7 (4.4)</td>
<td>34.1 (4.0)</td>
<td>33.4 (4.2)</td>
</tr>
</tbody>
</table>

*P<0.05 for group difference

Ht, height; BMI, body mass index; WC, waist circumference; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; HbA1c, glycosylated hemoglobin; HDL-C, high density lipoprotein cholesterol; APHV, age at peak height velocity; AAPHV, age away from peak height velocity; MVPA, moderate to vigorous physical activity; SE, Self Esteem; CDI, Children’s Depression Inventory Survey; PARTS-GA, Physical Appearance Related Teasing Scale -general appearance.
Table 2. Associations between physical activity and stress measures with metabolic syndrome score after adjustment for age and maturity offset.

<table>
<thead>
<tr>
<th>Physical Activity</th>
<th>Correlation Coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td>MVPA (counts)</td>
<td>-0.13</td>
</tr>
<tr>
<td>Vigorous PA (counts)</td>
<td>-0.09</td>
</tr>
<tr>
<td>Moderate PA (counts)</td>
<td>-0.13</td>
</tr>
<tr>
<td>Average counts/min</td>
<td>-0.07</td>
</tr>
<tr>
<td>Television (hrs/wk)</td>
<td>0.34*</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Selected Stress Variables</th>
<th>Correlation Coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td>AM Cortisol (ug/dl)</td>
<td>-0.04</td>
</tr>
<tr>
<td>SE-school</td>
<td>-0.46*</td>
</tr>
<tr>
<td>SE-sports</td>
<td>-0.31</td>
</tr>
<tr>
<td>Anxiety</td>
<td>0.29</td>
</tr>
<tr>
<td>PARTS-GA</td>
<td>-0.36*</td>
</tr>
</tbody>
</table>

*P<0.05

MVPA, moderate to vigorous physical activity; SE, Self Esteem; CDI, Children’s Depression Inventory Survey; PARTS-GA, Physical Appearance Related Teasing Scale - general appearance.
Table 3. Associations between stress measures with the metabolic syndrome score after adjustment for age and maturity offset in high and low physical activity groups. Values are low/high physical activity.

<table>
<thead>
<tr>
<th>Selected Stress Variables</th>
<th>Metabolic Syndrome Score</th>
<th>N</th>
<th>Difference in correlation</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>AM Cortisol (ug/dl)</td>
<td>-0.19/0.37</td>
<td>18/19</td>
<td></td>
<td>0.11</td>
</tr>
<tr>
<td>SE-school</td>
<td>-0.64*/-0.41</td>
<td>18/18</td>
<td></td>
<td>0.37</td>
</tr>
<tr>
<td>SE-sports</td>
<td>-0.53*/-0.09</td>
<td>18/18</td>
<td></td>
<td>0.16</td>
</tr>
<tr>
<td>Anxiety</td>
<td>0.53*/0.07</td>
<td>18/19</td>
<td></td>
<td>0.15</td>
</tr>
<tr>
<td>PARTS-GA</td>
<td>-0.39/-0.48</td>
<td>18/19</td>
<td></td>
<td>0.76</td>
</tr>
</tbody>
</table>

*P<0.05 for correlational significance
#P<0.05 for differences between correlations

SE, Self Esteem; CDI, Children’s Depression Inventory Survey; PARTS-GA, Physical Appearance Related Teasing Scale- general appearance.
Figure 1. Association between metabolic syndrome score and anxiety score in high and low physical activity groups.

Solid line and circles represent high physical activity group. Dashed line and triangles represent low physical activity group.
APPENDIX 1. ASSOCIATIONS BETWEEN METABOLIC SYNDROME COMPONENTS AND THE METABOLIC SYNDROME SCORE

<table>
<thead>
<tr>
<th>Metabolic Syndrome Composite Score</th>
<th>WC</th>
<th>MAP (mmHg)</th>
<th>HbA1c (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>WC</td>
<td>0.65*</td>
<td>.</td>
<td></td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>0.44*</td>
<td>0.26</td>
<td>.</td>
</tr>
<tr>
<td>HbA1c (%)</td>
<td>0.59*</td>
<td>0.27</td>
<td>0.06</td>
</tr>
<tr>
<td>HDL-C (mg/dL)</td>
<td>-0.60*</td>
<td>-0.07</td>
<td>0.44*</td>
</tr>
</tbody>
</table>

WC, waist circumference; MAP, mean arterial blood pressure; HbA1c, Glycosylated hemoglobin; HDL-C, high density lipoprotein cholesterol.
APPENDIX 2. ASSOCIATIONS BETWEEN SELF-REPORT MEASURES OF STRESS AND WAKING SALIVARY CORTISOL

<table>
<thead>
<tr>
<th>SE - peers</th>
<th>SE - peers</th>
<th>SE - school</th>
<th>SE - family</th>
<th>SE - body image</th>
<th>SE - sports</th>
<th>SE - global</th>
<th>CDI - interpersonal</th>
<th>CDI - ineffectiveness</th>
</tr>
</thead>
<tbody>
<tr>
<td>SE - peers</td>
<td>SE - peers</td>
<td>SE - school</td>
<td>SE - family</td>
<td>SE - body image</td>
<td>SE - sports</td>
<td>SE - global</td>
<td>CDI - interpersonal</td>
<td>CDI - ineffectiveness</td>
</tr>
<tr>
<td>0.58*</td>
<td>0.64*</td>
<td>0.71*</td>
<td>0.61*</td>
<td>0.75*</td>
<td>0.61*</td>
<td>0.61*</td>
<td>-0.33*</td>
<td>0.04</td>
</tr>
<tr>
<td>0.42*</td>
<td>0.70*</td>
<td>0.59*</td>
<td>0.45*</td>
<td>0.64*</td>
<td>-0.45*</td>
<td>0.57*</td>
<td>-0.45*</td>
<td>0.37*</td>
</tr>
<tr>
<td>0.76*</td>
<td>0.68*</td>
<td>0.71*</td>
<td>0.70*</td>
<td>0.59*</td>
<td>0.70*</td>
<td>0.59*</td>
<td>0.71*</td>
<td>0.70*</td>
</tr>
</tbody>
</table>

SE, Self Esteem; CDI, Children's Depression Inventory Survey; PSS, Perceived Stress Survey; PARTS-GA, Physical Appearance Related Teasing Scale-general appearance; PARTS-WT, Physical Appearance Related Teasing Scale-weight related.
## APPENDIX 3. ASSOCIATIONS BETWEEN SEDENTARY BEHAVIORS AND THE METABOLIC SYNDROME SCORE

<table>
<thead>
<tr>
<th></th>
<th>Metabolic Syndrome Composite Score</th>
<th>TV</th>
<th>Video Games</th>
<th>Computer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic Syndrome</td>
<td>.</td>
<td>.</td>
<td>.</td>
<td></td>
</tr>
<tr>
<td>Composite Score (hrs/wk)</td>
<td>0.34*</td>
<td>.</td>
<td>0.57*</td>
<td>.</td>
</tr>
<tr>
<td>Computer (hrs/wk)</td>
<td>-0.25</td>
<td>0.02</td>
<td>0.02</td>
<td></td>
</tr>
<tr>
<td>Total media (hrs/wk)</td>
<td>0.32</td>
<td>0.85*</td>
<td>0.84*</td>
<td>0.46*</td>
</tr>
</tbody>
</table>
ACKNOWLEDGEMENTS

I would like to thank my program of study committee members, not only for their academic guidance, but also for their kind words and encouragement. I would also like to extend special thanks to my supervisor, Joe Eisenmann. I am grateful to have studied under his mentorship and will take the lessons I’ve learned, both academic and of life, with me from here on. I am particularly appreciative of my lab group, with whom I’ve shared many insightful conversations and much laughter.

I would like to thank family as they have been a source of constant support and encouragement. I consider myself truly blessed to be so loved. My parents have been so supportive and loving. I also extend sincere thanks to my brother for his love and friendship. I am particularly grateful to my sister Kristi for her guidance, love, and support. I am fortunate to have such a good friend as my sister. Her own love of science and learning continues to be an inspiration to me.

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