PHYSICAL ACTIVITY, STRESS AND METABOLIC RISK SCORE IN 8-18 YR OLD BOYS

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Abstract

BACKGROUND: We examined whether physical activity modifies the relationship between stress and the metabolic risk score in 8-18 year old males (n=37). METHODS: Physical activity (PA) and television (TV)/video game (VG) use were assessed via accelerometer and questionnaire, respectively. Stress was determined from self-report measures. A metabolic risk score (MRS) was created by summing age-standardized residuals for waist circumference, mean arterial pressure, glycosylated hemoglobin, and high-density lipoprotein cholesterol. RESULTS: Correlations between PA and MRS were low ($r<-0.13$) while TV and VG were moderately associated with MRS ($r = 0.39$ and 0.43, respectively). Correlations between stress-related variables and MRS ranged from $r = 0.19$ to 0.64. After partitioning by PA, significant correlations were observed in the low PA group between school- and sports-related self-esteem and anxiety with the MRS. CONCLUSIONS: The results provide suggestive evidence that PA may modify the relationship between stress and MRS in male adolescents.

Background

Currently, an estimated 16.5% of United States (US) children and adolescents, 6 to 19 years of age, are obese and an additional 15% are overweight, which represents a three-fold increase over the past few decades [1]. These figures warrant attention since childhood obesity is adversely associated with cardiovascular disease (CVD) risk factors [2-4]. In addition to the immediate consequences, childhood obesity often tracks into adulthood [5] and has been linked to CVD morbidities in adulthood (coronary artery calcification [6], dyslipidemia and hypertension [7, 8], and carotid artery intima-media thickness [9]) and CVD mortality [10].

As mentioned, obesity, and more specifically abdominal or visceral obesity, is associated with elevated blood pressure (BP), an adverse blood lipid profile, and insulin resistance. The co-occurrence of these traits has been termed the metabolic syndrome [11]. 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 [12] and 6.4% among US adolescents [13]. Furthermore, about 43% of US adolescents possess at least one characteristic of the metabolic syndrome and 17% have two or more characteristics [14, 15].

Given the increased prevalence of pediatric obesity and the metabolic syndrome, there has been great interest in preventing these conditions during childhood and adolescence with considerable focus on diet and physical activity [16]. However, epidemiological studies indicate that physical activity and diet only explain a small-to-modest amount of the total variance in the adiposity-metabolic syndrome phenotype. Thus, there is reason to consider other possible contributing factors to 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 [17-20]. Chronically elevated levels of cortisol results in an up-regulation of lipoprotein lipase and subsequent storage of fat, specifically in the viscera [19]. Additionally, increased cortisol levels negatively affect insulin sensitivity [18]. Although the relationships between stress-related cortisol secretion and markers of the metabolic syndrome have been demonstrated in adults [21-25], little evidence is available in children [26]. Likewise, few studies have examined the relationship between physical activity and stress-related variables (i.e. perceived stress, anxiety, depression, self-esteem, etc.) in children and adolescents. In general, an inverse relationship has been demonstrated between physical activity and stress-related measures [27]. Parfitt and Eston [28] recently found that habitual physical activity was negatively related to anxiety and depression (r = -0.48 and -0.60, respectively,) and positively associated with global self esteem (r = 0.66) in children. A recent study also found that physical activity buffered the associations between chronic stress and adiposity [29]. However, both stress and physical activity were self-reported. Additionally, this study only examined the stress-physical activity interaction with adiposity. This relationship has yet to be established with the metabolic syndrome, a more comprehensive expression of overall metabolic health.

The purpose of this study was to examine if physical activity modifies the association between measures of stress (various self-report measures) and the components of the metabolic syndrome in adolescent males. We hypothesized that the correlation between stress and the metabolic syndrome would be stronger among subjects with lower levels of physical activity compared to those with higher levels of physical activity.

Methods

Subjects. Thirty-eight boys, ages 8-18 years, participated in the current study. Due to non-compliance with the physical activity assessment of one subject, 37 subjects were included in the analysis. All subjects signed assent forms and parental consent was obtained prior to data collection. This study was approved by the university Institutional Review Board.

General Procedures. The study protocol was reviewed with the subject upon arrival to the laboratory. The test session included an explanation of the physical activity monitors and assessment of CVD risk factors. Additionally, the subject received instruction on questionnaires aimed at assessing perceived stress, anxiety, depression, self-esteem, weight-related and general appearance-related teasing, and media time. A detailed description of each of these measures is provided below. Subject demographics and anthropometric data were assessed following the explanation of the study protocol. The subject was then seated for 5-10 minutes prior to the measurement of resting BP, blood lipids and glycosylated hemoglobin (HbA1c).

Assessment of stress.

Self-report measures of stress. Since stress is a ubiquitous term and is difficult to qualify with a single measure, the following surveys were used in an attempt to capture the various aspects of stress.

Physical Appearance Related Teasing Scale (PARTS). PARTS was used to determine weight- and size-related and general appearance-related teasing by peers [30]. The PARTS questionnaire consists of two scales with a total of 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 five-point Likert 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 [30].

Perceived Stress Scale (PSS). The PSS is a global measure of stress and was used to determine the subjects' overall perception of stress in their lives over the last month [31]. 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 [31].

Children's Depression Inventory (CDI). Depressive characteristics were examined using the CDI [32]. This instrument consists of 27 items assessing affective, cognitive, and behavioral symptoms of depression. Subjects were asked to choose the sentence that best describes them for the past two weeks (e.g. "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 [32].

State-Trait Anxiety Inventory for Children (STAI-C) The STAI-C [33] 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 [33].

Self-Esteem Questionnaire (SEQ). The SEQ [34] is composed of six sub-scales 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." Subjects 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 consistency [34].

Physical activity and media time. The Manufacturing Technology Inc. (MTI) uniaxial accelerometer (Shalimar, FL) was used to assess habitual physical activity. 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 is digitized and the magnitude 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 in this study. The unit was attached to a belt and worn at the mid-axillary line at the hip. The instrument was explained to the subject and worn for 4 days during the subsequent week (3 weekdays and 1 weekend day). The accelerometers were returned via mail in a standard, padded envelope. The Freedson age-specific MET equation [35] [MET = 2.757 + (0.0015 x counts/min) – (0.08957 x age (yr)) – (0.000038 x counts/min x age (yr))] was used to convert accelerometer counts to a metabolic equivalent. According to the Freedson protocol, moderate-to-vigorous physical activity (MVPA) was classified as being >3 METs.

TV viewing and video game playing time was assessed via questionnaire. The questionnaire asked participants to quantify average daily time spent watching TV and playing video games during the week and on weekend days. Specifically, the questionnaires asked subjects to quantify the time they spent with each form of media from the time they woke up until lunchtime, from lunchtime until dinner, and from dinner until bedtime. Subjects were asked these questions for both schooldays and weekend days.

Body size and maturity status. Stature and body mass were measured according to standard procedures [36]. 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 (WC) was assessed as a measure of central adiposity. Waist circumference was measured immediately above the iliac crest using a Gullick tape to the nearest 0.1 cm.

Since the age range of the subjects spans the period of puberty and numerous body size and physiological functions vary by pubertal status [37], 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. [38]. Anthropometric variables are used to calculate a value that is aligned to the estimated age of peak height velocity (e.g., -1.5 yrs from peak height velocity, etc.). This value was used as a covariate in the statistical analysis.

Assessment of CVD risk factors.

Resting blood pressure. Resting systolic and diastolic BP was measured using an automated monitor (Critikon Dinamap) in accordance with standard recommendations [39]. Mean arterial pressure (MAP) was calculated as: systolic BP – diastolic BP/3 + diastolic BP. 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 non-fasted 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 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 [40].

Derivation of the metabolic risk score. The metabolic risk score (MRS) was derived by first standardizing the individual MRS variables (WC, MAP, HbA1c, and HDL-C) by regressing them onto age to account for any age-related differences. The standardized HDL-C was multiplied by –1 since it is inversely related to metabolic risk. The standardized residuals (Z-scores) were summed to create the continuous MRS. These variables were chosen because they represent similar constructs used in the adult clinical criteria for the metabolic syndrome. We chose to include HDL-C as our sole indicator of dyslipidemia, because samples were collected in the non-fasted state and measures of triglycerides would be inaccurate. Likewise, HbA1c was chosen for similar reasons and fasting glucose is often normal in children, even those who are overweight. MAP was used since including both systolic and diastolic would load two blood pressure variables into the calculation, and MAP represents both SBP and DBP. 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 continuous value. A lower score is indicative of a better metabolic risk factor profile. The MRS has been used in recent work from our laboratory [41] and others [42].

Statistical analysis. Descriptive statistics were calculated for all variables in the total sample and in the high and low physical activity groups. The low and high physical activity groups were determined based on a median split (77 min MVPA). The associations between self-report measures of stress, physical activity/media time and the MRS were examined by partial correlation, controlling for chronological age and maturity offset, in the total sample and in the low and high PA groups. All statistical analyses were conducted using SPSS version 12.0.

Results

Table 1 provides the descriptive statistics for the total sample and the low and high physical activity groups. In the total sample, mean height, weight, and BMI approximated the 50th percentile on the CDC growth chart [43]. Approximately 27% and 16% of the participants were overweight or obese, respectively according to international cut points [44]. In addition to accumulating significantly less vigorous activity and MVPA, the participants in the low physical activity group were also significantly taller. Although not statistically significant, those in the low physical activity group were also noticeably older and heavier, had a higher BMI, WC, systolic BP, and MRS and lower HDL-C than the participants in the high physical activity group. None of the stress-related measures differed between the two groups.

Table 2a shows partial correlations between physical activity and the MRS. Correlations were low ($r < -0.13$), but in the expected direction (e.g., inverse). TV and video game playing were significantly related to the MRS ($r = 0.39$ and 0.43, respectively). Table 2b shows partial correlations between selected stress variables and the MRS. Of the self-reported markers of psychosocial stress, only school-related self-esteem ($r = -.46$, $p < .05$) and the general appearance scale of the PARTS ($r = -.36$, $p < .05$) were significantly related with the MRS. Of the remaining variables, the correlations of sports-related self-esteem and trait anxiety with the MRS approached significance ($p = .08$ and $p = .09$, respectively) and, therefore, were considered in subsequent analyses.

Table 3 shows partial correlations between selected markers of psychosocial stress and the MRS for the low and high physical activity groups. Both school- and sports-related self-esteem were significantly associated with the MRS ($r = -0.64$ and -0.53, respectively) in the low physical activity group. Trait-anxiety was also significantly associated with the MRS in the low physical activity group ($r = 0.53$). In contrast, none of the stress variables were associated with the MRS in the high physical activity group. As an example, a pictorial representation of the relationship between anxiety and MRS is shown in Figure 1.

Discussion

In an attempt to explain some of the unaccounted variance of obesity and related metabolic disorders, an emerging research trend is the examination of variables acting within a system of complex interactions rather than in univariate relations. For example, previous research has examined the relationship between physical activity and the metabolic syndrome [45] and the relationship between physical activity and stress [28, 29]; however, limited research has examined if physical activity modifies the relationship between stress and the metabolic syndrome in adolescents. The present study aimed to address this question in school-aged boys. To our knowledge, only one previous study [30] examined the relationship of self-reported personal and community stress and self-reported physical activity (assessed by the number of days per week that the respondents were active enough to "work up a sweat") with three measures of adiposity (waist circumference, sum of three skinfolds (tricep, subscapula, suprailiac), and BMI) in 303 12 and 24 year olds. Personal stress, but not physical activity, was significantly associated with BMI, after controlling for age, race, gender, socioeconomic status, and parental smoking. Moreso, the interaction of both personal and community stress with physical activity significantly predicted adiposity measures. It should be noted, however, that although statistically significant ($p < .05$), these interaction terms accounted for only 2- 3% of the variance in adiposity measures, with the total models accounting for no more than 15% and 22%.

The present study followed a considerably different conceptual and methodological approach that should enable readers to evaluate this issue from two different but complementary perspectives. First, instead of focusing only on adiposity, we chose to study the metabolic syndrome. Due to the fact that it reflects a broader spectrum of risk factors, the MRS is presumably a more robust indicator of overall metabolic and cardiovascular health than any single measure of adiposity. Second, given the lack of consensus in defining and operationalizing stress, as well as the absence of previous literature on the stress indices that might be most relevant to metabolic health among youth, we chose to adopt a broad-based approach to measuring stress. Thus, we thought it was important to assess a number of self-reported indices. We assessed both key variables known to be related to the appraisal of the demands of daily life (i.e., perceived stress, anxiety, depression, self-esteem), as well as variables known to influence the well-being of school-age youth (i.e., appearance-related teasing). The results from the present study are consistent with those found by Yin et al. [29] and provide suggestive evidence that physical activity may modify the relationship between stress and the metabolic syndrome in 8 to 18 year old boys. More specifically, low physical activity appears to enable psychosocial variables to impact metabolic health, or higher levels of physical activity may buffer the association of anxiety with the metabolic syndrome.

Given its novelty, the current study can be considered, in several respects, as preliminary findings on this topic. This is particularly important in the case of the methods used to operationalize "stress." First, among the self-reported stress-related variables assessed, only anxiety (positively) and aspects of self-esteem (negatively) were found to be related to the MRS. In contrast, depression and perceived stress were not related to MRS in this small sample. Surprisingly, appearance-related teasing was found to have a modest but significant relationship with the MRS, but the correlation was in the opposite-than-predicted direction (i.e., negative), albeit only in the whole sample and not among the low physical activity group. Presumably, this correlation might have been sample-specific and, therefore, unreliable. In our view, the challenge of identifying the stress marker or markers most closely related to the metabolic syndrome remains to be determined. For example, even though depression was unrelated to the MRS in the present investigation, it has previously been found related to BMI among school-age children [46].

The correlations between physical activity and MRS were low in the present study ($r < -0.13$), but were in the expected direction (i.e. negative). This finding is consistent with previous research which indicates that physical activity explains only a small amount of the variance in individual components of the metabolic syndrome among children and adolescents [47]. However, TV and video game playing time were significantly related with the MRS, which is also consistent with the literature. Previous research examining TV and other sedentary activities and adiposity provide convincing evidence to the role of physical inactivity/sedentary behavior in youth [48-52]. In a recent study by Heelan and Eisenmann [53], sedentary activities were better correlates of adiposity for both boys and girls (r = 0.31 and 0.51, respectively) compared to physical activity variables. The present investigation furthers the current body of literature by establishing a relationship between sedentary behavior and the metabolic syndrome in youth.

Despite recent investigations in adults [19, 54], limited attention has been given to the association between stress and physical health status in children and adolescents. Most research has focused on the bivariate relationship between stress and body mass. Sjörberg and colleagues [46] found higher BMI to be associated with depression. A low quality of life (QOL) has also been observed among obese children and adolescents [55]. Furthermore, the QOL in obese youth 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 [56]. Each of these stress-related constructs has the potential to perturb HPA axis functioning and possibly affect markers of metabolic 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 MRS. This finding may suggest that the stress associated with poor self-esteem, teasing, and anxiety is related to adverse health status in young people. Björntorp [19] 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 present example, increased stress due to poor self-esteem and teasing may result in chronic hypersecretion of cortisol and subsequent metabolic syndrome. Given the dynamic nature of adolescence on psychosocial function, further research should examine this hypothesis.

Although a relationship between stress and the MRS exists, the unexplained variance suggests that other factors contribute to the metabolic syndrome. It was hypothesized that physical activity may buffer the relationship between stress and MRS. The main finding in the current investigation provides preliminary, but suggestive evidence to support this hypothesis. Although cross-sectional correlations do not confer causal inference, the results suggest that adequate MVPA is important for individuals with poor self-esteem and high anxiety in order to maintain metabolic health. Because the manifestation of the metabolic syndrome typically does not occur until later in life, the prevalence is relatively low among adolescence and does not accurately express the severity of the problem. The use of a composite score such as the one created here allows each subject to have a value relative to a more healthy or diseased status. Several studies have shown that the combined components of the metabolic syndrome tracks from adolescence into adulthood [1, 8, 57]. An additional strength of this study is the objective measurement of physical activity by accelerometery.

Although the findings are based on a small sample, this study is distinct in its conceptual design. This is the first study to examine the associations between physical activity, stress-related measures, and the metabolic syndrome in children and adolescents. Further research is needed in larger samples before conclusive evidence is drawn. The results from the present study show preliminary but suggestive evidence that physical activity buffers the relationship between stress and the metabolic syndrome. Viewed from a broad perspective, this study may serve to usher in a new era in which the problems of obesity and the metabolic syndrome are seen as precipitated by a multitude of interacting etiologic factors that go beyond physical activity and diet. The consideration of stress in addition to physical activity and diet for prevention and treatment strategies of childhood obesity and metabolic syndrome may prove beneficial in that stress can influence factors associated with the metabolic syndrome [19], including obesity, and in turn, obesity has the propensity to influence stress levels via teasing and self-esteem [55, 58]. Because of the reciprocal relationship that exists between stress and metabolic disease it may be 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.

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References

1. Hedley A, Ogden C, Johnson C, Carroll M, Curtin L & Flegal K. Prevalence of overweight and obesity among US children, adolescents, and adults, 1999-2002. JAMA 291, 2847-2850 (2004).
2. Smoak CG, Burke GL, Webber LS, Harsha DW, Srinivasan S & Berenson GS. Relation of obesity to clustering of cardiovascular disease risk factors in children and young adults. Am J Epidemiol 125, 364-372 (1987).
3. Katzmarzyk PT, Srinivasan SR, Chen W, Malina RM, Bouchard C & Berenson GS. Body mass index, waist circumference, and clustering of cardiovascular disease risk factors in a biracial sample of children and adolescents. Pediatr 114, e198-205 (2004).
4. Gallistl S, Sudi KM, Borkenstein M, Troebinger M, Weinhandl G & Muntean W. Determinants of haemostatic risk factors for coronary heart disease in obese children and adolescents. Int J Obes 24, 1459-1464 (2000).
5. Power C, Lake JK & Cole TJ. Measurement and long-term health risks of child and adolescent fatness. Int J Obes 21, 507-526 (1997).
6. Mahoney LT, Burns TL, Stanford W, Thompson BH, Witt JD, Rost CA & Lauer RM. Coronary risk factors measured in childhood and young adult life are associated with coronary artery calcification in young adults: the Muscatine Study. J Am Coll Cardiol 27, 277-284 (1996).
7. Lauer R, Lee J & Clarke W. Factors affecting the relationship between childhood and adult cholesterol levels: The Muscatine Study. Pediatr 82, 309-318 (1988).
8. Eisenmann JC. Secular trends in variables associated with the metabolic syndrome of North American children and adolescents. Am J Hum Biol 15, 786-794 (2003).
9. Raitakari OT, Juonala M, Kahonen M, Taitonon L, Laitinen T, Maki-Torkko N, Jarvisalo MJ, Uhari M, Jokinen E, Ronnemaa T, Akerblom HK & Viikari JSA. Cardiovascular risk factors in childhood and carotid artery intima-media thickness in adulthood: The cardiovascular risk in the Young Finns study. JAMA 290, 2277-2283 (2003).
10. Nieto FJ, Szklo M & Comstock GW. Childhood weight and growth rate as predictors of adult mortality. Am J Epidemiol 136, 210-213 (1992).
11. Cleeman J. Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). 285, 2486-2497 (2001).
12. Cameron AJ, Shaw JE & Zimmet PZ. The metabolic syndrome: prevalence in worldwide populations. Endocrinol Metab Clin North Am 33, 351-375 (2004).
13. Duncan, G E, Li, S M & Zhou, X H. Prevalence and trends of a metabolic syndrome phenotype among U.S. adolescents, 1999-2000. Diabetes Care 27, 2438-43. (2004).
14. Laaksonen D, L H, Niskanen L, Kaplan G, Salonen J, Lakka T. Metabolic syndrome and development of diabetes mellitus: Application and validation of recently suggested definitions of the metabolic syndrome in a prospective cohort study. Am J Epidemiol 156, 1070-1077 (2002).
15. Ferranti S, G K, Ludwig D, Neufeld E, Newburger J, Rifai N. Prevalence of the metabolic syndrome in American adolescents: Findings from the third National Health and Nutrition Examination Survey. Circulation 110, 2494-2497 (2004).
16. Koplan J, Liverman C & Kraak V. Preventing childhood obesity: Health in the balance. National Academies Press (2005).

17. Björntorp P. Visceral fat accumulation: the missing link between psychosocial factors and cardiovascular disease? J Internal Med 230, 195-201 (1991).

18. Björntorp P. Neuroendocrine perturbations as a cause of insulin resistance. Diabetes Metab Res Rev 15, 427-441 (1999).

19. Björntorp P. Do stress reactions cause abdominal obesity and comorbidities? Obes Rev 2, 73-86 (2001).

20. Björntorp P & Rosmond R. Obesity and cortisol. Nutrition 16, 924-936 (2000).

21. Rosmond R, Chagnon YC, Holm G, Chagnon M, Perusse L, Lindell K, Carlsson B, Bouchard C & Bjorntorp P. A glucocorticoid receptor gene marker is associated with abnormal obesity, leptin, and dysregulation of the hypothalamic-pituitary-adrenal axis. Obes Res 8, 211-218 (2000).

22. Björntorp P, Holm G & Rosmond R. Hypothalamic arousal, insulin resistance and type II diabetes mellitus. Diabetic Medicine 16, 373-383 (1999).

23. Rosmond, R, Dallman, M & Björntorp P. Stress-related cortisol secretion in men: relationships with abdominal obesity and endocrine, metabolic and hemodynamic abnormalities. J Clin Endocrinol Metab 83, 1853-1859 (1998).

24. Fraser R, Ingram M, Anderson N, Morrison C, Davies E & Connell J. Cortisol effects on body mass, blood pressure, and cholesterol in the general population. Hypertension 33, 1364-1368 (1999).

25. Räikkönen K, Keitikangas-Järvinen L, Adlercreutz H & Hautanen A. Psychosocial stress and the insulin resistance syndrome. Metabolism 45, 1533-1538 (1996).

26. Dimitriou T, Maser-Gluth C & Remer T. Adrenocortical activity in healthy children is associated with fat mass. Am J Clin Nutr 77, 731-736 (2003).

27. Strong WB, Malina RM, Blimkie CJR, Daniels SR, Dishman RK, Gutin B, Hergenroeder AC, Must A, Nixon PA, Pivarnik JM, Rowland T, Trost S & Trudeau F. Evidence Based Physical Activity for School-age Youth. Pediatr 146, 732-737 (2005).

28. Parfitt G & Eston R. The relationship between children's habitual activity level and psychological well-being. Acta Paediatrica 94, 1-7 (2005).

29. Yin, Z, Davis, C, Moore, J & Treiber, F. Physical activity buffers the effects of chronic stress on adiposity in youth. Ann Behav Med 29, 29-36 (2005).

30. Thompson JK, Fabian LJ, Moulton DO, Dunn ME & Altabe MN. Development and validation of the Physical Appearance Related Teasing Scale. J Pers Assess 56, 513-521 (1991).

31. Cohen, S, Kamarck, T & Mermelstein, R. A global measure of perceived stress. J Health Social Behavior 15, 17-25 (1983).

32. Kovacs M. The Children's Depression Inventory Manual (Multi-Health Systems, Tonawanda, NY, 1992).

33. Spielberger CD, Edwards CD, Lushene RE, Montuori J & Platzek D. Preliminary manual for the State-Trait Anxiety Inventory for Children (Consulting Psychologists Press, Palo Alto, CA, 1973).

34. DuBois D, Felner RD, Brand S, Phillips RSC & Lease AM. Early adolescent self-esteem: A developmental-ecological framework and assesment strategy. Journal of Research on Adolescence 6, 543-579 (1996).

35. Freedson P, Pober D & Janz KF. Calibration of accelerometer output for children. Med Sci Sports Exerc 37, S523-S530 (2005).

36. Malina, R M. in Physiological Assessment of Human Fitness (eds. Maud, P. J. & Foster, C.) 205-219 (Human Kinetics, Champaign, IL, 1995).
37. Tanner, J M. Growth at Adolescence (Blackwell, Oxford, 1962).
38. Mirwald, R L, Baxter-Jones, A D, Bailey, D A & Beunen, G P. An assessment of maturity from anthropometric measurements. Med Sci Sports Exerc 34, 689-694 (2002).
39. National High Blood Pressure Education Program Working Group on Hypertension Control in Children and Adolescents. The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. Pediatr 114, 555-576 (2004).
40. Little, R. Glycated Hemoglobin Standardization - National Glycohemoglobin Standardization Program (NGSP) Perspective. Clin Chem Lab Med 41, 1191-1198 (2003).
41. Eisenmann JC, Katzmarzyk PT, Perusse L, Tremblay A, Després J-P & Bouchard C. Aerobic fitness, body mass index and CVD risk factors among adolescents: the Québec family study. Int J Obes 29, 1077-1083 (2005).
42. Anderson LB, Harro M, Sardinha LB, Froberg K, Ekelund U, Brage SW & SA, A. Physical activity and clustered cardiovascular risk in children: a cross sectional study (The European Youth Heart Study). Lancet 368, 261-262 (2006).
43. Ogden, C L, Kuczmarski, R J, Flegal, K M, Mei, Z, Guo, S, Wei, R, Grummer-Strawn, L M, Curtin, L R, Roche, A F & Johnson, C L. Centers for Disease Control and Prevention 2000 growth charts for the United States: improvements to the 1977 National Center for Health Statistics version. Pediatr 109, 45-60 (2002).
44. Cole TJ, Bellizzi MC, Flegal KM & Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. BMJ 320, 1240-1243 (2000).
45. Eisenmann, J. Physical activity and cardiovascular disese risk factors in children and adolescents. Can J Cardiol 20, 1-7 (2004).
46. Sjorberg RL, Nilsson KW & Leppert J. Obesity, Shame, and Depression in School-Aged Children: A Population-Based Study. Pediatr 116, 389-392 (2005).
47. Eisenmann, J C. Physical activity and cardiovascular disease risk factors in children and adolescents: an overview. Can J Cardiol 20, 295-301 (2004).
48. Eisenmann, J C, Bartee, R T & Wang, M Q. Physical activity, television viewing and weight status in U.S. adolescents: Results from the 1999 YRBS. Obes Res 10, 379-385 (2002).
49. Anderson, R E, Crespo, C J, Bartlett, S J, Cheskin, L J & Pratt, M. Relationship of physical activity and television watching with body weight and level of fatness among children: results from the Third National Health and Nutrition Examination Survey. JAMA 279, 938-942 (1998).
50. Crespo, C J, Smit, E, Troiano, R P, Bartlett, S J, Macera, C A & Anderson, R E. Televison watching, energy intake, and obesity in US children. Arch Pediatr Adolesc Med 155, 360-365 (2001).
51. Dowda, M, Ainsworth, B E, Addy, C L, Saunders, R & Riner, W. Environmental influences, physical activity, and weight status in 8- to 16-year-olds. Arch Pediatr Adolesc Med 155, 711-717 (2001).
52. Dietz, W H & Gortzmaker, S L. Do we fatten our children at the television set? obesity and television viewing in children and adolescents. Pediatr. 75, 807-812 (1985).

53. Heelan KA & Eisenmann, J. Physical activity, media time, and body composition in young children. J Phys Act Health 1, 1-14 (2006).
54. Rosmond R. The role of stress in he pathogenesis of the metabolic syndrome. Psychoneuroendocrinology 30, 1-10 (2005).
55. Schwimmer, J B, Burwinkle, T M & Varni, J W. Health-related quality of life of severely obese children and adolescents. JAMA 289, 1813-1819 (2003).
56. Varni, J W, Burwinkle, T M, Katz, E, Meeske, K & Dickinson, P. The PedsQOL in pediatric cancer: reliability and validity of the Pediactric Quality of Life Inventory Generic Cole Scales, Multidimensional Fatigue Scale, and Cancer Module. Cancer 94, 2090-2106 (2002).
57. Hickman TB, Briefel RR, Carroll MD, Rifkind BM, Cleeman JI, Maurer KR & Johnson CL. Distributions and trends of serum lipid levels among United States children and adolescents ages 4-19 years: data from the Third National Health and Nutrition Examination Survey. Prev Med 27, 879-890 (1998).
58. Janssen I, Craig WM, Boyce WF & Pickett W. Associations between overweight and obesity with bullying behaviors in school-age children. Pediatr 113, 1172-1174 (2004).

Table 1. Physical characteristics of the sample. Values are mean (SD) for low physical activity, high physical activity, and the total sample. The minimum-maximum values are also provided for the total sample.

	Low PA (n=18)	High PA (n=19)	Total (n=37)
Anthropometric Variables			
Age (yrs)	15.5 (2.1)	12.5 (2.4)	13.9 (2.7)
			9.0-18.9
Ht (cm)	173.1 (8.1)*	157.9 (16.2)	165.3 (14.9)
			136.7-189.6
Estimated APHV (yrs)	14.7 (0.8)	14.3 (0.6)	14.5 (0.7)
			13.1-16.5
Body mass (kg)	73.2 (14.3)	54.1 (21.7)	63.4 (20.7)
			27.1-111.1
BMI (kg/m^2)	24.4 (4.5)	21.0 (5.1)	22.7 (5.1)
			14.5-35.9
WC (cm)	83.8 (13.6)	72.8 (16.0)	78.1 (15.7)
			49.8-119.2
Overweight/Obese (%)	55.6%	31.6%	43%
Metabolic Variables			
SBP (mmHg)	126.0 (10.0)	116.3 (10.3)	121.0 (11.1)
			101-153
DBP (mmHg)	67.9 (3.8)	65.9 (3.7)	66.9 (3.8)
			59-74
MAP (mmHg)	89.0 (4.1)	86.0 (4.1)	87.4 (4.3)
			79-98
HbA1c (%)	5.4 (0.3)	5.5 (0.5)	5.5 (0.44)
			4.9-7.4
HDL-C (mg/dL)	39.0 (10.8)	49.5 (12.4)	44.4 (12.6)
			15-74
Metabolic Risk Score	0.18 (2.7)	-0.11 (2.3)	0.03 (2.5)
			-3.6-7.0

Physical Activity			
MVPA (min/day)	46.2 (15.9)*	109.7 (32.7)	78.8 (41.1) 22-205
Vigorous PA (min/day)	5.1 (7.1)*	18.0 (17.1)	11.7 (14.6) 0-65
Moderate PA (min/day)	41.2 (12.9)	91.7 (20.2)	67.1 (30.6) 21-140
Total PA (counts/min)	374.7 (130.5)	629.7 (241.1)	505.7 (232.0) 234-1164
Television (hrs/wk)	19.9 (12.7)	27.4 (19.7)	23.8 (16.9) 2-83
Video games (hrs/wk)	12.3 (23.0)	21.9 (24.9)	17.2 (24.2) 0-105
Selected Stress Variables			
SE-school	22.9 (6.2)	26.3 (3.9)	24.6 (5.4) 8-40
SE-sports	17.2 (3.6)	18.8 (3.7)	18.0 (3.7) 6-30
Anxiety	31.2 (5.2)	31.7 (5.1)	31.4 (5.1) 20-60
PARTS-GA	32.7 (4.4)	34.1 (4.0)	33.4 (4.2) 6-36

*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 2a. Partial correlations, controlling for age and maturity offset, between physical activity and the metabolic risk score in male adolescents.

	r
Physical Activity	
MVPA (min/d)	-0.13
Vigorous PA (min/d)	-0.09
Moderate PA (min/d)	-0.13
Total PA (counts/min)	-0.07
Television (hrs/wk)	0.34*
Video games (hrs/wk)	0.43*

*P<0.05

MVPA, moderate to vigorous physical activity

Table 2b. Partial correlations, controlling for age and maturity offset, between selected measures of stress and the metabolic risk score in male adolescents.

Selected Stress Variables	r
SE-school	-0.46*
SE-sports	-0.31
Anxiety	0.29
PARTS-GA	-0.36*

*P<0.05

SE, Self Esteem; CDI, Children's Depression Inventory Survey; PARTS-GA, Physical Appearance Related Teasing Scale - general appearance.

Table 3. Partial correlations, controlling for age and maturity offset, between stress measures and the metabolic risk score in low and high physical activity groups.

	Low PA	**High PA**
SE-school	-0.64*	-0.41
SE-sports	-0.53*	-0.09
Anxiety	0.53*	0.07
PARTS-GA	-0.39	-0.48

*P<0.05 indicates correlational significance

SE, Self Esteem; CDI, Children's Depression Inventory Survey; PARTS-GA, Physical Appearance Related Teasing Scale- general appearance.

Figure 1. Association between metabolic risk 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