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Accelerometer-determined physical activity and the cardiovascular response to mental stress in children

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ABSTRACT

Objectives: Cardiovascular reactivity has been associated with future hypertension and cardiovascular mortality. Higher physical activity (PA) has been associated with lower cardiovascular reactivity in adults, but little data is available in children. The purpose of this study was to examine the relationship between PA and cardiovascular reactivity to mental stress in children.
Design: Cross-sectional study.
Methods: This study sample included children from the Oswego Lead Study ($n = 79$, 46% female, 9–11 years old). Impedance cardiography was performed while children participated in a stress response protocol. Children were also asked to wear Actigraph accelerometers on their wrists for 3 days to measure intensity and duration of PA and sedentary time.
Results: In multivariable models, moderate to vigorous (MV) PA was associated with lower body mass index (BMI) percentile and lower total peripheral resistance (TPR) response to stress (beta = −0.025, $p = 0.02$; beta = −0.009, $p = 0.05$). After additional adjustment for BMI, MVPA was also associated with lower diastolic blood pressure response to stress (beta = −0.01, $p = 0.03$). Total PA and sedentary time were not associated with BMI or cardiovascular responses to stress.
Conclusions: A modest, inverse relation of PA to vascular reactivity to mental stress was observed in children. These data provide confirmatory evidence that the promotion of PA recommendations for children are important for cardiovascular health.

1. Introduction

Exaggerated cardiovascular (CV) reactivity to stress is associated with cardiovascular disease (CVD) risk in adults.[1] There is also consistent evidence showing that a high blood pressure (BP) response to stress is associated with a modest risk of future hypertension in both children and adults.[2,3] Physically inactive adults are generally observed to have higher CV reactivity to stress compared to more active peers,[4] which may partially explain the elevated hypertension and CVD risk attributed to physical inactivity. However, there is considerably less evidence supporting a relationship between physical activity (PA) and CV reactivity to stress in children.[5,6]

The underlying hemodynamics of the CV response may shed light on the relation of CV reactivity to CVD risk[7] as well as the influence of PA. An exaggerated BP response may be driven by a combination of elevated cardiac output (CO), insufficient vasodilation and low vascular compliance.[8] PA contributes to greater vascular compliance,[9] influencing the balance of BP to CO, thus reducing total peripheral resistance (TPR).

The main purpose of this investigation was to examine the relation between PA and the interplay among hemodynamic changes (primarily BP, heart rate [HR], and TPR) in response to mental stress in children. PA is a multidimensional factor that includes different intensity levels and sedentary behavior that may have independent effects on CV health, and we hypothesized that each may have independent effects on the CV response to mental stress tasks. The use of accelerometer-determined measures of PA in the current investigation has the potential to improve the sensitivity and specificity for detecting the influences of the volume of PA at different intensities on CV reactivity.

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2. Methods

Children between the ages of 9–11 years ($n = 100$) were recruited for participation in the current investigation as part of the Oswego Lead Study, as previously described.[10] Participant inclusion criteria involved willingness to undergo stress testing, agreeing to wear the PA monitor for 3 days and subjecting to a blood draw (for measures not included in the present investigation). Participants must have also reported no use of medication that could affect cardiovascular functioning (e.g., Ritalin) on the day of testing and have no significant developmental disorders that could affect task performance (e.g., severe intellectual disability). Written informed assent and consent was obtained from all participants and their parents, and children were paid $100 for their participation. The Human Research Committee of the State University of New York (SUNY) at Oswego approved this study.

Participants arrived at the laboratory in the morning, at which time height, weight and questionnaires were administered. A 10-minute (min) rest period was given directly before beginning the stress-reactivity protocol. Experimental tasks were developed and run using E-Prime™ (Psychology Software Tools, Pittsburgh, PA). This software is programmed to present stimuli and record timing of choices made by clicking the mouse or keypad and records positioning of the cursor. The order of acute stress tasks were counter balanced, as previously described.[10] Briefly, this protocol included: [1] a *mirror tracing task* (2 trials, 1.5-min each) in which participants use the mouse/cursor to trace a star (modified from [11]), with the mouse control inverted so that the cursor moves to the right when the mouse moves to the left; [2] an inter-task rest (8-min); [3] a *Go/No-Go task* (2 trials, 2.5-min each) in which the goal is to respond as quickly as possible to the target tone but not to a non-target tone, sounding on 10-second (s) intervals (8 targets and 7 non-targets); [4] an inter-task rest (8-min); and [5] a *continuous performance task* (5 trials, 75-s each) in which numerical stimuli (0–9) were shown on the screen in rapid, random sequence (250-ms stimulus interval, 500-ms inter-stimulus interval), with "9" as the target number. CV reactivity was measured using impedance cardiography, electrocardiogram (ECG), and BP cuff (non-dominant arm) during experimental tasks as described below.

This series of computer tasks was designed to be challenging for all participants and thereby reliably produce mild stress and a cardiovascular response, rather than major physiological stressors that may be less typical of everyday stressors. Our protocol is very similar to other protocols used to study cardiovascular responses to stress in children[2] and has been used extensively in the past.[10]

An Impedance Cardiograph (Model HIC-2000, Bio-Impedance Technology, Chapel Hill, NC) was used for the generation of the impedance wave forms using a tetra polar band electrode configuration. Electrodes for the ECG (to measure HR) were placed on each side of the abdomen below the impedance electrode bands, as well as a ground electrode beside the navel. This noninvasive technology measures changes in conductivity of the thorax. Calculations were performed as previously described[12] to derive stroke volume (SV) and CO.

Systolic (S)BP and diastolic (D)BP were monitored using the Vasotrac device (APM205A; Medwave, Danvers, MA), which automatically computes TPR using the formula: $TPR = ([(SBP - DBP)/3] + DBP)/CO \times 80$, where TPR is in dyne-s/cm^{-5}, CO is in liters/min (calculated by the Impedence Cardiograph), and SBP and DBP are in mm Hg. During the last 3-min of the initial rest period and during the entire stress task protocol, BP measurements were recorded every 30-s. During the same time periods, HR and data for the impedance-derived variables were collected using a 15-s inter-sample interval and 14-s ensemble average duration (allowing 1-s for storing data). Per previous recommendations,[12] measures based on volumetric calculations (i.e. SV, CO, and TPR) were only analyzed as change scores. The use of change scores has been reported to increase reliability of the measurements.[13]

SBP, DBP and HR change scores were computed by subtracting baseline levels of a variable from the task means. For impedance-derived variables involving volume measures (CO, SV, TPR), percent change from baseline to task was used because the accuracy of absolute levels of these variables is not reliable.[12] For all analyses of responses to acute stress, cardiovascular change scores were standardized within the three acute stress tasks and then averaged across tasks (zMean Δ or zMean %Δ).[2] This approach has been demonstrated to improve the reliability of cardiovascular reactivity assessment,[12] and has been shown to predict future BP in children.[2]

PA was measured with the Actigraph Model GT1M accelerometers (Actigraph Inc., Pensacola, FL), with a dynamic range of 0.05–2.5 g and frequency range of 0.25–2.5 Hz (Actigraph 2008). Participants were asked to wear accelerometers on their wrist from Friday morning when they arrived to the study visit (at 8 or 8:30 AM) until the next study visit (usually the following Monday) during all hours, except when bathing. Counts were collected in 1-min epochs.

Participants completed a sleep log, which was used as a general window to determine sleep and wake times. The actual sleep time was determined as starting with 10 consecutive minutes with no activity and wake time as 10 consecutive minutes of some activity, which was replicated successfully by independent coders ($r = 0.96$, $p < 0.001$). Sleep time was recoded as non-wear time for the current analysis. During waking hours, non-wear time was defined as 20 min or more 0 counts/min (allowing for 2 epoch interruptions). We received PA accelerometer data from 86 participants, of which 80 participants had 3 valid wear days with >10 h weartime per day, with sufficient wear time reported to provide high reliability in children.[14] Of these 80 participants, one was excluded for incomplete stress-test data ($n = 79$).

Sedentary time was defined as <420 counts per minute (cpm), moderate activity 4332–13,559 cpm, and vigorous activity ≥13,560 cpm, disseminating from a regression equation validated by Crouter et al. for wrist-worn Actigraph monitoring in children.[15] These cutpoints were chosen because they yielded better classification accuracy of activity intensity in previous studies.[16,17]

The following covariates were used in multivariate models: age, sex, socioeconomic status (SES) score (using an average of parents' education, occupation, and income; which were standardized prior to averaging), and season of accelerometer wear. Body mass index percentile (BMI%, age- and sex-adjusted by CDC standards[18]) was also added as a covariate in some models.

Results are reported as mean ± standard deviation. Comparisons by HR response were examined using *t* tests. PA variables were ranked to reduce the impact of outliers before regression models were run. Multivariable linear regression models were used to examine associations between PA and cardiovascular reactivity measures, adjusting for possible confounders. Interactions with BMI% were explored. All statistical analysis were performed using Statistical Analysis System (SAS, version 9.4, SAS Institute Inc., Cary, NC).

3. Results

There were no significant differences in baseline physiological or PA variables between boys and girls, aged 9–11 years in this study (Table 1). Mean resting BP was 106/57 mm Hg, increasing to 112/61 mm Hg during the mental stress tasks; mean HR increased from 86 to 87 bpm during the stress tasks (Table 1). Girls had a higher mean percent change in SV during the mental stress tasks ($p = 0.04$).

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