Objectively Measured Physical Activity and Cardiovascular Disease Risk Factors in African American Girls

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Objectively Measured Physical Activity and Cardiovascular Disease Risk Factors in African American Girls

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Abstract

Objective—This study examines the associations between objectively measured physical activity and cardiovascular disease (CVD) risk factors in preadolescent African American girls.

Methods—We conducted a cross-sectional analysis of data from Stanford Girls Health Enrichment Multisite Studies (GEMS) trial. Physical activity was assessed for four days by using an ActiGraph accelerometer and was correlated with anthropometric measures, blood pressure, blood lipids, glucose, and insulin. Associations between physical activity and CVD risk factors were computed using by Spearman correlations. Bonferroni adjustment \( \alpha = .003 \) was used to correct for multiple testing.

Results—A total of 261 girls participated, of which 208 had complete CVD risk measures (mean age 9.4 years, mean body mass index 20.7 kg/m\(^2\)). Average daily physical activity and time spent in moderate-to-vigorous physical activity (MVPA) were significantly correlated with body mass index \((r=-.23, P=.0008 \text{ and } r=-.29, P<.0001, \text{ respectively})\) and insulin \((r=-.27, P=.0001 \text{ and } r=-.30, P<.0001, \text{ respectively})\) but not to other CVD factors. After adjusting for age-adjusted pubertal stage of development, the association between MVPA and insulin remained significant.

Conclusions—Objective measures of both average daily physical activity and MVPA were inversely associated with body mass index and insulin levels in African American girls.

Keywords
African American; Preadolescent Girls; Physical Activity; CVD Risk Factors

Introduction

Associations between a physically active lifestyle and cardiovascular disease (CVD) risk factors have been widely documented in adults.\(^1\) However, less data are available on these associations in children, and what is reported has been inconsistent.\(^1\) Inconsistent results may be due to the wide variations in the methods used to assess physical activity.\(^2\) Epidemiologic studies have relied on self-report to quantify physical activity because of
their low cost and ease of use. Self-report relies on children’s abilities to report and recall past physical activity, so it does not provide an objective measure of physical activity. Because of these problems, more studies are using accelerometers to provide objective means to quantify physical activity. Only a few studies have assessed the associations between objectively measured physical activity and CVD risk factors in children, and the results are still inconsistent. Another reason for the inconsistency could be because the samples studied were heterogeneous. Correlates between physical activity and CVD risk factors sometimes vary across age, sex, and ethnic groups. While heterogeneous, representative samples are desirable to estimate population prevalence, heterogeneity could attenuate associations in correlational studies, which would threaten internal validity.

The prevalence of CVD risk factors and physical inactivity tends to be higher in African American children than in their Caucasian counterparts. However, only a small number of studies have examined the associations between physical activity and CVD risk factors in African American children. Of these studies, only one used objectively measured physical activity, and no associations were found; however, study participants were recruited differently from different regions of the United States. To address some of these potential limitations, we examined the associations between objectively measured physical activity and CVD risk factors in a homogeneous sample of African American preadolescent girls from a single city.

Methods
Participants
This study used baseline data from Stanford Girls Health Enrichment Multisite Studies (GEMS), a randomized, controlled, weight-gain prevention trial among lower socioeconomic status African American preadolescent girls. Girls were recruited from low-income areas of Oakland, California. Participants were required to be 8–10 years of age and have a BMI ≥50th percentile for their age or at least one parent or guardian with a BMI ≥25 kg/m². Girls were excluded from the study if they were taking any medications or had a medical condition that could affect their growth, if they had a condition that limited their participation in the interventions, if they were unable to understand or complete informed consent, or if they planned to move from the San Francisco Bay area in the next 24 months. A parent or guardian provided written informed consent and girls gave assent to participate in the study. The study was approved by the Stanford University Panel on the Protection of Human Subjects in Medical Research, was implemented as a cooperative agreement with the National Heart, Lung, and Blood Institute (NHLBI), and received oversight from an independent Data and Safety Monitoring Board formed by NHLBI.

Measures of Physical Activity
Participants’ physical activity levels were monitored by using the ActiGraph accelerometer (Model 7164, Manufacturing Technologies Inc. Health Services, Fort Walton Beach, Fla) for four days including one weekend day. The ActiGraph is a single-axis accelerometer designed to measure and record vertical accelerations ranging in magnitude from .05–2.00 G with a frequency response from .25–2.5 Hz. Within these parameters, the ActiGraph is capable of detecting normal human motion and rejecting motions associated with high-frequency vibrations. Accelerations were measured in one-minute epochs in this study.

Minute-by-minute accelerometer data were first reviewed visually to determine whether 1) the number of days with accelerometer data appeared sufficient and matched study protocol, 2) sleep and awake times were logical, 3) if any error codes indicated that the monitor might have malfunctioned (e.g., count per minute values of 32,767, which has been reported to
represent a voltage signal saturation within the ActiGraph,\textsuperscript{14} or 4) if any count per minutes values were $\geq 15,000$ (these were flagged, and participant’s parent or guardian was contacted to verify the participant was involved in an activity that might have resulted in such a high value). Data points with error codes and one minute on either side of those values were changed to missing data. Next, data were scanned to find periods of at least 20 consecutive minutes during which the ActiGraph measured only zeros, and these were considered periods when the monitor was not being worn and were changed to missing data. After the above procedures were completed, all remaining data were classified as acceptable data.

For all accelerometer data-reduction procedures, weekdays and weekend days were treated separately. Next, within the weekday/weekend day data, each participant’s accelerometer data were collapsed across days by averaging the counts per minute for each minute of the day for each day with data for that minute; this process resulted in a composite estimate for a weekday and weekend day. This data algorithm significantly maximizes the amount of usable data in this same sample, compared with more traditional algorithms.\textsuperscript{15} The composite weekday and weekend day were defined as 6:00 AM–10:00 PM (960 minutes) and 10:00 AM–10:00 PM (720 minutes), respectively. From the composite day, average daily physical activity levels (counts per minute) and percentage of time spent at different activity intensity levels were calculated. Intensity levels were based on the counts per minute intensity thresholds for adolescent girls.\textsuperscript{16}

**Measures of Cardiovascular Disease Risk**

Body weight was measured twice in light clothing, to the nearest 0.1 kg, using a calibrated portable digital scale (Scale-Tronix Model 5602 Scale, Scale-Tronix, White Plains, NY). Standing height was measured twice to the nearest millimeter with a portable direct reading stadiometer (Shorr height measuring board, Olney, Md). Waist circumference was measured twice to the nearest 0.1 cm at the level of the umbilicus, at end-expiration, using a nonelastic tape. Triceps skinfold thickness was measured three times with a Harpenden skinfold caliper (British Indicator, West Sussex, UK). For each assessment, the mean of the replicate measures was used in analysis. Sexual maturation status was self-assessed using drawings and descriptions of five standard stages of breast and pubic hair development,\textsuperscript{17} and the mean was used in the analysis.

Blood pressure was measured on the right arm supported at heart level using an automated blood pressure monitor (Dinamap Pro 100, GE Medical Systems, Tampa, Fla) with an appropriate-sized cuff, after three minutes of quite rest. Blood pressure measures were repeated two more times separated by one-minute rest intervals. The average of the three measures was used in the analysis.

Blood samples were collected in participants’ homes after an eight-hour fast. Blood samples were allowed to clot in a serum separator tube at room temperature for 30 minutes. Serum samples were then isolated by centrifugation at 1500\(\times\)g for 15 minutes, aliquoted into microcentrifugation tubes, and then placed on ice while in the field. Serum samples were then stored at $-70^\circ$C for analysis within 24 hours at the Stanford University Hospital Biochemistry Laboratory, which participates in the Centers for Disease Control and Prevention–NHLBI Lipid Standardization Program.

High-density lipoprotein cholesterol (HDL-C) was directly measured using a homogeneous assay that eliminates the need to first isolate HDL lipoprotein from serum sample. Total cholesterol, triglyceride, and HDL-C levels were analyzed using Beckman Synchron LX20 reagents (Beckman Coulter, Inc. Fullerton, Calif). Low-density lipoprotein cholesterol (LDL-C) was calculated using the Friedewald equation.\textsuperscript{18} Insulin was measured with an automated two-site chemiluminescent immunometric (sandwich) assay (DPC 2000...
Immulite, Diamond Diagnostics, Inc, Holliston, Mass). Glucose was determined by the oxygen rate method with a Beckman Oxygen electrode with a Beckman Synchron LX20 (Beckman Coulter, Inc).

**Statistical Analysis**

Associations between derived estimates of physical activity and CVD risk factors were computed using Spearman nonparametric correlations, making no assumptions about the distributions. A weighted average of weekday and weekend average daily physical activity (counts/minute) and time spent in moderate-to-vigorous physical activity (MVPA) was used in the analyses because weekday and weekend day activity variables were highly correlated (average counts/minute, \( r = .58, P < .0001 \); MVPA, \( r = .52, P < .0001 \)). The associations between physical activity estimates and CVD risk factors were adjusted for age-adjusted pubertal stage of development using partial correlation. Statistical analyses were performed with SAS version 9.1 (SAS Institutes Inc, Cary, NC). A Bonferroni adjustment \( \alpha = .003 \) was used to correct for multiple testing. For a sample of 261, we had 90% power to detect correlations \( \geq .2 \) magnitude at \( \alpha = .05 \).

**Results**

We enrolled 261 participants in the study. Complete ActiGraph data were obtained from 260, and blood samples were obtained from 208, who were included in this analysis. Descriptive data for selected CVD risk factors and physical activity variables are presented in Table 1. Participants’ mean BMI was 20.7 kg/m\(^2\) and their mean percentile BMI for their age was 74.7±26.2 (Table 1).\(^{19}\) Participants’ mean systolic and diastolic blood pressures were below the 50th percentile for their age and height.\(^{20}\) Mean total cholesterol was borderline elevated (>75th percentile), but mean LDL-C was within the normal range.\(^{21}\) All participants were normoglycemic (mean glucose <126 mg/dL). Participants spent 96.2% and 3.8% of their day engaged in sedentary to light and moderate to vigorous physical activity, respectively.

Average daily physical activity and percent of time spent in MVPA were significantly inversely related to BMI (average daily activity, \( r = -.23, P = .0008 \); MVPA, \( r = -.29, P < .0001 \)) and fasting insulin (average daily activity, \( r = -.27, P = .0001 \); MVPA, \( r = -.30, P < .0001 \)). No other correlations were statistically significantly different from zero. Age and pubertal stage of development were significantly correlated with physical activity estimates and BMI. After adjusting for age, all associations remained significant, with the exception of the association between average daily activity and BMI. However, after adjusting for age-adjusted pubertal stage of development, only the relationship between MVPA and fasting insulin remained significant (Table 2).

In addition, we examined the effects of different categories of physical activity (time spent in sedentary, light, moderate, and vigorous activity) and the selected CVD risk factors. When analyzed separately and adjusted for age-adjusted pubertal stage of development, time spent in moderate-intensity activity (\( r = -.23, P = .001 \)) was significantly correlated to insulin, while time spent in vigorous activity (\( r = -.13, P = .05 \)) was not correlated. Additionally, time spent in vigorous activity (\( r = -.28, P < .001 \)) was significantly correlated to BMI. No other significant correlations were observed between categorizes of physical activity and selected CVD risk factors.

As expected, waist circumference (\( r = .94, P < .0001 \)) and triceps skinfold (\( r = .85, P < .0001 \)) were highly positively correlated to BMI and were therefore not included separately in this analysis. Of the three, we chose to focus on BMI because it is more accurately assessed in children then either triceps skinfold thickness or waist circumference.\(^{22}\) Similarly, diastolic
blood pressure was significantly correlated to systolic blood pressure ($r=.47, P<.0001$) and consequently not included separately in the analysis.

**Discussion**

We found that average daily physical activity and time spent in MVPA are significantly inversely associated with BMI and insulin levels. After adjusting for age-adjusted pubertal stage of development, only the association between time spent in MVPA and insulin level remained significant after Bonferroni adjustment of the alpha level. These findings are similar to what others have found in European children when using objective measures of physical activity. For example, in a study of the association between objectively measured physical activity and indices of insulin resistance in 8- to 10-year-old Danish children ($N=384$), physical activity and insulin levels were negatively correlated.\(^4\) Similar findings were reported in a group of 9- to 15-year-old Swedish children ($N=668$).\(^5\) In both of these studies, physical activity was assessed with ActiGraph accelerometers, and the association between physical activity and insulin was still present after adjusting for potentially confounding variables.

In the present analysis, adjusting for age-adjusted pubertal stage of development attenuated the significant associations between physical activity measures and BMI. When examining the association between physical activity and BMI in children, we adjusted for age because age is positively associated with BMI and inversely associated with physical activity level.\(^23\) There is also a positive relationship between pubertal stage of development and BMI.\(^24\) However, the mechanism relating pubertal development and BMI is still unclear, and whether increased BMI leads to early pubertal development or vice versa is unclear. Similarly, after adjusting for age-adjusted pubertal stage of development, average daily physical activity was no longer significantly associated with fasting insulin level at a Bonferroni adjusted level of significance.

We did not find any associations between physical activity and blood lipids, blood pressure, or glucose levels. In general, the results regarding the associations between physical activity and these CVD risk factors in children and adolescents have been inconsistent. Inverse correlations between physical activity and total cholesterol, triglyceride, LDL-C, and glucose levels and a positive correlation with HDL-C level have been reported in some studies,\(^5,11,25,26\) but other studies have not found significant correlations between physical activity and these same CVD risk factors.\(^4,6\) These inconsistencies could be due to changes in age and maturation that occurs during pubertal development.\(^2\) However, it is also argued that the inconsistent associations between physical activity and CVD risk factors could be due to the methods used to assess physical activity.\(^2\) Studies that have used subjective measures of physical activity such as self-report have generally reported significant associations between physical activity and blood lipids, blood pressure, and glucose levels, even after controlling for confounding variables. Whereas in studies where physical activity levels were assessed objectively (eg., with accelerometers) no associations were found, with the exception of insulin levels.\(^4,6\) Therefore, caution should be taken when interpreting the results of studies that used self-report. If errors in self-reported physical activity are nonrandom, calculated associations with other measures may be biased.

We cannot rule out the possibility that the lack of significant associations between physical activity and some of the CVD risk factors is due to limited variation in both CVD risk factor values and physical activity levels in our sample. Attenuated correlations may be due to the fact that blood lipids and blood pressure levels are generally low in young people.\(^27\) Participants involved in the present study were apparently healthy. Although most participants had CVD risk factor levels in the normal range, with the exception of fasting
total cholesterol, relatively large standard deviations and interquartile ranges indicate substantial variation in many of these variables. Average accelerometer counts per minute and minutes of MVPA were rather low, potentially limiting the variation in those measures.

A strength of the present study is our participant population. Few studies of these relationships have involved African American children. In general, African American adults have been shown to have a higher prevalence of CVD risk factors and lower levels of physical activity than do their Caucasian counterparts and are therefore at increased risk for physical inactivity-related morbidities. Since physical inactivity-related morbidities begin during childhood, the associations between physical activity and CVD risk factors in African American children must be examined.

Because of the cross-sectional nature of the present analysis, we are unable to infer temporality or causality or determine whether an increase or decrease in physical activity would have any beneficial or negative effects on CVD risk factors. Although we found no significant associations between physical activity and fasting blood lipids or blood pressure at one point in time, changes in physical activity may still be associated with changes in CVD risk factors over time. Longitudinal analyses in African American girls will be needed to answer this question. We chose to perform this analysis in a sample of low-income, preadolescent, African American girls in a single city to enhance internal validity and avoid the potential problem of obscuring true associations through racial and geographic heterogeneity. Although our sample was an advantage for purposes of internal validity, whether our study results are generalizable to other groups of African American girls is unknown. Finally, while accelerometers provide a more objective measure of physical activity than self-report, accelerometers also have some limitations. For example, accelerometers do not capture upper body activities or activities that occur mostly in the horizontal plane with minimal vertical acceleration such as cycling. Accelerometers also have to be removed during water activities such as swimming. If any of these unmeasured activities influence CVD risk factors, correlations derived from accelerometer data may be attenuated or obscured. In addition, although accelerometers provide information on physical activity intensity and duration, they are not able to provide information on the types of physical activity being performed. Because of our accelerometer data reduction method, we were not able to examine the association between physical activity frequency and CVD risk factors. Regardless of these limitations, the use of accelerometers to measure physical activity is a distinct strength of this study.

Our study results indicate that physical activity is inversely associated with fasting insulin levels in apparently healthy, low socioeconomic status, African American girls. Other studies have found African American children to be less active and to have higher insulin levels than their Caucasian counterparts, which may put them at increased risk of type 2 diabetes and subsequent CVD. Therefore, future efforts should be geared toward developing and testing interventions to increase physical activity and decrease sedentary behaviors to determine their effects on reducing CVD risk factors in this at risk population.

Acknowledgments

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References


Table 1
Cardiovascular disease risk factors and physical activity measures in low-income, preadolescent, African American girls

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ± SD</th>
<th>Median</th>
<th>Interquartile Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>9.4±.9</td>
<td>9.5</td>
<td>8.7–10.1</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>40.4±12.6</td>
<td>38.2</td>
<td>31.2–46.9</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>138.8±8.8</td>
<td>138.4</td>
<td>132.7–144.1</td>
</tr>
<tr>
<td>Body mass index (kg/m^2)</td>
<td>20.7±4.8</td>
<td>19.6</td>
<td>17.0–23.0</td>
</tr>
<tr>
<td>Triceps skin fold (mm)</td>
<td>17.4±8.2</td>
<td>15.5</td>
<td>10.7–22.7</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>68.5±12.7</td>
<td>65.1</td>
<td>59.0–76.2</td>
</tr>
<tr>
<td>Resting heart rate (bpm)</td>
<td>81.1±9.8</td>
<td>80.8</td>
<td>74.0–87.5</td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>97.8±10.1</td>
<td>97.5</td>
<td>90.5–104.5</td>
</tr>
<tr>
<td>Diastolic blood pressure (mm Hg)</td>
<td>56.1±6.6</td>
<td>56.0</td>
<td>52.0–60.5</td>
</tr>
<tr>
<td>Total cholesterol (mg/dL)</td>
<td>173.6±30.8</td>
<td>171.5</td>
<td>151.5–195.5</td>
</tr>
<tr>
<td>LDL-C (mg/dL)</td>
<td>105.1±27.3</td>
<td>103.0</td>
<td>87.0–122.0</td>
</tr>
<tr>
<td>HDL-C (mg/dL)</td>
<td>55.6±12.7</td>
<td>54.0</td>
<td>47.0–64.5</td>
</tr>
<tr>
<td>Triglyceride (mg/dL)</td>
<td>64.4±32.0</td>
<td>57.5</td>
<td>44.0–72.0</td>
</tr>
<tr>
<td>Insulin (mU/L)</td>
<td>10.4±9.3</td>
<td>8.0</td>
<td>5.0–12.5</td>
</tr>
<tr>
<td>Glucose (mg/dL)</td>
<td>84.9±7.0</td>
<td>85.0</td>
<td>81.0–89.0</td>
</tr>
<tr>
<td>Average accelerometer (counts/minute)</td>
<td>642.6±189.0</td>
<td>608.5</td>
<td>525.8–738.3</td>
</tr>
<tr>
<td>Time spent in sedentary-to-light physical activity (%)</td>
<td>96.2±2.3</td>
<td>96.7</td>
<td>95.1–97.8</td>
</tr>
<tr>
<td>Time spent in moderate-to-vigorous physical activity (%)</td>
<td>3.8±2.3</td>
<td>3.3</td>
<td>2.2–4.9</td>
</tr>
</tbody>
</table>

SD, standard deviation; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol.

Note: To calculate average minutes per day of sedentary-to-light or moderate-to-vigorous physical activity, multiply by 891.4.
Table 2
Spearman correlation for physical activity and selected CVD risk factors among low-income, preadolescent, African American girls, adjusted for age-adjusted pubertal stage of development

<table>
<thead>
<tr>
<th>Variable</th>
<th>Average Daily Physical Activity (counts/minute)</th>
<th>Percentage of Time Spent in Moderate-to-Vigorous Activity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$r$</td>
<td>$P$ value</td>
</tr>
<tr>
<td>Body mass index (kg/m$^2$)</td>
<td>$-0.11$</td>
<td>0.13</td>
</tr>
<tr>
<td>Resting heart rate (bpm)</td>
<td>$-0.03$</td>
<td>0.63</td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>$-0.01$</td>
<td>0.86</td>
</tr>
<tr>
<td>Total cholesterol (mg/dL)</td>
<td>$-0.05$</td>
<td>0.46</td>
</tr>
<tr>
<td>Low density lipoprotein cholesterol (mg/dL)</td>
<td>$-0.05$</td>
<td>0.51</td>
</tr>
<tr>
<td>High density lipoprotein cholesterol (mg/dL)</td>
<td>$-0.02$</td>
<td>0.78</td>
</tr>
<tr>
<td>Triglyceride (mg/dL)</td>
<td>$-0.02$</td>
<td>0.74</td>
</tr>
<tr>
<td>Insulin (mU/L)</td>
<td>$-0.17$</td>
<td>0.02</td>
</tr>
<tr>
<td>Glucose (mg/dL)</td>
<td>$-0.05$</td>
<td>0.45</td>
</tr>
</tbody>
</table>

* Significant at Bonferroni adjustment $\alpha=0.003$. 

Significant at Bonferroni adjustment $\alpha=0.003$. 

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