The Influence of Physical Activity on Cardiometabolic Biomarkers in Youths: A Review

Bernard Gutin
University of North Carolina-Chapel Hill, Medical College of Georgia, and Columbia University

Scott Owens
University of Mississippi

The purposes of this article were to (1): review recent studies of relations between physical activity and cardiometabolic biomarkers of youths (2); highlight areas in which additional research is needed; and (3) make recommendations for preventive interventions. Observational studies show that youths who engage in high amounts of moderate-vigorous physical activity display a more favorable cardiometabolic biomarker profile than youths who engage in lesser amounts of moderate-vigorous physical activity. Intervention studies in obese youths show that favorable changes in biomarkers are produced by moderate-vigorous physical activity doses of 150–180 min/week. However, for nonobese youths, intervention studies suggest that such doses are not effective; higher moderate-vigorous physical activity doses of approximately 300 min/week seem necessary. Continuing a physically active lifestyle from childhood into the adult years will enable people to maintain less end-organ damage and lower rates of morbidity and mortality from cardiovascular disease and type 2 diabetes.

Although morbidity from obesity-related disorders (i.e., cardiovascular disease and type 2 diabetes) is ordinarily manifested in the adult years, some of the pathophysiologic processes underlying the disorders actually begin during the pediatric years (13). Because the global prevalence of obesity has increased markedly over the last few decades, it is likely that the prevalence of these disorders will be manifested at younger and younger ages (19). Such a scenario has potentially enormous implications for public health; therefore, preventive interventions are needed. A substantial body of recent research has highlighted the important role of physical activity (PA) in such interventions. The potential role of physical activity may be
examined in terms of its relation to cardiometabolic (CM) biomarkers that underlie cardiovascular disease and type 2 diabetes, such as insulin resistance, lipid profile, blood pressure, inflammation, left ventricular geometry, parasympathetic activity, and endothelial function.

In this paper we review recent studies (focusing especially on publications since the year 2000) of relations between PA and CM biomarkers of youths, highlight areas in which additional research is needed and make recommendations for preventive interventions.

**General Model**

Figure 1 is a schematic representation of potential relations among lifestyle behaviors (i.e., PA and diet), CM biomarkers, and “adult diseases” with consideration of mediating factors (i.e., fatness and fitness) and predisposing factors. CM biomarkers associated with premature acceleration of obesity-related disorders are often present in obese youths and frequently track into adulthood (49). The influence of lifestyle on cardiometabolic biomarkers is to a large degree mediated by physiologic factors such as fatness and fitness.

Because of the important role of fatness in this model, two issues need to be mentioned. First, recent research suggests that prevention of excess fatness in growing youths is primarily dependent on adequate volumes of vigorous PA, rather than PA of lower intensities or restriction of energy intake (22).

With respect to measurement of fatness, PA interventions can increase fat-free mass (FFM) at the same time that they reduce fat mass, thereby improving body composition without necessarily reducing weight (15). Thus, Body Mass Index

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**Figure 1** — Schematic representation of potential relations among lifestyle behaviors, CM biomarkers, and adult diseases with consideration of mediating and predisposing factors.
Influence of Physical Activity on Cardiometabolic Biomarkers

(BMI), which is useful for epidemiologic studies of obesity prevalence in populations, is not always appropriate for PA studies.

Another reason to focus on body composition rather than weight, especially in growing youths, is illustrated by a provocative new line of research dealing with stem cell differentiation. There are reciprocal relationships in the processes through which stem cells differentiate into different tissues, such that mechanical signals that stimulate deposition of energy and nutrients into bone and muscle tend to direct them away from fat; moreover, the mechanical stimulation which is effective in preventing the development of fatness, is not necessarily effective in reducing the fatness of already obese animals. This emphasizes the importance of focusing our efforts on prevention, rather than treatment, of obesity. Although these studies have generally been conducted in rodent models, some confirming human data are becoming available (41). It is not yet known if the mechanical stimulation of tissues used in these studies (a vibrating platform) is comparable to the mechanical stimulation imparted by vigorous PA.

As a result of the emerging evidence of the importance of body composition rather than weight, this paper will emphasize studies that have measured indices of body composition such as fat mass, FFM, percent body fat (%BF) or visceral adipose tissue (VAT).

There are two general research strategies that can be used to test deductions from this model. In observational designs the variables are measured and the relations observed, either at one point in time or as they evolve over time. Although such designs can provide evidence that is consistent or inconsistent with causal hypotheses (e.g., PA causes a decrease in fatness and improvement in CM biomarkers), it is often difficult to know which way the causal arrow should be pointed. For example, a situation in which high levels of PA are associated with lower fatness and better CM risk profile would be consistent with the hypothesis that PA causes lower fatness and better CM health. But it is also possible that an inherited tendency to be fat causes fatter children to be inactive and to have poor CM risk profiles. In this case, increasing PA would not necessarily have a positive impact on body composition or CM health. From a public health perspective, we need to know what kind of interventions might improve matters; i.e., if youths increase their PA, will it lead to a reduction in fatness and related CM disorders?

A clearer way to test causal hypotheses is to conduct experimental interventions in which youths are randomly assigned to different PA regimens and followed over time to see if the physiologic and CM biomarkers are influenced. This approach requires that the interventions be administered and controlled over periods of time that are long enough to produce substantial changes in the mediating factors of fatness and fitness, which in turn may produce beneficial CM changes. Scientists who have tried to “control” the behavior of youths over long periods know that studies of this kind are logistically daunting. Moreover, the cost of facilities and staff to administer and document the interventions is very high. Consequently, relatively few such studies have been reported. At the current state of knowledge, we need to examine data from both observational and experimental studies to inform our public health recommendations.

A number of criteria were applied in selecting articles to be included in this review. First, the studies had to report data on human youths; although some animal studies were mentioned to support underlying physiologic mechanisms, they
were not included in the summarizing tables. Second, if more than one paper was published on the same cohort, only one of the papers was used unless the papers included somewhat different information. Third, it was necessary that relations during childhood or adolescence were reported; thus, studies that reported relations between childhood PA and adult CM biomarkers were omitted from the summarizing tables, even though they may have been mentioned in the text to emphasize the enduring effects of the childhood PA. Fourth, intervention studies had to report differences between the intervention group and a nonintervention control group.

Observational Studies

PA may exert much of its influence on CM biomarkers by having favorable effects on fitness and fatness, which in turn influence the underlying processes leading to cardiovascular disease and type 2 diabetes mellitus. Thus, studies typically have focused on risk factors for these two diseases, namely fasting levels of insulin, lipids, and inflammatory markers. Some recent studies have gone further to determine whether PA influences other mechanisms and end-organ parameters such as: cardiac parasympathetic activity, endothelial function, left ventricular geometry and function, arterial stiffness, and carotid intima-media thickness (IMT). Because so little information is currently available on these variables, and because of the interrelationships among them and the CM biomarkers, we have tried to draw generalizations across the entire CM biomarker profile.

Observational studies have generally shown that youths who engage in relatively large amounts of PA have more favorable biomarker profiles than youths who engage in relatively little PA (1,8,16,20,40,48,56).

Because intervention trials have shown that PA decreases %BF and VAT (4,24,45), which are themselves related to poor CM biomarker levels (26), it is noteworthy that, to some degree, the association of PA to favorable biomarker profile is retained even after controlling for the possible mediating effect of body fatness (9,10,14,34,52). Because of the difficulty of measuring free-living PA accurately, some studies have used aerobic fitness as a surrogate index and then examined the separate and joint influence of fitness and fatness on CM biomarkers. Both fitness and fatness explain significant proportions of the variance in CM biomarkers (7,23,25,33) with the correlations being especially high for insulin status (29,38,50). Because fatness and fitness are inversely correlated (27), it is difficult to disentangle their separate influences in observational studies. Nonetheless, it appears that fatness explains more of the variance than fitness (7,28). With respect to insulin resistance, both fatness and fitness explain independent proportions of the variance (29,38). Some evidence indicates that the relationships of PA, fitness and fatness to insulin sensitivity are clearer in boys than in girls (29,35).

In recent years, a CM syndrome underlying cardiovascular disease and type 2 diabetes mellitus has been identified in adults, and the concept of risk factor clustering as being especially detrimental has been extended to youth (21). Youths who engage in more PA have better clustering scores than inactive youth (1), even after adjustment for fatness and fitness (2). Sustained participation in sports during youth decreases the risk for the CM syndrome during adulthood (57). Moreover, studies that have followed the subjects have shown that subjects with the metabolic
syndrome in the adult years had increased more in fatness, and decreased more in fitness and vigorous PA since adolescence (18).

Investigations of PA and biomarker profiles have generally focused on aerobic PA. However, some evidence is available that youths with substantial muscle strength, as a proxy for strength-building PA, have good insulin sensitivity (6). This subject deserves further study.

Taken together, the observational results support the hypothesis that PA is associated with a favorable CM biomarker profile. Table 1 summarizes observational studies that examined the relationship of PA to CM biomarkers in youths.

**Intervention Studies**

Because of the potential role of fatness as a mediator of the relationship between PA and risk profile, many intervention trials have used as subjects youths who were obese at baseline. Such studies show that PA programs lasting 2–8 months have favorable effects on many CM biomarkers, including insulin sensitivity, lipid profile, indices of inflammation, endothelial function, cardiac parasympathetic activity, and carotid IMT (3,17,37,43). As to whether certain CM biomarkers are more sensitive to changes than others in obese youths, data from high-quality randomized controlled trials suggest that support is strongest for favorable changes in fasting insulin and triglyceride levels (36). Indices of inflammation, such as C-reactive protein (CRP), appear less sensitive to increased PA (39). A study of 44 obese youths found that PA interventions led to improvements in insulin sensitivity, lipid profile, or blood pressure that were to some degree independent of changes in body fatness (17). These results are consistent with the hypothesis that PA can have a direct effect on some CM biomarkers without necessarily influencing total body fatness.

In contrast to results from intervention studies in obese youths, some research with youths who varied over the spectrum of fatness before the intervention has failed to provide evidence that PA reduced fatness or improved risk profiles (54). One way to explain this discrepancy between the results of observational and intervention studies is to consider that the observational studies provide information about a relationship that has evolved over a long period of time; e.g., youths who have been sedentary over several years before the measurements have gradually worsened their CM biomarker profile, with the result that a relationship between low levels of free-living PA and poor biomarker profile is clearly observed. Similarly, prospective observational studies that are carried out over several years time into the adult years may show that changes in PA are accompanied by worsening biomarker profile (18). Because of the difficulty of conducting randomized controlled trials over such long periods of time, conclusive evidence from intervention trials is quite difficult to obtain.

To give guidelines to the public, it is important to determine what dose of PA is associated with favorable biomarker levels. However, relatively little clear information is available on this matter. The limited information available from observational studies suggests that approximately 360 min per week or more of moderate-vigorous physical activity (MVPA) is associated with a good biomarker profile (1). With respect to a desired intensity of PA, some evidence indicates that vigorous PA, such as that found in sports (57), may be more closely related to a
Table 1 Relationship of Physical Activity (PA) and Cardiometabolic (CM) Biomarkers in Youths: Observational Studies

<table>
<thead>
<tr>
<th>Ref</th>
<th>N</th>
<th>Sex</th>
<th>Ages (years)</th>
<th>PA Measurements</th>
<th>CM Biomarkers</th>
<th>PA/CM Relationship</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1732</td>
<td>both</td>
<td>15 &amp; 19</td>
<td>PA quintiles from accelerometry (4 days)</td>
<td>Composite Risk Factor Score (SBP, TG, TC/HDL-C, HOMA-IR, body fat from skinfolds)</td>
<td>Increased clustering of risk factors in 3 lowest PA quintiles</td>
</tr>
<tr>
<td>2</td>
<td>1769</td>
<td>both</td>
<td>15 &amp; 19</td>
<td>PA quartiles from accelerometry (4 days)</td>
<td>Clustered Risk Factor Score (SBP, TG, TC/HDL-C, HOMA-IR, body fat from skinfolds)</td>
<td>PA independently associated with clustered risk even after adjustment for fitness and fatness</td>
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<tr>
<td>9</td>
<td>589</td>
<td>both</td>
<td>range = 8–10</td>
<td>PA from accelerometry (3 days)</td>
<td>Metabolic Syndrome Risk Score (SBP, DBP, insulin, glucose, TG, TC/HDL-C, body fat from skinfolds)</td>
<td>Metabolic risk inversely related to PA</td>
</tr>
<tr>
<td>10</td>
<td>90</td>
<td>both</td>
<td>5 and again at 10</td>
<td>Number of activities per week from questionnaire</td>
<td>Insulin Sensitivity Index (fasting insulin and glucose), body fat from DXA</td>
<td>Remaining relatively active associated with smaller decreases in insulin sensitivity</td>
</tr>
<tr>
<td>14</td>
<td>49</td>
<td>girls</td>
<td>range = 8–11</td>
<td>Daily nonresting EE from doubly-labeled water and self-reported moderate or greater PA</td>
<td>SBP, DBP, TG, TC, HDL-C, LDL-C, glucose, insulin, body fat from O dilution</td>
<td>Moderate or greater PA more likely associated with improved CM profile than is EE</td>
</tr>
<tr>
<td>16</td>
<td>771</td>
<td>both</td>
<td>range = 10–19</td>
<td>3-day PA record</td>
<td>TG, TC, LDL-C, HDL-C, body fat from skinfolds</td>
<td>Greater PA associated with increased HDL-C</td>
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Table 1 (continued)

<table>
<thead>
<tr>
<th>Ref</th>
<th>N</th>
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<tbody>
<tr>
<td>20</td>
<td>624</td>
<td>both</td>
<td>range = 8–10 year-1 mean = 9.6</td>
<td>MET score from 7-day PA recall measured over 3 years</td>
<td>SBP, LDL-C, BMI measured over 3 years</td>
<td>Significant association between increased MET score and lower SBP over 3 years</td>
</tr>
<tr>
<td>25</td>
<td>304</td>
<td>both</td>
<td>range 14–18 mean = 16.2</td>
<td>Moderate-to-vigorous PA (MVPA) from accelerometry (7 days)</td>
<td>Heart rate variability (HRV) as a measure of cardiac autonomic modulation</td>
<td>Greater MVPA associated with more favorable HRV</td>
</tr>
<tr>
<td>27</td>
<td>412</td>
<td>both</td>
<td>mean = 16</td>
<td>Moderate and vigorous PA from accelerometry (5 days)</td>
<td>% body fat from DXA</td>
<td>Lower % body fat associated with greater vigorous, but not moderate, PA</td>
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<tr>
<td>32</td>
<td>129</td>
<td>both</td>
<td>range = 10–11 mean = 10.3</td>
<td>PA intensity from accelerometry (7 days)</td>
<td>Vascular function from flow mediated dilation</td>
<td>High intensity PA was the only predictor of impaired vascular function</td>
</tr>
<tr>
<td>34</td>
<td>224</td>
<td>both</td>
<td>range = 7–10</td>
<td>PA intensity from accelerometry (4 days)</td>
<td>BMI, waist circumference</td>
<td>Body composition inversely related to PA intensity in boys, but not girls</td>
</tr>
<tr>
<td>35</td>
<td>1783</td>
<td>both</td>
<td>range = 12–19 mean = 15.4</td>
<td>MET hours/week from questionnaire</td>
<td>Insulin sensitivity using Quantitative Insulin Sensitivity Check Index</td>
<td>High levels of PA associated with high insulin sensitivity in boys, but not girls</td>
</tr>
<tr>
<td>40</td>
<td>54</td>
<td>both</td>
<td>range = 9–11.5</td>
<td>PA from accelerometry (4 days)</td>
<td>Insulin resistance using HOMA-IR</td>
<td>Low levels of PA associated with insulin resistance</td>
</tr>
<tr>
<td>47</td>
<td>878</td>
<td>both</td>
<td>range = 11–15 mean = 12.7</td>
<td>Minutes of moderate and vigorous PA from accelerometry (7 days)</td>
<td>BMI categories (normal or at risk for overweight)</td>
<td>More minutes of vigorous PA decreased likelihood of being at risk for overweight or overweight</td>
</tr>
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Table 1  (continued)

<table>
<thead>
<tr>
<th>Ref</th>
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<th>PA Measurements</th>
<th>CM Biomarkers</th>
<th>PA/CB Relationship</th>
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<tbody>
<tr>
<td>48</td>
<td>640</td>
<td>both</td>
<td>mean = 11.5</td>
<td>MET hours/week from questionnaire</td>
<td>Metabolic Syndrome variables (TG, HDL-C, glucose, SBP, and waist circumference), insulin resistance using HOMA-IR, inflammatory makers (IL-6, CRP), body fat from BIA</td>
<td>PA inversely related to insulin resistance and IL-6, independent of adiposity</td>
</tr>
<tr>
<td>51</td>
<td>780</td>
<td>both</td>
<td>range = 9–10</td>
<td>PA intensity from accelerometry (4 days)</td>
<td>Body fat from skinfolds</td>
<td>Vigorous PA, but not PA of lower intensity, inversely related to body fatness</td>
</tr>
<tr>
<td>52</td>
<td>357</td>
<td>both</td>
<td>range = 10–16</td>
<td>PA assessed with Paffenbarger Physical Activity Survey</td>
<td>SBP, DBP, TG, TC, HDL-C, LDL-C, insulin sensitivity with euglycemic hyperinsulinemic clamp, body fat from skinfolds</td>
<td>PA associated with increased insulin sensitivity</td>
</tr>
<tr>
<td>53</td>
<td>661</td>
<td>both</td>
<td>range = 14–18</td>
<td>PA from 24-hr activity recall</td>
<td>% body fat from DXA and visceral adipose tissue (VAT) from MRI</td>
<td>Vigorous PA was a negative predictor of % body fat but not of VAT</td>
</tr>
<tr>
<td>56</td>
<td>969</td>
<td>both</td>
<td>range = 8–16</td>
<td>PA from accelerometry (4 days)</td>
<td>BMI, TG, TC, HDL-C, LDL-C, glucose, insulin</td>
<td>Total PA was inversely associated with insulin</td>
</tr>
</tbody>
</table>

Abbreviations: SBP = systolic blood pressure, DBP = diastolic blood pressure, TG = triglycerides, TC = total cholesterol, IR = insulin resistance, WC = waist circumference, DXA = dual-energy x-ray absorptiometry, EE = energy expenditure, MET = metabolic equivalent, HOMA-IR = Homeostasis Model Assessment for Insulin Resistance, BIA = bioelectrical impedance analysis, MRI = magnetic resonance imaging, IL-6 = interleukin -6, CRP = C-reactive protein
favorable biomarker profile than is moderate PA (8,32). This is consistent with studies showing that vigorous PA, more so than moderate PA, is associated with lower levels of fatness and higher levels of fitness (27,47,51,53).

In intervention studies using obese youth, the dose of PA that has proved effective has been 150–180 min per week of MVPA. There appears to be a dose-response relationship, such that youths who engage in greater doses of PA have clearer beneficial effects than those who have smaller doses (37). This is an area in which research is needed.

In the general population of youth who are not preselected to be obese at baseline, few experimental data exist to show a beneficial effect of PA on fatness or CM biomarker profile, perhaps because the few investigations available used relatively small doses of PA. It is likely that doses of controlled MVPA greater than 300 min per week are needed for such youths to prevent accretion of general and visceral fat, suggesting that such doses may be needed to also influence fatness-related CM biomarkers (4). Especially high intensity resistance exercise may also elicit favorable changes in body composition (5); thus it seems sensible to incorporate strength-building exercise into a total PA program. Even when interventions have produced favorable effects on fatness or fitness, the changes in biomarker profile of the intervention and control groups did not reach significance (5,58). These studies suggest that it may be necessary for youths to maintain the PA-induced improvements in fatness or fitness for years to see clear effects on the fatness-related CM biomarker profiles.

The importance of maintaining exposure to PA on a continuing basis is illustrated by a study showing that obese youths who participated in a school-based PA intervention and who improved in fitness, fatness and fasting insulin concentration (12), lost their gains over the subsequent summer when they were not engaged in regular PA (11). The Medical College of Georgia FitKid project also showed beneficial effects in fatness and fitness during two school years, with beneficial changes lost during the subsequent summers (30).

Table 2 summarizes intervention studies that examined the effects of PA and CM biomarkers in youths.

### Developmental and Demographic Influences

Very few studies have investigated interactions of PA with age, developmental status, gender, race/ethnicity or socioeconomic status. Of those that have investigated such interactions, some have reported contradictory findings. For example, a study of Danish children found that PA was inversely related to insulin resistance in girls, but not in boys (9) whereas studies of American youth found the opposite result; i.e., greater PA and fitness were associated with better insulin sensitivity in boys, but not in girls (29,35). Thus, interactions of PA with these factors are an important topic for future investigations.

### Needed Research

High quality observational studies and randomized controlled trials are needed to clarify the dose-response relationships between PA and CM biomarkers. Inclusion of the less studied CM biomarkers (e.g., cardiac parasympathetic activity,
### Table 2  Effects of Physical Activity (PA) on Cardiometabolic (CM) Biomarkers in Youths: Intervention Studies

<table>
<thead>
<tr>
<th>Ref</th>
<th>N</th>
<th>Sex</th>
<th>Age</th>
<th>Other Conditions</th>
<th>Design</th>
<th>Type</th>
<th>Freq. (d/wk)</th>
<th>Duration (min/day)</th>
<th>Length</th>
<th>Intensity</th>
<th>CM Biomarkers</th>
<th>Significant Treatment Effects vs Control</th>
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<tbody>
<tr>
<td>3</td>
<td>15</td>
<td>both</td>
<td>age = 15</td>
<td>BMI &gt; 30, insulin resistant</td>
<td>RCT</td>
<td>aerobic</td>
<td>3</td>
<td>45</td>
<td>3 mon</td>
<td>not reported</td>
<td>IL-6, CRP, TG, TC, HDL-C, LDL-C, insulin resistance, % body fat from DXA</td>
<td>IL-6, CRP, insulin resistance, % body fat</td>
</tr>
<tr>
<td>4</td>
<td>201 girls</td>
<td>range = 8–12 mean = 9.5</td>
<td>African American</td>
<td>RCT</td>
<td>aerobic + resistance + flexibility + skills</td>
<td>5</td>
<td>35 min aerobic, 20 min resistance + flexibility, 25 min skills</td>
<td>10 mon</td>
<td>aerobic = hr &gt; 150 bpm</td>
<td>% body fat from DXA, VAT from MRI, BMI, waist circumference</td>
<td>% body fat, VAT, BMI</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>78</td>
<td>both</td>
<td>mean = 12.2</td>
<td>RCT</td>
<td>resistance</td>
<td>2</td>
<td>2 sets of 11 exercises</td>
<td>2 mon</td>
<td>15–18 on 6–20 RPE scale</td>
<td>TC, HDL-C, LDL-C, insulin resistance with HOMA2-IR, waist circumference, % body fat from BIA</td>
<td>Waist circumference, % body fat</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>5</td>
<td>both</td>
<td>mean = 12.0</td>
<td>RCT</td>
<td>aerobic</td>
<td>2–3</td>
<td>42</td>
<td>9 mon</td>
<td>not reported</td>
<td>Insulin, glucose, % body fat from DXA</td>
<td>Insulin, % body fat</td>
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<tr>
<th>Ref</th>
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<th>CM Biomarkers</th>
<th>Significant Treatment Effects vs Control</th>
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<td>17</td>
<td>44</td>
<td>both</td>
<td>mean = 8.9</td>
<td>BMI = 97th percentile</td>
<td>RCT</td>
<td>aerobic + resistance</td>
<td>3</td>
<td>30 aerobic + 20 resistance</td>
<td>3 mon</td>
<td>aerobic = 55–65% VO₂max</td>
<td>24-hr SBP, 24-hr DBP, TG, TC, HDL-C, LDL-C, CRP, FMD, IMT, BMI, % body fat and abdominal fat from DXA</td>
<td>24-hr SBP, % body fat, abdominal fat</td>
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<tr>
<td>30</td>
<td>206</td>
<td>both</td>
<td>mean = 8.5</td>
<td>BMI = 97th percentile</td>
<td>RCT</td>
<td>aerobic + resistance</td>
<td>5</td>
<td>80 min</td>
<td>3 years</td>
<td>MVPA</td>
<td>% body fat from DXA</td>
<td>% body fat</td>
</tr>
<tr>
<td>37</td>
<td>40</td>
<td>both</td>
<td>range = 13–16</td>
<td>triceps skinfold &gt;85th percentile</td>
<td>RCT</td>
<td>aerobic</td>
<td>5</td>
<td>29–43</td>
<td>8 mon</td>
<td>55–85% VO₂max</td>
<td>SBP, DBP, TG, TC, HDL-C, LDL-C, LDL particle size, VLDL-C, apo AI, apo B, Lp(a), insulin, glucose, % body fat from DXA, VAT</td>
<td>TG, VLDL-C, LDL particle size, % body fat, VAT</td>
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<th>Design</th>
<th>Type</th>
<th>Freq. (d/wk)</th>
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<tbody>
<tr>
<td>39</td>
<td>20</td>
<td>both</td>
<td>mean = 10.9</td>
<td>BMI &gt; 97th percentile</td>
<td>RCT</td>
<td>aerobic</td>
<td>4</td>
<td>50</td>
<td>2 mon</td>
<td>70–80% VO2peak</td>
<td>SBP, DBP, TG, TC, HDL-C, LDL-C, glucose, insulin, CRP, % body fat, FMD</td>
<td>HDL-C, FMD</td>
</tr>
<tr>
<td>43</td>
<td>67</td>
<td>both</td>
<td>mean = 14.7</td>
<td>BMI &gt; 97th percentile</td>
<td>RCT</td>
<td>aerobic</td>
<td>3</td>
<td>60</td>
<td>6 mon</td>
<td>not reported</td>
<td>24-hr SBP, 24-hr DBP, TC, HDL-C, LDL-C, insulin, glucose, CRP, FMD, IMT, % body fat from BIA</td>
<td>24-hr SBP, FMD, IMT</td>
</tr>
<tr>
<td>54</td>
<td>38</td>
<td>girls</td>
<td>range 13–14</td>
<td>RCT aerobic</td>
<td>3</td>
<td>20</td>
<td>75–85% VO2max</td>
<td>None</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: IL-6 = interleukin-6, CRP = C-reactive protein, DXA = dual-energy x-ray absorptiometry, TC= total cholesterol, TG = triglycerides, RPE = rating of perceived exertion, BIA = bioelectrical impedance analysis, HOMA2-IR= Homeostasis Model Assessment for Insulin Resistance, FMD = flow mediated dilation, IMT = intima-media thickness, MVPA = moderate-to-vigorous physical activity, VAT = visceral adipose tissue, MRI = magnetic resonance imaging, apo = apolipoprotein, HR = heart rate, BPM = beats per minute
endothelial function, left ventricular geometry and function, arterial stiffness, and carotid intima-media thickness) in these interventions may provide important insights not currently recognized. In addition, a better understanding is needed on how vigorous PA, such as seen with sports play and resistance training, relates to CM biomarkers, and ultimately to PA recommendations.

Insight into the underlying genetic mechanisms that orchestrate favorable physical activity-related changes in CM biomarkers is emerging from DNA transcription studies. These studies demonstrate that PA induces specific gene-protein interactions resulting in improved insulin signaling (31), fatty acid oxidation (42), and aerobic fitness (46). Additional studies of gene-PA interactions are needed to enhance our understanding of the causal pathways and optimal PA doses.

Investigations of how PA influences stem cells to differentiate into muscle and bone cells, rather than fat cells, can also help us understand the basic processes that underlie the development of healthy bodies during youth.

Finally, accurate measurement of free-living PA is an on-going research need. Advances in accelerometry, GPS, and thermal transduction appear promising, but lack standardization.

General Conclusion

Taken together, the observational and experimental evidence supports the hypothesis that maintaining high amounts and intensities of PA starting in childhood and continuing into the adult years will enable people to maintain a favorable CM biomarker profile, less end-organ damage, and lower rates of morbidity and mortality from cardiovascular disease and type 2 diabetes mellitus. The research suggests that MVPA for at least 1 hr per day would help youths to maintain a healthy CM biomarker profile. Higher volumes or intensities of PA may have greater benefit, but little is known about such high doses of PA.

Practical Implications

From a clinical perspective, pediatricians and family practitioners interact with parents and children on a regular basis; thus they should incorporate the topic of PA into their health assessments and be prepared to make appropriate recommendations to their patients. From a community perspective, “fitogenic” environments should be provided; such environments would tend to encourage better fitness, as contrasted with the obesogenic environments to which our youths are commonly exposed. Fitogenic environments should include physical education classes and recess periods during school hours, as well as programs for the after-school hours, weekends and vacation periods. Although moderate-intensity activities like walking may be valuable for especially unfit youths starting an exercise program, it seems that vigorous activities such as running, basketball, soccer, tennis, resistance training, swimming and dance, that provide greater mechanical stimulation to the tissues, are more likely to help a youth develop a healthy body composition. Of course, to avoid injury or undue fatigue, it is necessary for an unfit and obese child to build up gradually from lower to higher PA doses. It is also important that the activities be age-appropriate. For example, younger children prefer games that involve stop and go movements rather than exercising on machines at constant loads, whereas
some adolescents may do well with the adult model of exercising on machines or jogging at a constant pace. These ideas are consistent with the recommendations of a consensus report that youths should have at least 60 min/day of age-appropriate MVPA for obesity prevention (55).

In previous generations, it was common for youths to obtain substantial amounts of vigorous PA simply by playing outside without supervision. However, fears for safety have led many present-day parents to reject this degree of freedom for their children. Thus, communities need to step forward and provide safe and supervised fitogenic environments, especially in low SES neighborhoods where obesity is most prevalent. The main problem is that the facilities and staffing for such programs are quite expensive, thereby putting an added strain on already stretched budgets. However, the long-term benefits to public health are potentially enormous and there are few societal activities more worthy of our financial investment than the health of our children.

Acknowledgment

The initial literature review on which this paper is based was carried out by the CDC in connection with the development of physical activity guidelines for the USA by the U.S. Department of Health and Human Services. One of us (B.G.) was involved in the preparation of the scientific report that served as the foundation for the guidelines.

References


