Aerobic Fitness, Not Energy Expenditure, Influences Subsequent Increase in Adiposity in Black and White Children

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ABSTRACT. Background. Low levels of energy expenditure and aerobic fitness have been hypothesized to be risk factors for obesity. Longitudinal studies to determine whether energy expenditure influences weight gain in whites have provided conflicting results. To date, no studies have examined this relationship in blacks or whether aerobic fitness influences weight gain in white or black children.

Methods. One hundred fifteen children, 72 white (55 girls and 17 boys) and 43 black (24 girls and 19 boys) were recruited for this study. Aerobic fitness, resting, total, and activity-related energy expenditure and body composition were measured at baseline. The children returned annually for 3 to 5 repeated measures of body composition. The influence of the initial measures of energy expenditure and fitness on the subsequent rate of increase in adiposity was examined, adjusting for initial body composition, age, ethnicity, gender, and Tanner stage. Because 20 children did not attain maximum oxygen consumption, the sample size for the combined analysis was 95.

Results. Initial fat mass was the main predictor of increasing adiposity in this cohort of children, with greater initial fat predicting a higher rate of increase of adiposity. There was also a significant negative relationship between aerobic fitness and the rate of increasing adiposity (F coefficient = 3.92). With every increase of .1 L/minute of fitness, there was a decrease of .081 kg fat per kg of lean mass gained. None of the measures of energy expenditure significantly predicted increasing adiposity in white or black children.

Conclusions. Initial fat mass was the dominant factor influencing increasing adiposity; however, aerobic fitness was also a significant independent predictor of increasing adiposity in this cohort of children. Resting, total, or activity-related energy expenditure did not predict increasing adiposity. It seems that aerobic fitness may be more important than absolute energy expenditure in the development of obesity in white or black children.

Reduced energy expenditure has been hypothesized to be a predisposing factor for the development of obesity in infants, children, and adults.1-4 In addition, some cross-sectional studies in children and adults have shown that blacks have a lower resting energy expenditure (REE) than do whites, even after adjusting for body composition.5-9 This has been suggested as a possible reason for the increased prevalence of obesity observed in black individuals, particularly women, compared with white individuals.5,6,8 However, not all studies have shown this ethnic difference in REE. Sun et al10 found no difference in any component of energy expenditure between black and white prepubertal children. Regardless of whether cross-sectional differences exist, there is no evidence to link any difference in energy expenditure in blacks with the increased prevalence of obesity observed in blacks. To date, no longitudinal studies have examined whether reduced energy expenditure leads to increased weight gain in blacks.

Only a few longitudinal studies have been performed to examine the relationship between energy expenditure and obesity risk, with conflicting results.4,11-14 Studies on white infants13 and adult Pima Indians4 found that low total energy expenditure (TEE) resulted in greater weight gain during follow-up. However, other longitudinal studies on white infants14 and children12,13 failed to find any link between energy expenditure and future weight gain, including a previous study from our group of white children in Burlington, Vermont.12

Aerobic fitness may also play an important role in the development of obesity, because of its significant association with physical activity in young children.15 Aerobic fitness is also a marker of later cardiovascular disease (CVD), with greater aerobic fitness being associated with a reduction in risk of later CVD.16-18 Maximum oxygen consumption (VO2max) is one estimate of aerobic fitness that has been associated with lower levels of risk factors in a longitudinal study from adolescence to early adulthood.19 Cross-sectional studies have shown that blacks have a lower VO2max than do whites, even after adjusting...
for body composition. It is possible that this difference in aerobic fitness may help to explain the difference in obesity between the 2 ethnic groups. However, these studies have been cross-sectional in design and have not examined whether this observed difference in VO$_{2\text{max}}$ is correlated with future increased weight gain.

The aims of this study were, therefore, to determine whether TEE, REE, or activity-related energy expenditure (AEE) or VO$_{2\text{max}}$ predicted the rate of increasing adiposity in white and black children over a 3- to 5-year period. To separate the accumulation of fat in excess of that associated with normal growth, the increase in fat mass (FM) adjusted for the increase in lean mass was used as the major dependent variable. We hypothesized that energy expenditure and/or aerobic fitness would be negatively related to increased fat relative to lean mass, implicating these factors as causal in the development of obesity.

**METHODS**

**Subjects**

This study consisted of 115 children, 72 white (55 girls and 17 boys) and 43 black (24 girls and 19 boys) between 4.6 and 11.0 years of age at the start of the study. The children were recruited from Birmingham, Alabama, and had been free of any major illnesses since birth. Cross-sectional data from these children have been reported previously. Studies were performed during the school year (fall and spring). The nature, purpose, and possible risks of the study were carefully explained to the parents before consent was obtained. This study was approved by the Institutional Review Board at the University of Alabama at Birmingham. All measurements were performed at the General Clinical Research Center (GCRC) and the Department of Nutrition Sciences at the University of Alabama at Birmingham between 1994 and 1999.

**Protocol**

Children were admitted to the GCRC in the late afternoon for an overnight visit. On arrival a baseline urine sample was collected and subjects were dosed with doubly-labeled water. Anthropometric measurements and a physical examination by a pediatrician for assessment of sexual maturation were obtained. After 8 PM, only water and energy-free, noncaffeinated beverages were permitted until after the morning testing. On the following morning after an overnight fast, resting metabolic rate was assessed by indirect calorimetry on awakening of the subjects, and 2 timed urine samples were collected for the doubly-labeled water analysis. Two weeks later the children arrived at the Energy Metabolism Research Unit at 07:00 AM in the fasted state. Body composition was determined by dual-energy X-ray absorptiometry (DXA) and VO$_{2\text{max}}$ measured during a treadmill test. Two additional timed urine samples were collected for the doubly-labeled water analysis. All of the above tests were performed on the children’s initial visit, and body composition was also measured annually for 3 to 5 years after the initial visit.

**Assessment of Sexual Maturation**

Tanner’s criteria were used to estimate sexual maturation on the scale of 1 to 5, with stage 1 being prepubertal and 5 being adult. The same qualified paediatrician (R.F.-C.) assessed Tanner stage in all of the children.

**Measurement of Energy Expenditure Components**

TEE was measured over 14 days under free-living conditions with the doubly-labeled water technique, using a protocol with a theoretical error of <5%, as previously described. Carbon dioxide production was determined using equation R2 of Speakman et al., assuming a fixed dilution space ratio of 1.0427, and energy expenditure was calculated using equation 12 of de Weir. Mean values for the food quotient from triplicate 24-hour recalls of the children’s diet were .90 in whites and .87 in blacks.

REE was measured in the early morning after an overnight fast, using a Deltatrac Metabolic Monitor (Sensormedics, Yorba Linda, CA) as previously described. An adult-size canopy hood was used to collect the expired air. After a 10-minute equilibration period, data on oxygen consumption and carbon dioxide production were collected continuously for 20 minutes. Energy expenditure was calculated using the equation of de Weir.

Physical AEE was estimated from the difference between TEE and REE after reducing TEE by 10% to account for the thermic effect of feeding.

**Assessment of Body Composition**

FM and lean tissue mass (LTM; not including bone) were measured by DXA using a Lunar DPX-L densitometer (Lunar, Madison, WI) that we have previously validated in the pediatric body weight range. Subjects were scanned in light clothing while lying flat on their backs. DXA scans were performed and analyzed using pediatric software (Version 1.5c). On the day of each test, the DPX-L was calibrated using the procedures provided by the manufacturer.

**Exercise Testing**

Subjects followed an all-out, progressive walking treadmill protocol appropriate for children as described previously. The children walked for 4 minutes at 0% grade and 4 km/hour, after which the treadmill grade was raised to 10%. Each ensuing work level lasted 2 minutes, during which the grade was increased by 2.5%. The speed remained constant until a 22.5% grade was reached, at which time the speed was increased by .6 km/hour until the subject reached exhaustion.

Oxygen consumption and carbon dioxide production were measured continuously and analyzed using a Sensormedics metabolic cart (Model 2900, Sensormedics, Yorba Linda, CA). Heart rate was monitored by a Polar Vantage XL heart rate monitor (Model 62104, Polar Electro Inc, Woodbury, NY). Three criteria were used to determine a successful maximal test: 1) a leveling or plateauing of VO$_2$ (defined as an increase of oxygen uptake < 2 mL/kg/minute); 2) heart rate > 195 bpm; and 3) respiratory exchange ratio > 1.0. VO$_{2\text{max}}$ was defined by the attainment of at least 2 of the 3 criteria. Of the 20 children not successfully attaining VO$_{2\text{max}}$, 8 were white (7 girls and 1 boy) and 12 were black (8 girls and 4 boys). These were excluded from the data analysis involving VO$_{2\text{max}}$.

**Statistics**

The individual rate of increase in adiposity was calculated for each child using the annual measurements of FM and LTM. A regression equation was obtained for each child using the annual measurements of FM and LTM (3–5 measures per subject). For each child, FM was plotted against LTM for all visits, and the gradient of this relationship was used as the individual rate of increasing adiposity adjusted for the increase in LTM. These values ranged from –31.2 to 2.19 kg fat/kg lean. All measures of energy expenditure, including VO$_{2\text{max}}$, were regressed against LTM, and data > 3 standard deviations away from the means were excluded as physiologic outliers (n = 2). REE, VO$_{2\text{max}}$, and initial FM and LTM were not normally distributed and were log-transformed. A generalized linear model was used to determine the effects of each energy expenditure component and VO$_{2\text{max}}$ on the rate of increasing adiposity, with initial FM, LTM, and age as covariates, and Tanner stage and ethnicity as main-effect variables. Data were not stratified by age, but rather age was included as a covariate to adjust for differences in age at the start of the study. To determine the direction of the relationships, a multiple regression analysis was performed, with the rate of increasing adiposity as the dependent variable. Data were analyzed using SAS software, Version 6.10 (SAS, Cary, NC), with a significance level of P < .05.

**RESULTS**

**Descriptive Results**

The mean values for the variables measured at the initial visit and the rate of increasing adiposity are shown in Table 1, together with the results of the
2-way analysis of variance. There were no significant differences between white and black children in any of the variables measured (Table 1); however, boys had a higher LTM ($P = .02$), REE ($P < .001$), and VO$_{2\text{max}}$ ($P = .01$) than did girls.

**Generalized Linear Model (Table 2)**

The overall model significantly predicted the rate of increasing adiposity ($F_{9,82} = 3.49; P < .001$) and explained 27.7% of the variation. Initial FM was the primary predictor of increasing adiposity ($P < .001$), with high initial FM resulting in a greater rate of increase in adiposity (Fig 1).

There was also a significant but negative relationship between baseline VO$_{2\text{max}}$ and increasing adiposity ($P = .05$; Fig 2). Children with a higher VO$_{2\text{max}}$ at the start of the study had a lower rate of increase of adiposity over the course of the study. None of the baseline measurements of energy expenditure significantly predicted increasing adiposity in the children (TEE: $P = .72$; REE: $P = .79$; AEE: $P = .74$). There were no significant interactions between VO$_{2\text{max}}$ or the measurements of energy expenditure and ethnicity.

Ethnicity was significantly negatively related to increasing adiposity ($P = .03$), with black children having a lower increase in fat relative to lean than white children after adjusting for the other variables in the model.

**DISCUSSION**

The dominant factor influencing the rate of increasing adiposity was the initial amount of adipose tissue. Aerobic fitness was significantly and negatively related to increased adiposity, with increased initial fitness resulting in less adipose tissue gain. None of the measures of energy expenditure were

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**TABLE 1.** Mean and Standard Deviations for All the Variables Measured on the Initial Visit and the Rate of Increasing Adiposity Over the Course of the Study*

<table>
<thead>
<tr>
<th>Variable</th>
<th>White Boys ($n = 17$)</th>
<th>White Girls ($n = 55$)</th>
<th>Black Boys ($n = 19$)</th>
<th>Black Girls ($n = 24$)</th>
<th>Gender ANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>8.70 ± 1.76</td>
<td>8.10 ± 1.38</td>
<td>7.59 ± 1.5</td>
<td>8.05 ± 1.73</td>
<td>NS</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>32.63 ± 11.97</td>
<td>28.58 ± 7.41</td>
<td>31.11 ± 8.73</td>
<td>32.16 ± 11.37</td>
<td>NS</td>
</tr>
<tr>
<td>FM (kg)</td>
<td>9.38 ± 6.51</td>
<td>8.33 ± 4.19</td>
<td>8.13 ± 5.21</td>
<td>10.62 ± 6.68</td>
<td>NS</td>
</tr>
<tr>
<td>Lean mass (kg)</td>
<td>22.00 ± 5.77</td>
<td>19.18 ± 3.58</td>
<td>21.66 ± 3.94</td>
<td>20.25 ± 4.84</td>
<td>Gender $P = .016$</td>
</tr>
<tr>
<td>Tanner</td>
<td>1.06 ± .24</td>
<td>1.02 ± .13</td>
<td>1.00 ± .00</td>
<td>1.13 ± .34</td>
<td>NS</td>
</tr>
<tr>
<td>TEE (kcal/d)</td>
<td>1783 ± 377</td>
<td>1566 ± 399</td>
<td>1799 ± 480</td>
<td>1715 ± 428</td>
<td>NS</td>
</tr>
<tr>
<td>REE (kcal/d)</td>
<td>1307 ± 250</td>
<td>1172 ± 150</td>
<td>1257 ± 178</td>
<td>1192 ± 192</td>
<td>Gender $P = .006$</td>
</tr>
<tr>
<td>AEE (kcal/d)</td>
<td>307 ± 195</td>
<td>238 ± 312</td>
<td>355 ± 324</td>
<td>346 ± 254</td>
<td>NS</td>
</tr>
<tr>
<td>VO$_{2\text{max}}$ (L/min)</td>
<td>1.46 ± .39</td>
<td>1.12 ± .26</td>
<td>1.21 ± .29</td>
<td>1.22 ± .24</td>
<td>Gender $P = .014$</td>
</tr>
<tr>
<td>Rate of increasing adiposity</td>
<td>.18 ± .39</td>
<td>.45 ± .57</td>
<td>.36 ± .32</td>
<td>.29 ± .40</td>
<td>NS</td>
</tr>
</tbody>
</table>

* These are absolute values and have not been adjusted for any of the other variables. A 2-way ANOVA was performed to determine whether any of the above variables differed with gender and ethnicity.

NS indicates not significant.

* $P > .05$

**TABLE 2.** Results of the Generalised Linear Model and Multiple Regression Analysis on the Relationship Between Baseline Energy Expenditure and Fitness Measurements

<table>
<thead>
<tr>
<th>Variable</th>
<th>$F_{9,82}$</th>
<th>$\beta$</th>
<th>$P$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Log initial FM (kg)</td>
<td>14.29</td>
<td>1.2</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Log VO$_{2\text{max}}$ (L/min)</td>
<td>3.92</td>
<td>-2</td>
<td>.05</td>
</tr>
<tr>
<td>Ethnicity (1 = white, 2 = black)</td>
<td>4.84</td>
<td>-2.7</td>
<td>.03</td>
</tr>
<tr>
<td>TEE (kcal/d)</td>
<td>.13</td>
<td>0</td>
<td>.72</td>
</tr>
<tr>
<td>AEE (kcal/d)</td>
<td>.11</td>
<td>0</td>
<td>.74</td>
</tr>
<tr>
<td>Log REE (kcal/d)</td>
<td>.07</td>
<td>1.69</td>
<td>.79</td>
</tr>
<tr>
<td>Log initial LTM (kg)</td>
<td>2.76</td>
<td>2.69</td>
<td>.1</td>
</tr>
<tr>
<td>Age (y)</td>
<td>3.06</td>
<td>-0.9</td>
<td>.08</td>
</tr>
<tr>
<td>Tanner stage (1–5)</td>
<td>2.81</td>
<td>-3.7</td>
<td>.1</td>
</tr>
</tbody>
</table>

Overall model $r^2 = .277$.

* The rate of increasing adiposity (kg fat/ kg lean) is the dependent variable.
inversely related to increasing adiposity in white or black children.

Measurements of Energy Expenditure (REE, AEE, and TEE)

In this cohort, REE was not found to influence the rate of increasing adiposity in black or white children. This supports our previous findings, where we found no relationship between REE and future weight gain in a different cohort of white children living in Burlington, Vermont, in addition to those of Roberts et al and Stunkard et al in white infants. The only study which has observed a significant relationship between REE and weight gain was in Pima Indian adults, which differ in both age and ethnicity to our study group and those that have also failed to observe a significant relationship. In this study, children with low REE were not predisposed to gaining more fat than were those with high REE.

In addition to calculating REE, oxygen consumption and carbon dioxide production was used to calculate the respiratory quotient (RQ). However, as with REE, there was no relationship between RQ and increasing adiposity (P = .957), which seems to indicate that the level of fasting fat or carbohydrate oxidation does not play a role in the increase in adiposity in this cohort of children, in contrast to studies in Pima Indians.

There was no relationship between AEE or TEE and increasing adiposity in both whites and blacks, as we have previously found in a different cohort of white children. In some other studies, TEE has previously been found to influence weight gain in both white infants and Pima-Indian adults, in that those subjects with low TEE gained more weight than those with high TEE. It is important to note that low levels of energy expenditure can only lead to an increase in adiposity if accompanied by an energy intake that exceeds expenditure. In a previous cross-sectional study of prepubertal children, we found that FM was inversely related to hours per week of physical activity but not to energy expended in physical activity, indicating that time spent being physically active, and not necessarily the amount of energy expended (AEE) may be more important in the regulation of body fat.

All 3 components of energy expenditure were positively related to increasing adiposity before they were adjusted for initial body composition. This was attributable to the heavier children having higher energy expenditures and also having the greatest increase in adiposity. Once initial body composition was included in the analyses, the positive relationships disappeared.

**VO\textsubscript{2\textmax}**

Aerobic fitness measured by VO\textsubscript{2\textmax} on a treadmill was inversely related to increasing adiposity (Fig 2). There was no significant interaction between ethnicity and VO\textsubscript{2\textmax} which indicates that the relationship between VO\textsubscript{2\textmax} and increasing adiposity was the same for both white and black children.

From the relationship between adjusted VO\textsubscript{2\textmax} and the rate of increasing adiposity, an increase in VO\textsubscript{2\textmax} of .1 L/min would result in a decrease of .081 kg of fat per kg of lean mass. The potential results of this increase in fitness on the change in body composition of this cohort are shown in Table 3. In this study the children gained an average of 10 kg of lean mass and 4 kg of FM over an average of 3.7 years. An increase in baseline VO\textsubscript{2\textmax} of just .1 L/minute (8%) at the start of the study would have resulted in a reduced rate of increasing adiposity (.32 vs .4), .8 kg less fat gained and a decrease of 1.3% in body fat over the course of the study. The resulting annual decrease in adiposity would be .22 kg per year, which is nearly identical to the secular increase in weight observed by Freedman et al of .2 kg per year. Our data seem to suggest that an 8% increase in aerobic fitness (measured by VO\textsubscript{2\textmax}) would be sufficient to offset this secular increase of .2 kg per year.

This is one of the first articles to provide strong evidence in favor of reduced levels of physical activity as a cause of obesity using a longitudinal study

### Table 3. Comparison of the Average Initial (8 Years of Age) and Final (11 Years of Age) Body Composition of the Children (\(n = 95\))

<table>
<thead>
<tr>
<th>Results With Average VO\textsubscript{2\textmax} (1.22 L/Minute)</th>
<th>Increase VO\textsubscript{2\textmax} by .1 to 1.32 L/Minute</th>
</tr>
</thead>
<tbody>
<tr>
<td>8 Years of Age</td>
<td>11 Years of Age</td>
</tr>
<tr>
<td>Lean mass (kg)</td>
<td>20.6</td>
</tr>
<tr>
<td>FM (kg)</td>
<td>9.5</td>
</tr>
<tr>
<td>Total mass (including bone; kg)</td>
<td>31.3</td>
</tr>
<tr>
<td>Percent body fat</td>
<td>30.4</td>
</tr>
</tbody>
</table>

* The average VO\textsubscript{2\textmax} at 8 years of age and the predicted final body composition were given an increase of .1 L/minute in VO\textsubscript{2\textmax} at the start of the study.
design in a reasonable sample size and using advanced measurement techniques. We believe that VO2max serves as a strong proxy indicator of habitual levels of moderate to vigorous physical activity, because this level of activity results in greater changes in VO2max than do low intensity activities.34,35 VO2max is a more robust and less variable measure of physical activity than AEE for several reasons. First, VO2max is less variable over time (CV < 10%; unpublished data), whereas AEE can vary greatly from day-to-day and week-to-week in the order of up to 60% (unpublished data). AEE is far more variable because of inherent physiologic variation and also because the AEE value is an estimate that is generated from the mathematical difference of 2 numbers (TEE – RER). This computation results in a great deal of error propagation, as we have previously discussed.34 Second, the influence of AEE on increased adiposity is confounded by the positive loading effect of FM on AEE attributable to the fact that greater FM increases the energy cost of movement and AEE.36 This may be why we observe a positive relationship between AEE and increased adiposity. In contrast, VO2max is completely independent of all measures of adiposity,37 and, therefore, may be a cleaner measure to examine changes in adiposity. Finally, VO2max and AEE may represent distinct aspects of physical activity, which have different impacts on susceptibility to weight gain. This seems likely given the lack of relationship between AEE and VO2max after adjusting for fat-free mass.

Our findings in terms of susceptibility to obesity in black children are also significant. Our results suggest that the impact of differences in baseline VO2max on subsequent increased adiposity is similar in black and white children. However, as we20 and others21 have shown, VO2max is significantly lower in black children, independent of body composition. Thus, a lower VO2max in black children at baseline would explain the greater subsequent adiposity gain over time and the higher prevalence of obesity in this group in general.

CONCLUSION

Our data provide strong evidence that reduced physical activity, expressed as lower aerobic fitness, results in greater adiposity gain in growing prepubertal children. This finding contrasts with the lack of an inverse relationship between any component of energy expenditure and subsequent fat gain, as we have now demonstrated in 2 independent studies.3,11 Previous studies that have found lower energy expenditure in blacks or other subgroups of the population at greater risk of obesity should, therefore, be interpreted with caution, because they do not prove that reduced energy expenditure results in increased adiposity. These findings emphasize the importance of increasing or maintaining aerobic fitness as an intervention for preventing the development of obesity in children. Moderate to vigorous physical activity should be encouraged both within families and at schools. In addition to being protective against fat gain, aerobic fitness may also reduce risk for CVD and diabetes. Additional longitudinal studies are needed to assess if high levels of aerobic fitness are maintained throughout childhood, and if not, then examine the subsequent effect of reduced fitness on body composition.

ACKNOWLEDGMENTS

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