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Physical Fitness, Activity, and Insulin Dynamics in Early Pubertal Children

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Abstract

The objectives of this study were to identify the independent effect of physical activity and fitness on insulin dynamics in a cohort of European-, African-, and Hispanic-American children ($n = 215$) aged 7–12 years and to determine if racial/ethnic differences in insulin dynamics could be statistically explained by racial/ethnic differences in physical activity or fitness. An intravenous glucose tolerance test and minimal modeling were used to derive the insulin sensitivity index (SI) and acute insulin response to glucose (AIRg). Fitness was assessed as $\text{VO}_2\text{-170}$ and physical activity by accelerometer. Multiple regression models were tested for contributions of fitness and physical activity to SI and AIRg. Fitness was a stronger predictor of SI and AIRg than physical activity regardless of ethnicity; racial/ethnic differences in insulin dynamics were not accounted for by differences in fitness and/or physical activity.

Although estimates suggest that approximately 16–33% of the general pediatric population is overweight or obese (12), the incidence of becoming overweight at any age during childhood and adolescence is approximately twice as high among African- and Hispanic Americans when compared with European Americans (33). Inherent racial/ethnic differences in the physiology of insulin action in combination with an obesogenic environment may put African- and Hispanic-American children at greater risk for the development of type 2 diabetes than their European American counterparts. We, as well as others, have previously reported racial/ethnic differences in insulin dynamics among children (14,15). Newly diagnosed type 2 diabetes as well as subclinical features of type 2 diabetes (i.e., decreased insulin sensitivity, hyperinsulinemia) in children have drastically increased and have become an emerging public health issue. In addition, studies have indicated that differences in insulin response and action may track into adulthood (6,28). The mechanism driving the differences in insulin-related outcomes is unclear. However, it has been suggested that regardless of race/ethnicity, increased physical activity and/or fitness has the potential to improve metabolic parameters including insulin response and action.

Research strongly suggests a major role of physical activity and/or fitness in improving insulin response and action. For example, in adults, it has been well documented that fitness and physical activity are positively related to improvements in insulin sensitivity and inversely related to insulin secretion (23,36); however, the evidence in children is not quite as clear. Although some researchers have noted an association, others have not; thus limiting

evidence for the role of fitness among children. In a small study using a biethnic cohort, researchers from our group demonstrated that neither self-reported physical activity nor fitness explained racial differences in insulin sensitivity or acute insulin response to glucose (AIRg; 22). Ball et al. (4) showed that neither fitness (as measured by VO_2max) nor physical activity (hours of recreational activity per week) were independently related to measures of insulin sensitivity or secretion in Hispanic American children. On the other hand, in a recent study of primarily European American children, Allen and colleagues (1) demonstrated that cardiovascular fitness was a significant predictor of fasting insulin. Similarly, in a cohort of 589 Danish children, Brage and colleagues (8) demonstrated that physical activity correlated with indices of insulin resistance, and the variance in fasting insulin in the children was significantly associated with the variance in physical activity, independent of body mass index.

The association between insulin sensitivity and physical activity and/or fitness is most likely attributable to the enhanced insulin action involving muscle, adipose tissue, and key proteins of the insulin cascade (23) that results from exercise. Reports that African American children, in general, participate in less physical activity (17,35) and have a lower VO_2max than their European American (34) peers suggest that reduced levels of physical activity/fitness may decrease insulin sensitivity, increase fasting insulin, and, consequently, contribute to racial/ethnic differences in insulin response and action. African Americans and Hispanic Americans have lower insulin sensitivity and greater acute insulin response than age- and BMI-matched European Americans (16). It is important to note that there is a distinction between physical activity and fitness; while physical activity is dynamic and varies on a daily basis, fitness remains relatively static and takes time (and training) to change. Differential measurement precision, partially related to precision of measurements and partially due to acute day-to-day changes also may influence the ability to illustrate relationships with metabolic outcomes.

The purpose of this study was twofold: to investigate, in a multiethnic cohort of early pubertal children, (1) the effect of cardiovascular fitness and physical activity (independently) on insulin dynamics in the entire sample and (2) whether race/ethnicity modifies the association between physical activity and fitness with insulin dynamics.

Methods

Participants

A total of 215 children self-identified as African American ($n = 68$), European American ($n = 92$), or Hispanic American ($n = 55$) age 7–12 years were recruited from the Birmingham, Alabama area through advertisements, community health fairs, school presentations and word-of-mouth. The children were pubertal stage ≤ 3 as assessed by a pediatrician according to the criteria of Marshall and Tanner (24), and had no medical diagnosis or medications known to affect body composition or contraindicated for study participation. Before participating in the study, the nature, purpose, and possible risks of the study were carefully explained to the parents and children. The children and parents provided informed consent to the protocol, which was approved by the Institutional Review Board for human subjects at the University of Alabama at Birmingham (UAB). All measurements were performed at the General Clinical Research Center (GCRC) and the Department of Nutrition Sciences at the UAB between 2005 and 2007.

Protocol

Participants completed two testing sessions. In the first session, pubertal status, body composition, and fitness level were assessed. At the second session, participants were

admitted to the GCRC in the late afternoon for an overnight visit. All participants consumed the same meal and snack foods. After 2000h, only water and/or noncaloric decaffeinated beverages were permitted until after morning testing. After the overnight fast, resting energy expenditure and an insulin-modified, frequently sampled intravenous glucose tolerance test (IVGTT) were performed.

Assessment of Body Composition

Children arrived at the Department of Nutrition Sciences after a minimum 3 hr fast for the first testing session. Body composition (total body fat mass and non-bone lean tissue mass) was measured by dual-energy x-ray absorptiometry (DXA) using a GE Lunar Prodigy densitometer (GE LUNAR Radiation Corp., Madison, WI) located in the Department of Nutrition Sciences at UAB. Subjects were scanned in light clothing while lying flat on their backs with arms at their sides. DXA scans were performed and analyzed. DXA has been found to be highly reliable for body composition assessment in children. In our laboratory, the coefficient of variation (c.v.) for repeated measures of total body fat mass was 6.55%.

Intravenous Glucose Tolerance Test (IVGTT)

Following the overnight fast, a topical anesthetic (Emla cream, AstraZeneca, Wilmington, DE) was applied to the antecubital space of both arms, and flexible intravenous catheters were placed in both arms. At time zero, glucose (25% dextrose; 11.4 g/m²) was administered intravenously. Blood samples (2 mL) were collected at the following times relative to glucose administration at 0 min: -15, -5, -1, 2, 3, 4, 5, 6, 8, 10, 14, 19, 22, 25, 30, 40, 50, 70, 100, 140, and 180 min. Human specific insulin (HI-14k; 0.02 units/kg) was infused intravenously for 5 min at 20–25 min. The acute insulin response to glucose (AIRg), an approximation of first phase insulin secretion, was calculated as the incremental area under the curve for insulin during the first 10 min after glucose injection using trapezoidal methodology (25). Blood draws obtained at times -15, -5 and -1 were pooled and fasting insulin was assessed. Glucose and insulin values were entered into the MINMOD computer program for determination of the insulin sensitivity index (SI) as described elsewhere (26).

Assay of Glucose and Insulin

Glucose was measured in 10 µl sera using an Ektachem DT System (Johnson and Johnson Clinical Diagnostics). The intraassay c.v. for this analysis was 0.61% and the mean interassay c.v. was 1.45%. Insulin was assayed in duplicate 100 µl aliquots using double-antibody radioimmunoassay (Linco Research Inc., St. Charles, MO). This assay has a sensitivity of 3.35 µIU/ml in the Core Laboratory, and a mean intra- and interassay c.v. of 3.49% and 5.57%, respectively. Commercial quality control sera of low, medium, and high insulin concentration (Lymphochek, Bio-Rad Laboratories, Inc., Anaheim, CA) were included in every assay to monitor variation over time.

Physical Fitness

At the first testing session, VO₂-170 was determined by indirect calorimetry on a treadmill, as described by Gutin et al. (18). The first 4 min of the test, as a mode of standardization, was at 2.5 miles per hour with no incline. Measurements were taken following the standardization period (at 4 min) and served as baseline for heart rate, VO₂, and VCO₂; participants then began exercising at 3 mph. The incline was subsequently increased by 2% every 2 min. Heart rate was measured with the Polar Vantage XL HR monitor (Polar Beat, Port Washington, NY). Based on this protocol, a measure of fitness was established for each participant. For fitness, volumes of O₂ and CO₂ were measured continuously using open circuit spirometry until recording the VO₂ level at a heart rate of 170 beats/min. Data were analyzed with a Max-II metabolic testing system (PHYSIO-DYNE, Quogue, NY). VO₂-170

was derived by regression of heart rate-VO₂ relationship, solved for heart rate equal to 170 beats per minute. When we compared a small sample of data using both methods, results did not differ.

Fitness variables were expressed as follows: (a) VO₂-170 in mL/min (VO₂-170); (b) VO₂-170 divided by kilograms body weight (VO₂-170/kg); (c) VO₂-170 divided by kilograms lean mass (VO₂-170/kg lean).

Physical Activity by Accelerometer

The MTI Actigraph accelerometer (Actigraph GT1M—Standard Model 198–0100–02, ActiGraph LLC, Pensacola, FL) was used to measure physical activity levels and patterns for 7 days before participant's inpatient visit at the GCRC. Epoch length was set at one minute and data expressed as counts per minute (counts min⁻¹). Children wore the monitor on an elastic belt at the waist. It was assumed that the monitor was worn at all times except during sleeping, bathing, and swimming. Actigraph monitors have previously been demonstrated to exhibit a high degree of interinstrument reliability (8). Daily counts per minute were summed. Minutes spent in light, moderate, hard or very hard activity were determined using the following cut-off values in metabolic equivalents (METs): Light: ≤ 2.99; Moderate: 3–5.9; Hard: 6–8.99; Very hard: ≥9. Sedentary time (0 METs) was also computed.

Socioeconomic Status (SES)

Socioeconomic status (SES) was measured with the Hollingshead 4-factor index of social class (13), which combines the educational attainment and occupational prestige for the number of working parents in the child's family. Scores ranged from 8 to 66, with the higher score indicating higher theoretical social status.

Statistical Analyses

Racial/ethnic differences in descriptive statistics were examined using ANOVA with Duncan's posthoc analysis.

To develop the statistical models to test our hypotheses, exploratory stepwise regression analyses were conducted to evaluate, from a series of covariates, those that would contribute the most to each outcome variable. The cutoff point for inclusion of the model was a *p*-value < 0.10. These results were used to develop and test all regression models. Racial/ethnic differences in insulin related outcomes, were performed using ANCOVA adjusting for age, pubertal stage, sex, body composition, and SES. Three multiple regression models (*n* = 215) were used to test for the contributions of the independent variables (fitness and physical activity) to the dependent variables (fasting insulin, SI, and AIRg). Age, sex, pubertal stage, and body composition are potential confounding variables and were adjusted for in each model. In addition, models for AIRg also included SI. Variables were added systematically to test the research question. The first set of models evaluated the effect of fitness and physical activity on insulin dynamics using the entire sample. In the second set of models, the effect of ethnicity was evaluated, adding an interaction term. Significant interaction terms justified stratification according to racial/ethnic group for a third set of models.

For models in which fitness was tested as an independent contributor, fitness parameters (VO₂-170, VO₂-170/kg, VO₂-170/kg lean) were independently evaluated as predictors. Physical activity measures, calculated from accelerometer counts (total daily physical activity, total daily moderate and hard activity, and sedentary activity) were independently evaluated as predictors in separate models.

To conform to the assumptions of linear regression, all statistical models were evaluated for residual normality, constant variance and outliers, and logarithmic transformations were performed when appropriate. Those residuals that deviated above and below three standard deviations were removed in the final models. Models analyzed with and without outliers. In particular, there was only one outlier removed. Removal of outlier did not affect determination of significance. All data were analyzed using SAS 9.1 software.

Results

Descriptives

There were no differences in age, sex, weight, or height between the racial/ethnic groups. However, as illustrated in Table 1, European Americans were in a higher grade in school and reported a higher SES compared with African- and Hispanic Americans. African Americans were in a higher grade in school and reported higher SES than Hispanic Americans. African Americans had greater lean mass and were assessed at a higher pubertal stage than both European- and Hispanic Americans. Hispanic Americans had a greater BMI and adiposity than both European- and African Americans.

Table 2 presents a comparison by ethnic group for fitness and physical activity measures. African Americans had a lower fitness than European- and Hispanic Americans regardless of using absolute, body weight or lean mass adjusted VO_2 -170. Hispanic Americans were the most fit as assessed by VO_2 -170, albeit differences were significantly greater than European Americans only when fitness relative to lean mass was used. There were no differences in total physical activity or sedentary activity between the racial/ethnic groups. However, when the sum of moderate and hard activity (light activity excluded) was analyzed, European Americans engaged in more moderate and hard physical activity than Hispanic Americans, while minutes per day moderate and hard activity was not different between European- and African Americans or African and Hispanic Americans.

Table 3 illustrates the racial/ethnic differences in insulin dynamics after adjusting for sex, SES, body composition, pubertal stage, and age. European Americans had greater SI than both Hispanic- and African Americans. African Americans had the greatest AIRg. There were no differences in fasting insulin levels between the three groups. When fitness and physical activity measures were added, the racial/ethnic differences persisted; however, the differences were attenuated. In fact, the difference in AIRg between Hispanic- and African American disappeared with the addition of either physical activity or fitness.

Fitness and Physical Activity as Predictors of Insulin Response and Action

Using multiple linear regression analyses, several models were tested to determine the independent contribution of fitness and physical activity on the dependent insulin-related parameters. All measures of fitness were significant predictors of both SI and AIRg in the total sample. The race/ethnicity by fitness interaction terms were significant for SI in all models, for AIRg in models with absolute VO_2 -170, and VO_2 -170 adjusted by body weight, and for fasting insulin when fitness relative to lean mass was analyzed (Table 4). However, the results were not significant when participants were analyzed according to race. Total physical activity was not a significant predictor of any of the insulin-related parameters nor was the interaction term significant. However, the interaction term with the sum of moderate and hard physical activity and race/ethnicity was significant. When analyzed by race, the sum of moderate and hard activity was a significant predictor of SI in African Americans, such that increased moderate and hard physical activity was associated with greater SI. Sedentary behavior did not contribute to differences in insulin dynamics, nor was there an interaction between race/ethnicity and time spent in sedentary behavior.

The results remained significant after controlling for the effect of multiple comparisons via permutation tests (1,000 simulations) to generate empirical *p*-values under the null hypothesis of no association between insulin dynamics and physical activity and/or fitness (data not shown).

Discussion

Physical activity and fitness have been directly linked to physiological, metabolic, and gene regulatory responses/adaptations although the exact mechanism by which activity and/or fitness induce changes in insulin response and action remain unclear (19). Nevertheless, in adults numerous reports of improvements in insulin response and action in association with fitness and/or physical activity are abundant in the literature. This study sought to evaluate the contribution of fitness and physical activity to racial/ethnic differences in insulin dynamics and test the hypotheses that racial/ethnic differences in insulin dynamics would be statistically explained by differences in fitness and/or physical activity in a multiethnic cohort of children aged 7–12 years. Fitness was a stronger predictor of SI and AIRg than physical activity; however racial/ethnic differences in insulin dynamics were not accounted for by differences in fitness or physical activity.

This study adds to the body of scientific evidence indicating that insulin response and action differs between racial/ethnic groups, with Hispanic Americans (14) and African Americans (3,16) having lower SI and higher AIRg than their European American counterparts. This study also demonstrated that, just as in previous studies (22,30), African Americans present with lower fitness compared with their European American counterparts. Interestingly, there was no difference in any physical activity measure between European Americans and African Americans after controlling covariates. Several previous studies have suggested that European Americans engage in more daily physical activity than African Americans (17,30,35), but results may vary (22). Our results indicate that the differences in fitness between European- and African Americans, were not mediated by differences in physical activity, anthropometry, body composition or socioeconomic status.

Research from our laboratory in African Americans adults suggests racial/ethnic differences in fitness may be at least partially attributed to racial/ethnic differences in muscle oxidative capacity, oxygen delivery capabilities, hemoglobin, and/or African genetic admixture (20,27) and lower muscle mitochondrial function among African Americans may in part explain lower SI (32). Others have demonstrated an association between Spanish ancestry and VO_2max (9). In addition, work from our laboratory has demonstrated that individuals having greater African genetic admixture had a lower SI and a higher fasting insulin concentration (15).

Scientific evidence documenting the differences in fitness between European American and Hispanic American youth is limited and have been met with equivocal findings (5,31). Although our results suggest that Hispanic Americans are expending less energy than their European- and African American counterparts, similar to the study by Beet and colleagues, we found little difference in fitness between European Americans and Hispanic Americans. Our observations indicate that although have higher levels of adiposity, in our normal weight Hispanic American youth, neither body composition nor daily physical activity had an independent effect on fitness. Although fitness and physical activity tend to be highly correlated, higher $\text{VO}_2\text{-170}$, but lower physical activity coupled with greater adiposity in Hispanic Americans poses a conundrum. We have no explanation as to whether there are genetic or physiologic factors that may have confounded our results, if there are metabolic implications associated or if differential precision measurement may play a role and further investigation may be warranted.

We found that fitness measures were related to SI and AIRg in the total sample after controlling for covariates. This finding is consistent with previous reports indicating that fitness is positively associated with SI and negatively associated with postchallenge insulin concentration (1,6). When analyses were conducted within each ethnic group, no associations were observed among fitness and insulin variables. Although we observed racial/ethnic differences in insulin dynamics and fitness, our results indicate that in this cohort the contribution of fitness to insulin dynamics exists regardless of race/ethnicity.

It has been widely suggested that physical activity is positively associated with insulin sensitivity and inversely related to insulin response (2,7,10,11) suggesting that an active lifestyle may be beneficial with respect to diabetes risk. Based on the results of our study, the effect of physical activity on insulin dynamics was not strong enough to be detected in this sample. This finding is in contrast to previous results suggesting that physical activity is positively associated with insulin sensitivity and negatively associated with AIRg in children (22,29) and adults (19,23).

When activity was analyzed by intensity, it appeared as if the metabolic benefit from increased moderate to hard daily physical activity may exist only among African American children. African Americans who engaged in more minutes per day of moderate and hard physical activity had greater insulin sensitivity. This is in accordance with findings of the College of Sports Medicine indicating that for physical activity to be effective, the intensity should be at least moderate (21); however, longitudinal studies are needed to determine if adopting a physically active lifestyle contributes to decreased overweight/obesity.

We cannot fully explain why a similar pattern was not demonstrated in European- or Hispanic Americans. However, SI is consistently reported to be lower among African Americans vs. European- and Hispanic Americans, independent of BMI and adiposity. Thus, the threshold for improvement in SI via increased physical activity may be lower among African Americans. In addition, the contribution of regular physical activity to increased oxygen consumption may differ with ethnic background. Maximal oxygen consumption has consistently been found to be lower among African Americans vs European Americans (20,27,32), suggesting a strong genetic component. It is possible that the large genetic component of oxygen consumption among African Americans may override the flexible component that reflects regular physical activity. Thus, it may be difficult to observe relationships between “fitness,” as reflected in oxygen consumption, and other metabolic variables. As such, it is plausible that accelerometry may be a better reflection of routine physical activity, and accompanying skeletal muscle contraction, than oxygen consumption among African Americans.

Although we used robust measures to determine the insulin sensitivity index and body composition and had direct measures of habitual physical activity from accelerometry, this was a cross-sectional study with a relatively small sample size. Measures of submaximal oxygen consumption (VO_2 , VO_2 -170) are often used as an accurate measure of cardiovascular fitness, but have been criticized as being not as indicative of fitness as use of VO_2 max. In the interest of achieving complete data on as many subjects as possible, we chose to use VO_2 -170, in our population of children due to the difficulty in getting this age group to exercise to their maximum oxygen consumption. Unlike self-reported physical activity, use of an accelerometer is an objective measure of recent physical activity patterns. However, the device used is limited in its ability to capture output while engaging in some activities (e.g., swimming; 8). Such inaccuracies could potentially bias the relationship between insulin response and action and measures of physical activity. We attempted to capture regular daily activity as much as possible by using an average over 7 days. We did not estimate the reproducibility of fitness, but assumed it would vary minimally when

measured over a relatively short period of time, which is comparable with the reproducibility of regular daily activity. Therefore, differential measurement precision is unlikely to explain the apparent independence and greater strength of the association between fitness than physical activity. The sample size when stratified according to ethnicity was limited. Although the ethnicity by fitness and the ethnicity by physical activity interaction did not yield highly significant associations, we do not rule out the possibility that differences according to ethnicity may have been found using a larger sample size.

It is unclear whether higher AIRg and lower SI among Hispanic- and African Americans are genetically or environmentally determined or whether these differences in what are considered to be subclinical features of type 2 diabetes lead to differences in the development of clinical type 2 diabetes. In our sample, fitness was a stronger predictor of insulin dynamics than physical activity regardless of race/ethnicity. Although increased engagement in physical activity in this cohort was limited, it is plausible that improvements in fitness and increased engagement in physical activity may respond to and influence insulin response and action differently in population of diverse racial/ethnic backgrounds. Longitudinal studies are needed to determine if modification of lifestyle habits could improve insulin dynamics and reduce risk factors for overweight/obesity and type 2 diabetes.

List of Abbreviations

AIRg	Acute Insulin Response to Glucose
IVGTT	Intravenous Glucose Tolerance Test
SES	Socioeconomic Status
SI	Insulin Sensitivity Index
VO ₂ -170	Volume of oxygen used at heart rate of 170 beats per minute

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Table 1Descriptive Statistics Compared Using ANOVA (Mean $\pm$ SD)

	European Americans (<i>n</i> = 92)	Hispanic Americans (<i>n</i> = 55)	African Americans (<i>n</i> = 68)
Age (years)	9.7 $\pm$ 1.7	9.3 $\pm$ 0.21	9.7 $\pm$ 1.4
Sex (% female)	40.4	54.1	45.0
Grade in school	6.2 ^a $\pm$ 0.8	3.7 ^b $\pm$ 1.8	5.2 ^c $\pm$ 1.1
Pubertal stage	1 ^a $\pm$ 0.6	1 ^a $\pm$ 0.7	2 ^b $\pm$ 0.9
SES	49.5 ^a $\pm$ 9.6	26.1 ^b $\pm$ 12.2	38.3 ^c $\pm$ 10.6
BMI	17.9 ^a $\pm$ 2.6	19.4 ^b $\pm$ 2.7	18.5 ^a $\pm$ 3.3
BMI percentile	60.2 ^a $\pm$ 26.6	78.1 ^b $\pm$ 18.6	62.9 ^a $\pm$ 27.4
Weight (kg)	35.6 $\pm$ 8.6	37.1 $\pm$ 9.6	37.6 $\pm$ 10.1
Height (cm)	140.2 $\pm$ 10.5	137.1 $\pm$ 10.8	141.7 $\pm$ 10.1
Lean mass (kg)	25.3 ^a $\pm$ 5.0	24.3 ^a $\pm$ 4.8	27.3 ^b $\pm$ 5.4
Fat mass (kg)	8.2 ^a $\pm$ 5.0	10.6 ^b $\pm$ 5.4	8.2 ^a $\pm$ 6.2
Percent fat	22.4 ^a $\pm$ 8.4	28.2 ^b $\pm$ 8.4	20.5 ^a $\pm$ 9.6

*Note.**a,b,c* superscripts indicate a similarities/differences between racial/ethnic group for descriptive variable (*p* < .05).

Table 2Comparison of Fitness and Physical Activity Measures by Racial/Ethnic Group by ANCOVA (Mean $\pm$ SEM)

	European Americans (<i>n</i> = 92)	Hispanic Americans (<i>n</i> = 55)	African Americans (<i>n</i> = 68)
VO ₂ -170 (mL/min)	1069.2 ^a $\pm$ 26.8	1136.2 ^a $\pm$ 37.3	969.6 ^b $\pm$ 29.1
VO ₂ -170/kg body weight (mL/min)	29.6 ^a $\pm$ 0.7	31.9 ^a $\pm$ 0.9	27.7 ^b $\pm$ 0.7
VO ₂ -170/kg lean mass (mL/min)	40.9 ^a $\pm$ 0.7	46.6 ^b $\pm$ 1.0	36.1 ^c $\pm$ 0.8
Total physical activity (min/d)	291.7 $\pm$ 6.1	276.4 $\pm$ 9.1	282.5 $\pm$ 7.5
Moderate and hard activity (min/d)	282.5 ^a $\pm$ 1.5	19.6 ^b $\pm$ 2.3	23.2 ^{ab} $\pm$ 1.9
Sedentary behavior (min/d)	163.0 $\pm$ 2.8	160.7 $\pm$ 4.5	19.6 $\pm$ 2.3

Note.

^{a,b,c} superscripts indicate differences between ethnic groups ($p < .05$). All measures adjusted for sex, SES, age, pubertal stage. VO₂-170 also adjusted for total fat and lean mass.

Table 3

Racial/Ethnic Differences Attenuated, but Persisted After Fitness and Physical Activity Are Taken Into Account (Mean $\pm$ SEM)

	European Americans (<i>n</i> = 92)	Hispanic Americans (<i>n</i> = 55)	African Americans (<i>n</i> = 68)	<i>p</i> -values
SI* ($\times 10^{-4} \text{ min}^{-1}/(\mu\text{IU}/\text{ml})$)	6.9 ^a $\pm$ 0.4	5.6 ^b $\pm$ 0.5	4.7 ^b $\pm$ 0.4	EA:AA <i>p</i> = .001
AIRg** ($\mu\text{IU}/\text{ml} \times 10 \text{ min}$)	701.2 ^a $\pm$ 67.2	795.1 ^a $\pm$ 70.4	1154.6 ^b $\pm$ 70.4	EA:AA <i>p</i> < .001 HA:AA <i>p</i> = .008
Fasting Insulin [#] ($\mu\text{IU}/\text{ml}$)	11.9 $\pm$ 0.5	12.5 $\pm$ 0.8	13.1 $\pm$ 0.6	
VO ₂ -170 added				
SI* ($\times 10^{-4} \text{ min}^{-1}/(\mu\text{IU}/\text{ml})$)	6.4 ^a $\pm$ 0.36	5.5 ^b $\pm$ 0.39	4.5 ^b $\pm$ 0.29	EA:AA <i>p</i> = .05
AIRg** ($\mu\text{IU}/\text{ml} \times 10 \text{ min}$)	734.1 ^a $\pm$ 95.5	978.9 ^a $\pm$ 90.6	1113.1 ^b $\pm$ 71.3	EA:AA <i>p</i> = .01
Fasting Insulin [#] ($\mu\text{IU}/\text{ml}$)	12.1 $\pm$ 0.6	12.9 $\pm$ 0.9	12.3 $\pm$ 0.8	
Total Activity added				
SI* ($\times 10^{-4} \text{ min}^{-1}/(\mu\text{IU}/\text{ml})$)	6.9 ^a $\pm$ 0.4	5.7 ^a $\pm$ 0.6	5.1 ^b $\pm$ 0.5	EA:AA <i>p</i> = .02
AIRg** ($\mu\text{IU}/\text{ml} \times 10 \text{ min}$)	690.1 ^a $\pm$ 78.2	681.3 ^a $\pm$ 93.9	1139.5 ^b $\pm$ 78.2	EA:AA <i>p</i> < .001
Fasting Insulin [#] ($\mu\text{IU}/\text{ml}$)	12.1 $\pm$ 0.6	11.8 $\pm$ 0.9	12.7 $\pm$ 0.7	

Table 4

Multiple Regression Analyses Indicated That Fitness, but Not Physical Activity Contributes to Insulin Dynamics in a Multiethnic Sample of Early Pubertal Children

	Fasting Insulin		SI		AIRg	
	β	p-value	β	p-value	β	p-value
VO₂-I70						
Race* fitness interaction	-0.163	0.064	0.277	0.003	-0.208	0.026
VO₂I70/kg						
Race* fitness interaction	-0.114	0.085	0.243	0.004	-0.113	0.104
		0.201		0.019		0.041
VO₂-I70/ kg lean tissue mass						
Race* fitness interaction	-0.111	0.071	0.186	0.004	-0.140	0.023
		0.001		0.003		0.758
Total PA (min/day)						
Race* activity interaction	0.086	0.146	0.056	0.368	-0.093	0.170
		0.172		0.201		0.179
Moderate + hard PA (min/day)						
Race* activity interaction	0.078	0.229	0.118	0.0754	0.089	0.237
		0.735		0.019		0.115
Sedentary activity (min/day)						
Race* activity interaction	-0.036	0.592	-0.022	0.754	0.102	0.191
		0.468		0.310		0.298

β = Standardized parameter estimate

Note. All models adjusted for sex, pubertal stage/age, SES and body composition. AIRg models also adjusted for SI.