

Association of Physical Activity and Body Composition With Insulin Sensitivity in a Community Sample of Adolescents

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Objective: To examine how body composition and physical activity are related to insulin sensitivity and secretion in adolescents.

Design: Cross-sectional.

Setting: Baltimore, Maryland.

Participants: Fifty-six healthy adolescents (34 boys and 22 girls; mean [SD] age, 13.3 [1.3] years; 95% were African American) who had been recruited at infancy from a health care clinic serving a low-income, urban community.

Main Exposures: Physical activity was measured for 5 to 7 days by a uniaxial accelerometer placed on the right ankle. Proportion of time spent in play-equivalent physical activity (PEPA) was defined as 1800 or more counts per minute. Body mass index (BMI) (calculated as weight in kilograms divided by height in meters squared) was converted to an age- and sex-specific z score.

Main Outcome Measures: Insulin sensitivity, insulin secretion, and disposition index calculated from a fasting oral glucose tolerance test.

Results: Thirty-nine percent of the adolescents had a BMI in the 85th percentile or higher; half of those were overweight (BMI \geq 95th percentile). Play-equivalent physical activity and BMI z score were not correlated. In multivariate analyses, BMI z score and time spent in PEPA together explained 21% of the variance in insulin sensitivity and 18% in insulin secretion. Independent of each other, high BMI z score and low proportion of PEPA were significantly associated with low insulin sensitivity (partial $r^2=0.14$ and 0.10 , respectively) and high insulin secretion (partial $r^2=0.10$ and 0.10 , respectively), but not with disposition index.

Conclusions: In a cohort of urban, predominantly African American adolescents, both body composition and physical activity were independently associated with insulin sensitivity. At this point, insulin secretion is appropriately regulated.

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WITH THE INCREASING prevalence of overweight among children,¹ type 2 diabetes mellitus has become “an evolving epidemic in the young.”² Case studies from clinic-referred samples have documented that as many as 8% to 45% of the children with diabetes mellitus have type 2 diabetes.³⁻¹¹ The prevalence of type 2 diabetes is disproportionately high among African American individuals and other ethnic and racial minority groups.¹²

Little is known about the determinants of type 2 diabetes in children.² Although some studies suggest strong similarities between the pathogenesis in children and adults,¹³ others have failed to extend well-documented findings in adults to children.^{14,15}

The objective of the present investigation was to examine how adiposity, physical activity, heritability, and other factors relate to insulin sensitivity, insulin secretion, and disposition index (DI) (the arithmetic product of insulin sensitivity and secretion) in a community sample of urban, mostly African American adolescents. This report used accelerometry for objective assessment of physical activity.

METHODS

SUBJECTS

The subjects were 56 adolescents (34 boys and 22 girls) between 11 and 16 years of age (mean [SD] age, 13.3 [1.3] years) who were participating in a longitudinal investigation of child growth and development, having been enrolled as infants from a primary health care clinic

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servicing a low-income, urban community. All youths had been born at term (>37 weeks) with birth weight appropriate for gestational age and had no documented congenital or disabling conditions. Seventeen percent of the youths had experienced growth faltering in the first 2 years of life (weight for age or weight for height <fifth percentile based on age- and sex-adjusted growth charts), but by 6 years of age, all faltering children had experienced growth recovery, their growth parameters being higher than the fifth percentile.¹⁶ None of the youth had a chronic disease associated with an increased risk of type 2 diabetes and none had been referred for services related to overweight. Among ethnic and racial groups defined by the investigators with a write-in category, 95% of the adolescents identified themselves as African American and 5%, as white. This ethnic/racial information is reported because the prevalence of diabetes differs between ethnic and racial groups.¹⁷ Those who identified as white are included in the report despite their low number, making up a racially mixed but socioeconomically homogeneous sample.

The protocol was approved by the institutional review board of the University of Maryland. Signed consent was obtained from parents and signed assent, from youths. Parents and adolescents were compensated for transportation and participation.

PROCEDURES

Anthropometric and Sociodemographic Measures

The adolescents and parents participated in a baseline evaluation at our clinical laboratory. Body weight was measured to the nearest 0.1 lb and converted to kilograms; height was measured with a wall-mounted stadiometer to the nearest 0.1 cm. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. The BMI values were converted to *z* scores and percentiles based on the 2000 Centers for Disease Control and Prevention age- and sex-specific tables¹⁸ using algorithms obtained from and explained at the Centers for Disease Control and Prevention Web site.¹⁹ In brief, the *z* score for a given BMI was calculated from empirical age and sex LMS parameters where *M* is the median; *S*, the generalized coefficient of variation; and *L*, the power in the Box-Cox transformation. Subjects were divided into the following categories based on their age-adjusted, sex-specific BMI percentiles: normal weight (<85th percentile), at risk for overweight (\geq 85th percentile and <95th percentile), and overweight (\geq 95th percentile).

Parents completed questionnaires on demographic information and identified family members with diabetes. Using the poverty ratio equation provided by the US Census Bureau,²⁰ the family's poverty ratio was calculated and compared with the Poverty Index.

Physical Activity

At the conclusion of the baseline evaluation, a uniaxial accelerometer (Actiwatch; Mini Mitter Co Inc, Bend, Oregon) was placed on the right ankle with a nonremovable, reinforced hospital band. The accelerometer was worn next to the skin, under socks. The accelerometer summarized activity as "counts" per 1-minute epoch. Discarding data from the first and last days of wear, which were incomplete, data were available for 5 (*n*=3), 6 (*n*=27), or 7 consecutive days (*n*=26), respectively. Although the 5- and 6-day periods disproportionately represented weekends, during which the adolescents were less active—approximately 13% fewer daily counts on Saturdays and Sundays compared with nonweekend days—the resulting overall systematic underestimation in subjects wearing the accelerometers for 5 and 6 days was calculated to be a modest 1.6% and 0.8%, respectively. When the adolescents returned to the

clinic to have the accelerometers removed, data were extracted from the accelerometer's memory and processed with a program written in SPSS (SPSS Inc, Chicago, Illinois) as follows. Each hour with a mean activity of less than 55 counts/min was considered "sleep." The proportion of nonsleep time spent in play-equivalent physical activity (PEPA), defined as more than 1800 counts/min (see next paragraph), was calculated. In addition, we calculated mean daily activity counts as a measure of total activity.

The Actiwatch accelerometer worn on the knee has been validated as a measure of energy expenditure against indirect calorimetry in children.²¹ We placed the accelerometer on the right ankle, rather than the knee, because our experience indicated better acceptability. To identify an appropriate threshold for PEPA and to compare ankle and knee placement, we conducted a separate substudy with 25 participants (10 boys and 15 girls; mean [SD] age, 14.6 [1.7] years) who wore an Actiwatch accelerometer on the right knee and ankle during a 20-minute free-play session in a gymnasium furnished with age-appropriate exercise equipment and toys. The mean (SD) ankle counts per minute were 3003 (1358). All individual means were higher than 1800 counts/min (range, 1941-5924 counts/min), except 3 individuals who had mean values ranging from 255 to 670 counts/min and stood out by having spent the entire session riding stationary bicycles. Therefore, we chose 1800 counts/min as a threshold for PEPA. The ankle and knee placement counts were highly correlated ($r=0.94$; $P<.001$), knee counts amounting to approximately two-thirds of ankle counts, including in those who rode stationary bicycles. In summary, the methodological substudy showed that (1) the 1800 counts/min PEPA threshold consistently identified a wide range of free-play activities, with the exception of stationary bicycling, and (2) ankle counts during free play were highly correlated with knee counts, shown by others²⁰ to be correlated with energy expenditure.

Each accelerometer was used multiple times (median number of uses, 11). The accelerometers were delivered calibrated to the manufacturer's standards. For each accelerometer, we tested for subsequent calibration drift by (1) visually examining plots of mean daily activity counts (7-day mean for each subject) against recording start date and (2) testing, by multiple linear regression adjusting for age and sex, whether recording start date was a determinant of mean daily activity counts. We did not identify any drift.

Body Composition

When the youths returned to have the accelerometer removed, they participated in tests at the General Clinical Research Center, following a standardized protocol. First, a total-body dual energy x-ray absorptiometry (DEXA) scan was performed on a Hologic QDR 4500 W scanner (Hologic Inc, Bedford, Massachusetts).

Percentages of body fat and abdominal fat were calculated using software provided by the scanner manufacturer. Abdominal fat was defined as the percentage of fat in an abdominal region-of-interest rectangle. The horizontal sides of the rectangle were drawn at the upper edge of the body of the second lumbar vertebra and the lower edge of the body of the fourth lumbar vertebra, respectively. The vertical sides connected the lateral inferior borders of the rib cage and the iliac crest. The usefulness of abdominal region-of-interest analysis of DEXA in predicting intra-abdominal fat has been independently validated.^{22,23}

Oral Glucose Tolerance Test

Immediately following the DEXA scan, a fasting 2-hour oral glucose tolerance test (OGTT) was performed according to

American Diabetes Association guidelines observing the pediatric dosing regimen.²⁴ Determination of plasma glucose concentration was performed by the glucose oxidase method, using automated equipment. Plasma insulin concentration (in microunits per milliliter) was determined by an enzyme-linked immunosorbent assay method using commercial kits (Linco Research Inc, St Charles, Missouri). Impaired glucose tolerance was defined as a fasting plasma glucose level lower than 126 mg/dL and a 2-hour plasma glucose level of 140 mg/dL or higher and lower than 200 mg/dL.²⁴

Insulin sensitivity (Si) was calculated as per Matsuda and DeFronzo²⁵ as 10 000 divided by the square root of (fasting insulin level × fasting glucose level × mean OGTT insulin × mean OGTT glucose) where mean OGTT insulin and glucose are the mean levels at the 30-, 60-, 90-, and 120-minute points of the OGTT. The Matsuda and DeFronzo index has been validated in children against the hyperinsulinemic, euglycemic clamp.²⁶

Insulin secretion was quantified as the corrected insulin response (CIR) based on glucose and insulin levels at the 30-minute point of the OGTT (CIR₃₀), calculated as insulin₃₀ / (glucose₃₀ [glucose₃₀ × (glucose₃₀ - 70 mg/dL)]). This index has been validated against the acute insulin response to glucose, determined 3 to 5 minutes after the administration of a 25-g intravenous glucose bolus.²⁷ None of the subjects had glucose₃₀ values lower than 70 mg/dL. Si and CIR₃₀ will be reported without their units in this article.

The DI was calculated as Si × CIR₃₀.

DATA ANALYSIS

Comparisons of variables by BMI category were done using analysis of variance for continuous variables and χ^2 tests for categorical variables. Bivariate relationships among variables were examined using Pearson correlation. Multiple regression analysis was used to find the determinants of insulin sensitivity. Insulin sensitivity index and other nonnormally distributed variables were log transformed to approximate a normal distribution. With a sample size of 56, we had 81% power to detect a (true) effect size of $r=0.36$, as determined with Power and Precision software (Biostat, Englewood, New Jersey). Fewer than 5% of the data points were missing, primarily because of clerical or administrative error. Imputation by mean substitution within BMI category was used to replace missing values. With the exception of the earlier-mentioned uses of SPSS and Power and Precision, the analyses were performed using SAS 9.1 (SAS Institute, Cary, North Carolina).

RESULTS

GROUP DIFFERENCES

Table 1 shows physical characteristics by BMI category. Of all 56 youths, 23 had a family history of diabetes, among either parents (n=3), grandparents (n=18), or both (n=2). Being overweight was associated with having a history of diabetes in the parents ($P=.02$) or both the parents and the grandparents ($P=.02$), but not in the grandparents alone. Neither age, sex, time spent in PEPA, fasting glucose level, household income, poverty level, or history of failure to thrive differed by BMI category. Four subjects had impaired glucose tolerance. Three of these had a BMI higher than the 95th percentile. Thus, of the 11 adolescents in the overweight (≥ 95 th percentile) BMI category, 3 (27%) had impaired glucose tolerance. Boys and girls did not differ by BMI z score, but on average, boys had a lower per-

Table 1. Sample Characteristics by BMI Category^a

Characteristic	Mean (SD)		
	BMI < 85th Percentile (n = 34)	BMI \geq 85th and < 95th Percentile (n = 11)	BMI \geq 95th Percentile (n = 11)
Male sex, %	68	55	45
Age, y	13.3 (1.3)	13.6 (1.3)	12.8 (1.0)
Height, cm	158 (10)	162 (5)	159 (6)
Weight, kg	46.9 (9.2)	62.6 (6.3)	80.0 (18.0) ^b
BMI	18.5 (1.9)	24.0 (1.7)	31.2 (5.7) ^b
BMI z score	-0.25 (0.85)	1.30 (0.16)	2.14 (0.31) ^b
Total body fat, %	15.2 (4.8)	28.9 (4.3)	36.4 (4.1) ^b
Abdominal fat, %	11.4 (4.6)	25.9 (6.2)	35.8 (5.3) ^b
Time in PEPA, of nonsleep time	6.6 (2.7)	5.6 (2.4)	6.5 (2.6)
Fasting plasma glucose level, mg/dL	86.6 (5.1)	86.4 (6.2)	85.9 (4.6)
Fasting plasma insulin level, μ U/mL ^c	6.4 (2.2)	7.3 (2.3)	13.5 (1.7) ^d
Glucose level at 120-min point of OGTT, mg/dL	108.2 (17.3)	104.0 (21.0)	116.5 (24.8)
Insulin sensitivity ^{c,e}	5.6 (1.8)	5.1 (2.0)	3.1 (2.0) ^d
Insulin secretion ^{c,e}	0.8 (2.7)	1.3 (2.8)	1.1 (2.7)
Disposition index ^{c,e}	4.7 (3.1)	6.4 (2.7)	3.4 (1.8)

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); OGTT, oral glucose tolerance test; PEPA, play-equivalent physical activity.

^a Variables with no notation did not differ significantly between groups.

^b $P < .01$ for all 3 comparisons.

^c Geometric means.

^d $P < .01$ for BMI in the 95th percentile or higher vs BMI lower than the 85th percentile.

^e See "Oral Glucose Tolerance Test" subsection in the "Methods" section for explanation.

centage of body fat (mean [SD], 19.1% [10.0%] vs 26.6% [8.2%]) and abdominal fat (mean [SD], 16.4% [11.5%] vs 23.0% [10.1%]) than girls, spent 36% more time in PEPA, and collected 14% more accelerometer counts overall (for all sex comparisons, $P < .05$). Insulin sensitivity did not differ by sex, but girls had lower fasting plasma glucose levels and higher insulin secretion.

BIVARIATE CORRELATIONS

Table 2 shows bivariate correlations between the variables. When girls and boys were considered separately, PEPA declined with age in girls ($r=-0.52$; $P=.01$) but not in boys ($r=-0.24$; $P=.16$). **Figure 1** shows the relation between insulin secretion and insulin sensitivity. This relation is hyperbolic, with values clustering around the curve representing the median DI (4.38). Eight of the 11 overweight teens had values lower than the median DI but the difference in the proportion of overweight youths vs youths with a BMI lower than the 95th percentile who were below the median DI did not reach statistical significance ($P=.20$). Two normal-weight girls stood out as having very high CIR₃₀ insulin secretion values (>14). Mathematically, their high CIR₃₀ insulin secretion values were explained by the fact that they had the lowest glucose₃₀ values of the whole sample (75 mg/dL and 80 mg/dL, respectively) accompanied by typical insulin₃₀ values.

Table 2. Bivariate Correlations

	<i>r</i> Value (<i>P</i> value)						
	Age	BMI	BMI z score	Total Body Fat, %	Abdominal Fat, %	Mean Daily Activity Counts	PEPA
BMI	0.06 (.65)						
BMI z score	-0.15 (.27)	0.87 (<.001) ^a					
Total body fat, %	-0.18 (.18)	0.85 (<.001) ^a	0.86 (<.001) ^a				
Abdominal fat, %	-0.16 (.25)	0.86 (<.001) ^a	0.85 (<.001) ^a	-0.97 (<.001) ^a			
Mean daily activity counts	-0.22 (.11)	-0.16 (.23)	-0.08 (.58)	-0.23 (.09)	-0.18 (.20)		
PEPA	-0.18 (.16)	-0.09 (.51)	0.007 (.96)	-0.19 (.17)	-0.13 (.35)	0.91 (<.001) ^a	
Fasting glucose level	0.06 (.64)	0.03 (.84)	0.04 (.75)	0.05 (.70)	-0.007 (.96)	0.11 (.42)	0.21 (.13)
Fasting insulin level	-0.19 (.15)	0.28 (.03) ^a	0.33 (.01) ^a	0.31 (.02) ^a	0.33 (.02) ^a	-0.15 (.25)	-0.21 (.13)
2-h glucose	0.02 (.90)	0.09 (.52)	0.05 (.71)	-0.002 (.99)	0.07 (.62)	0.05 (.71)	0.11 (.40)
Log (insulin sensitivity)	0.07 (.63)	-0.36 (.006) ^a	-0.36 (.007) ^a	-0.34 (.01) ^a	-0.35 (.008) ^a	0.24 (.07)	0.29 (.03) ^a
Log (insulin secretion)	-0.12 (.35)	0.23 (.09)	0.30 (.02) ^a	0.30 (.03) ^a	0.30 (.02) ^a	-0.30 (.02) ^a	-0.30 (.02) ^a
Disposition index	-0.01 (.92)	-0.04 (.78)	0.03 (.80)	0.009 (.95)	-0.02 (.87)	-0.21 (.11)	-0.18 (.43)

Abbreviations: BMI, body mass index; 2-h glucose, glucose level at 120-min point of OGTT; log (insulin secretion), log-transformed corrected insulin response at the 30-minute point of OGTT; log (insulin sensitivity), log-transformed Matsuda and DeFronzo index²⁵; OGTT, oral glucose tolerance test; PEPA, play-equivalent physical activity.

^aStatistically significant.

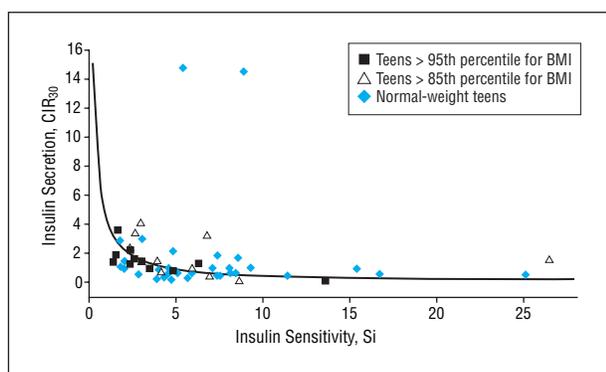


Figure 1. Hyperbolic relationship between insulin secretion and insulin sensitivity. The line represents the median disposition index (insulin secretion \times insulin sensitivity) of 4.38. BMI indicates body mass index; CIR₃₀, corrected insulin response at the 30-minute point of the oral glucose tolerance test; Si, insulin sensitivity calculated as per Matsuda and DeFronzo.²⁵

MULTIVARIATE RELATIONSHIPS

Table 3 shows multiple regression models that include the significant determinants of insulin sensitivity and insulin secretion. Neither age nor sex was a significant determinant in any tested model. The models with BMI z score and time spent in PEPA as the sole determinants of insulin sensitivity explained 21% of the variance in insulin sensitivity ($r^2=0.21$; partial $r^2=0.14$ and 0.10, respectively) and 18% of the variance in insulin secretion, ($r^2=0.18$; partial $r^2=0.10$ and 0.10, respectively). Models in which BMI z score was replaced by other measures of adiposity (BMI, percentage of total body fat, or percentage of abdominal fat) provided an almost identical fit. Models that included more than 1 measure of adiposity were not tested because of collinearity and high variance inflation estimates (eg, for total fat and abdominal fat, $r=0.95$; variance inflation estimate=16.9). When PEPA was replaced by mean daily activity counts to evaluate the importance of cumulative activity rather than intensity, insulin sensitivity was no longer significantly correlated with activ-

Table 3. Multivariate Regressions^a

Independent Variables	Dependent Variable	
	Model 1: Insulin Sensitivity	Model 2: Insulin Secretion
BMI z score	$P = .005$; $-198 \pm 68 \times 10^{-3}$	$P = .02$; $255 \pm 104 \times 10^{-3}$
PEPA	$P = .02$; 7.40 ± 3.09	$P = .02$; -11.61 ± 4.73
r^2	0.21	0.18

Abbreviations: BMI, body mass index; PEPA, play-equivalent physical activity.

^a Each column corresponds to a multivariate model whose dependent variable is log-transformed insulin sensitivity or log-transformed insulin secretion and whose independent variables are BMI z score and proportion of time spent in PEPA. Numbers are *P* values and regression coefficients \pm SE.

ity ($P=.09$), only with BMI z score ($P=.009$). Insulin secretion, on the other hand, was correlated with mean daily activity counts ($P=.03$) and BMI z score ($P=.03$). Neither history of failure to thrive, family income, poverty, or family history of type 2 diabetes nor the adiposity \times PEPA interaction was a significant determinant in any model.

In a linear regression model in which the dependent variable was DI, neither PEPA nor BMI z score (or any other measure of activity or body composition) was a significant independent determinant. However, as shown in **Figure 2**, where the markers are coded to identify subjects higher and lower than the median of PEPA, teens higher than the median for PEPA were preferentially toward the right end of the curve (higher than the median insulin sensitivity) (18 of 28; $P<.05$ by χ^2 test; ratio identical after adjustment for body composition).

COMMENT

Based on studies in adults, the disease process leading to type 2 diabetes is classically described in 2 stages.²⁸ The first stage is characterized by a decline in the sensi-

tivity of peripheral insulin receptors to insulin. This decline, referred to as *insulin resistance*, is observable long before the onset of diabetes²⁹⁻³¹ and is associated with a high percentage of body fat,³² especially abdominal body fat,³³ and low levels of physical activity.^{34,35} The insulin resistance is initially accompanied by a compensatory increase in insulin secretion by the pancreatic beta cells. In the general nondiabetic population with intact beta-cell function, insulin secretion and sensitivity are therefore related to obesity and physical activity in a converse fashion.³⁶ The arithmetic product of insulin secretion and insulin sensitivity tends to equal a constant, termed the *disposition index*.³⁷ Consequently, in a coordinate system with the abscissa representing insulin sensitivity and the ordinate representing insulin secretion, the curve connecting individual values of insulin secretion and sensitivity takes the shape of a hyperbola, as confirmed in multiple populations.³⁷ Relatively insulin-resistant individuals may be on the hyperbolic curve derived from the general population if their insulin secretion is high enough to compensate for the insulin resistance and maintain glucose homeostasis. With intact beta-cell function, perturbations in insulin sensitivity are accompanied by complementary changes in insulin secretion that fit the hyperbolic model, as observed in studies of pregnancy, lipid infusion, weight gain, or weight loss.³⁷

The second stage of the 2-stage model ensues when the beta cells are unable to maintain sufficient insulin secretion to compensate for the declining insulin receptor sensitivity. As demonstrated by Weyer et al³⁸ in prospective studies of subjects who gained weight, subjects who progress to diabetes do not move leftward along the hyperbolic curve (ie, experience a concomitant decrease in insulin sensitivity and increase in insulin secretion) but in a direction perpendicular to and below the curve (ie, experience a concomitant decrease in insulin sensitivity and insulin secretion) toward a lower DI. Consistent with these observations, the most immediate indicator of impending beta-cell failure and diabetes is a decrease in DI.³⁷ Independently of insulin sensitivity, defects in insulin secretion (a low first-phase insulin response) predict the development of diabetes.³⁹

In our study, both adiposity and level of physical activity were significant, independent determinants of insulin sensitivity and secretion, such that a high degree of adiposity and a small proportion of time spent in PEPA were associated with low insulin sensitivity and high insulin secretion, in keeping with the DI concept.

Prospective studies showing that insulin resistance predicts diabetes^{29-31,40,41} suggest that if left unchecked, insulin resistance and consequent increased insulin secretion will often lead to beta-cell failure. In the present study, analysis by BMI category or PEPA level did not identify any group whose DI was diminished in a statistically significant fashion, suggesting that at the group level, beta-cell insulin secretion was appropriate. Inference from Weyer et al⁴² suggests that as long as DI has not deteriorated, an effective behavioral intervention to increase physical activity and reduce weight might achieve the goal of averting diabetes.

The multivariate models were practically identical regardless of the measure of adiposity (BMI z score, BMI,

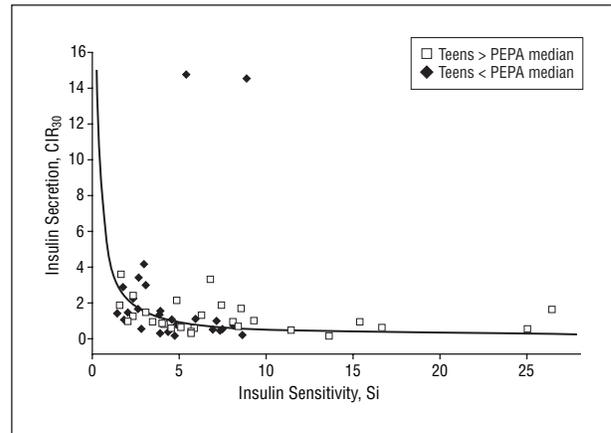


Figure 2. Hyperbolic relationship between insulin secretion and insulin sensitivity. The line represents the median disposition index (insulin secretion \times insulin sensitivity) of 4.38. Teens higher than the median for play-equivalent physical activity (PEPA) were preferentially toward the right side of the curve ($>$ median insulin sensitivity) ($P < .05$ by χ^2 test). CIR₃₀ indicates corrected insulin response at the 30-minute point of the oral glucose tolerance test; Si, insulin sensitivity calculated as per Matsuda and DeFronzo.²⁵

percentage of total body fat, or percentage of abdominal fat). If anything, relations were stronger with BMI z score than with percentages of total body or abdominal fat, which is surprising because accretion of body fat,³² and especially abdominal fat,³³ is thought to underlie the association between insulin resistance and a high BMI. We speculate that BMI z score has extra leverage in the models compared with the DEXA-derived measures because BMI z scores express individual deviation from the norm as a multiple of the empirical age- and sex-specific standard deviation. The DEXA-derived measures were not transformed to z scores because of a lack of normative data.

Of the 11 adolescents in the overweight (≥ 95 th percentile) BMI category, 3 (27%) had impaired glucose tolerance, a prevalence close to the 21% reported in a multiethnic cohort of 11- to 18-year-olds referred to an obesity treatment clinic¹³ and the 27% reported in 8- to 13-year-old Hispanic children with a BMI higher than the 85th percentile.⁴³

A history of failure to thrive was not related to physiological precursors of type 2 diabetes. Thus, although there is evidence that reduced prenatal growth is associated with metabolic disturbances,⁴⁴ our study does not provide evidence that reduced growth in the immediate postnatal period influences insulin sensitivity later in childhood.

Analogous to other cross-sectional studies in children,³ we did not demonstrate an association between physical activity levels and body composition. Although one might expect a correlation based on the notion that physical activity leads to weight loss or, as supported by prospective studies in adults,⁴⁵ that overweight leads to inactivity, the absence of a correlation between activity level and body composition in this or any other cross-sectional study does not refute cause-and-effect relations between physical activity and body composition. As other investigators,⁴⁶ we found that physical activity in girls declined in adolescence.

In addition to the cross-sectional nature of the study, which limits our ability to make inferences about cau-

salinity and temporality, several methodological issues should be considered in interpreting these findings. First, a larger sample size would have yielded increased statistical power to uncover associations. Second, we did not use a rigorous selection strategy to achieve a statistically representative sample of a particular defined population. Although we cannot rule out some selection bias, our community sample compares favorably with referred samples in being representative of mostly African American, urban youth. Because the subjects were infrequently in contact with the research team throughout their lives, it is unlikely that having been observed from infancy influenced behavior. Third, the age group studied (11-16 years) coincides with the onset of puberty, which influences insulin sensitivity and fat accretion. Self-reported Tanner staging of pubertal development was part of the protocol but approximately two-thirds of subjects chose to skip the questions. However, the typical time curve and direction of adiposity changes that differ between boys and girls during puberty were accounted for by the age and sex standardization incorporated in BMI z score.

In addition to the use of a community sample as opposed to a referred sample, the strengths of the study include the sophistication of the measurements (ie, the use of an OGTT as opposed to fasting measurements to explore insulin dynamics and the use of accelerometry to objectively assess activity).

In conclusion, the study shows that in a community sample of African American adolescents, both adiposity and lack of PEPA are independent statistical determinants of low insulin sensitivity and high insulin secretion, both of which have been identified as precursors of type 2 diabetes. Together, adiposity and PEPA, both of which are influenced by the environment, explained 18% to 21% of the variance in insulin sensitivity and secretion. Further analysis involving the DI, a measure of beta-cell secretory capacity, was consistent with these observations.

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Announcement

Submissions. The Editors welcome contributions to Picture of the Month. Submissions should describe common problems presenting uncommonly, rather than total zebras. Cases should be of interest to practicing pediatricians, highlighting problems that they are likely to at least occasionally encounter in the office or hospital setting. High-quality clinical images (in either 35-mm slide or electronic format) along with parent or patient permission to use these images must accompany the submission. The entire discussion should comprise no more than 750 words. Articles and photographs accepted for publication will bear the contributor's name. There is no charge for reproduction and printing of color illustrations. For details regarding electronic submission, please see: <http://archpedi.ama-assn.org>.