Rate of Change in Adiposity and Its Relationship to Concomitant Changes in Cardiovascular Risk Variables Among Biracial (black-white) Children and Young Adults: The Bogalusa Heart Study

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To assess the annual rate of change in adiposity and its relationship to concomitant changes in cardiovascular risk variables during childhood and young adulthood, serial data on black and white children (n = 3,459; initial and follow-up mean age, 8.1 and 14.4 years) and young adults (n = 1,263; initial and follow-up mean age, 22.5 and 30.9 years) enrolled in the Bogalusa Heart Study were examined. Body mass index (BMI) and sum of subscapular and triceps skinfolds were used as indicators of adiposity. In addition, measurements were made of systolic and diastolic blood pressure and fasting levels of low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, triglycerides, insulin, and glucose. Annualized rate of change for each variable was estimated. The rate of increase in adiposity was significantly more pronounced during childhood versus adulthood. Race difference (blacks > whites) in the rate of increase in adiposity was seen only among females. Females, black females in particular, displayed greater rate of increase in adiposity than males. In a multivariate analysis, the rate of increase in adiposity was related independently of baseline age and baseline adiposity to adverse changes in measured cardiovascular risk variables, except glucose. Many of these associations were modulated significantly by race, sex, and age group. The impact was relatively greater for blood pressure and LDL cholesterol in adults and for triglycerides in children. The changes in blood pressure, LDL cholesterol, and HDL cholesterol were greater in whites, while the rate of increase in insulin was greater in blacks. Females displayed greater changes in blood pressure, HDL cholesterol, and insulin. On the other hand, the rate of increase in triglycerides was greater in males. These results indicate that increases in adiposity regardless of initial status of body fatness alter cardiovascular risk variables towards increased risk beginning in childhood, and that this deleterious trend underscores the importance of weight control early in life.

THAT OBESITY IS A risk factor for atherosclerotic cardiovascular disease, type 2 diabetes, dyslipidemia, and hypertension is now well recognized.[1] Further, obesity is commonly associated with insulin resistance/hyperinsulinemia, which is thought to mediate the above disorders.[2,3] The adverse impact of obesity on cardiovascular risk variables has been found both in children and adults.[1,4-9] Fat mass and fat pattern change adversely with growth and maturation and aging.[4,5,10-12] Because cardiovascular risk factors, including obesity in youth, are associated with coronary atherosclerosis,[13,14] there is a need to examine the association between longitudinal changes in body fatness and concurrent changes in cardiovascular risk variables in youth.

While most of the previous studies on this subject have used differences between 2 measurements made at baseline and follow-up as longitudinal measure of change,[15-18] a few studies have used consecutive measurements.[19,20] In particular, observations from the Fels Longitudinal Study underscore the advantage of using serial measurements in examining the age- and sex-specific relationships between changes in body fatness during adulthood period and concurrent changes in cardiovascular risk variables in terms of lipoproteins.[20] However, no corresponding information is available during childhood versus young adulthood.

The Bogalusa Heart Study, a long-term biracial (black-white) community-based study, provides periodic cross-sectional data on cardiovascular risk factor variables during childhood and young adulthood.[21] The objective of the present analysis was to examine the race- and sex-specific associations between annual rate of change in adiposity and concomitant changes in blood pressure, lipoprotein variables, insulin and glucose during childhood versus young adulthood.

MATERIALS AND METHODS

Study Population

The biracial (65% white and 35% black) population of the Bogalusa Heart Study consists of all school children and eligible young adults living in the semirural community of Bogalusa, LA. Between 1978 and 1996, 5 cross-sectional surveys of school children aged 5 to 17 years and 4 surveys of young adults aged 19 years or above, who were examined previously as children and residing in the community, were conducted approximately every 3 years. The oldest adult during the last screening cycle was 38 years of age. The participation rate was approximately 80% for the school children and 60% for the young adult cohort. Children and young adults of both races were eligible for inclusion in this study if they participated in at least 2 surveys, with a minimum of 3-year intervals between the first and last surveys. Accordingly, 41% (3,459 of 8,404) of children and 44% (1,263 of 2,843) of young adults were selected for the study.

The number of subjects examined multiple times, number of examinations, midage (average age across screenings), and follow-up period of children and young adults are given in Table 1 by race and sex. Of the 3,459 selected children (40.5% black, 50.7% female), 48%, 35%, and 17% of white males, 42%, 35%, and 23% of black males, 47%, 35%, and 18% of white females, and 44%, 33%, and 23% of black females, respectively, were examined 2, 3, and 4 times. With respect to young adults (n = 1,263; 28% black; 59% female), 55%, 29%, and 16% of white males, 60%, 32%, and 8% of black males, 47%, 29%, and 14% of white females, and 58%, 29%, and 13% of black females had

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Table 1. Sample Size and Mean (± SD) of Age and Follow-up Period of the Study Cohorts by Race and Sex: The Bogalusa Heart Study

	White Male	Black Male	White Female	Black Female
Children				
No. of subjects examined				
Two times	499	290	481	318
Three times	355	235	369	234
Four times	170	156	183	169
Total	1,024	681	1,033	721
No. of examinations	2,744	1,914	2,793	2,016
Midage (yr)*	11.2 ± 1.8	11.4 ± 1.8	11.2 ± 1.8	11.3 ± 1.8
Follow-up period (yr)	6.1 ± 1.9	6.6 ± 2.1	6.3 ± 2.1	6.6 ± 2.1
Adults				
No. of subjects examined				
Two times	214	77	241	133
Three times	112	42	172	67
Four times	64	10	102	29
Total	390	129	515	229
No. of examinations	1,020	320	1,406	583
Midage (yr)*	26.8 ± 2.9	27.1 ± 3.1	26.6 ± 2.7	26.3 ± 2.8
Follow-up period (yr)	8.3 ± 3.4	8.1 ± 3.2	8.8 ± 3.4	7.9 ± 3.2

* Average age of the subject across screenings.

2, 3, and 4 examinations, respectively. The average age at initial screening was 8.1 years for children; 22.5 years for adults.

General Examination

Identical protocols were used by trained examiners across all surveys. Subjects were instructed to fast for 12 hours before venipuncture, and compliance was determined by interview on the morning of examination.

Height and weight were measured in triplicate to the nearest 0.1 cm and 0.1 kg, respectively. As a measurement of overall adiposity, the body mass index (BMI, weight in kilograms divided by the square of the height in meters) was used. Subscapular and triceps skinfolds were measured 3 times to the nearest 1.0 mm, and the sum of these 2 skinfolds was used as an another measure of body fatness. Systolic and diastolic (fourth phase) blood pressure levels were measured in 6 replicates by 2 randomly assigned nurses on the right arm of subjects in a relaxed, sitting position. Diastolic blood pressure at the fourth phase rather than the fifth phase was used because both overall variance and interobserver variability during childhood were smaller, and the predictability of adult hypertension was better with the former.[22] Among the study subjects, fourth phase diastolic blood pressure was registered in 99.6% of children (3,459 subjects; 9,467 examinations) and 99.8% of adults (1,263 subjects; 3,329 examinations). Means of replicates were used in all analyses.

Laboratory Analyses

From 1978 to 1986, cholesterol and triglyceride levels were measured using chemical procedures on Technicon AutoAnalyzer II (Technicon Instrument, Tarrytown, NY), according to the Laboratory Manual of the Lipid Research Clinics Program.[23] Since then, these variables were determined by enzymatic procedures on the Abbott VP instrument (Abbott Laboratories, North Chicago, IL). Serum lipoprotein cholesterols were analyzed by a combination of heparin-calcium precipitation and agar-agarose gel electrophoresis procedure.[24] Both chemical and enzymatic procedures met the performance requirements of the Lipid Standardization Program of the Centers for Disease Control and Prevention, Atlanta, GA. The laboratory has been monitored for precision and accuracy of lipid measurements by the agency's surveillance program since 1973.

Plasma immunoreactive insulin levels were measured by a commercial radioimmunoassay kit (Phadebas; Pharmacia Diagnostics, Piscataway, NJ). The intra and interassay coefficients of variation of insulin assay were 5.7% and 6.5%, respectively. Plasma glucose levels were measured by a glucose oxidase method using a Beckman glucose analyzer (Beckman Instrument, Fullerton, CA).

Statistical Analyses

Interval of follow-up was defined as difference in age at the first and last surveys. Midage was defined as the mean of the ages at the first and last screening. Annualized rates of change for each variable were computed following the method of Siervogel, et al.[20] Briefly, for each subject a series of regression equations were generated predicting individual risk factors from age. The slope coefficient is the annualized rate of change. This approach maximizes the information for each individual by using all their serial data and requires the assumption that the relationship between levels of a specific risk factor and age can be approximated by a linear function. Mean levels of risk factors for each subject were defined by using the midage as the predictor in the appropriate regression equation.

Data, including rates of change, were summarized by race/sex/age groups. Linear regression methods were used to predict change in levels of risk factor variables from change in adiposity (BMI or sum of skinfolds). Baseline age and adiposity, age group, race, sex, and the interaction of race and sex were included as covariates in all regression models. Of interest was to determine whether the relationship between change in risk variables and change in adiposity was the same for both sexes, races, and age groups; therefore, predictors of interest in the regression models included the annualized change in adiposity, as well as an interaction of change in adiposity and the demographic covariates. Significance was defined as $P < .05$.

RESULTS

Baseline levels of measured risk factor variables adjusted for race, sex, age, and study year were compared between the study subjects and those who were examined only once. There were no significant differences between the groups with respect to BMI, sum of skinfolds, triglycerides, high-density lipoprotein (HDL) cholesterol, and insulin among children and any of the risk factor variables among adults. However, children with repeat examinations had small, but significantly ($P < .01$), lower systolic blood pressure (99.8 *v* 101.2 mm Hg) and

diastolic blood pressure (59.8 *v* 60.7 mm Hg) and higher low-density lipoprotein (LDL) cholesterol (103.0 *v* 99.9 mg/dL) and glucose (82.0 *v* 80.7 mg/dL) than those with single examination. Such differences in baseline characteristics may not influence the outcome, because in assessing rates of change in risk variables, baseline levels were used as covariates.

Mean levels of obesity indices, blood pressure, lipoprotein variables, insulin, and glucose calculated at the midage over the follow-up period in children and young adults are presented in Table 2 by race and sex, as background. The race-, sex- and age-related trends for these variables were in the expected directions based on the earlier data from US populations of children and young adults.[6,21,22,25-28] In general, mean levels of risk variables changed adversely in young adults compared with children. As children, whites compared with blacks had higher levels of sum of skinfolds, triglycerides, and glucose, and lower levels of HDL cholesterol and insulin (females only). In adulthood, higher levels of systolic and diastolic blood pressure, HDL cholesterol, sum of skinfolds (females only), BMI (females only), and insulin (females only) and lower levels of LDL cholesterol and triglycerides were seen in blacks than in whites. With respect to sex-related differences, as children, females versus males had higher levels of BMI, sum of skinfolds, LDL cholesterol, triglycerides, and insulin and lower levels of systolic blood pressure and glucose. As adults, males compared with females displayed higher systolic and diastolic blood pressure, triglycerides, HDL cholesterol (whites only), and glucose and lower sum of skinfolds. In addition, adulthood levels of BMI were higher in white males (*v* white females) and black females (*v* black males).

Annual rate of changes (Δ) of adiposity indices in children versus adults are shown in Fig 1 by race and sex. The statistical significance ($P < .05$) of the race-sex differences are not denoted in Fig 1 for the sake of simplicity. Both Δ BMI and Δ sum of skinfolds were markedly higher in children versus adults, with the exception of Δ sum of skinfolds in black males. In children and adults, race differences (blacks $>$ whites) in rates of both adiposity measures were seen only among females. Females as children and adults had higher Δ sum of skinfolds than males. However, such sex differential with respect to ΔBMI was seen only among blacks.

The relationship of Δ adiposity measures to Δ cardiovascular risk variables were examined in a multivariate analysis, and the results are given in Tables 3 through 5. Both Δ BMI and Δ sum of skinfolds were related to adverse changes in risk variables (except Δ glucose), independently of baseline age and baseline adiposity. Further, many of these associations were modulated significantly by race, sex, or age group (children *v* adults), as can be seen from the interaction terms retained in the regression model. The impact of both Δ BMI and Δ sum of skinfolds on Δ systolic blood pressure, Δ diastolic blood pressure, and Δ LDL cholesterol were greater in adults than children. Further, Δ

Table 2. Mean (± SD) Levels of Cardiovascular Risk Variables Over the Follow-up Period in Children and Adults by Race and Sex: The Bogalusa Heart Study

Variable*	White Male	Black Male	White Female	Black Female
BMI (kg/m^2)				
Children	19.3 ± 3.6	19.0 ± 3.5	19.4 ± 3.9^{x}	19.8 ± 4.1^{x}
Adults	26.2 ± 4.5^{x}	25.6 ± 6.0	24.0 ± 5.2	27.3 ± 7.1^{a,x}
Sum of skinfolds (mm)†				
Children	25.1 ± 12.3^{a}	20.7 ± 11.6	31.1 ± 13.0^{a,x}	29.3 ± 14.0^{x}
Adults	32.6 ± 13.5	29.9 ± 18.5	41.8 ± 15.1^{x}	46.8 ± 18.8^{a,x}
Systolic BP (mm Hg)				
Children	102.9 ± 8.1^{x}	102.7 ± 8.0^{x}	101.8 ± 7.7	101.8 ± 8.1
Adults	114.4 ± 8.5^{x}	118.2 ± 11.5^{a,x}	107.8 ± 8.4	111.2 ± 8.8^{a}
Diastolic BP (mm Hg)				
Children	61.5 ± 6.5	61.1 ± 6.3	62.9 ± 6.7	62.2 ± 7.1
Adults	74.0 ± 6.8^{x}	75.8 ± 9.0^{a,x}	70.5 ± 6.4	71.9 ± 7.1^{a}
LDL cholesterol (mg/dL)				
Children	95.6 ± 22.1	96.0 ± 23.4	99.1 ± 23.5^{x}	100.7 ± 24.8^{x}
Adults	122.5 ± 29.6^{a}	109.6 ± 31.1	117.2 ± 29.7^{a}	111.1 ± 26.4
HDL cholesterol (mg/dL)				
Children	56.3 ± 14.4	62.4 ± 15.1^{a}	54.3 ± 14.8	62.0 ± 15.0^{a}
Adults	41.8 ± 12.8	55.1 ± 16.0^{a}	50.9 ± 13.8^{x}	56.0 ± 16.5^{a}
Triglycerides (mg/dL)				
Children	69.5 ± 32.1^{a}	56.4 ± 18.6	76.5 ± 32.7^{a,x}	62.0 ± 21.8^{x}
Adults	121.8 ± 73.4^{a,x}	101.1 ± 77.0^{x}	111.4 ± 162.3^{a}	79.2 ± 31.7
Insulin (μU/mL)				
Children	8.8 ± 5.0	9.2 ± 6.1	10.6 ± 5.8^{x}	12.3 ± 8.4^{a,x}
Adults	11.2 ± 7.6	11.5 ± 10.1	10.0 ± 4.9	14.1 ± 11.1^{a}
Glucose (mg/dL)				
Children	83.6 ± 5.9^{a,x}	82.9 ± 6.3^{x}	81.6 ± 5.8^{a}	80.5 ± 6.5
Adults	84.0 ± 6.9^{x}	86.0 ± 25.3^{x}	80.5 ± 12.2	83.9 ± 27.4

NOTE. Race-sex difference: the superscripts a (for race) and x (for sex) denote significantly ($P < .05$) higher value compared with other race or sex group.

* Mean values calculated at midage.

† Subscapular and triceps.

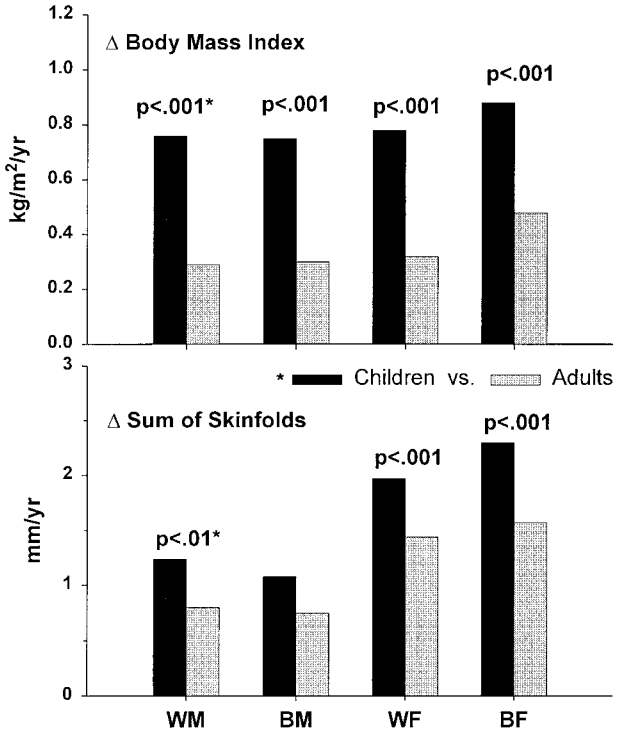

Fig 1. Estimated annual rate of change of obesity measures in children *v* adults: the Bogalusa Heart Study.

BMI affected Δ triglycerides more in children than adults. The impact of Δ BMI on diastolic blood pressure and Δ LDL cholesterol was greater in whites than blacks. Δ sum of skinfold affected Δ HDL cholesterol more in whites than blacks, while its impact on Δ insulin was more in blacks than whites. The effect of Δ BMI on Δ HDL cholesterol was greater in females than males, while its influence on Δ triglycerides was greater in males than females. The influence of Δ sum of skinfolds on Δ systolic blood pressure, Δ diastolic blood pressure, and Δ insulin was greater in females than males.

The percent variance (R^2) of risk variables explained by Δ obesity measure was determined separately for children and adults in the regression model that included race, sex, race-sex interaction, age at baseline, and obesity measure at baseline. In children, the R^2 values with respect to Δ BMI were 13% for systolic blood pressure, 6% for diastolic blood pressure, 7% for LDL cholesterol, 5% for HDL cholesterol, 8% for triglycerides, 7% for insulin, and 4% for glucose. In adults, the corresponding R^2 values were 8%, 6%, 9%, 4%, 1%, 12%, and 2%. Similar results were obtained with respect to Δ sum of skinfolds (data not shown).

DISCUSSION

The present community-based study demonstrates that the annual rate of increase in adiposity was accompanied by adverse changes in blood pressure, lipoprotein variables and insulin, and that this relationship was independent of baseline adiposity and baseline age and modulated by developmental period (children *v* young adults), race and sex. These observations are noteworthy in that, to our knowledge, no comparable data describing the association between rate of change in adiposity and concurrent changes in multiple cardiovascular risk variables during childhood versus young adulthood are available to establish this relationship in the general population of youth.

It is well known that the body fatness increases with age in the US population.[4,5] In this study, the annual rate of increase in adiposity (BMI or sum of skinfolds) was more pronounced during childhood versus adulthood. That the fat mass increases markedly during periods of growth and maturation is known,[10-12] and the present results are in agreement with these findings. The observed accelerated rate of increase in adiposity in childhood may have a bearing on adult obesity and its prevention, because the genesis of adult obesity is thought to begin in childhood.[29-31] It is of interest that as children and young adults, females, especially black females, displayed a greater rate of increase in adiposity compared with males in this study. The observed highest rate of increase in adiposity in black females since childhood may reflect the high prevalence of obesity and type 2 diabetes in this group.[32-34] In this regard, the temporal association between obesity and hyperinsulinemia is of particular interest. In a related study involving cohorts of

Table 3. Relationship of Annual Rate of Change of Obesity Measures to Annual Rate of Change of Blood Pressure: The Bogalusa Heart Study

	Δ BMI*		Δ Sum of Skinfolds*	
Dependent Variable/Interaction Terms	Regression Coefficient†	*P* Value	Regression Coefficient†	*P* Value
Δ Systolic blood pressure				
Interaction	.62	.001	.06	.0001
Age group	.37	.001	.41	.0001
Sex	−.01	NS	.23	.0001
Race	−.16	NS	.10	NS
Δ Diastolic blood pressure				
Interaction	.49	.01	.06	.0001
Age group	.36	.001	.40	.001
Sex	.06	NS	.14	.05
Race	−.22	.05	−.10	NS

Abbreviation: NS, not significant.

* Independent variable.

† Linear regression model included baseline age, baseline obesity measure, age group (children, 0; adults, 1), sex (male, 0; female, 1), race (white, 0; black, 1), and race-sex interaction as covariates, in addition to parameters listed in the table.

Table 4. Relationship of Annual Rate of Change of Obesity Measures to Annual Rate of Change of Lipoprotein Variables: The Bogalusa Heart Study

Dependent Variable/Interaction Terms	Δ BMI*		Δ Sum of Skinfolds*	
	Regression Coefficient†	*P* Value	Regression Coefficient†	*P* Value
Δ LDL cholesterol				
Interaction	2.96	.0001	.11	.05
Age group	1.40	.0001	1.54	.0001
Sex	−.49	NS	.27	NS
Race	−.59	.05	.23	NS
Δ HDL cholesterol				
Interaction	−1.76	.001	−.06	.05
Age group	−.38	NS	−.47	NS
Sex	.55	.05	.03	NS
Race	−.06	NS	−.57	.01
Δ Triglycerides				
Interaction	10.77	.0001	.67	.001
Age group	−2.85	.05	−2.32	NS
Sex	−3.58	.001	−.71	NS
Race	−1.23	NS	1.28	NS

Abbreviation: NS, not significant.

* Independent variable.

† Linear regression model included baseline age, baseline obesity measure, age group (children = 0, adult = 1), sex (male = 0, female = 1), race (white = 0, black = 1), and race × sex interaction as covariates, in addition to parameters listed in the table.

children, adolescents, and young adults, we found a significant association between baseline BMI and incidence of hyperinsulinemia at follow-up among these age groups independent of race, sex, and baseline insulin levels.[35] That no such temporal relationship was noted between baseline insulin and incidence of obesity underscore the role of obesity in the development of hyperinsulinemia/insulin resistance in youth.

Results of multivariate analysis show that the rate of increase in adiposity was adversely related to concurrent changes in cardiovascular risk variables, with the exception of glucose. Because these relationships were independent of baseline age and baseline adiposity, the adverse effect of rate of increase in adiposity on changes in risk variables seem to occur across the distribution of body fatness in the study population, as previously reported.[15,16,19,20] The current study also supports the longitudinal evidence that adverse changes in cardiovascular risk variables are a function, in part, of the increment in adiposity.[15-20] Of note is our finding that the impact of rate of increase in adiposity on adverse changes in systolic blood pressure, diastolic blood pressure, and LDL cholesterol was greater in adults than children; whereas the reverse (children > adults) was true in the case of triglycerides.

Race-sex differences in associations between longitudinal changes in adiposity and cardiovascular risk variables have been noted previously.[15-20] In this study cohort, the detrimental associations were relatively stronger for blood pressure, LDL cholesterol, and HDL cholesterol in whites and for insulin in blacks. The changes in blood pressure, HDL cholesterol, and insulin were relatively greater in females than males. On the other hand, the rate of increase in triglycerides was relatively greater in males, although females displayed a greater rate of increase in adiposity than males. The reason for the observed

Table 5. Relationship of Annual Rate of Change of Obesity Measures to Annual Rate of Change of Plasma Insulin and Glucose: The Bogalusa Heart Study

Dependent Variable/Interaction Terms	Δ BMI*		Δ Sum of Skinfolds*	
	Regression Coefficient†	*P* Value	Regression Coefficient†	*P* Value
Δ Insulin				
Interaction	.80	.01	.08	.001
Age group	−.10	NS	−.06	NS
Sex	.09	NS	.25	.05
Race	.13	NS	.28	.05
Δ Glucose				
Interaction	−.17	NS	−.04	NS
Age group	.31	NS	.29	NS
Sex	.06	NS	.02	NS
Race	.13	NS	.11	NS

Abbreviation: NS, not significant.

* Independent variable.

† Linear regression model included baseline age, baseline obesity measure, age group (children = 0, adults = 1), sex (male = 0, female = 1), race (white = 0, black = 1), and race × sex interaction as covariates, in addition to parameters listed above.

variability by race and sex cannot be explained from this observational study.

Previous studies have shown race-sex differences in the association of obesity and body fat distribution with hyperinsulinemia/insulin resistance and related metabolic processes and cardiovascular risk factors,[36-43] which may have a bearing on the current findings. For example, it has been reported that decreased triglyceride levels associated with female sex are independently accounted for by the increased ability of insulin to suppress circulating plasma free fatty acids in females,[44,45] the major substrate for very low-density lipoprotein synthesis and secretion by the liver.[46,47] Moreover, in general, blacks and premenopausal females compared with their race-sex counterparts have higher lipoprotein lipase and lower hepatic triglyceride lipase,[48-52] key enzymes in the metabolism of triglyceride-rich lipoprotein and HDL.[53] The metabolic characteristics of black females who showed the highest rate of increase in body fatness in the current study is of particular interest. It has been found that excess body fatness in black females, unlike other race-sex groups, is not associated with resistance to insulin's antilipolytic activity in the adipose tissue and the attendant hypertriglyceridemia.[43] Such a condition in the presence of resistance to insulin's glucoregulatory action and increased lipoprotein lipase in black females could contribute to their high prevalence of obesity and type 2 diabetes.[43,54]

The present study did not examine the issue of body fat distribution in terms of visceral fat deposition, an important mediator of metabolic aberrations.[55-57] Visceral fat accumulates with age and is greater in men than women and in whites than blacks.[36,56-60] It has been shown that in blacks, adipocytes from both upper- and lower-body obesity groups, unlike those from their white counterparts, are equally sensitive to insulin in terms of glucose uptake and suppression of lipolysis.[61] These findings suggest a racial dimorphism in the pathophysiology of obesity. Obviously, the relationship of the rate of increase in visceral fat deposition to concurrent changes in cardiovascular risk variables among the race-sex groups needs to be established.

In the United States, the prevalence of obesity is increasing among adults and children alike,[62,63] and our results indicate that increases in adiposity are associated with detrimental changes in cardiovascular risk variables, irrespective of baseline age and baseline adiposity. Although the strength of adiposity-risk variable associations showed some variability by race, sex, and developmental period, as a public health issue, our results underscore the importance of prevention and intervention of excessive weight gain early in life in the general population.

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