6 Racial Differences in Childhood Obesity: Pathogenesis and Complications

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Key Words: Race, ethnicity, insulin resistance, prediabetes, fat distribution, socioeconomic status, NAFLD, lipid, type 2 diabetes

INTRODUCTION

Race and ethnicity are terms used to categorize populations on the basis of shared characteristics. Race is traditionally used to categorize populations on the basis of shared biological characteristics such as skin color, other observable features, and the genetic determinants of such differences *(1)*. Ethnicity is used to categorize individuals on the basis of cultural characteristics such as shared language, ancestry, religious traditions, dietary preferences, and history. Although ethnic groups can share a range of phenotypic characteristics due to shared ancestry, the term is typically used to highlight cultural and social characteristics instead of biological ones *(1)*. Both race and ethnicity are frequently used interchangeably and are constantly evolving concepts, especially in the United States, making the task of comparing groups or following the same group over time quite challenging. There is an emerging number of Americans who describe their race as "mixed" or "other," and there are changes in ethnic self-identification across generations. Such emerging patterns make it difficult to assign

From: *Contemporary Endocrinology: Pediatric Obesity: Etiology, Pathogenesis, and Treatment*
Edited by: M. Freemark, DOI 10.1007/978-1-60327-874-4_6,

individuals to invariant categories of race or ethnicity. Nevertheless, we continue to use the terms race and ethnicity because there is social importance given to these constructs, and because such classifications have revealed important biological differences in disease predisposition and complications as well as inequalities in access to health care *(1)*.

Obesity is a worldwide problem affecting all ethnic and social groups; however, there are important racial differences in the prevalence as well as the health complications of obesity. Although childhood obesity is increasing in all ethnic and racial groups, there is a disproportionate rise among black Americans and Hispanic/Mexican Americans. The reasons are complex, involving interactions among genetic, biological, cultural, socioeconomic, environmental, and other influences *(1,2)*. This chapter will focus primarily on the biological differences between black and white children in risk of obesity and its complications.

OBESITY AND RACE

Data from the National Health and Nutrition Examination Survey (NHANES) demonstrate that the prevalence of overweight in American youth has increased in all age and racial groups during each of the survey periods during the past 20 years. Between 1999 and 2004, the prevalence of "at risk of overweight" (BMI $\geq$ 85th percentile) or "overweight" (BMI $\geq$ 95th percentile) in youth ages 12–19 years has increased from 30 to 34.4% and 14.8 to 17.4%, respectively *(3)*. However, the increases in childhood overweight were considerably greater in non-Hispanic blacks (13.4–23.6%) and Mexican Americans (13.8–23.4%) than in American whites; 25% of black girls (12–19 years) are now overweight compared with 15% of white girls of similar age *(3)*. The trends are similar in younger children and adults. On the other hand, among a sample of preschool children drawn from 20 large US cities, the prevalence of obesity was 25.8% among Hispanics, 16.2% among blacks, and 14.8% among whites *(4)*. These differences were not explained by racial/ethnic differences in socioeconomic indicators.

The National Heart, Lung, and Blood Institute Growth and Health Study shows that the racial divergence in adiposity begins during adolescence and relates temporally to puberty, with the prevalence increasing in blacks during pubertal maturation *(5)*. Although the mechanisms responsible for these differences are not fully understood, environmental factors such as high energy intake *(6)*, low-physical activity level *(7)*, and sedentary behavior (e.g., increased television watching) have been reported in black vs. white girls. However, in adult women, racial differences in the prevalence of obesity persist after accounting for these environmental factors *(8)*, suggesting that inherent metabolic/physiological differences exist between the two racial groups *(9)*. Thus, unmodifiable and modifiable risk factors, together or independent of each other, could explain the racial disparity in obesity. Among the former would be genetic/biological/inherent factors predisposing to obesity, involving appetite regulation, energy expenditure, and metabolic alterations; among the latter would be environmental/behavioral/sociocultural factors conducive to a positive energy balance. While the former may not be amenable to therapy until such time that specific genetic alterations and their mechanism of action are identified and lead to specific pharmacogenomic therapies, the latter could be targeted to correct the racial disparity in childhood obesity and its consequences.

RACE AND GENETIC/BIOLOGICAL DIFFERENTIAL IN RISK OF OBESITY

Race, Obesity, and Genetics

Human adiposity is highly heritable, but only a few of the genes that predispose to obesity in most humans are known (see the chapters in Part III). Less well understood are race-specific genes that could modulate the risk of obesity in any group. Because the prevalence of higher obesity rates in

minority populations persists even after adjusting for socioeconomic factors, genetic factors have been implicated to explain some of the differences.

To identify genetic loci influencing BMI, a pooled analysis of genome-wide admixture mapping scans was performed in 15,280 African-American adults from 14 epidemiologic studies *(10)*. After adjusting for age, sex, and study, BMI was analyzed both as a dichotomized and a continuous trait. The results revealed that a higher percentage of European ancestry correlated with lower BMI. In addition, in obese individuals there were two loci with increased African ancestry on chromosome X (Xq13.1 and Xq25) and one locus with increased European ancestry on chromosome 5 (5q13.3).

The 5q13.3 and Xq25 regions both contain genes that are known to be involved in appetite regulation; thus it is possible that genetic differences in the hormonal regulation of appetite and energy intake play a role in race-related differences in obesity. Ghrelin is a "hunger" peptide whose levels increase before meals and decrease postprandially *(11)*. Conversely, peptide YY (PYY) is a "satiety" hormone that is low preprandially and increases after meals *(11)*. Therefore, impaired regulation of ghrelin and PYY might lead to impaired hunger and/or satiety. We demonstrated that suppression of ghrelin following an oral glucose load was attenuated in black children relative to whites. Additionally, PYY levels were lower in blacks *(11)*. Thus, lesser suppression of ghrelin and lower levels of PYY after a meal could in theory promote subsequent food intake and thereby contribute to the higher risk of obesity in black youth. However, there are no epidemiological data or well-controlled studies that demonstrate differences in food intake between black and white children.

Other hormones and peptides likely contribute to obesity risk. A genome-wide association study in a racially and ethnically diverse sample of 24,722 adults from four cohorts observed no variation in the frequencies of the three insulin-induced gene 2 (*INSIG2*) single nucleotide polymorphism (SNP) genotypes between white, Hispanic, and black American obese adults and non-obese individuals *(12)*. Association analysis of the *FTO* gene with obesity in children of Caucasian and African ancestry revealed a common tagging SNP with some differences in allele frequencies *(13)*. In contrast, an association between obesity and a SNP of the *MC4R* locus in European American children was not found in African-American obese youth *(14)*. Thus, much work remains to be done to identify potential genetic differences which could explain the higher rates of obesity in minority children.

RACE, OBESITY, AND BIOLOGY

Hyperinsulinemia/Insulin Resistance

There are convincing epidemiological data that black children are hyperinsulinemic compared with their white peers. The Bogalusa Heart Study was the first to show that black children, 5–17-years-old, have higher insulin responses than their white counterparts during an oral glucose tolerance test (OGTT) *(15)*. In another study, the higher insulin levels in black children remained significant after controlling for adiposity differences *(16)*. In well-controlled patient-oriented research in which healthy black and white children were matched for age, puberty, total and visceral adiposity, and physical fitness (variables that impact insulin sensitivity and secretion), black children were found to have ~20% lower in vivo insulin sensitivity *(17)*. This was accompanied by ~150% higher first-phase insulin secretion in blacks, which was over and beyond the expected compensatory response to lower insulin sensitivity (Fig. 1).

Using genetic admixture analysis, others demonstrated that these differences in insulin sensitivity and secretion could be explained both on genetic and environmental bases *(19)*. In favor of the former is the observation that low adiponectin levels in black vs. white children may be a biological marker that predisposes them to a greater risk of insulin resistance *(18)*. Adiponectin is an adipocytokine that is exclusively expressed and secreted from adipose tissue. Its levels are low in obesity, states of

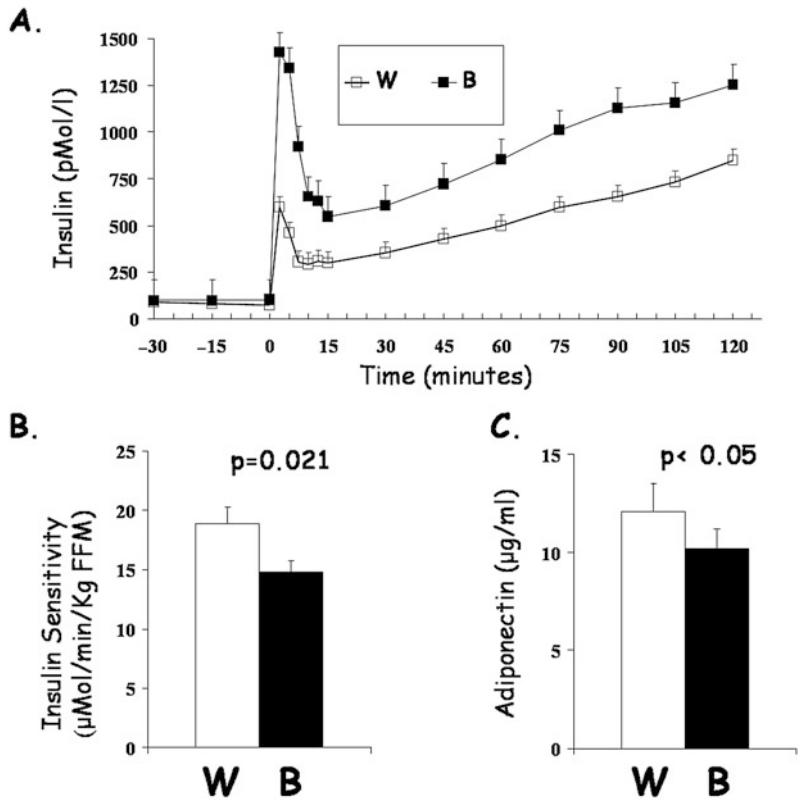

Fig. 1. (**a**) Insulin concentrations during a 2-h hyperglycemic clamp (225 mg/dl). (**b**) In vivo insulin sensitivity measured during a 40 mU/m^2/min hyperinsulinemic-euglycemic clamp. (**c**) Adiponectin levels, in black (B) and white (W) youth. Adapted with permission *(17,18)*.

insulin resistance, type 2 diabetes, and cardiovascular disease. The antidiabetogenic and antiatherogenic properties of adiponectin are evident early in life because hypoadiponectinemia is a strong and independent correlate of insulin resistance and β-cell dysfunction in youth. The low adiponectin level in black youth is independent of visceral adiposity. Moreover, the race-related difference in insulin sensitivity disappears after controlling for the lower adiponectin levels in blacks.

Lifestyle differences in dietary habits may also contribute to racial differences in insulin sensitivity. High-fat diets have been implicated in the pathogenesis of insulin resistance in adults. Correlation analysis reveals an inverse relationship between the higher fat/carbohydrate ratio in the diets of black children and insulin sensitivity *(17)*. Black children are reported to have high-fat intake in some but not all studies *(20)*. Low levels of physical activity and physical fitness might also play roles. However, in one study, neither physical activity nor fitness could explain the racial difference in insulin sensitivity *(21)*.

Even when black and white youth are matched for degree of insulin sensitivity, insulin levels and insulin secretion are higher in blacks *(22)*. Although the mechanisms responsible for the up-regulation of β-cell function in black youth are unknown, potential causes include dietary/lifestyle factors, genetic differences, and inherent differences in neuronal and/or metabolic signaling for insulin secretion. Increased dietary fat relative to carbohydrate was found to correlate positively with first-phase insulin levels in children *(17)*. On the other hand, genetic admixture was independently related to acute insulin response to glucose, indicating that hyperinsulinemia in black youth may have a genetic basis *(19)*.

Thus, much work remains to be done to decipher the underlying causes for race-related differences in insulinemia.

Lipid Metabolism

Irrespective of what causes the hyperinsulinemia in black youth, it is plausible that it plays a role in the increased risk of obesity in blacks. Hyperinsulinemia inhibits lipolysis and leads over time to progressive fat accretion under conditions of excess energy intake or diminished physical activity. Indeed, the rate of whole body lipolysis, measured by [$^2H_5$]glycerol stable isotope, was 40% lower in black vs. white healthy prepubertal children (Fig. 2) *(23)*. This may constitute an early metabolic phenotype in blacks that may mediate fat trapping and susceptibility to obesity in a specific environmental context of energy excess. Thus, both genetics and environment together may play a pathophysiological role in the excess risk of development of obesity in blacks.

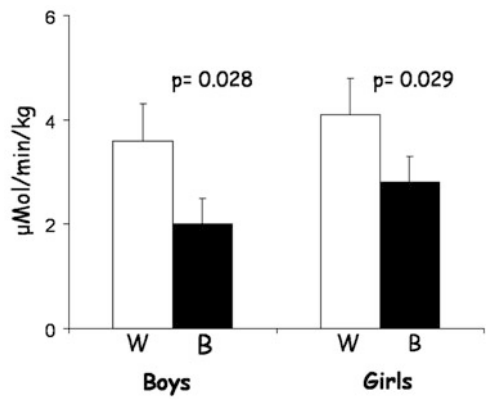

Fig. 2. Rates of total body lipolysis in black (B) and white (W) boys and girls. Adapted with permission *(23)*. Copyright 2001, The Endocrine Society.

Debate continues in the literature as to whether or not hyperinsulinemia is a consequence or cause of obesity. In 1962, the so-called thrifty genotype hypothesis was proposed by Neel *(24)*. Fundamental to this theory is the hypothesis that hyperinsulinemia precedes obesity, with a differential in insulin action on glucose vs. fat metabolism *(23)*; the antilipolytic effect of insulin is increased and mobilization of stored fat is inhibited, leading to lipid storage. In favor of this theory, fasting insulin levels in 5- to 9-year-old Pima Indian children were associated with higher rates of weight gain during a 10-year follow-up period. In a cohort of black and white children, fasting insulin levels were positively associated with the rate of increase in fat mass *(25)*.

Reduced postabsorptive fat oxidation also contributes to positive energy balance and, therefore, future weight gain and obesity *(26)*. Puberty is associated with increased fat oxidation. However, the puberty-associated increase in fat oxidation is diminished in black girls compared with their white peers *(9)*. In addition we and others have shown reduced resting metabolic rates in black vs. white youth *(9)*. A two-year longitudinal study in 9- to 11-year-old black and white youth indicates that total daily energy expenditure, resting metabolic rate, and substrate oxidation are predictors of gain in body fatness *(27)*. Collectively, these observations suggest that the lower metabolic rate and the reduced fat oxidation during puberty in black girls may predispose them to future weight gain and obesity in the context of an obesogenic environment. This metabolic phenotype could potentially explain the divergence in adiposity in black girls during adolescence *(5)*.

Several factors influence resting substrate oxidation; these include genetic factors, the amount of adipose tissue, level of activity, caloric intake, and composition of diet *(28)*. However, the underlying mechanisms of racial differences in fat oxidation are still unclear. A single recent study in adults showed that the mass of metabolically active organs (e.g. brain, liver, heart, kidney, and spleen) was significantly smaller in blacks than in whites, and the racial difference in resting metabolic rate disappeared once the mass of these organs was accounted *(29)*. It is currently unknown whether this holds true in children. On the other hand, it is also plausible that higher inactivity levels and lower cardiorespiratory fitness in black vs. white children *(30)* may contribute to lower metabolic rate and fat oxidation.

It remains to be investigated whether the smaller mass of metabolically active organs, the lower fat oxidation, and the lower cardiorespiratory fitness are biologically/genetically driven, environmentally determined, or both. The skeletal muscles of obese or type 2 diabetic adults are characterized by fewer mitochondria, smaller mitochondrial size, and reduced mitochondrial oxidative capacity *(31)*; these findings suggest that there could be race-related biological differences in mitochondrial characteristics that underlie differences in fat oxidation, resting metabolic rate, and insulin sensitivity. In a study of young adult black and white men, we demonstrated race-related variations in skeletal muscle oxidative metabolism using ^{31}phosphorous nuclear magnetic resonance spectroscopy. The results suggested a lower proportion of type 1 oxidative fibers and a higher proportion of type 2 glycolytic fibers in blacks, which explained their lower peak oxygen consumption *(32)*. It remains to be determined if similar black vs. white differences in skeletal muscle oxidative metabolism are present early in childhood.

RACE, OBESITY, AND BODY FAT DISTRIBUTION

Racial differences in body composition and body fat topography are well documented in adults. When matched for BMI, waist/hip ratio, or total adiposity, black adults have less visceral adipose tissue than their white counterparts *(33)*. This racial dimorphism in visceral adipose tissue accumulation is evident in prepubertal children *(34)*, and over time the growth in visceral fat is greater in white children compared with blacks with a difference of $\sim 1.9 \pm 0.8$ cm^2/year *(35)*. We and others have shown that black obese adolescents have significantly less visceral fat, measured by CT, than white peers with similar BMI and total body adiposity *(36,37)*. Furthermore, for a given BMI, whites have higher waist circumferences than blacks. Additionally, for a given waist circumference, blacks have more subcutaneous adipose tissue than white peers, with the magnitude of the difference increasing with increasing waist circumference *(37)*. Such information is of importance, given that visceral obesity plays an important role in the metabolic complications of obesity including insulin resistance, hypertension, dyslipidemia, type 2 diabetes, nonalcoholic fatty liver disease, and the metabolic syndrome *(36,38)*. Thus, racial differences in body composition and body fat topography could result in race-related differences in obesity co-morbidities and the metabolic syndrome.

Our research shows that for similar degrees of BMI and total body adiposity, white obese adolescents have a more atherogenic lipid risk profile, because of increased visceral adiposity, than blacks. Conversely, blacks have a heightened diabetogenic risk *(36)*. Triglyceride levels are substantially lower in black children and adults than in whites, making the application of a uniform cutoff of triglycerides for the metabolic syndrome questionable. Thus, using BMI alone may be misleading when children of different racial groups are evaluated, and future studies are needed to develop race-specific anthropometric criteria for obesity-related co-morbidities and health outcomes *(37)*.

RACE AND ENVIRONMENTAL/SOCIOCULTURAL DIFFERENTIAL IN RISK OF OBESITY

Obesity and Socioeconomic Status (SES)

Socioeconomic status (SES) is a risk factor for obesity. There is an inverse association between SES and obesity prevalence in children which is independent of parental weight status. The higher prevalence of obesity in lower SES children is assumed to be due to greater exposure to risk factors for positive energy balance both within the home (e.g., sedentary lifestyles, energy-dense diet, and permissive parental-feeding styles) and in the local neighborhood (e.g., availability of fast foods, limited access to safe, and pleasant areas for physical activity) *(39,40)*. In environments that facilitate positive energy balance – as is likely to be true in lower SES environments – genetically susceptible individuals will show even higher weight gain.

Parental SES is associated inversely with childhood obesity among whites: 40% of poor white women and 34% of poor white men were obese in the 1999–2002 NHANES survey, compared with 23% of white women and 14% of men with family incomes greater than 400% of the poverty line. In contrast, higher SES does not appear to protect black children against obesity. In this group, childhood obesity is not associated significantly with parental income and education *(41)*. Nevertheless, obesity rates in whites vary inversely with educational status and are significantly higher in high school dropouts than in high school graduates and twice as high as those in college graduates *(42,43)*.

Obesity and Sociocultural Differences

Sociocultural factors play an important role in shaping body image and desirable weight and may contribute to racial/ethnic differences in obesity. Body image is a multidimensional concept thought to influence the desire to lose weight and the self-perception of body size *(44)*. Racial/ethnic differences in weight misperception have been reported; misperception among overweight people (that is belief among overweight people that they are healthy weight) was more common in blacks than in whites and more common in men than women *(45)*. In addition, several studies demonstrate black/white differences in consumption of regular soda, high-fat foods, and fast-food restaurant use, which could be culturally mediated, enhancing the risk of obesity in black children (Table 1) *(46,47)*. Limited evidence suggests that blacks are more consistently exposed to food promotion and distribution patterns that promote adverse health effects *(48)*. Thus, ethnic, cultural, environmental, and economic factors might enhance the inherent/genetic predisposition of black youth to obesity.

Table 1
Cultural Differences in Black Adolescents

Heavy black adolescents do not perceive themselves heavy
More black girls express a desire to be on the fat side
Black children have higher total fat and cholesterol intake
Black children prefer sweet taste in liquids
Black girls have higher total energy intake
Black children are physically less active
Black girls spent more time watching television
Black high school students have the highest consumption of sugar-sweetened beverages, high-fat foods, and the highest frequency of fast-food restaurant use

RACE AND CO-MORBIDITIES OF OBESITY

Numerous co-morbidities are associated with childhood obesity, which serves as a segway to adult morbidity and mortality *(49)*. Here we provide information on race-related differences, mostly focusing on black vs. white children.

Race and Nonalcoholic Fatty Liver Disease

Nonalcoholic fatty liver disease (NAFLD) encompasses a wide range of liver damage, from simple steatosis to steatohepatitis, advanced fibrosis, and cirrhosis *(50)* and is the most common cause of pediatric liver disease *(51)*. The prevalence of NAFLD increases with age and varies by race *(52)*. NAFLD prevalence is lowest in blacks, followed by whites, then Asians (variability among the subgroups), and is highest in Hispanics *(52,53)*. The odds of a Hispanic child having fatty liver is five times greater than that of a black child *(52)*. The prevalence of cryptogenic cirrhosis is 3.1-fold higher in Hispanic Americans compared with European Americans and 3.9-fold lower in black Americans compared with European Americans *(53,54)*. It is felt that NAFLD may play an under-recognized role in cryptogenic cirrhosis *(53,55)*. The lower prevalence of NAFLD in blacks is counterintuitive given their increased risk for obesity and insulin resistance *(51)*. However, it might be explained in part by the smaller visceral adipose tissue compartment and the lower triglyceride levels (see later).

Race and Lipids

Black–white differences in lipids and lipoproteins occur early in childhood and are clearly established by 9 years of age *(56)*. Few studies have been carried out in neonates; these have not found significant differences between blacks and whites *(56,57)*. By age 2, total serum cholesterol approaches young adult levels and further dynamic changes occur with sexual maturation, establishing adult patterns *(56,57)*. During sexual maturation, the most striking difference occurs in white males, with progressive increases in the ratio of LDL-C to HDL-C *(56)*. In both childhood and adolescence, blacks have lower triglyceride and higher HDL levels compared with whites *(57)*. Increased prevalence of hypertriglyceridemia is also seen in Mexican Americans *(1,58,59)*.

The elevated triglyceride level in whites has been related to an increased large VLDL subclass in comparison to blacks *(60)*; however, racial differences in particle sizes may be attributed to levels of triglyceride and HDL cholesterol *(61)*. Our research demonstrates that there are significant black/white differences in lipoprotein particle size and concentrations in childhood. However, after adjusting for visceral adiposity differences between black and white children, only VLDL size and concentration remain significantly favorable in blacks *(62)*. Moreover, analysis of lipoprotein particle size and concentration across in vivo insulin sensitivity quartiles revealed that in both racial groups, the most insulin-resistant children had higher concentrations of small dense LDL, small HDL, large VLDL, and smaller LDL and HDL sizes than their more insulin-sensitive counterparts *(62)*.

Race and Cardiovascular Disease

Though the repercussions of cardiovascular disease are not usually seen until adulthood, the initiation of cardiovascular disease occurs early in life and childhood risk factors are associated with adult cardiovascular disease *(63)*. The fatty streak is the earliest lesion of atherosclerosis and has been found in aortas of children as young as 9 months of age and invariably after the age of 3 years *(64)*. By the age of 20, raised lesions have already appeared in the coronary arteries and are implicated in clinical disease *(57,65)*. In adolescence and early adulthood, blacks have more aortic fatty streaks than whites, with 1.5 times greater surface involvement; this cannot be attributed to antemortem cardiovascular risks *(64–66)* and is not as obvious in the coronary arteries *(66)*. Interestingly, white adults have more extensive raised lesions in the aorta than blacks *(65)* and have significantly higher rates of coronary

heart disease *(57)*. Thus the progression to advanced lesions from fatty streaks likely differs between races; the mechanisms are yet to be elucidated *(65)*.

Race and Prediabetes and Type 2 Diabetes (T2DM)

Based on population-based registries, the rates of type 1 diabetes are typically lower in black than white children *(67)*. On the other hand T2DM is disproportionately higher among blacks, Hispanics, American Indians, and Asians/South Pacific Islanders *(1,67–71)*. The earliest reports from pediatric diabetes clinics found that 68–100% of children with T2DM were black *(71–73)*. In the more recent SEARCH for Diabetes in Youth Population Study, T2DM in 10- to 19-year-olds varied by ethnicity: 33% of patients were black, 76% American Indian, 40% Asians/Pacific Islanders, 22% Hispanic youth, and 6% white *(74)*. The TODAY (Treatment Options for type 2 Diabetes in Adolescents and Youth) study found that 36% of adolescents with type 2 diabetes are black, 35% Hispanic, and 19% white *(75)*.

Reported rates of *prediabetes* [i.e., impaired fasting glucose (IFG) *and/or* impaired glucose tolerance (IGT)] in obese children vary tremendously, from 2 to 40%, depending on the population. Adolescent data from the NHANES 2005–2006 using fasting and 2-h glucose during an oral glucose tolerance test revealed unadjusted prevalence rates of IFG, IGT, and prediabetes of 13.1, 3.4, and 16.1%, respectively *(76)*. Blacks had lower rates than non-Hispanic whites and Mexican Americans (IFG: 9.7, 14.1, and 14.3%, respectively; IGT: 0.9, 3.7, and 3.5% respectively, prediabetes: 10.3, 17.2, and 16.9%, respectively). The IFG prevalence of 13.1% among US adolescents in 2005–2006 was ~87% higher than the 7% estimated from NHANES 1999–2000 (13% in Mexican Americans, 7% in whites, and 4.2% in blacks) *(77)*. The lower prevalence rate in blacks is surprising considering their increased risk of obesity and insulin resistance. However, the data included adolescents who were normal weight and overweight without separation for only obese adolescents of different racial groups. The results of the Studies to Treat or Prevent Pediatric Type 2 Diabetes (STOPP-T2D) revealed that the prevalence of IGT was 4.1% among overweight eighth graders in four middle schools *(78)*. In contrast to the NHANES findings, the prevalence rates of IGT were highest in Native Americans and Hispanics, followed by blacks and lowest in whites (7.3, 3.2, 1.3, and 0.9%, respectively). Prevalence of IFG was lowest in blacks (32.4%) and highest in Native Americans and Hispanics (~45%). In the Princeton School District of Cincinnati rates of IGT were much lower, detected among only 0.5% of 5th–12th graders and more in non-Hispanic whites than blacks or whites *(79)*. These highly variable estimates, whether overall or race specific, may stem from sampling and geographic variations.

Race and the Metabolic Syndrome

The prevalence of the metabolic syndrome varies depending on the definition used in the literature. Applying four different criteria to a well-characterized population of children, we demonstrated that the prevalence of the metabolic syndrome was significantly higher in overweight youth compared with non-overweight black and white youth *(80)*. The prevalence of metabolic syndrome was 31.3% in overweight black and 42.9% in overweight white youth, compared with 0.7% in non-overweight blacks and 2.8% in non-overweight whites. In overweight Hispanic children age 8–13 years, the prevalence of three or more features of the metabolic syndrome was 30% *(81)*. Path analyses of metabolic syndrome components in black and white children, adolescents, and adults revealed that path coefficients were generally greater in whites than blacks; this may account for the greater prevalence of metabolic syndrome in whites *(82)*. Importantly, individual components of the metabolic syndrome differ between races, especially abdominal adiposity and triglycerides (see previous sections for further details) *(83)*. Triglyceride levels increase significantly with enlarging waist circumference in whites but not in blacks (Fig. 3) *(84)*.

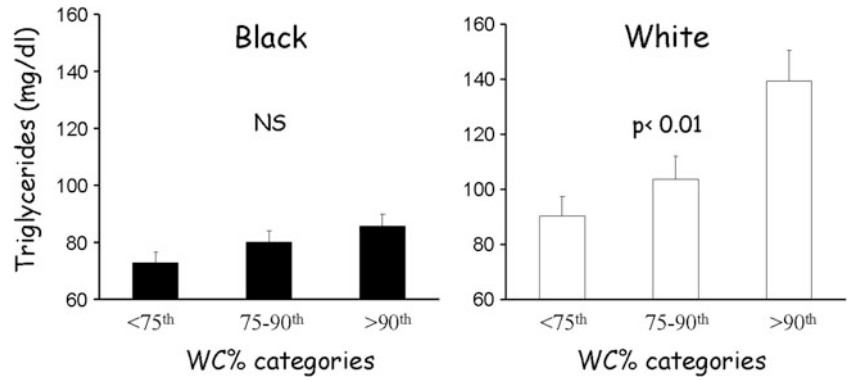

Fig. 3. Triglyceride concentrations according to waist circumference (WC) categories in black and white youth; NS, not significant. Adapted with permission *(84)*.

In a study by Weiss et al., the prevalence of metabolic syndrome was lower in blacks than whites when the groups were analyzed using the same criteria for lipid levels; however, when lipid thresholds specific to blacks were used in the analysis, the prevalence of metabolic syndrome in blacks was comparable to that in whites *(85)*. Independent of race, however, our research demonstrates that visceral obesity, insulin resistance, hyperinsulinemia, and hypoadiponectinemia are the common characteristics of youth with the metabolic syndrome *(80)*. Irrespective of race, the prevalence of large waist circumference, high triglycerides, low HDL, high blood pressure, and prediabetes is highest among youth in the lowest quartile of in vivo insulin sensitivity (Fig. 4). However, in white but not black children, the metabolic syndrome is associated with increased inflammatory markers (Fig. 5) *(83)*. The translation of such race-related differences remains to be determined based on long-term longitudinal outcome studies in different racial groups. Further research in childhood metabolic syndrome is needed not only to unify the definition of the syndrome, but also to test the validity of race-specific criteria.

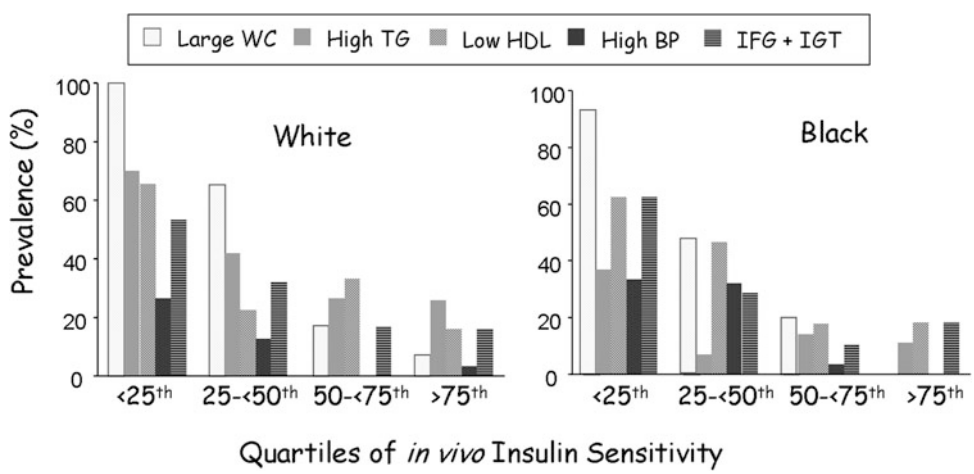

Fig. 4. The prevalence of the individual components of metabolic syndrome by quartiles of in vivo insulin sensitivity in black and white youth. (WC, waist circumference; TG, triglycerides; BP, blood pressure; IFG, impaired fasting glucose; IGT, impaired glucose tolerance.) Adapted with permission *(83)*.

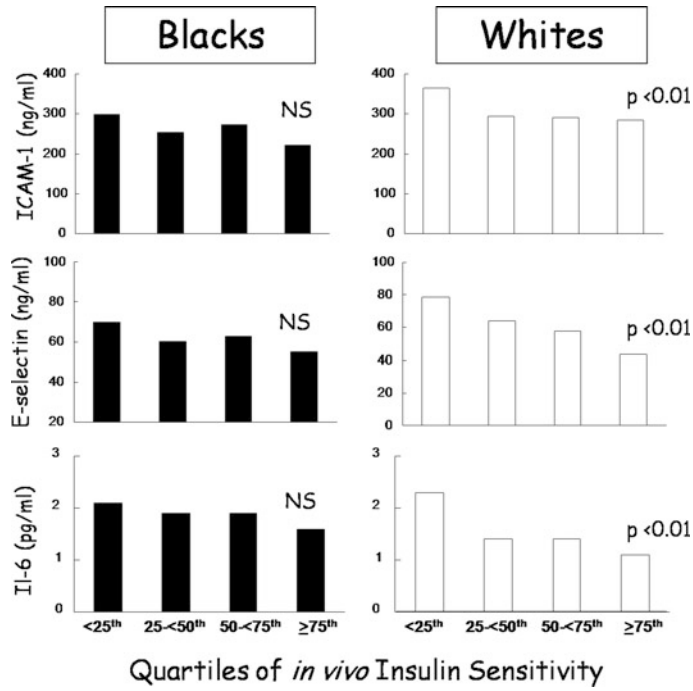

Fig. 5. Circulating biomarkers of endothelial dysfunction and IL-6 in black and white youth according to quartiles of insulin sensitivity. Adapted with permission *(83)*.

RACE AND THE TREATMENT OF OBESITY

Considering that racial differences (biological, environmental, or both) influence the risks of childhood obesity and its complications, it may seem intuitive that obesity treatments be individualized to specific racial/ethnic groups. Though guidelines exist for the treatment of obese children (see *(86)* and treatment sections of this book), there is currently no justification for recommending race-specific treatment *(1)*. Limited data suggest that white adolescents may have more weight loss with either metformin or the combination of sibutramine (an appetite suppressant that inhibits the reuptake of norepinephrine and serotonin) and behavioral therapy than black adolescents *(1)*. In adults, blacks lose less weight after gastric bypass surgery than whites but have comparable improvements in cardiovascular risk factors; these differences in weight loss post-gastric bypass may be related to differences in energy expenditure rather than dietary intake *(1)*. It is unclear whether these racial differences would be replicated in large randomized, controlled studies or whether they reflect the influence of genetics as opposed to cultural and environmental factors. In any case, providers caring for obese children must consider many factors when making treatment decisions, including differences in the perception of obesity, which may play a role in motivation; access to healthcare; availability and affordability of treatment for obesity (lack of insurance is more common in Hispanics than blacks and more common in blacks than whites); dietary preferences; and physical activity preferences *(1)*.

CLOSING REMARKS AND FUTURE DIRECTIONS

Increases in obesity in childhood and adulthood will result in major morbidity and mortality and will inflate health care costs at a time of severe financial deficits in the US economy. The medical profession and its allies may very well be able to advance research in the field of obesity, but what is needed to

stop the epidemic of obesity is societal change. The latter will require major policy change, akin to the smoking campaign, combined with economic change and incentives and education. Racial/ethnic factors must be considered in the overall approach to prevention and treatment of childhood obesity.

ACKNOWLEDGMENTS

This work was supported by the United States Public Health Service grant RO1 HD27503 (SA), K24 HD01357 (SA), MO1 RR00084 (GCRC) and UL1 RR024153 (CTSA), T32 DK063686 (CD), and T32 DK007729 (JH).

Editor's Questions and Authors' Response

- **Do you think that differences in rates of pubertal maturation among ethnic groups could contribute to differential rates of obesity or co-morbidities?**
- No. Actually I believe that differences in adiposity are important determinants of the onset of puberty and its tempo. Differences in rates of pubertal maturation exist among ethnic groups with black girls tending to enter puberty and experience menarche about 1 year before white girls. Additionally, increased body mass index is associated with earlier sexual maturation. It is difficult to delineate cause and effect, especially with numerous confounding variables. Irrespective, however, divergence in adiposity begins during adolescence and relates temporally to puberty. In one of our studies we showed that lower increases in fat oxidation between prepubertal and pubertal black girls (puberty is characterized by higher rates of fat oxidation) could predispose them to faster weight gain compared with their white peers. The definitive answer to your question requires longitudinal studies to follow children from prepuberty to completion of puberty in different racial groups.

- **Do we have any data that compares the caloric intake or macronutrient composition of diets of the various ethnic groups?**
- Dietary intake trends are affected by many variables including socioeconomic status, education, environment, food availability, individual preference, and culture. In children and adolescents, the National Health and Nutrition Examination Surveys (NHANES) revealed no clear difference in overall energy intake among racial groups; however, the percentage of energy from total fat was highest among non-Hispanic blacks followed by Mexican Americans then non-Hispanic whites without significant difference in saturated fatty acids (Troiano et al., 2000). According to self-reported dietary recalls, Mexican American children aged 9 years ate higher than recommended fat servings and had higher percent energy from fat and saturated fat and their daily fruit and vegetable intake was half of that recommended by national guidelines (Trevino et al., 1999).

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