Lipids, Lipases, and Obesity
Does Race Matter?

Peter W.F. Wilson

For >20 years, researchers have reported that American blacks tend to have lower triglyceride and higher HDL cholesterol (HDL-C) levels compared with whites at a similar level of corpulence. Early studies focused on potential environmental explanations and concluded that differences in exercise, alcohol intake, leanness, and undernutrition did not readily account for the higher HDL-C levels. It was also noted that the propensity toward more favorable HDL-C and triglyceride levels was absent in highly educated blacks. More recent reports have shown that socioeconomic status was positively correlated with HDL-C in white men and women, a negative association was present in black men, and no association was evident in black women.

Autopsy reports have also demonstrated differences in atherosclerosis in blacks versus whites. The Bogalusa Study reported that young blacks had more fatty streaks than did whites but that middle-aged blacks had less evidence of fibrous plaques. The authors concluded that the transition of fatty streaks to advanced atherosclerotic lesions may differ in whites and blacks. Similar results were obtained in the Pathobiological Determinants of Atherosclerosis in Youth study, an autopsy investigation of young adults who died from trauma between the ages of 15 to 34 years. Adolescent blacks in that study had a greater extent of fatty streaks than did whites, but older blacks and whites had a similar extent of raised lesions.

The associations of sex, race, obesity, and fat metabolism have been under intense study as coronary heart disease mortality has declined, but Americans have ballooned in size over the past 2 decades. This article in this issue of Arteriosclerosis, Thrombosis, and Vascular Biology by Després et al highlights metabolic differences in a systematic comparison of lipids, obesity, and lipase activity in a well-designed multicenter North American study that included blacks and whites.

Després et al demonstrate effectively that blacks are less likely than whites to be viscerally obese. In addition, plasma hepatic lipase activity is less and lipoprotein lipase activity is greater in blacks after intravenous heparin infusion. These results suggest indirectly that a fat load can be cleared more efficiently by blacks than by whites. The findings are muted by multivariate regression analysis, but the primary result is unassailable: lipolytic activity is greater and visceral obesity is less in the black participants.

Cardiovascular risk factor reports in obese individuals have recently demonstrated a remarkable number of metabolic abnormalities that embrace differences in lipids, glycemia, insulin, blood pressure, and hematologic function. We now have several names for syndromes that are similar and include the clustering of metabolic risk factors. In fact, Sniderman et al have suggested that hyperapolipoproteinemia B, familial combined hyperlipidemia, syndrome X, the plurimetabolic syndrome, the visceral fat syndrome, familial dyslipidemic hypertension, the atherogenic lipoprotein phenotype, and the deadly quartet share a common pathophysiology.

There is a need for improved metabolic profiling of obese persons that participate in larger surveys. We already have dynamic testing for glycemic responses to oral and intravenous glucose loads, and a variety of techniques are available to assess glucose and insulin response. Population research suggests that a 2-hour oral glucose tolerance is adequate for many studies, and fasting insulin is key in the study of insulin resistance.

Fat-load studies are a traditional way to characterize fat metabolism, but protocols are somewhat cumbersome and typically take at least 4 hours after the initial fat load. Fat-loading data suggest differences between whites and blacks. For instance, carotid intimal medial thickness was positively associated with postprandial responses of triglycerides and triglyceride-rich lipoproteins in white men and women but not in blacks in the Atherosclerosis Risk in Communities study. These results were obtained only in persons with a body mass index <30 kg/m². The postprandial lipid associations were not statistically significant after adjustment for fasting lipids, and the authors concluded that postprandial triglycerides must be accompanied by the accumulation of triglyceride-rich lipoprotein remnants to be atherogenic. A recent fat clearance study in young adults found that blacks cleared a fat load more efficiently than did whites. In that investigation, postprandial triglycerides were negatively correlated with lipoprotein lipase in blacks and whites, whereas postprandial triglyceride response was positively correlated with hepatic lipase activity in whites and negatively correlated in blacks.

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Newer assays can determine the densities for a spectrum of lipid particles by nuclear magnetic resonance testing or gradient gel electrophoresis. These techniques have the promise to be helpful in characterizing population groups. Also available are assays of triglyceride-rich lipid remnants, which can be measured in the fasting state. Preliminary studies suggest that higher levels of triglyceride-rich remnant particles are positively associated with a greater prevalence of coronary disease.

Després et al. used postheparin lipase profiling in their study and compared hepatic lipase and lipoprotein lipase activities after a heparin infusion. Because the study highlights differences in visceral obesity and lipase activity that appear to be based on ethnicity, it is natural to consider the importance of the genetic underpinnings for such findings. Are blacks more likely to have specific genetic variations in lipases that might help to explain the lipid and visceral tendencies? Several polymorphisms are under study, and research is focusing on the cholesteryl ester transfer protein, apolipoprotein AI/CI/II/AIV, and hepatic lipase loci. A polymorphism within the hepatic lipase promoter (−514C→T), which is present in ≈36% of middle-aged Framingham Heart Study participants, has been relatively consistently associated with higher HDL-C, and it has been suggested the HDL3 fraction is particularly increased. In addition, Vega et al. have reported that the −514T hepatic lipase allele is more common in blacks than in whites. This genetic variant, and potentially others, may help to explain the relative advantage that blacks have for higher HDL-C, although this polymorphism may not be key to the effects. Linkage disequilibrium with another gene or another polymorphism may be responsible for the phenotypic expression.

Although coronary mortality has declined over the past 3 decades, our society is still unsettled by a large burden of coronary artery disease and stroke. There have been declines in blood pressure, cholesterol, and cigarette smoking, but as we have rallied forces to battle these risk factors, we have been outflanked by the adipocyte. Recently published National Health and Nutrition Examination Survey III data, undertaken between 1988 to 1994, demonstrate obesity (>30 kg/m²) in ≈22% of white and black men and in ≈24% and 38% of white and black women, respectively. These national survey data, taken with the results from the report of Després et al., suggest that environmental factors may be removing any natural advantage that blacks may have toward more favorable lipid profiles. Efforts to control lipid levels and vascular disease risk in blacks and whites need to consider the current obesity epidemic, recognizing that the age-adjusted prevalence of obesity in the United States is still on the rise.

References


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