DYSLIPIDEMIA AND INSULIN RESISTANCE IN RELATION TO GENETIC ADMIXTURE AMONG HISPANICS AND NON-HISPANIC BLACKS OF CARIBBEAN ORIGIN

Eugene S. Tull, DrPH, MT and Ann Thurland, MPH
Pittsburgh, Pennsylvania and St. Croix, U.S. Virgin Islands

The tendency to develop insulin resistance and dyslipidemia varies between black Africans, white Europeans, and Amerindian racial groups. Genetic admixture of these three racial groups has resulted in hybrid populations of Hispanics and non-Hispanic blacks. The current study was undertaken to examine the relationship of white European admixture to insulin resistance and dyslipidemia among Hispanics and non-Hispanic blacks of Caribbean origin. The study population included 224 Hispanics and 684 non-Hispanic blacks without a history of diabetes who were recruited between 1995 and 1999 on the island of St. Croix in the U.S. Virgin Islands. For each participant, anthropometric measurements were performed, and a fasting blood sample was analyzed for glucose, insulin, and serum lipids (triglycerides, HDL cholesterol). Genetic admixture was determined from grandparent race data. Hispanics were more likely than non-Hispanic blacks to have dyslipidemia and insulin resistance (measured by HOMA-IR method). White European admixture was significantly and inversely associated with insulin resistance among Hispanics. There was no significant relationship between white European admixture and insulin resistance or dyslipidemia among non-Hispanic blacks. Individuals who classified themselves as Hispanic blacks appeared to have a greater risk for cardiovascular disease than Hispanic whites or non-Hispanic blacks. (*J Natl Med Assoc.* 2004;96:332–340.)

Key words: dyslipidemia ♦ insulin resistance

INTRODUCTION

In the United States, African Americans and Mexican Americans are more insulin resistant than non-Hispanic whites[1,2]. Consistent with their greater insulin resistance, Mexican Americans have a higher fasting level of triglycerides and lower level of HDL cholesterol (HDLc), compared to non-Hispanic whites.[3-5] However, despite greater insulin resistance, African Americans (particularly men) have a lower level of triglycerides and higher level of HDLc than non-Hispanic whites[4-6]. Similarly, people of African-Caribbean descent have a higher plasma level of HDL cholesterol and lower level of triglycerides compared to white Europeans[7,8]. These findings suggest that genetic factors may influence the extent to which insulin resistance contributes to dyslipidemia in the various ethnic groups.

Non-Hispanic blacks and Hispanic groups in the Caribbean are hybrid populations with gene pools derived from the mixture of genes from black African, white European, and Amerindian parent

 From the Virgin Islands Center for Chronic Disease Research, Department of Epidemiology, Graduate School of Public Health, University of Pittsburgh, Pittsburgh, PA (Tull) and the Division of Health Education and Prevention, Virgin Islands Department of Health, St. Croix, U.S. Virgin Islands (Thurland). Send correspondence and reprint requests for *J Natl Med Assoc.* 2004;96:332–340 to: Eugene S.Tull, 512 Paran Hall, 130 DeSoto Street, Pittsburgh, PA 15261; phone: (412) 624-2961; fax: (412) 624-2961; e-mail: drmirt@yahoo.com

populations.[9,10] Large numbers of these Caribbean-born Hispanics and non-Hispanic blacks have emigrated to the United States and reside in many of the large metropolitan areas. Little is known about the extent to which genetic admixture in these individuals influences their degree of insulin resistance or their plasma lipid profile. This report presents the results of a study that examined the association of white European genetic admixture with insulin resistance and the relationship of insulin resistance to dyslipidemia among Hispanics and non-Hispanic blacks of Caribbean origin.

MATERIALS AND METHODS

Study Population

The population for this study consisted of a randomly selected sample of individuals age ≥20 who participated in the Virgin Islands Diabetes Study (VIDS), a cross-sectional study of diabetes and associated risk factors that was conducted from February 1995 through December 1999 on the island of St. Croix in the U.S. Virgin Islands (USVI). A description of the sample selection and recruitment procedures for the VIDS has been recently published.[11] Study participants were recruited by in-home interview. The participation rate for the VIDS was ~77%. Each participant signed a consent form approved by the Biomedical Institutional Review Board of the University of Pittsburgh.

A total of 1,080 Caribbean-born persons participated in the VIDS. They were distributed according to the following ethnic groupings: 258 Hispanics, 781 non-Hispanic blacks, six non-Hispanic whites, three persons of Asiatic background, and 32 persons with various other ethnicities. For this report, persons with a prior history of diabetes were excluded from further analyses. The non-Hispanic whites were also excluded because of their small sample size. In addition, because the focus of the study was on Hispanics and non-Hispanic blacks, all other ethnic groups were excluded. A total of 71 nondiabetic non-Hispanic blacks who did not provide enough information for determining racial admixture were also excluded. A comparison of demographic and metabolic characteristics of these persons with those who were included showed that the group with missing information was older and had a significantly higher mean triglyceride level. However, after adjustment for age, the difference in mean triglyceride level was no longer significant between the two groups. Additionally, a small sample of Hispanic individuals (n=15) who did not classify themselves into a racial group were also excluded. The final consisted of 209 Hispanics and 613 non-Hispanic blacks.

Lifestyle Risk Factors

Face to face interviews were used to collect demographic data and information about lifestyle practices. Lifestyle variables included alcohol consumption, fast-food consumption pattern, smoking status, and physical activity. The measurement of alcohol consumption was based on a "yes" or "no" response to having consumed at least one alcoholic beverage during the past month. Smoking status was measured as a "yes" or "no" response to being a current smoker. Physical activity was assessed as the presence or absence of leisure time physical activity. Persons without leisure activities were characterized as having a "sedentary lifestyle".

Clinical Variables

Weight and height were measured with participants wearing light clothing and no shoes. Body mass index (BMI), a measure of obesity, was calculated as weight in kilograms (kg) divided by height in meters squared (m^2). The waist circumference was measured with a Gullic tape using standardized procedures.[12] Blood samples were drawn after an overnight fast of 10–12 hours and were measured for serum glucose, lipids, and insulin. Serum glucose was measured with a Kodak Ektachem 700 Analyzer (Eastman Kodak Company, Rochester, NY) using a glucose oxidase colorimetric method. Insulin was measured using an RIA procedure (Linco Research Inc.). Cross-reactivity with proinsulin was under 0.02%. Triglyceride level was determined enzymatically using the procedure of Bucolo et al.[13] HDL cholesterol (HDLc) was determined using selective precipitation of non-HDL lipoproteins by heparininmanganese chloride.[14] In the current study, values for HDL cholesterol and triglycerides were based on the following values vels of triglycerides and HDLc, including the following parameters: triglycerides ≥1.7 mmol/l (≥150 mg/dl) and HDL cholesterol level <1.0 mmol/l (<40 mg/dl) in men, or <1.3 mmol/l (<50 mg/dl) in women. Insulin resistance was estimated by the Homeostasis Assessment Model (HOMA-IR), which is calculated as {fasting glucose (mmol/L) x fasting insulin (μU/ml)}/22.5[16]. Hyperinsulinemia was defined as a fasting insulin value in the upper 25th percentile of the

distribution of insulin values in the study. This method of identifying hyperinsulinemia has been used in other studies[17]. The simultaneous presence of high triglycerides and low HDLc was classified as dyslipidemia.

Racial Admixture

Each participant was asked to identify the race of his/her grandparents, and this information was used to calculate a racial score (RSCORE). The lowest possible RSCORE was "1," which indicated that all grandparents were listed as being nonwhite, whereas the highest RSCORE was "4," which indicated that all grandparents were of white European ancestry. This method of estimating the proportion of white European admixture in nonwhites of Caribbean origin has been used in other studies in the USVI[18], and it has been shown that grandparent race can be used as a reliable estimate of the degree of racial admixture at the population level in epidemiological studies.[19]

Statistical Analysis

Statistical analyses were conducted using Statistical Analysis System (SAS) software[20]. Pair-wise comparisons of variables were performed with the χ^2 tests or t tests. Spearman correlation analyses were used to examine the strength of the relationship of the RSCORE to metabolic and anthropometric variables. When correlations were significant, partial correlation analyses were used to examine the degree to which other lifestyle or demographic factors might contribute to the relationship between RSCORE and the metabolic or anthropometric variable. The logarithm of the HOMA-IR scores was used in all analyses because of the skewed distribution of insulin values.

The distribution of HOM Analysis of covariance was used to assess the variation in mean level of serum lipids and potential confounders across levels of HOMA-IR.

RESULTS

Table 1 shows demographic characteristics and age-adjusted mean levels of metabolic variables for Hispanics and non-Hispanic blacks. The mean age, proportion of females, percent consuming at least

Table 1. Demographic Characteristics and Age- and Sex-Adjusted Means With 95% Confidence Intervals for Metabolic Variables Among Hispanics and Non-Hispanic Blacks Without a Prior History of Diagnosed Diabetes

Variable	Hispanic	Non-Hispanic Black	p Value
N	224	684	—
Age (mean ± SD)	47.2±15.5	46.1±15.2	0.3647
Female (%)	66.1	68.5	0.5593
Education ≥ high school (%)	52.7	63.3	0.0073
Current smoker (%)	13.5	4.6	<0.0001
Consumed alcohol in the past month (%)	35.0	33.9	0.8432
Sedentary lifestyle (%)*	27.5	28.6	0.7629
RSCORE †	1.53 (1.44–1.61)	1.17 (1.12–1.22)	<0.0001
BMI (kg/m^2)	28.3 (27.5–29.1)	28.9 (28.4–29.3)	0.2236
Waist (cm)	87.4 (85.3–89.6)	89.1 (87.8–90.3)	0.1906
Fasting glucose (mmol/l)	5.33 (5.12–5.55)	5.30 (5.18–5.43)	0.8223
Log fasting insulin (pmol/l)	2.86 (2.80–2.93)	2.80 (2.77–2.84)	0.1244
HDL cholesterol (mmol/l)	1.19 (1.16–1.23)	1.27 (1.24–1.29)	0.0010
Triglycerides (mmol/l)	3.16 (2.97–3.35)	2.33 (2.22–2.44)	<0.0001

* Percent of persons who report having no leisure time physical activity; † RSCORE estimates the level of white European admixture based on self-reported grandparental data.

one alcoholic beverage in the past month, and percent with a sedentary lifestyle were similar for the two groups. The Hispanic group had a lower percentage of persons completing high school and a higher percentage of current smokers. Among the metabolic variables, Hispanics had a lower mean HDLc level and a higher mean level of triglycerides than non-Hispanic blacks.

Association of Racial Admixture with Metabolic Variables

The results of Spearman correlation analyses showed that among Hispanics, the RSCORE was significantly and inversely correlated with fasting insulin (r=-0.155, p=0.0454) and lnHOMA-IR (r=-0.184, p=0.0171). For the non-Hispanic blacks, RSCORE was not significantly correlated with insulin resistance (r=-0.035, p=NS) or any other metabolic variable. After adjusting for age, gender, education, smoking, alcohol consumption, and sedentary lifestyle among Hispanic persons, RSCORE remained significantly correlated to lnHOMA-IR (r=-0.183, p=0.0268) but not to fasting insulin (r=-0.123, p=0.1124).

Relationship of Insulin Resistance to Dyslipidemia

To examine the association of insulin resistance with triglycerides and HDLc levels among individuals with varying degrees of white European admixture, the Hispanic and non-Hispanic black groups were each divided into two sub-groups. The Hispanics were divided on the basis of self-reported race into Hispanic blacks (n=151) and Hispanic whites (n=58), and the non-Hispanic blacks were divided into those who said they had some white admixture (n=156) and those who said they had no white admixture (n=457).

Table 2 shows the age-adjusted mean values for RSCORE and metabolic variables, together with the frequency of hyperinsulinemia and dyslipidemia. The mean level of self-reported white European admixture was higher among Hispanic whites and the non-Hispanic blacks with admixture (NHBwA) than Hispanic blacks. The Hispanic blacks had a higher mean lnHOMA-IR score and a higher frequency of hyperinsulinemia than the other groups. Individuals with Hispanic ethnicity were more than twice as likely to have dyslipidemia, compared to non-Hispanic blacks.

Figure 1 shows the mean level of triglycerides, (adjusted for age, gender, education, alcohol consumption, smoking, sedentary lifestyle, and BMI) across tertiles of lnHOMA-IR score. In all groups, the mean triglyceride level increased ($p<0.05$) within each ascending tertile of lnHOMA-IR but remained significantly higher for Hispanics within each tertile. However, the percentage increase in mean triglyceride level from the first to the third tertile of lnHOMA-IR values was not significantly different between the groups. At the lowest tertile of HOMA-IR, the mean HDLc level for Hispanic blacks was

Table 2. Age-Adjusted Mean Values With 95% Confidence Intervals for White Caucasian Admixture* (RSCORE) and Metabolic Variables Among Hispanics and Non-Hispanic Blacks

Variable	Hispanic Individuals†		Non-Hispanic Black Individuals	
	White	Black	With Admixture*	Without Admixture*
N	58	151	156	437
RSCORE	1.88 (1.74–2.01)[b,d]	1.40 (1.32–1.48)[a,c,d]	1.83 (1.74–1.91)[b,d]	1.00[a,b,c]
HDLc (mmol/l)	1.13 (1.06–1.21)[c,d]	1.20 (1.16–1.25)[c,d]	1.28 (1.23–1.33)[a,b]	1.26 (1.24–1.29)[a,b]
Triglycerides (mmol/l)	3.20 (2.2–3.58)[c,d]	3.14 (2.90–3.37)[c,d]	2.21 (1.98–2.45)[a,b]	2.33 (2.19–2.46)[a,b]
Log of HOMA-IR	1.23 (1.08–1.38)[b]	1.46 (1.37–1.55)[a,c,d]	1.27 (1.18–1.36)[b]	1.33 (1.28–1.39)[b]
Hyperinsulinemia (%)	22.4	32.4[c,d]	18.5[b]	22.3[b]
Dyslipidemia (%)	20.7[c,d]	18.5[c,d]	3.9[a,b]	7.0[a,b]

*Admixture estimates based on self-reported race of grandparents; a higher RSCORE score is indicative of a greater degree of white caucasian admixture; † Racial classification among Hispanics was based on self-report; a = values are significant at $p<0.05$, compared to white Hispanics; b = values are significant at $p<0.05$, compared to black Hispanics; c = values are significant at $p<0.05$, compared to non-Hispanic blacks with self-reported white European admixture; d = values are significant at $p<0.05$, compared to non-Hispanic blacks without self-reported white admixture.

similar to that of non-Hispanic blacks and higher ($p<0.05$) than that of Hispanic whites (Figure 2). Among the Hispanic blacks, HDLc levels declined dramatically and significantly across increasing tertiles of HOMA-IR. There was a slight but nonsignificant decline in mean HDLc level across increasing tertile of HOMA-IR for non-Hispanic blacks. The mean HDLc level for Hispanic whites was lowest among the groups in each tertile of HOMA-IR but did not decline significantly as HOMA-IR increased.

DISCUSSION

The results of the current study show that Caribbean-born non-Hispanic blacks have higher fasting plasma levels of HDLc and lower levels of triglycerides compared to Caribbean-born Hispanic individuals. These findings are consistent with other reports[5,6]. The reason for the more favorable lipoprotein profile of non-Hispanic blacks is not clear. The biochemical processes that make individuals with black African ancestry less responsive to an atherogenic diet appear to be present at birth.[21] Zoratti and colleagues[22] examined lipoprotein metabolism in African-Caribbean, European, and South Asian men and found that the ethnic differences in HDLc and triglycerides were not accounted for by differences in nonesterified fatty acid (NEFA) levels, the sensitivity of suppression of NEFA levels to insulin, or body composition. They concluded that the more favorable lipoprotein profile of African Caribbeans may relate to more effective VDRL metabolism. Compared to non-Hispanic whites, African Americans have been shown to have a three-fold increased frequency of the -514T hepatic lipase allele, which is associated with higher levels of HDL cholesterol[23]. Polymorphisms of the apolipoprotein D (APOD) gene that are unique to populations with black African ancestry are associated with plasma levels of HDLc and triglycerides[24]. Additional studies are needed to determine if these genetic factors can account for the decreased tendency toward dyslipidemia in non-Hispanic blacks, compared to other ethnic groups.

Individuals of white European ancestry are less insulin resistant and have lower levels of fasting glucose compared to non-Hispanic blacks and Amerindian groups, like the Pima Indians[25]. A recent study showed that white European genetic admixture was significantly and inversely associated with plasma glucose level among Pima Indians[26]. In the current study, white European genetic admixture was inversely correlated with the level of insulin resistance in Hispanic individuals and non-Hispanic blacks, but the correlation coefficient was significant only for the Hispanic group. One explanation for this finding is that the two groups differ in their perceptions of who should be classified as being white. It may be that non-Hispanic blacks were more likely to list lighter complexion grandparents (who may have been less insulin resistant) as being black, thereby reducing the likelihood of finding a significant relationship between admixture and insulin resistance. Another possibility is that the significant relationship between white European genetic admixture and insulin resistance in Hispanic individuals is due to a relatively stronger association of nonwhite admixture with insulin resistance in that group.

The assessment of the relationship of insulin resistance to plasma lipids among individuals with varying degrees of self-reported genetic admixture highlights the complex nature of the expression and interaction of metabolic factors that result from genetic admixture of several ethnic groups. In the current study, fasting levels of triglycerides among individuals who characterized themselves as Hispanic blacks and Hispanic whites were similar and significantly different from non-Hispanic blacks. However, Hispanic blacks were more insulin resistant than Hispanic whites or non-Hispanic blacks. Furthermore, after adjusting for the degree of insulin resistance, the plasma HDLc levels for Hispanic blacks were higher than those of Hispanic whites and similar to those for non-Hispanic blacks. Yet, Hispanic blacks differed from non-Hispanic blacks in that their HDLc levels declined significantly and more dramatically with increasing insulin resistance. The majority of Hispanic individuals in the USVI have ancestral origins on the island of Puerto Rico where one estimate[10] suggests that 45%, 37%, and 18% of the gene pool are Spanish, African, and Amerindian derived, respectively. The degree of Amerindian genetic admixture in Hispanic groups has been linked to their higher risk of diabetes, compared to non-Hispanic whites.[27,28] It might also be the case that the greater insulin resistance in Hispanic blacks reflects a higher degree of Amerindian genetic admixture; whereas, compared to Hispanic whites, their higher HDLc level is indicative of more African ancestry. There was no apparent difference in the relationship of insulin resistance to

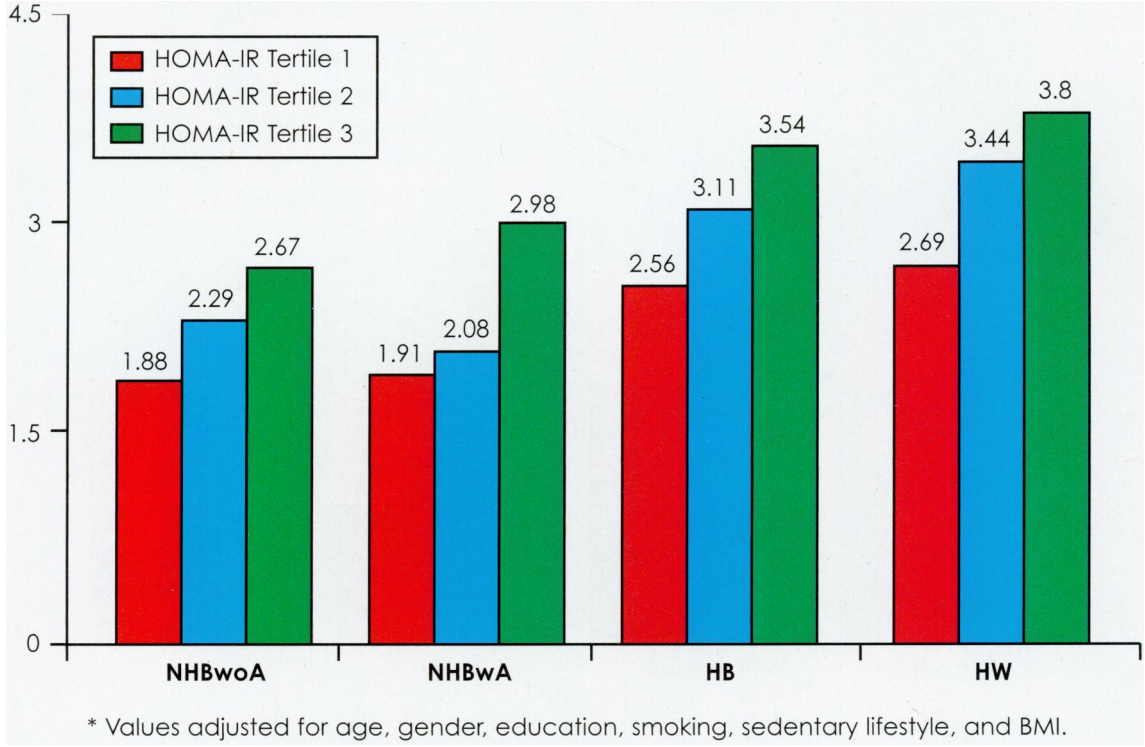
plasma levels of triglycerides and HDLc between non-Hispanic blacks with and without self-reported white European admixture. However, this finding does not exclude a relationship between white European admixture and dyslipidemia in non-Hispanic blacks, since it is possible that the use of other methods of assessing genetic admixture—such as skin reflectance or genetic alleles—may have produced contrasting results[19].

As reported elsewhere,[21,29,30] the more favorable lipid profile of non-Hispanic blacks was superimposed on the background of insulin resistance. This reduced tendency to dyslipidemia, even in the presence of greater insulin resistance, contributes to the lower frequency of the metabolic syndrome seen in African Americans compared to European Americans[4]. However, despite their favorable lipoprotein profile, African Americans have higher rates than European Americans of cardiovascular disease (CVD).[31,32] In contrast, African Caribbeans in the United Kingdom are reported to have lower rates than white Europeans of CVD.[33,34] A higher prevalence of traditional CVD risk factors, including hypertension, diabetes, physical inactivity, and smoking, is thought to contribute to the excess CVD risk in African Americans.[35] It would be interesting to know if CVD rates are lower for non-Hispanic blacks of Caribbean origin who reside in the United States, compared to native-born African Americans or European Americans.

The current study has some limitations and strengths. A limitation of this study is the cross-sectional design, which does not allow for inferences about causal relationships between study variables. Another limitation is the small sample size of the various admixed groups, which prevented more extensive analyses by gender. The use of grandparent information to determine genetic admixture is also a limitation.

The classification of race based on phenotypical characteristics is subjective, and each person may have his/her own criteria for assigning a grandparent to a particular racial group. Notwithstanding the limitations, in the absence of more specific bio-

Figure 1. Mean Level of Plasma Triglycerides by Tertiles of HOMA-IR Among Non-Hispanic Blacks Without Self-Reported Admixture (NHBwoA), Non-Hispanic Blacks with Self-Reported Admixture (NHBwA), Hispanic Blacks (HB), and Hispanic Whites (HW)



logical markers, the determination of admixture by grandparental race is a useful tool that can provide clues about the involvement of genetic factors in disease. Additional studies of the current populations using allele frequencies to assess admixture are recommended. Strengths of the study include the population-based sampling strategy and the presentation of the data on Hispanics by categories based on racial classifications.

Clinical Implications

Because of the close association between overweight or obesity and adverse health outcomes like type-2 diabetes and coronary heart disease, individuals who fall into these body weight categories are usually targeted for risk-reduction therapy. Lifestyle modification or medication designed to facilitate weight loss is often the key component of the therapeutic regimen. However, studies suggest that weight loss may produce the greatest metabolic benefits in overweight people who are insulin resistant.[36] Reaven[37] has argued that individuals who are overweight and insulin resistant may be the best candidates for weight reduction programs and that they can be identified in clinical settings by screening for the presence of dyslipidemia and hypertension. The current study has shown that individuals who classify themselves as Hispanic blacks have a greater tendency to be insulin resistant, compared to other groups. Therefore, in the USVI, the overweight patient who is an Hispanic black may be a candidate of choice for initiation of intensive weight loss and CVD risk-reduction therapy.

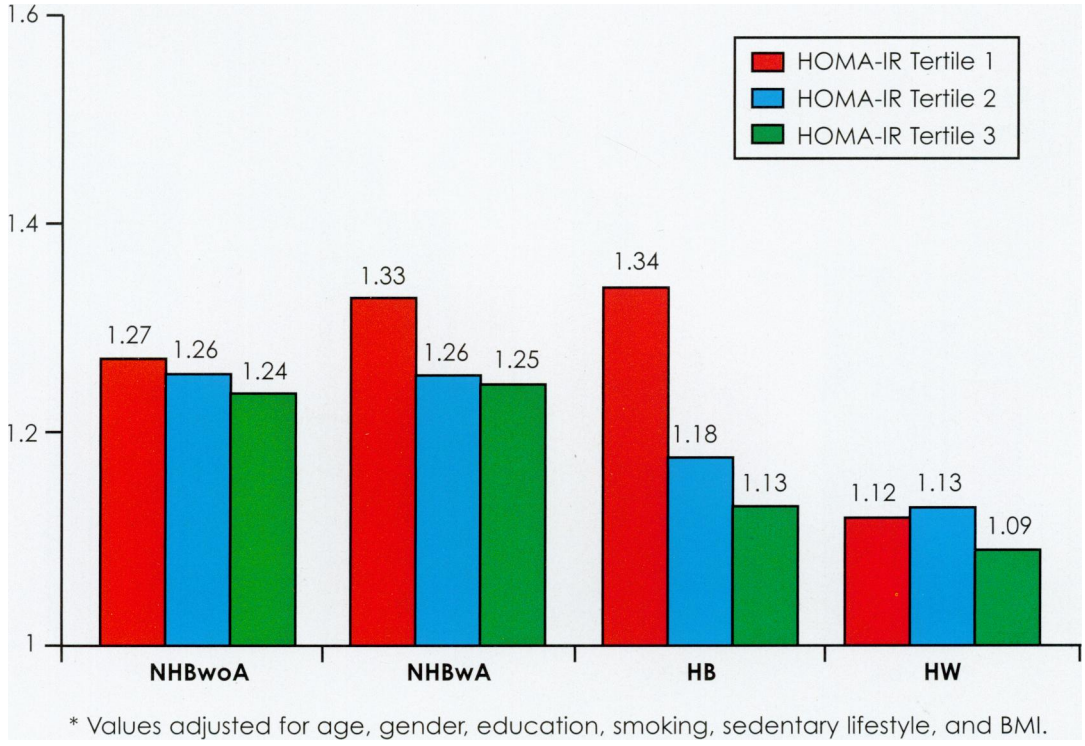
ACKNOWLEDGEMENT

This study was supported by a grant from the National Institute for Diabetes, Digestive, and Kidney Diseases (1 RO1 DK46502). Sincere thanks to the staff of the Virgin Islands Center for Chronic Disease Research and the St. Croix Hospital Laboratory for their assistance, and to the people of the U.S. Virgin Islands for their participation.

REFERENCES

1. Haffner SM, D'Agostin R, Saad MF, et al. Increased insulin resistance and insulin secretion in nondiabetic African-Americans and Hispanics compared with non-Hispanic whites. The Insulin Resistance Atherosclerosis Study. *Diabetes.* 1996;45:742-748.

Figure 2. Mean Level of HDL Cholesterol by Tertiles of HOMA-IR Among NHBwoA, NHBwA, HB, and HW



2. Chui KC, Chuang LM, Yoon C. Comparison of measured and estimated indices of insulin sensitivity and β-cell function: impact of ethnicity on insulin sensitivity and β-cell function in glucose-tolerant and normotensive subjects. *J Clin Endocrinol Metab.* 2001;86:1620-1625.

3. Batey LS, Goff Jr DC, Tortolero SR, et al. Summary measures of the insulin resistance syndrome are adverse among Mexican-American versus non-Hispanic white children: the Corpus Christi Child Heart Study. *Circulation.* 1997;96:4319-4325.

4. Park YW, Zhu S, Palaniappan L, et al. The metabolic syndrome: prevalence and associated risk factor findings in the U.S. population from the Third National Health and Nutrition Examination Survey, 1998–1994. *Arch Intern Med.* 2003;163:427-436.

5. Haffner SM, D'Agostino R, Goff D, et al. LDL Size in African Americans, Hispanics, and Non-Hispanic Whites. *Arterioscler Thromb Vas Biol.* 1999;19:2234-2240.

6. Sorlie PD, Sharrett AR, Patsch W, et al. The relationship between lipids/lipoproteins and atherosclerosis in African Americans and Whites: The Atherosclerosis Risk in Communities Study. *Ann Epidemiol.* 1999;9:149-158.

7. U.K. Prospective Diabetes Study Group. U.K. Prospective Diabetes Study. XII: Differences among Asian, Afro-Caribbean and white caucasian type-2 diabetic patients at diagnosis of diabetes. *Diabet Med.* 1994;11:670-677.

8. Chaturvedi N, Mckeigue PM, Marmot MG. Relationship of glucose intolerance to coronary risk in Afro Caribbeans compared to Europeans. *Diabetologia.* 1994;38:630-631.

9. Parra EJ, Marcini A, Akey J, et al. Estimating African-American admixture proportions by use of population specific alleles. *Am J Hum Genet.* 1998;63:1839-1851.

10. Hannis CL, Hewett-Emmett D, Bertin TK, et al. Origins of U.S. Hispanics. Implications for diabetes. *Diabetes Care.* 1991; 14:618-627.

11. Tull ES, LaPorte RE, Kriska A, et al. Glucose intolerance by race and ethnicity in the U.S. Virgin Islands. *J Natl Med Assoc.* 2002;9:135-142.

12. Lohman TG, Roche AF, Martorell R, eds. Anthropometric Standardization Reference Manual. Champaign, IL: Human Kinetics, 1988.

13. Bucolo G, David H. Quantitative determination of serum triglycerides by use of enzymes. *Clin Chem.* 1973;19:476-482.

14. Warnick GR, Albert JJ. A comprehensive evaluation of heparin-manganese precipitation procedure for estimating high lipoprotein cholesterol. *J Lipid Res.* 1978;19:65-76.

15. The Expert Panel. Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *JAMA.* 2001;285: 2486-2499.

16. Matthews DR, Hosker JP, Rudenski AS, et al. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia.* 1985;28:412-419.

17. Balkau B, Charles MA. Comment on the provisional report from the WHO consultation. European Group for the Study of Insulin Resistance (EGIR). *Diabet Med.* 1999;16:442-443.

18. Tull ES, Roseman JM, Christian CL. Epidemiology of childhood IDDM in the U.S. Virgin Islands from 1979 to 1988. Evidence of an epidemic in the early 1980s and variation by degree of racial admixture. *Diabetes Care.* 1991;14:558-564.

19. Mitchel BD, Williams-Blangero S, Chakraborty R, et al. A comparison of three methods for assessing Amerindian admixture in Mexican Americans. *Ethn Dis.* 1993;3:22-31.

20. SAS Institute. SAS Procedures Guide Version 6, 3rd Ed., Cary, NC, SAS Inst., 1990.

21. Vermaak WJ, Ubbink JB, Delport R, et al. Ethnic immunity to coronary heart disease? *Atherosclerosis.* 1991;89:155-162.

22. Zoratti R, Godsland IF, Chaturvedi N, et al. Relation of plasma lipids to insulin resistance, nonesterified fatty acid levels, and body fat in men from three ethnic groups: relevance to variation in risk of diabetes and coronary disease. *Metabolism.* 2000;49: 245-252.

23. Vega GL, Clark LT, Tang A, et al. Hepatic lipase activity is lower in African-American men than in white American men: effects of 5' flanking polymorphism in the hepatic lipase gene (LIPC). *J Lipid Res.* 1998;39:228-232.

24. Desai PP, Bunker CH, Ukoli FAM, et al. Genetic variation in the apolipoprotein D gene among African blacks and its significance in lipid metabolism. *Atherosclerosis.* 163:329-338.

25. Saad MF, Lillioja S, Nyomba BL, et al. Racial differences in the relation between blood pressure and insulin resistance. *N Engl J Med.* 1991;324:733-739.

26. Williams RC, Long JC, Hanson RL, et al. Individual estimates of European genetic admixture associated with lower body-mass index, plasma glucose, and prevalence of type-2 diabetes in Pima Indians. *Am J Hum Genet.* 2000;66:527-538.

27. Chakraborty R, Ferrell RE, Stern MP, et al. Relationship of prevalence of noninsulin-dependent diabetes mellitus to Amerindian admixture in the Mexican Americans of San Antonio, Texas. *Genet Epidemiol.* 1986;3:435-454.

28. Garza-Chapa R, De Los Angeles Rojas-Alvarado M, Cerda-Flores RM. Prevalence of NIDDM in Mexicans with paraphyletic and polyphyletic surnames. *Am J Human Biol.* 2000;12:721-728.

29. Fontbonne A, Papoz L, Eschwege E, et al. Features of insulin-resistance syndrome in men from French Caribbean islands. The Telecom Study. *Diabetes.* 1992;41: 1385-1389.

30. Howard BV, Mayer-Davis EJ, Goff D, et al. Relationships between insulin resistance and lipoproteins in nondiabetic African Americans, Hispanics, and non-Hispanic whites: the Insulin-Resistance Atherosclerosis Study. *Metabolism.* 1998;47:1174-1179.

31. Griffin FR. Cardiovascular disease in the United States: an epidemiological overview. *Cardiovasc Clin.* 1991;21:3-16.

32. Jones DW, Chambless LE, Folsom AR, et al. Risk factors for coronary heart disease in African Americans: the atherosclerosis risk in communities study, 1987–1997. *Arch Intern Med.* 2002;162:2565-2571.

33. Wild S, Mckeigue P. Cross-sectional analysis of mortality by country of birth in England and Wales, 1970–1992. *Br Med J.* 1997;14:705-710.

34. U.K. Prospective Diabetes Study Group. The incidence of myocardial infarction in white, South Asian, and Afro-Caribbean patients with type-2 diabetes (U.K. Prospective Diabetes Study 32). *Diabetes Care.* 1998;21:1271-1277.

35. Sundquist J, Winkleby MA, Pudaric S. Cardiovascular disease risk factors among older black, Mexican-American, and white women and men: an analysis of NHANES III, 1988–1994. Third National Health and Nutrition Examination Survey. *J Am Geriartr Soc.* 2001;49:109-116.

36. Reaven G, Segal K, Hauptman J, et al. Effect of orlistat-assisted weight loss in decreasing coronary heart disease risk in patients with syndrome X. *Am J Cardiol.* 2001;87: 827-831.

37. Reaven GM. Importance of identifying the overweight patient who will benefit most by losing weight. *Ann Intern Med.* 2003;138:420-423.

CAREER OPPORTUNITIES