

# DIFFERENTIAL IMPACT OF CARDIOVASCULAR DISEASE (CVD) RISK FACTOR CLUSTERING ON CVD AND RENAL DISEASE AMONG AFRICAN-AMERICAN AND WHITE PATIENTS WITH TYPE 2 DIABETES MELLITUS

**Objectives:** To determine if clustering of cardiovascular disease (CVD) risk factors has a differential impact on CVD and renal disease among African Americans compared to Whites with type 2 diabetes.

**Design:** Cross-sectional.

**Methods:** Prevalent CVD, macroalbuminuria, and CVD risk factors were measured in 323 African-American and White adult patients with type 2 diabetes. CVD risk factors were dichotomized according to standard guidelines. Data were analyzed by race according to the presence of any 3 or more CVD risk factors.

**Results:** Despite a similar prevalence of hypertension, the prevalence of macroalbuminuria in the presence of 3 or more CVD risk factors tended to be higher among African Americans compared to Whites (28.9% vs 13.6%,  $P=0.05$ ). The presence of 3+ CVD risk factors was associated with an odds ratio (OR) of 2.5 ( $P=0.001$ , 95% CI, 1.44–4.27) for macroalbuminuria in African Americans compared to an OR of 1.4 ( $P=0.25$ , 95% CI, 0.78–2.53) in Whites. The race/3+ CVD risk factors interaction was statistically significant ( $P=0.007$ ). Conversely, the presence of 3+ risk factors was associated with an OR of 1.6 ( $P=0.019$ , 95% CI, 1.08–2.28) for CVD in Whites compared to an OR of 0.8 ( $P=.287$ , 95% CI, 0.54–1.20) in African Americans. The prevalence of any CVD in the presence of 3+ risk factors was 61% and 49% in Whites and African Americans respectively ( $P=.217$ ). The race/3+ CVD risk factors interaction was statistically significant ( $P=0.029$ ).

**Conclusions:** These findings suggest that among persons with diabetes, a clustering of 3+ CVD risk factors is more predictive for renal disease among African Americans, and more predictive for CVD in Whites. Further research should clarify the impact of CVD risk factor clustering on the incidence of vascular disease among African Americans and Whites with type 2 diabetes. (*Ethn Dis.* 2002;12:530–534)

**Key Words:** Diabetes Mellitus, Cardiovascular Disease, Risk Factors

From the Department of Family and Community Medicine (JHS, JCS), Depart-

John H. Summerson, MS;  
Ronny A. Bell, PhD, MS; Joseph C. Konen, MD, MSPH;  
John G. Spangler, MD, MPH

## INTRODUCTION

In addition to being at increased risk for type 2 diabetes mellitus, African Americans suffer disproportionately from the disease, with an increased mortality rate and a greater prevalence of end-stage renal disease (ESRD).<sup>1–4</sup> However, while overall cardiovascular disease (CVD) mortality appears to be greater among African Americans compared to Whites, among individuals with diabetes, CVD and ischemic heart disease mortality (IHD) rates are lower for African Americans.<sup>1</sup> Both African-American males and females with diabetes have lower death rates from both IHD and CVD, compared to Whites.<sup>1</sup> This phenomenon may be due to higher competing mortality in African Americans from other diabetes-related conditions such as nephropathy.

CVD risk factors tend to cluster together, with CVD morbidity/mortality risk increasing proportionately with the number of risk factors present.<sup>5–7</sup> Individuals with type 2 diabetes typically have one or more additional CVD risk factors, and the major cause of death in this group is CVD.<sup>8</sup> However, neither the prevalence of CVD risk factor clus-

tering, nor its association with vascular disease, has been adequately described, especially in African Americans with diabetes. We hypothesized that the effect of CVD risk factor clustering on risk of CVD and nephropathy may differ between African Americans and Whites. This study examines CVD risk factor clustering and its association with CVD and nephropathy by race in type 2 diabetes.

## METHODS

Three hundred and twenty-three individuals with a diagnosis of type 2 diabetes mellitus were recruited from the Family Medicine ambulatory care unit at Wake Forest University School of Medicine and a community health clinic. Potential participants were identified through a computerized database and invited by mail to receive a free screening to determine the prevalence and number of risk factors for both CVD and renal disease. Those interested in the screening called the clinic and were scheduled for an initial appointment. Thirty-one percent of those contacted agreed to participate, with similar response rates from African Americans and Whites. This study was approved by our institutional review board (IRB), and written informed consent was obtained from each participant.

Height and weight were measured during a physical examination, and a body mass index (BMI=wt in kg/ht in m<sup>2</sup>) was derived for each subject. Obesity was defined as a BMI $\geq$ 28. Blood pressure (BP) was measured on the left arm of each subject after a 5-minute

ment of Public Health Sciences, Wake Forest University School of Medicine, Winston-Salem (RAB); Department of Family Medicine, Carolinas Medical Center, Charlotte (JCK), North Carolina.

Address correspondence and reprint requests to John H. Summerson, MS; Department of Public Health Sciences; Wake Forest University School of Medicine; Winston-Salem, NC 27104; 336-716-2125; 336-713-4300 (fax); jsummers@wfubmc.edu

*This study examines CVD risk factor clustering and its association with CVD and nephropathy by race in type 2 diabetes.*

rest, with the subject in a seated position. The presence of hypertension was determined by the subject having a systolic BP $\geq$ 140 mm Hg, a diastolic BP $\geq$ 90 mm Hg, or currently receiving anti-hypertensive medications. Smoking status was determined by questionnaire.

After a 12-hour fast, each patient had venous blood drawn for determination of lipid levels and glycosylated hemoglobin (HbA<sub>1c</sub>). Total cholesterol was determined by enzymatic assay. High-density lipoprotein (HDL) cholesterol was measured using a precipitation technique.<sup>9</sup> Low-density lipoprotein cholesterol (LDL) was estimated using the Friedewald formula.<sup>10</sup> Hyperlipidemia was defined as either a total cholesterol  $\geq$ 240 mg/dL, LDL  $\geq$ 160 mg/dL, or HDL <35 mg/dL. HbA<sub>1c</sub> was determined using the BioRad column procedure (Richmond, Calif). Urinary albumin excretion was assessed from an overnight urine collection and analyzed by radioimmunoassay to determine urinary albumin excretion ratios (UAER).<sup>11</sup> An overnight urine collection involves collecting all urine voided after going to bed, as well as the first morning void. Macroalbuminuria was used as a marker for nephropathy and was defined as a UAER >0.2 g albumin/g creatinine. Cardiovascular disease was determined from patient interview, medical history, and chart review, and was defined as either a previous abnormal electrocardiogram, significant findings on coronary angiography, history of bypass surgery, angioplasty, classic angina, stroke, signs of asymmetric carotid pulses or bruits, the presence of a neurologic abnormal-

**Table 1. Demographic and clinical characteristics of 323 White and African-American patients with type 2 diabetes mellitus**

Variables	White (N=170)	African American (N=153)	P Value
Age (mean $\pm$ SD)	58.9 $\pm$ 12.1	57.2 $\pm$ 12.1	.211
Female (%)	48.2	69.9	<.001
Diabetes duration (years)	8.4 $\pm$ 7.7	8.7 $\pm$ 8.0	.702
Glycosylated hemoglobin (% $\pm$ SD)	6.9 $\pm$ 1.8	7.7 $\pm$ 2.4	.001
Current cigarette smoker (%)	18.2	22.0	.372
Diabetes treatment			
Insulin (%)	13.5	31.1	<.001
Oral agents (%)	24.1	23.2	.768
Hypertensive (%)	57.6	65.4	.155
Body mass index (% $\geq$ 28.0)	61.2	71.9	.052
Total cholesterol (% $\geq$ 240 mg/dl)	24.1	29.4	.282
HDL-cholesterol (% <35 mg/dl)	30.6	10.5	<.001
LDL-cholesterol (% $\geq$ 160 mg/dl)	34.7	29.4	.309
Macroalbuminuria (%)	9.4	15.0	.122
CVD* (%)	48.2	44.4	.495

\* CVD=cardiovascular disease.

ity on memory, verbal, motor, or sensory examination, asymmetric or diminished dorsalis pedes or posterior tibial pulses, or a history of claudication. Medical histories were verified by examination of medical records.

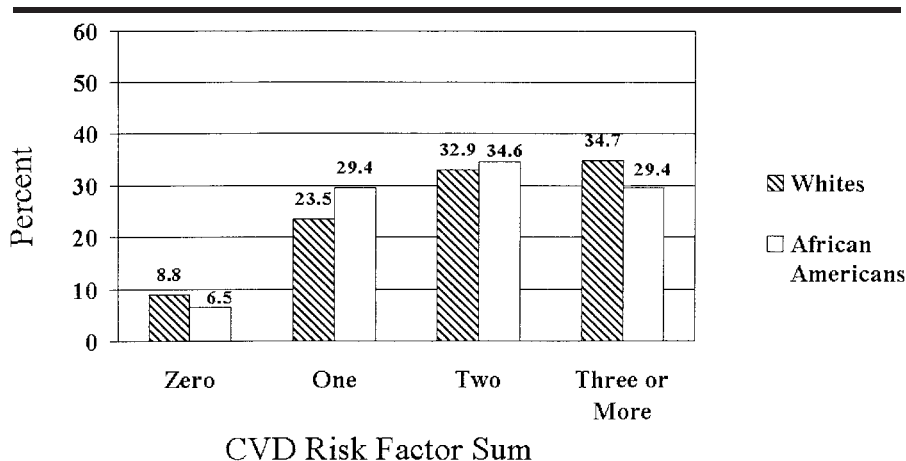
Means and standard deviations were calculated for each study variable. Chi-square and analysis of variance procedures were used to evaluate differences in demographic and clinical measures between groups. Logistic regression analysis was used to identify independent predictors of macroalbuminuria and CVD. Three models were used to adjust for the following: 1) unadjusted; 2) sex and duration of diabetes; 3) sex, diabetes duration, insulin treatment, and HbA<sub>1c</sub>, with separate analyses performed by race. Significance was established as  $P<.05$ . All statistical procedures were carried out using the Statistical Package for the Social Sciences for Personal Computers (SPSS, Inc, Chicago, Ill).

## RESULTS

Table 1 presents demographic and clinical variables by race for the study sample. Compared to Whites, African

Americans were more likely to be female, to be treated with insulin, to be obese and to have higher HbA<sub>1c</sub> levels, and were less likely to have low HDL levels. No racial differences were found for age, diabetes duration, smoking, or elevated total or LDL cholesterol, or BP. Risk factor sums ranged from 0 to 5, and were equally common among African Americans and Whites (Figure 1). The prevalence of macroalbuminuria in the presence of any 3 or more CVD risk factors tended to be higher among African Americans compared to Whites (28.9% vs 13.6%, respectively;  $P=0.05$ ) (Table 2). Conversely, the prevalence of CVD in the presence of 3+ risk factors was 61% and 49% in Whites and African Americans respectively, although this difference was not statistically significant ( $P=.217$ ).

Table 3 gives odds ratios (OR) for macroalbuminuria and CVD in the presence of 3+ CVD risk factors. After adjustment for all covariates, regression analyses indicated that the presence of 3+ risk factors was associated with an OR of 2.5 ( $P=.001$ , 95% CI, 1.44–4.27) for macroalbuminuria in African Americans compared to an OR of 1.4 ( $P=.25$ , 95% CI, 0.78–2.53) in Whites. The race/3+ CVD risk factors



**Fig 1. Prevalence of CVD risk factors among patients with type 2 diabetes mellitus, by race (all comparisons non-significant)**

interaction was significant ( $P=.007$ ) for macroalbuminuria. The presence of 3+ risk factors was associated with an OR of 1.6 ( $P=.019$ , 95% CI, 1.08–2.28) for CVD in Whites compared to an OR of 0.8 ( $P=0.287$ , 95% CI, 0.54–1.20) in African Americans. The race/3+ CVD risk factors interaction was also significant ( $P=.029$ ) for CVD.

**DISCUSSION**

Individuals with CVD typically exhibit more than one risk factor for the disease, and risk factor clustering has been observed frequently in the general population. Seventeen percent of participants in the Framingham Offspring Study,<sup>5</sup> and 14.4% of the participants in the First National Health and Nutrition Examination (NHANES I) Epidemiologic Follow-up Study<sup>6</sup> were found to have at least 3 known CVD risk factors.

Others have found that factors proposed to make up the insulin resistance syndrome (abdominal obesity, hyperlipidemia, hyperglycemia, and hypertension) are found together in 25% to 80% of the general population, depending on the age and ethnicity of the sample.<sup>12–15</sup> Due to an increased risk of CVD, one might expect that persons with diabetes would also be likely to have multiple risk factors for the disease. The present study found that the clustering of 3 or more CVD risk factors is common in this population. These risk factors occurred in isolation only 7.7% of the time. Approximately 33% of subjects had any 2 risk factors while 32.2% had any 3 or more. The risk factor prevalence rates were similar for African-American and White participants.

The tendency of these CVD risk factors to cluster suggests a common pathogenesis, although other environmental and/or genetic factors are in-

involved, as each risk factor can occur in isolation. Insulin resistance and abnormal sympathoadrenal activity have been suggested as causal factors for risk factor clustering.<sup>16</sup> Insulin resistance is often accompanied by hyperinsulinemia and a down regulation of lipoprotein lipase activity, leading to dyslipidemia.<sup>17</sup> Hyperinsulinemia has also been postulated to stimulate the sympathetic nervous system, which may lead to adverse vascular and renal effects, thereby contributing to the pathogenesis of hypertension.<sup>18</sup>

Coronary heart disease morbidity and mortality, and mortality from CVD, occur less frequently in African Americans with diabetes compared to Whites. The 2nd National Health and Nutrition Examination Study indicated that, compared to African Americans, Whites with newly diagnosed diabetes were 2.3 times as likely to have a history of angina, and 3 times as likely to have suffered a myocardial infarction.<sup>8</sup> In addition, African Americans with diabetes are less likely to die from either IHD or CVD, compared to their White counterparts, a pattern that has existed at least since the 1980s.<sup>19,20</sup> Among men, the age-adjusted mortality rates with IHD listed as the underlying cause of death (from 1996) are 354.5 vs 642.2/100,000 for African Americans and Whites, respectively.<sup>1</sup> Among women, age-adjusted rates are 316.4 vs 495.9/100,000 for African Americans and Whites, respectively.<sup>1</sup> A similar pattern exists for mortality from CVD. It is possible that competing mortality due to other diabetes-related conditions, such as nephropathy, account for these ethnic differences, as African Americans are significantly more likely to develop diabetic ESRD compared to Whites.<sup>3,4</sup> Our results support this hypothesis, as clusters of 3 or more CVD risk factors were associated with an increased risk of macroalbuminuria only in African Americans.

The question of why risk factor clustering may be associated with renal

**Table 2. Prevalence of renal disease and cardiovascular disease by race and number of cardiovascular disease risk factors among persons with type 2 diabetes**

Risk Factors	Macroalbuminuria			Cardiovascular Disease		
	Whites	African Americans	P Values	Whites	African Americans	P Values
Zero or any one	9.1%	1.8%	.093	34.5%	41.8%	.432
Any two	5.4%	17.0%	.053	48.2%	43.4%	.620
Any three or more	13.6%	28.9%	.054	61.0%	48.9%	.217

**Table 3. Odds ratios (95% CI) of cardiovascular disease and renal disease according to presence or absence of three or more cardiovascular disease risk factors**

	Macroalbuminuria		Cardiovascular Disease	
	Whites	African American	Whites	African American
Model 1	1.4 (0.86–2.39)	2.0 (1.26–3.15)	1.5 (1.08–2.05)	0.9 (0.62–1.25)
Model 2*	1.5 (0.87–2.65)	2.4 (1.42–4.10)	1.6 (1.11–2.29)	0.8 (0.56–1.21)
Model 3†	1.4 (0.78–2.53)	2.5 (1.44–4.27)	1.6 (1.08–2.28)	0.8 (0.54–1.20)

\* Model 1 plus gender and diabetes duration.

† Model 1 plus gender, diabetes duration, glycosylated hemoglobin, and insulin treatment. Race/3+ CVD risk factors interaction term for presence of macroalbuminuria,  $P=.007$  (Model 3). Race/3+ CVD factors interaction term for presence of CVD,  $P=.029$  (Model 3).

function in African Americans is unanswered. Although hypertension increases the risk of developing renal disease in most populations, its effect seems to be enhanced in African Americans.<sup>21,22</sup> Perhaps high BP and/or its interaction with other risk factors produces renal impairment prior to CVD in African Americans. It is also possible that genetic or biologic factors are responsible as there is evidence suggesting that African Americans are genetically more susceptible to developing renal disease.<sup>23</sup> Other possible explanations could include differences in socioeconomic status; lack of access to, and/or lack of utilization of, healthcare services; diet; stress; or other environmental variables, although these factors would also tend to increase the risk of CVD in this population.

Risk for CVD has been found to increase proportionately with the number of risk factors.<sup>5-7</sup> While we found this to be true in Whites with diabetes, clusters of 3 or more risk factors were not associated with CVD in African Americans. It has been suggested that the lower CVD mortality rate in African Americans with diabetes may involve insulin resistance. African Americans may be less likely than Whites to have the insulin resistant variant of type 2 diabetes which may lead to a lower CVD risk factor profile.<sup>24,25</sup> In addition, metabolic abnormalities may have a weaker association with insulin resistance in this population.<sup>26</sup> These factors may impact CVD risk in African Americans with type 2 diabetes. However, much of the

more favorable risk factor profile found between those with the insulin sensitive and insulin resistant variants of type 2 diabetes involve more favorable lipid and lipoprotein levels.<sup>24,25</sup> While the observed favorable lipid profile could be cardio-protective, a more favorable lipid profile is a consistent finding in African Americans compared to Whites, both with<sup>27-29</sup> and without<sup>30-32</sup> diabetes. This more favorable lipid profile would tend to reduce CVD mortality among African Americans compared to Whites in samples without diabetes as well, a finding which has not been documented as African Americans in the general population appear to suffer similar or greater overall mortality from CVD.<sup>33</sup> Our results suggest that the lower CVD mortality found in African Americans with diabetes compared to Whites is partially due to a more detrimental, or earlier, effect of CVD risk factor clustering on renal function in this population.

While these data concentrate on differences by race there is not consensus within the scientific community or from federal health statistics regarding the meaning of concepts such as race and ethnicity.<sup>34</sup> Given this lack of consensus, the absence of explicit race definitions may be a limitation to our data although self-report agreement appears to be very high among African Americans and Whites.<sup>35</sup> Other limitations include possible selection bias, confounding by variables not controlled for or measured, limitations of statistical power and the

*The tendency of these CVD risk factors to cluster suggests a common pathogenesis, although other environmental and/or genetic factors are involved, as each risk factor can occur in isolation.*

generalizability of our sample population.

In conclusion, multiple CVD risk factors were equally common in both African Americans and Whites with diabetes. Although the prevalence of certain risk factors, such as hypertension and high cholesterol, has declined in the United States over the past 2 decades, the prevalence of obesity and diabetes has increased,<sup>36-38</sup> underscoring the importance of risk factor prevention and control. The differential association of risk factor clustering found in the present study may partially explain the higher ESRD rates and lower IHD and CVD mortality among African Americans compared to Whites with diabetes. Additional research should be directed toward the elucidation of the pathophysiological basis for the ethnic differences in the impact of CVD risk factors on the occurrence of renal disease and CVD in patients with diabetes mellitus.

**ACKNOWLEDGMENTS**

This study was supported in part by a cooperative agreement from the Centers for Disease Control and Prevention (U32CCU-403318).

**REFERENCES**

- Centers for Disease Control and Prevention. *Diabetes Surveillance 1999*. Atlanta Ga: US Dept of Health and Human Services, National Center for Chronic Disease Prevention and Health Promotion, Division of Diabetes Translation; 1999.
- Moritz DJ, Ostfeld AM, Blazer D, Curb D,

## RACIAL DIFFERENCES IN CVD RISK FACTOR IMPACT - Summerson et al

- Taylor JO, Wallace RB. The health burden of diabetes in elderly in 4 communities. *Public Health Rep.* 1994;109:782-790.
3. Cowie CC, Port FK, Wolfe RA. Disparities in incidence of diabetic end-stage renal disease according to race and type of diabetes. *N Engl J Med.* 1989;321:1074-1079.
  4. Pao CI, Whittle JC, Whelton PK, Seidler AJ. The excess incidence of diabetic end-stage renal disease among African Americans: a population-based study of potential explanatory factors. *JAMA.* 1992;268:3079-3084.
  5. Wilson PW, Kannel WB, Silbershatz H, D'Agostino RB. Clustering of metabolic risk factors and coronary heart disease. *Arch Intern Med.* 1999;159:1104-1109.
  6. Yusuf HR, Giles WH, Croft JB, Anda RF, Casper ML. Impact of multiple risk factor profiles on determining cardiovascular disease risk. *Prev Med.* 1998;27:1-9.
  7. Stamler J, Dyer AR, Shekelle RB, Neaton J, Stamler R. Relationship of baseline risk factors to coronary and all-cause mortality, and to longevity: findings from long-term follow-up of Chicago cohorts. *Cardiology.* 1993;82:191-222.
  8. Harris MI. Non-insulin dependent diabetes mellitus in Black and White Americans. *Diabetes Metab Rev.* 1990;6:71-90.
  9. Albers JJ, Russell-Warnick GR, Chenng MC. Quantitation of high-density lipoprotein. *Lipids.* 1978;13:926-932.
  10. Friedewald WT, Levy RI, Frederickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of preparative ultracentrifuge. *Clin Chem.* 1972;18:499-502.
  11. McCormick CP, Shihabi ZK, Kone JC. Micro-transferrinuria and microalbuminuria: enhanced immunoassay. *Ann Clin Lab Sci.* 1989;19:444-451.
  12. Genest J, Cohn JS. Clustering of cardiovascular risk factors: targeting high risk individuals. *Am J Cardiol.* 1995;76:8A-20A.
  13. Julius S, Gudbrandsson T, Jamerson K, Andersson O. The interconnection between sympathetics, microcirculation, and insulin resistance in hypertension. *Blood Pressure.* 1992;1:9-19.
  14. Reaven GM. Syndrome X: 6 years later. *J Intern Med Suppl.* 1994;736:13-22.
  15. Depres JP, Marette A. Relation of components of insulin resistance syndrome to coronary disease risk. *Curr Opin Lipidol.* 1994;5:274-289.
  16. Depres JP. Abdominal obesity as an important component of insulin-resistance syndrome. *Nutrition.* 1993;9:452-459.
  17. Blomhoff JP. Lipoproteins, lipases, and the metabolic cardiovascular syndrome. *J Cardiovasc Pharmacol.* 1992;20(suppl 8):S22-S25.
  18. Reaven GM, Chen YD. Insulin resistance, its consequences, and coronary heart disease: must we choose one culprit? *Circulation.* 1996;93:1780-1783.
  19. Centers for Disease Control. *Diabetes Surveillance, 1991.* Atlanta Ga: Centers for Disease Control; 1992.
  20. DeStefano F, Neuman J. Comparison of coronary heart disease mortality risk between Black and White people with diabetes. *Ethn Dis.* 1993;3:145-151.
  21. Shulman NB, Ford CE, Hall WD. Prognostic value of serum creatinine and effect of treatment of hypertension on renal function: results from the Hypertension Detection and Follow-up Program. *Hypertension.* 1989;13(suppl 1):180-193.
  22. Powers DR, Wallin JD. End-stage renal disease in specific ethnic and racial groups. *Arch Intern Med.* 1998;158:793-800.
  23. Rostand SG. Hypertension and renal disease in African Americans: role of genetic and/or environmental factors? *Adv Nephrol.* 1992;21:99-113.
  24. Banerji MA, Lebovitz HE. Insulin action in Black Americans with NIDDM. *Diabetes Care.* 1992;15:1295-1302.
  25. Chaiken RL, Banerji MA, Pasmantier R, Huey H, Lebovitz HE. Patterns of glucose and lipid abnormalities in Black NIDDM subjects. *Diabetes Care.* 1991;14:1036-1042.
  26. Chaiken RL, Banerji MA, Huey H, Lebovitz HE. Do Blacks with NIDDM have an insulin-resistance syndrome? *Diabetes.* 1993;42:444-449.
  27. Cowie CC, Howard BV, Harris MI. Serum lipoproteins in African Americans and Whites with NIDDM in the US population. *Circulation.* 1994;90(3):1185-1193.
  28. Summerson JH, Konecny JC, Dignan MB. Racial differences in lipid and lipoprotein levels in diabetes. *Metabolism.* 1992;41:851-855.
  29. Pacy PJ, Dodson PM, Kubicki AJ, Fletcher RF. Differences in lipid and lipoprotein levels in White, Black, and Asian non-insulin dependent (type 2) diabetics with hypertension. *Diabetes Res.* 1987;4:187-193.
  30. Friday KE, Srinivasan SR, Elkasabany A, et al. Black-White differences in postprandial triglyceride response and postheparin lipoprotein lipase and hepatic triglyceride lipase among young men. *Metab Clin Exp.* 1999;48(6):749-754.
  31. Glueck CJ, Gartside P, Laskarzewski PM, Khoury P, Tyroler HA. High-density lipoprotein cholesterol in Blacks and Whites: potential ramifications for coronary heart disease. *Am Heart J.* 1984;108:815-825.
  32. Metcalf PA, Sharrett AR, Folsom AR, et al. African-American-White differences in lipids, lipoproteins, and apolipoproteins, by educational attainment, among middle-aged adults: The Atherosclerosis Risk in Communities Study. *Am J Epidemiol.* 1998;148:750-760.
  33. American Heart Association. *1999 Heart and Stroke Facts.* Dallas, Tex: AHA; 1999.
  34. Hahn RA. The state of federal health statistics on racial and ethnic groups. *JAMA.* 1992;267:268-271.
  35. Kelly JJ, Chu SY, Diaz T, Leary LS, Buehler JW. Race/ethnicity misclassification of persons reported with AIDS. *Ethn Health.* 1996;1(1):87-94.
  36. National Center for Health Statistics. *Health, United States, 1994.* Hyattsville, Md: Public Health Service; 1995.
  37. National Center for Health Statistics and the National Heart, Lung, and Blood Institute Collaborative Lipid Group. Trends in serum cholesterol levels among US adults aged 20 to 74 years: data from the National Health and Nutritional Surveys, 1960-1980. *JAMA.* 1987;257:937-942.
  38. Harlan WR, Landis JR, Flegal KM, Davis CS, Miller ME. Secular trends in body mass in the United States, 1960-1980. *Am J Epidemiol.* 1988;128:1065-1074.

### AUTHOR CONTRIBUTIONS

*Design and concept of study:* Summerson, Konecny, Spangler

*Acquisition of data:* Summerson, Konecny

*Data analysis and interpretation:* Summerson, Bell, Konecny, Spangler

*Manuscript draft:* Summerson, Bell, Spangler

*Statistical expertise:* Summerson

*Acquisition of funding:* Konecny

*Administrative, technical, or material assistance:* Konecny

*Supervision:* Konecny