Mortality Among Low-Income African Americans and Whites With Diabetes

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OBJECTIVE—To estimate mortality rates and risk factors for mortality in a low-socioeconomic status (SES) population of African Americans and whites with diabetes.

RESEARCH DESIGN AND METHODS —We determined mortality among African Americans and whites aged 40–79 years with (n = 12,498) and without (n = 49,914) diabetes at entry into a cohort of participants recruited from government-funded community health centers. Multivariable Cox analysis was used to estimate mortality hazard ratios (HRs) (95% CI) among those with versus those without diabetes and among those with diabetes according to patient characteristics.

RESULTS—During follow-up (mean 5.9 years), 13.5% of those with and 7.3% of those without diabetes died. All-cause mortality risk was higher among those with versus without diabetes for both African Americans (HR 1.84 [95% CI 1.71–1.99]) and whites (1.80 [1.58–2.04]), although among those with diabetes, mortality was lower among African Americans than whites (0.78 [0.69–0.87]). Mortality risk increased with duration of diabetes and was greater among patients on insulin therapy and reporting histories of cardiovascular disease (CVD), hypertension, and stroke. The HRs associated with these multiple risk factors tended to be similar by sex and race, with the exception of a differentially higher impact of prevalent CVD on mortality among African Americans (interaction P value = 0.03), despite a lower baseline prevalence of CVD.

CONCLUSIONS—In this population with similarly low SES and access to health care, strong and generally similar predictors of mortality were identified for African Americans and whites with diabetes, with African Americans at a moderately but significantly lower mortality risk.

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Population studies have consistently shown increased all-cause mortality among individuals with type 2 diabetes (1–4). Several reports have also shown that the impact of diabetes on mortality varies by socioeconomic status (SES) (5–7), race (8–10), and/or sex (4,11). However, scant literature exists on the mortality experience of diabetic patients within low-SES populations in the U.S. and whether within these vulnerable populations mortality varies by sex or race. We have previously reported that the prevalence of diabetes is only slightly higher among African Americans than whites once SES and other risk factors are accounted for (12). We now describe mortality patterns and risk factors for mortality by sex and race in a large, low-SES population of southern U.S. African Americans and whites with type 2 diabetes.

RESEARCH DESIGN AND METHODS

—The Southern Community Cohort Study (SCCS) is a population-based, prospective study designed to investigate causes of disparities among African Americans and whites in the incidence of and mortality from cancer and other chronic diseases. Details of the rationale, study design, and methods have previously been described (13). Briefly, between 2002 and 2008, 64,096 participants aged 40–79 years were recruited from community health centers from twelve states in the southeast U.S. Community health centers are government-funded health care facilities offering basic health care and preventative services to the medically underserved. Persons receiving services at the centers are typically of low income and without health insurance. All study procedures were approved by the institutional review boards of Vanderbilt University and Meharry Medical College.

After providing informed consent, participants completed a 40- to 60-min in-person interview, through which data were collected on medical history, lifestyle, and socioeconomic factors. If a participant answered "yes" to the question "Has a doctor ever told you that you have had diabetes or high blood sugar?" he/she was asked questions about age at diabetes diagnosis and medications prescribed to treat the disease. Women were specifically asked not to include gestational diabetes mellitus in their reporting. For the current analysis, we excluded participants missing information on diabetes status (N = 339) or age at first diabetes diagnosis (N = 196), as well as those reporting a first diagnosis of diabetes before the age of 30 years (N = 1,149). Participants diagnosed with diabetes at or after the age of 30 years (N = 12,498) thus formed our type 2 diabetes cohort.

Mortality status was determined from linkages of the entire SCCS cohort with the Social Security Administration (SSA) Death Master File and the National Death Index (NDI). SSA is the most current and captures the large majority of mortality cases in the U.S. However, in order to be recorded in SSA one has to have a social security number, and thus a small number not covered by SSA will be covered by NDI. Our NDI files were current only through 2008, while SSA files included deaths through April 2011. Both sources were used to ensure the maximal amount of mortality ascertainment. Follow-up extended from date of entry into the

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SCCS until the earlier of 28 February 2011 (2 months prior to the most recent SSA linkage) or date of death. Since cause-of-death information, available only from NDI and not SSA, was not available for deaths after 2008, only all-cause mortality data are presented in this report.

General linear models were used to test for differences in continuous variables, and χ^2 tests were used for categorical data. Race was self-reported based on prespecified categories. Because of sample size limitations in other racial/ethnic groups, race-specific analyses and evaluations of effect modification by race were conducted in African Americans and whites only.

Cox proportional hazards modeling, using age as the time scale, was used to determine hazard ratios (HRs) (95% CI) for all-cause mortality risk in those with diabetes relative to those without diabetes. In additional analyses restricted to those with diabetes, Cox modeling was used to determine predictors of mortality among those with diabetes overall and by race and sex group. All baseline covariate data were self-reported. Baseline data on other complications and comorbidities such as ischemic heart disease (heart attack or bypass surgery, herein referred to as cardiovascular disease [CVD]) and hypertension (positive response to the question "Has a physician ever told you that you have had high blood pressure?") were based on self-reported physician diagnosis. Included covariates in the Cox models are presented in Tables 1–3. Tests for interaction of these variables with either sex or race (African American and white) were conducted by adding the corresponding cross-product terms to the models, and significant interactions are listed in the RESULTS section. All tests for statistical significance were two tailed. Statistical analysis was conducted using SAS version 9.2 (SAS, Cary, NC).

RESULTS—A total of 12,498 individuals, nearly 20% of the SCCS community health center–enrolled participants, reported having a physician diagnosis of diabetes at or after the age of 30 years, with a mean duration of diabetes of 7.7 years at study baseline. Seventy-nine percent reported taking either insulin or an oral antihyperglycemic agent, with 51% taking oral hypoglycemic drugs alone, 17% insulin alone, 11% oral hypoglycemic drugs plus insulin, 6% unspecified medication, and 15% no medication. Baseline characteristics of these 12,498 individuals versus the 49,914 without diabetes are presented in Table 1.

Several of the baseline characteristics of the SCCS study participants with diabetes differed according to sex and/or race (Table 2). Compared with their male counterparts, African American and white women had a slightly longer duration of diabetes and a lower use of insulin. Women also had a lower use of medications other than statins to treat high cholesterol and a slightly lower prevalence of a history of CVD and were less likely to be smokers or to have a history of smoking. When data were characterized by race, compared with their white counterparts African Americans were younger at age of diabetes diagnosis and had a slightly longer duration of the illness and greater use of insulin therapy alone. African Americans were more likely to report a history of hypertension (women only) but less likely to report a history of high cholesterol or CVD.

During an average of 5.9 years of follow-up, 13.5% of the diabetic population and 7.3% of those without diabetes died. Those with diabetes had an 87% increased multivariable-adjusted mortality risk (HR 1.87 [95% CI 1.76–2.00]).

Table 1—*Baseline characteristics of SCCS participants by type 2 diabetes status*†

	Case subjects	Noncase subjects	*P*
N	12,498	49,914	
Age (years)	55.4 (0.08)	51.1 (0.04)	<0.0001
Age at diagnosis (years)	47.2 (9.3)		
Diabetes duration (years)	7.7 (7.3)		
Sex (female)	66.7 (8,332)	59.2 (29,524)	<0.0001
Race			0.25
African American	71.8 (8,978)	71.5 (35,705)	
White	24.3 (3,041)	25.0 (12,479)	
Other race	3.8 (479)	3.5 (1,730)	
BMI at age 21 years (kg/m^2)	23.5 (20.6–27.3)	22.2 (20.0–24.9)	<0.0001*
Current BMI (kg/m^2)	32.9 (28.3–38.5)	28.1 (24.2–33.1)	<0.0001*
Insulin therapy use only**	17.9 (2,099)		
Oral hypoglycemic therapy use only**	54.7 (6,394)		
Insulin and oral hypoglycemic therapy use**	11.5 (1,347)		
No hypoglycemic medication**	15.9 (1,860)		
Hypertension	81.6 (10,190)	49.8 (24,842)	<0.0001
High cholesterol	54.1 (6,744)	25.8 (12,848)	<0.0001
Cholesterol-lowering medication			<0.0001
None	62.2 (7,745)	88.0 (43,794)	
Statin	30.5 (3,794)	9.7 (4,850)	
Other cholesterol medication	7.3 (911)	2.3 (1,127)	
CVD	12.9 (1,609)	5.2 (2,597)	<0.0001
Stroke/TIA	11.2 (1,401)	5.6 (2,788)	<0.0001
Glaucoma	7.7 (955)	2.8 (1,382)	<0.0001
Cataract	18.0 (2,242)	7.0 (3,503)	<0.0001
History of smoking	59.0 (7,368)	67.1 (33,486)	<0.0001
Current smoker	30.3 (3,779)	48.3 (24,092)	<0.0001
Education			<0.0001
<High-school graduate	37.6 (4,693)	30.8 (15,364)	
High-school graduate/vocational school	37.8 (4,723)	40.8 (20,343)	
≥Some college	24.6 (3,078)	28.5 (14,199)	
Annual household income (USD)			<0.0001
<15,000	64.9 (8,021)	60.2 (29,731)	
15,000–24,999	21.4 (2,643)	22.6 (11,145)	
≥25,000	13.7 (1,693)	17.2 (8,496)	
Have health insurance	65.3 (8,111)	53.6 (26,651)	<0.0001

Data are presented as means (SD), median (interquartile range), or % (*n*) unless otherwise indicated. †Type 2 diabetes defined as diabetes diagnosed at or after the age of 30 years. *Nonparametrically tested. **Three individuals had missing data on diabetes medication use, and 795 individuals had missing data on type of diabetes medication used.

Table 2—*Baseline characteristics of SCCS participants with type 2 diabetes by sex and race†*

	Males		Females				
	Whites	African Americans	Whites	African Americans	*P* for sex differences	*P* for race differences	*P* for global test of differences
N	987	3,004	2,054	5,974			
Age (years)	55.7 (8.9)	54.1 (8.5)	56.4 (9.1)	55.7 (9.1)	<0.0001	<0.0001	<0.0001
Age at diagnosis (years)	48.4 (9.5)	46.2 (8.7)	48.5 (9.6)	47.0 (9.3)	0.0002	<0.0001	<0.0001
Diabetes duration (years)	6.8 (6.5)	7.4 (6.8)	7.4 (7.1)	8.1 (7.6)	<0.0001	<0.0001	<0.0001
BMI at age 21 years (kg/m^2)	25.2 (22.2–28.8)	24.3 (21.7–27.8)	22.9 (20.2–27.5)	22.8 (20.2–26.6)	<0.0001	<0.0001	<0.0001
Current BMI (kg/m^2)	32.5 (28.0–37.2)	30.1 (26.2–34.7)	34.6 (29.7–40.7)	34.3 (29.3–39.8)	<0.0001	<0.0001	<0.0001
Hyperglycemic therapy					<0.0001	<0.0001	<0.0001
Insulin therapy use only*	15.4 (140)	24.7 (663)	10.8 (216)	17.9 (1,007)			
Oral hyperglycemic therapy use only*	53.0 (483)	49.1 (1,319)	57.2 (1,146)	56.7 (3,201)			
Insulin and oral hyperglycemic therapy use*	12.1 (110)	8.9 (240)	12.2 (245)	12.4 (699)			
No hyperglycemic medication*	19.5 (178)	17.3 (465)	19.8 (396)	13.0 (736)			
Hypertension	79.6 (786)	79.9 (2,399)	77.2 (1,585)	84.4 (5,042)	0.0003	<0.0001	<0.0001
Hypercholesterolemia	60.3 (592)	47.9 (1,433)	64.7 (1,325)	52.0 (3,098)	<0.0001	<0.0001	<0.0001
Cholesterol-lowering medication					<0.0001	<0.0001	<0.0001
None	54.9 (538)	67.0 (2,003)	53.2 (1,088)	64.6 (3,845)			
Statin	34.3 (336)	23.5 (704)	39.9 (816)	30.0 (1,784)			
Other cholesterol medication	10.8 (106)	9.5 (284)	6.9 (142)	5.5 (326)			
CVD	27.4 (270)	12.7 (381)	14.4 (295)	9.7 (581)	<0.0001	<0.0001	<0.0001
Stroke/TIA	13.5 (133)	11.5 (344)	11.6 (238)	10.5 (625)	0.05	0.03	0.03
Glaucoma	3.8 (37)	7.5 (224)	5.3 (109)	9.3 (552)	0.001	<0.0001	<0.0001
Cataract	17.6 (174)	12.2 (364)	22.7 (465)	19.3 (1,150)	<0.0001	<0.0001	<0.0001
History of smoking	73.5 (725)	73.7 (2,211)	58.2 (1,196)	48.9 (2,916)	<0.0001	<0.0001	<0.0001
Current smoker	35.0 (345)	41.9 (1,258)	28.9 (594)	24.0 (1,430)	<0.0001	0.34	<0.0001
Education					0.26	0.0007	0.0007
<High-school graduate	35.7 (352)	39.2 (1,178)	34.5 (709)	38.5 (2,297)			
High-school graduate/vocational school	37.6 (371)	38.7 (1,161)	40.5 (832)	37.1 (2,213)			
≥Some college	26.8 (264)	22.1 (664)	25.0 (513)	24.5 (1,461)			
Annual household income (USD)					<0.0001	0.0002	<0.0001
<15,000	62.7 (616)	61.7 (1,830)	63.6 (1,290)	67.8 (4,001)			
15,000–24,999	20.4 (200)	22.1 (656)	21.2 (431)	21.1 (1,248)			
≥25,000	16.9 (166)	16.2 (482)	15.2 (309)	11.1 (656)			
Have health insurance	63.4 (624)	62.3 (1,862)	62.5 (1,277)	68.2 (4,046)	<0.0001	0.0006	<0.0001

Data are presented as mean (SD), median (interquartile range), or % (*n*) unless otherwise indicated. †Type 2 diabetes defined as diabetes diagnosed at or after the age of 30 years. *Three individuals had missing data on diabetes medication use, and 795 individuals had missing data on type of diabetes medication used.

Even in those taking no antihyperglycemic medication, mortality risk was still 39% higher than in those without diabetes (1.39 [1.23–1.57]). Compared with the population without diabetes, African Americans and whites with diabetes had similar increased mortality risks (1.84 [1.71–1.99] for African Americans with versus without diabetes; 1.80 [1.58–2.04] for whites with versus without diabetes) in analyses adjusted for sex, BMI, CVD, hypertension, stroke/transient ischemic attack (TIA), high cholesterol, smoking history, education, income, and health insurance status. The excess risk in mortality was slightly higher in women (1.94 [1.76–2.13]) than in men (1.77 [1.62–1.94]) with versus without diabetes.

Table 3 shows the multivariate-adjusted HRs of all-cause mortality according to various characteristics of those with diabetes. Risk of death rose with duration of diabetes, overall being 23% higher among those with diabetes of ≥20 years compared with those with <10 years of diabetes duration; in analyses linearly modeling mortality risk in relation to years of duration of diabetes, the HR increased by 1% per year of diabetes (HR 1.01 [95% CI 1.00–1.02], $P = 0.003$). However, as suggested in Fig. 1, which shows survival curves of crude mortality rates in African Americans and whites with diabetes, African Americans had a 22% lower multivariable-adjusted mortality risk than whites, a pattern that held among both men and women. Overall, women with diabetes had a lower mortality risk than men, and this was similar in both African Americans and whites. Mortality was highest among those reporting insulin therapy use, while there was no significant difference in mortality between those taking no antihyperglycemic agents and those taking only oral hypoglycemic agents. Other predictors of increased mortality among those with diabetes included prevalent CVD, stroke/TIA, and hypertension. Table 3 also shows that predictors for mortality were generally similar for both men and women and African Americans and whites with diabetes. The exceptions were for prevalent CVD and hypertension at entry into the SCCS among those with diabetes, where a greater risk was observed for hypertension in men than in women (P value for interaction by sex = 0.01) and where the significantly increased mortality risk associated with baseline CVD was observed only among African Americans (P value for interaction by race = 0.03). When this was further examined in each specific sex-race group (Supplementary Table 1), this differential mortality impact of hypertension and CVD by sex and race, respectively, appeared to be driven by African American men.

CONCLUSIONS—In this large cohort study, the overall mortality risk was approximately 90% higher among those with versus without diabetes, which is similar to findings from other studies of type 2 diabetes (1,3,14). The excess risk associated with diabetes was nearly identical among African Americans and whites. We have also shown that after adjustment for multiple covariates, African Americans with diabetes had a slightly more favorable survival than socioeconomically similar whites. We have previously reported that, within the SCCS, the prevalence of diabetes was only slightly (<15%) greater among African Americans than among whites after controlling for BMI and other risk factors (12)—in contrast to higher excesses reported nationally (12). Our findings suggest that diabetes prevalence and survival are similar for African Americans and whites who have similar social and health care characteristics. Thus, the reported twofold-higher national diabetes mortality rates observed among African Americans compared with whites (15,16) do not appear to be predominantly biologically based.

Among those with diabetes, we observed a moderately better survival over the follow-up period among African Americans compared with whites. National diabetes mortality rates, such as data from the National Center for Health Statistics, are generally based on death certificate data listing diabetes as the underlying cause of death and are usually only adjusted for age and sex. Mortality rates are a function of both the combined incidence of and survival from disease. One primary reason that some other studies showed higher mortality rates among African Americans and we did not was that they (e.g., U.S. cause-of-death reports) were reporting death rates attributable to diabetes in the general population where African Americans are more likely to have and die of diabetes. In our cohort, we were conditioning on having diabetes and looking at survival among African Americans and whites with this disease. Thus, we have removed the effect of a higher incidence of diabetes among African Americans. Furthermore, both by study design and statistical analysis, we have also removed the effect of SES. This is important, since rate ratios based on national population data, such as from the National Center for Health Statistics, do not account for SES or having health insurance in their estimates—factors that may also account for some of the observed increased risk. In the Multiple Risk Factors Intervention Trial (MRFIT), African American men were at >2.5-fold greater age-adjusted risk of having diabetes listed as the underlying cause of death on death certificates (17). When mortality was investigated only in those with diabetes at study baseline, this risk in African American men in MRFIT was attenuated to a 19% increased age-adjusted mortality risk compared with white men (18). Furthermore, the age-adjusted RR for all-cause mortality was reduced from 1.19 to a nonsignificant 0.94 in African Americans compared with whites after additionally controlling for income and other risk factors. In a Medicare-for-fee beneficiaries study, the age- and sex-adjusted mortality rate was significantly higher in African Americans compared with whites without diabetes; however, among those with diabetes there was no significant difference in mortality by race (19). A Veterans Affairs study (20) showed a lower mortality rate among African American men with diabetes compared with white men. Similar to the SCCS participants, who were recruited from government-funded community health centers, the individuals in the Veterans Affairs study likely had similar access to health care. We also controlled for duration of diabetes and antihyperglycemic medications as proxies for disease severity, and the lower mortality rate among African Americans persisted. In summary, our data add to a growing literature suggesting that once SES and other risk factors are controlled for, mortality is no higher among African American than white patients with diabetes. Reasons for the slightly lower mortality risk observed among African Americans than whites in our population may in part be related to a lower risk of coronary artery disease in African Americans (18,21,22). Other studies may not have been able to show this because they were not able to as fully control for SES and having similar access to health care as we were in our study.

As expected, the coexistence of other chronic illnesses was associated with increased mortality. Comorbidities, especially

Table 3—*Predictors of all-cause mortality in individuals with type 2 diabetes† overall and by race and sex*

		Sex		Race	
	Total	Males	Females	African Americans	Whites
Mortality rate (events per 1,000 person-years)‡	23.8	38.7	18.4	22.5	27.6
Duration category					
<10 years	Ref.	Ref.	Ref.	Ref.	Ref.
10–19 years	1.19 (1.06–1.34)	1.06 (0.89–1.25)	1.32 (1.12–1.54)	1.22 (1.06–1.39)	1.09 (0.86–1.38)
≥20 years	1.23 (1.05–1.44)	1.32 (1.04–1.68)	1.17 (0.94–1.46)	1.26 (1.04–1.53)	1.13 (0.81–1.57)
P¶	0.003	0.09	0.02	0.004	0.16
Sex (female)	0.60 (0.54–0.66)			0.63 (0.56–0.72)	0.53 (0.43–0.64)
P	<0.0001			<0.0001	<0.0001
Race					
White	Ref.	Ref.	Ref.		
African American	0.78 (0.69–0.87)	0.76 (0.64–0.90)	0.81 (0.69–0.96)		
Other race	1.03 (0.79–1.33)	1.05 (0.74–1.49)	1.01 (0.69–1.47)		
P§	0.0001	0.008	0.08		
Hyperglycemic medication					
None	Ref.	Ref.	Ref.	Ref.	Ref.
Insulin only	2.21 (1.85–2.64)	2.21 (1.73–2.84)	2.26 (1.77–2.91)	2.26 (1.82–2.80)	2.03 (1.44–2.85)
Insulin plus oral agent	1.81 (1.48–2.22)	2.05 (1.53–2.77)	1.66 (1.25–2.19)	1.80 (1.39–2.33)	1.89 (1.31–2.72)
Oral agent only	1.14 (0.96–1.35)	1.25 (0.98–1.59)	1.05 (0.83–1.32)	1.19 (0.96–1.47)	1.02 (0.76–1.38)
Missing data on medication	1.28 (1.01–1.63)	1.29 (0.94–1.78)	1.36 (0.94–1.97)	1.34 (1.01–1.78)	1.15 (0.69–1.91)
P§	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
CVD	1.47 (1.30–1.67)	1.55 (1.30–1.85)	1.37 (1.14–1.64)	1.61 (1.38–1.89)	1.22 (0.97–1.54)
P	<0.0001	<0.0001	0.0007	<0.0001	0.08
Stroke/TIA	1.45 (1.27–1.65)	1.50 (1.25–1.80)	1.42 (1.19–1.71)	1.50 (1.29–1.75)	1.38 (1.07–1.78)
P	<0.0001	<0.0001	0.0002	<0.0001	0.01
Hypertension	1.37 (1.18–1.59)	1.53 (1.25–1.88)	1.16 (0.95–1.42)	1.46 (1.22–1.76)	1.12 (0.88–1.44)
P	<0.0001	<0.0001	0.14	<0.0001	0.36
High cholesterol					
No high cholesterol	Ref.	Ref.	Ref.	Ref.	Ref.
High cholesterol, not treated	0.94 (0.81–1.08)	0.95 (0.77–1.18)	0.93 (0.77–1.13)	0.94 (0.79–1.11)	0.85 (0.64–1.14)
High cholesterol, statin	0.75 (0.67–0.85)	0.70 (0.58–0.84)	0.79 (0.67–0.92)	0.76 (0.66–0.89)	0.70 (0.55–0.89)
High cholesterol, other cholesterol-lowering medication	0.85 (0.71–1.02)	0.81 (0.63–1.03)	0.90 (0.68–1.19)	0.93 (0.74–1.15)	0.62 (0.42–0.90)
P§	<0.0001	0.002	0.03	0.004	0.009

Data are HR (95% CI) unless otherwise indicated. †Type 2 diabetes defined as diabetes diagnosed at or after the age of 30 years. ‡Age-adjusted mortality rate using the total population as the age standard. ¶*P* value for linear trend with duration of diabetes. §*P* value for overall differences between categories. Cox models included the variables listed in the table plus BMI, BMI squared, smoking (never/former/current), education (<12 years, 12 years/vocational school, and ≥some college), income (<15,000, 15,000–24,999, and ≥25,000 USD), and having health insurance as covariates.

hypertension, were common among those with diabetes. Hypertension was reported by nearly 80% of the patients. The risk of all-cause mortality associated with comorbid illnesses tended to be similar for African Americans and whites, except for a differential impact of preexisting CVD. The baseline prevalence of reported CVD was considerably lower in African Americans compared with whites, consistent with the literature (18,21,22). However, among those with diabetes, having CVD was associated with a 60% increased mortality risk in African Americans and only a 17% increased risk in whites. The disparity in clinical outcomes between African Americans and whites who have diabetes and CVD has been described previously (23,24). Patient behavior and preferences, access to care, and physician bias have all been put forth as potential explanations (23,24). In a review by Fincher et al. (24), access to care, patient preferences, and patient behaviors only partially accounted for racial disparities in the treatment of coronary artery disease. Our findings in a population with similar access to care appear to support this.

In the general population, CVD is the leading cause of death (25). Women with or without CVD have a longer life expectancy than their male counterparts (26). CVD is also the leading cause of death among both men and women with diabetes (27); however, the male excess in CVD mortality has been reported to be eliminated or attenuated in those with diabetes (28). Whether diabetes eliminates the male excess in all-cause mortality is less clear (11,29,30). In our population, this was not the case. Women with diabetes had nearly half the risk of all-cause mortality as men. The relative increase in mortality risk among those with diabetes compared with those without diabetes, stratified by sex, was only slightly higher in women. A more adverse

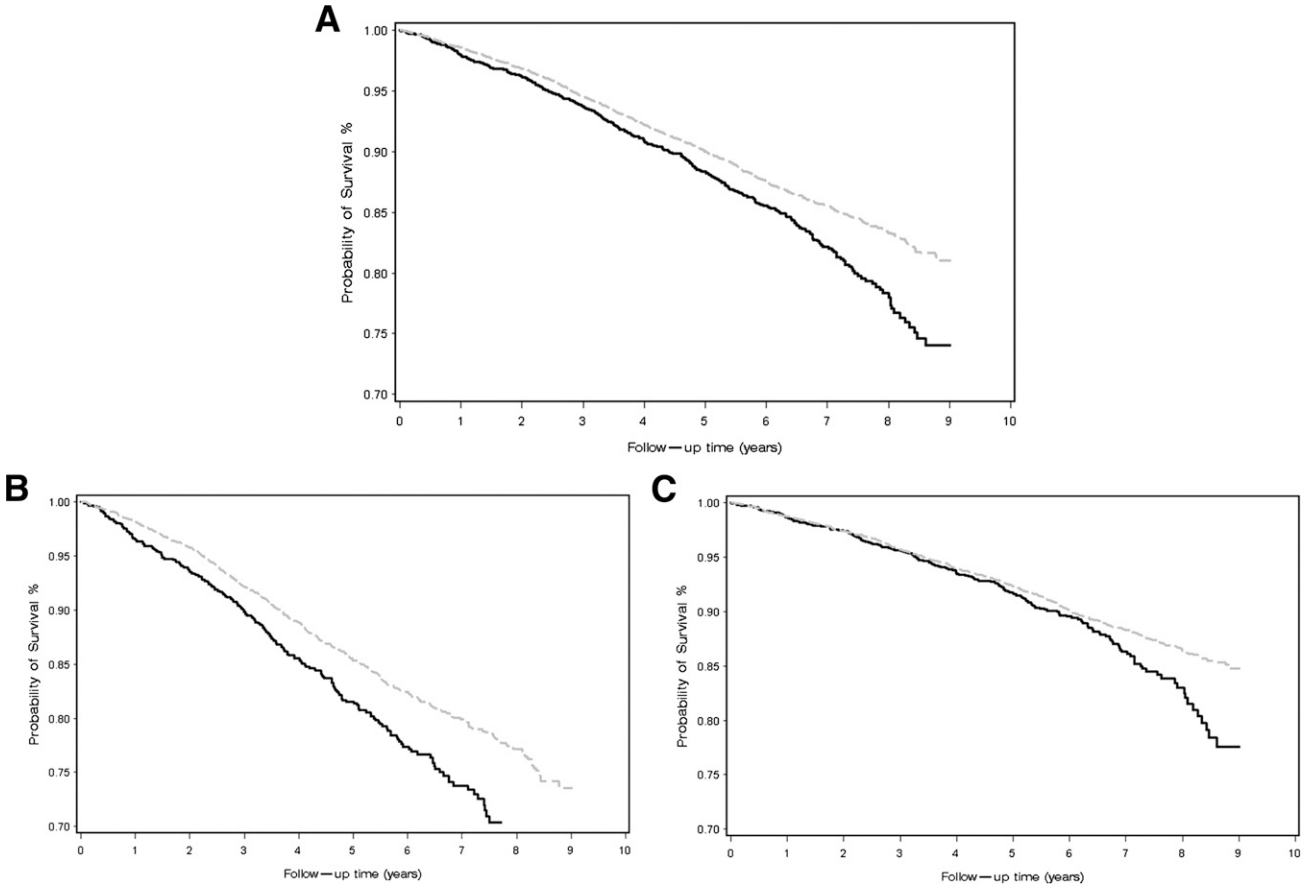

Figure 1—*Kaplan-Meier analysis curve of mortality in African Americans* (broken lines) *and whites* (solid lines) *with type 2 diabetes.* A*: Survival curves in both sexes combined.* B*: Survival curves in men.* C*: Survival curves in women.*

risk factor profile, factors related to CVD risk factor management, and CVD diagnosis and treatment have been posited as reasons for a greater relative impact of diabetes on mortality in women than in men seen in some studies (28,29,31). We did not observe a more adverse CVD risk factor profile among women than men in our population, which may explain why we did not observe a loss of the protective effect of being female on mortality that is sometimes observed in diabetes.

A major strength of this study is its large sample size. To our knowledge, this is the largest cohort study of African Americans with diabetes to date. By including African Americans and whites of similarly low SES backgrounds and similar access to health care and then further adjusting for SES-related factors, we were able to more accurately investigate the impact of diabetes, and risk factors within diabetes, on mortality both overall and by race specifically. Nevertheless, this study does have important limitations that need to be highlighted. Data on renal function and damage, major predictors of mortality in diabetes, were not available. Additionally, information on the severity of diabetes was limited so that we had to rely on surrogate indicators, namely, the duration of diabetes and the types of antihyperglycemic medications. We also had no histories of medications—only information on the drugs reported being used at entry into the cohort. However, the differences between African Americans and whites for type of diabetes therapy did not explain differences in overall mortality rates between African Americans and whites. Data as to type of antihyperglycemic agent were missing on 6% of the participants, but omission of these participants did not materially affect the results. Finally, diabetes status was based on self-reported physician diagnosis of diabetes and self-reported age at diagnosis; thus, misclassification is possible. Validation efforts based on review of medical records and/or HbA_{1c} levels for samples of SCCS participants, however, confirmed over 97% of the self-reports (32).

In conclusion, in low-SES individuals with diabetes and enrolled in a long-term prospective cohort study from community health centers, all-cause mortality was increased by nearly 90% among those with versus without diabetes. This excess risk was seen among both African Americans and whites. However, among diabetic patients mortality was lower among African Americans compared with whites. Our findings suggest no intrinsic poorer survival among African Americans with diabetes. Racial disparities in diabetes survival are small after accounting for SES and other risk factors for mortality.

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B.N.C. wrote the manuscript and analyzed data. M.E.M. reviewed the manuscript and contributed to the discussion. W.J.B. collected data, contributed to the methods and discussion, and reviewed and edited the manuscript for scientific content. B.N.C. is the guarantor of this work and, as such, had full access to all

the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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