

# Socioeconomic Status and Type 2 Diabetes in African American and Non-Hispanic White Women and Men: Evidence From the Third National Health and Nutrition Examination Survey

## ABSTRACT

**Objectives.** This study examined the associations of poverty income ratio (PIR), education, and occupational status with type 2 diabetes prevalence among African American and non-Hispanic White (White) women and men aged 40 to 74 years.

**Methods.** We analyzed cross-sectional data from the Third National Health and Nutrition Examination Survey, controlling for age and examination-related variables.

**Results.** Among African American women, there was a strong, graded association between PIR and diabetes, which remained significant after other risk factors were adjusted for. All 3 variables were significantly associated with diabetes among White women. Among White men, only PIR was significantly associated with diabetes. Controlling for risk factors substantially attenuated these associations among White women. There were no significant associations for African American men.

**Conclusions.** Socioeconomic status is associated with type 2 diabetes prevalence among women, but not consistently among men. Diabetes prevalence is more strongly associated with PIR than with education or occupational status. These associations are largely independent of other risk factors, especially among African American women. Economic resources should be addressed in efforts to explain and reverse the increasing prevalence of diabetes in the United States. (*Am J Public Health.* 2001;91:76–83)

Jessica M. Robbins, PhD, Viola Vaccarino, MD, PhD, Heping Zhang, PhD, and Stanislav V. Kasl, PhD

The prevalence of type 2 diabetes varies with socioeconomic status (SES) within populations. In industrialized countries, diabetes is associated with low SES.<sup>1,2</sup> SES is a complex construct, and different variables used to measure it (most often education, income, and occupation) can reflect different specific exposures. Income is a direct (although incomplete) measure of economic resources, but it may reflect feedback effects, because poor health can limit income. Educational status reflects childhood and adolescent SES as well as partially determining adult occupations and income. Occupation carries with it specific environmental exposures, and it may be of limited value in measuring SES for women who have not been in the paid workforce for much of their adult lives. Each of these variables provides somewhat different information, and differentiating their effects should advance our efforts to understand—and address—socioeconomic inequalities in diabetes in the United States.

We examined the association of diabetes with each of these measures of SES within each of 4 strata—African American women, non-Hispanic White (hereafter called “White”) women, African American men, and White men—because there are indications that the association may vary with sex as well as race/ethnicity.<sup>3</sup> In addition, we examined the role of a number of risk factors in mediating these associations, including obesity, physical activity, diet, and alcohol and tobacco use.

## Methods

The Third National Health and Nutrition Examination Survey (NHANES III) collected cross-sectional data between 1988 and 1994 from a sample representative of the civilian noninstitutionalized population of the United States.<sup>4</sup> Among Whites and African Americans aged 40 to 74 years who were identified as sample persons, basic demographic informa-

tion was obtained for 100% (where necessary, from interviews with at least 2 neighbors), and 77% were interviewed successfully. Interview rates for African Americans (women, 81%; men, 79%) were slightly higher than for Whites (women, 78%; men, 76%).<sup>5</sup>

All subjects were also invited to receive physical examinations in mobile examination centers; for subjects aged 40 to 74 years, a 2-hour, 75-g oral glucose tolerance test (OGTT) was conducted unless a subject refused the test or reported using insulin. Among those interviewed, physical examinations were completed and diabetic status was ascertained (on the basis of the use of antidiabetic medication or OGTT) for 77% of African American men, 79% of African American women and White women, and 82% of White men. Overall, there was little variation of missing information according to SES or self-rated health. Within strata, the widest variation by any of the SES variables in rates of diabetes ascertainment was among White men, with 84% diabetes ascertainment among those with incomes at least 5 times the poverty level and 75% among those with incomes below the poverty line.

There were 4993 White and African American women and men aged 40 to 74 years who were examined in the mobile examination center and who either reported use of antidiabetic medication or completed an OGTT. Fifteen subjects who reported a physician's diagnosis of diabetes before age 40 and use of insulin within 1 year of diagnosis were excluded

At the time of the study, all of the authors were with the Department of Epidemiology and Public Health, Yale University, New Haven, Conn. Jessica M. Robbins is now with the Population Studies Center, University of Pennsylvania, Philadelphia.

Requests for reprints should be sent to Jessica M. Robbins, PhD, Population Studies Center, University of Pennsylvania, 3718 Locust Walk, Philadelphia, PA 19104-6298 (e-mail: jessica\_robbins@pop.upenn.edu).

This article was accepted June 26, 2000.

**TABLE 1—Characteristics of the Type 2 Diabetes Study Sample: Third National Health and Nutrition Examination Survey, 1988–1994**

	African American Women (n=961)	Non-Hispanic White Women (n=1641)	African American Men (n=839)	Non-Hispanic White Men (n=1537)
Poverty income ratio, mean (SE)	2.04 (0.05)	3.38 (0.05)	2.30 (0.06)	3.62 (0.05)
Years of education, mean (SE)	11.03 (0.10)	12.19 (0.07)	10.22 (0.13)	12.44 (0.08)
Duncan SEI score, mean (SE)	29.65 (0.54)	36.06 (0.42)	27.19 (0.45)	35.77 (0.43)
Diabetic, %	20.1	13.2	18.8	13.9
Age, mean (SE)	54.16 (0.33)	57.87 (0.26)	55.60 (0.36)	58.21 (0.27)
Mean length of fast, h (SE)	10.37 (0.15)	10.35 (0.10)	10.21 (0.17)	10.45 (10)
Weekend examination, %	31.4	26.6	28.2	28.2
Season of examination, %				
Summer	21.9	35.6	21.2	33.3
Winter	27.6	19.0	29.9	20.9
Spring/fall	50.6	45.4	48.9	45.8
Region, %				
Northeast	20.0	17.8	14.2	19.0
South	52.5	37.3	56.0	41.1
Midwest	20.4	28.5	21.1	25.7
West	7.1	16.5	8.7	14.2
Body mass index, mean (SE)	30.41 (0.24)	27.49 (0.15)	26.99 (0.17)	27.41 (0.12)
Waist–hip ratio, mean (SE)	0.908 (0.003)	0.895 (0.002)	0.969 (0.002)	0.996 (0.002)
Weight (kg) at age 25, mean (SE)	60.61 (0.42)	57.74 (0.25)	74.26 (0.45)	74.76 (0.33)
Exercise score, mean (SE)	60.40 (3.22)	92.58 (2.68)	94.43 (4.30)	113.21 (3.04)
Total kilocalories, mean (SE)	1559 (23)	1651 (16)	2108 (38)	2361 (24)
Current cigarette smoker, %	27.4	20.6	42.7	24.8
Current alcohol use, %	24.1	36.8	54.2	56.0

Note. SEI = socioeconomic index.

as possibly having type 1 diabetes, leaving a study sample of 4978: 961 African American women, 1641 White women, 839 African American men, and 1537 White men.

### Definition of Variables

Subjects whose race was identified by the respondent as Black or a mixed racial category including Black<sup>6</sup> and whose ancestry was not identified as Mexican/Mexican American or other Latin American or Spanish were classified as non-Hispanic Blacks (referred to here as African Americans). Non-Hispanic Whites (referred to here as Whites) were defined as subjects whose race was reported as White (or more than 1 racial category, not including Black, with White mentioned first)<sup>6</sup> and whose ancestry was not identified by the respondent as Mexican/Mexican American or other Latin American or Spanish. This study examines only the White and African American groups.

Income was modeled as categories of poverty income ratio (PIR), which is annual family income divided by the federal poverty line. This line is adjusted each calendar year for inflation and varies with the size of the household. The categories used in the analyses of African American women and men were less than 1 (poor), 1 to 1.999 (near poor), and 2 and above. In analyses of White women and men, there was a much larger proportion

of higher-income families, and the highest income group was divided into the categories 2 to 2.999, 3 to 4.999, and 5 and above. In all cases, the lowest income group, comprising those with family incomes below the poverty line, was used as the reference category. Education was categorized as less than 9 years, 9 to 11 years, 12 years, and 13 years and above. Occupations were coded to 1 of the 13 major occupational groups assigned by the Census Bureau in 1980 and assigned the average Duncan socioeconomic index score for that grouping<sup>7</sup>; these scores were modeled as tertiles.

Diabetes prevalence was ascertained by OGTT and by the use of antidiabetic medications. Subjects who were examined in the morning were instructed to fast for 12 hours preceding the test. Those who were examined in the afternoon or evening were instructed to fast for 6 to 8 hours. Glucose levels were measured at the Diabetes Reference Laboratory at the University of Missouri–Columbia by a modified hexokinase enzymatic method.<sup>8</sup> Following criteria developed by the National Diabetes Data Group, subjects with fasting glucose levels of  $\geq 7.8$  mmol/L (140 mg/dL) (all examinees) or 2-hour glucose levels of  $\geq 11.1$  mmol/L (200 mg/dL) (for morning examinees) or  $\geq 13.9$  mmol/L (250 mg/dL) (for afternoon and evening examinees) were classified as type 2 diabetes cases. All subjects who reported use of antidiabetic medications

were classified as type 2 diabetes cases, regardless of whether or not they completed the OGTT.

Covariables examined as possible mediators of the SES–diabetes association included body size, physical activity, diet, and alcohol and tobacco use. Stratum-specific tertiles of 3 measures of body size were used: body mass index (BMI) (weight in kilograms divided by height in meters squared), waist–hip ratio (waist circumference divided by hip circumference), and self-reported weight at age 25. On the basis of a series of questions about walking and recreational activities in the past month and standard intensity ratings for these activities,<sup>9</sup> we calculated an exercise score for each subject, and the log of these scores was used along with a dichotomous variable for any participation in vigorous exercise and a variable indicating whether subjects reported that they were more or less active than 10 years previously. Dietary variables included were total kilocalories consumed and total fat and saturated fat as percentages of total kilocalories. Alcohol use was based on subjects' reports of the frequency of drinking and average amount consumed on each occasion during the 12 months preceding the interview. Variables for ever having been a smoker, being a current smoker, number of cigarettes smoked daily at the time of the interview, and lifetime number of cigarettes smoked were included as measures of tobacco use. Some analyses

**TABLE 2—Odds Ratios (95% Confidence Intervals) for Type 2 Diabetes Prevalence Among African American Women, by Socioeconomic Status (SES): Third National Health and Nutrition Examination Survey, 1988–1994**

Control Variable	SES Variables		
		Poverty Income Ratio	
	<1 <sup>a</sup>	1–1.999	≥2
Age and confounders <sup>b</sup>	1.00	0.62 (0.36, 1.06)	0.42 (0.25, 0.70)**
Body size	1.00	0.79 (0.42, 1.49)	0.51 (0.28, 0.96)*
Physical activity	1.00	0.60 (0.34, 1.05)	0.44 (0.26, 0.74)**
Diet	1.00	0.60 (0.34, 1.04)	0.43 (0.25, 0.74)**
Cigarette smoking	1.00	0.67 (0.42, 1.09)	0.41 (0.25, 0.66)***
Alcohol use	1.00	0.59 (0.34, 1.04)	0.41 (0.23, 0.74)**
All mediators	1.00	0.81 (0.45, 1.47)	0.53 (0.29, 0.96)*
	Years of Education		
	0–8 <sup>a</sup>	9–11	12
Age and confounders <sup>b</sup>	1.00	1.18 (0.71, 1.97)	0.90 (0.42, 1.93)
Body size	1.00	1.29 (0.69, 2.41)	0.97 (0.41, 2.30)
Physical activity	1.00	1.30 (0.77, 2.21)	1.02 (0.46, 2.24)
Diet	1.00	1.19 (0.70, 2.02)	0.96 (0.45, 2.04)
Cigarette smoking	1.00	1.13 (0.69, 1.84)	0.86 (0.40, 1.85)
Alcohol use	1.00	1.04 (0.65, 1.68)	0.81 (0.39, 1.68)
All mediators	1.00	1.21 (0.62, 2.38)	0.84 (0.36, 1.99)
	Duncan Socioeconomic Index Score		
	<21 <sup>a</sup>	21–32	>32
Age and confounders <sup>b</sup>	1.00	0.56 (0.32, 0.96)	0.84 (0.43, 1.65)
Body size	1.00	0.53 (0.27, 1.04)	1.11 (0.51, 2.41)
Physical activity	1.00	0.56 (0.32, 0.96)	0.94 (0.47, 1.88)
Diet	1.00	0.53 (0.31, 0.91)	0.84 (0.43, 1.63)
Cigarette smoking	1.00	0.52 (0.30, 0.92)	0.80 (0.45, 1.45)
Alcohol use	1.00	0.55 (0.31, 0.98)	0.86 (0.44, 1.72)
All mediators	1.00	0.46 (0.21, 1.01)	1.04 (0.46, 2.35)

<sup>a</sup>Reference category.

<sup>b</sup>All models are adjusted for age and the following confounders: time of day (morning, afternoon, or evening); day of the week (weekday or weekend); season of the year (June–August, December–March, or other); number of minutes between the time glucose was administered and the “2-hour” venipuncture (categorized as high or low when it deviated from 120 minutes by more than 1 standard deviation [6.575 minutes]); and reported length of fast before the oral glucose tolerance test (categorized as high [ $>13$  hours], medium, or low [ $<6$  hours for morning examinees or  $<4.5$  hours for afternoon or evening examinees]).

\*.01  $< P < .05$ ; \*\*.001  $< P < .01$ ; \*\*\* $P < .001$ .

also included a variable for self-rated health (rated from 1 = excellent to 5 = poor).

All analyses were controlled for age and for variables related to the conditions of the examination, including time of day, day of the week, season of the year, minutes between the time glucose was administered and the “2-hour” venipuncture, and reported length of fast before the OGTT.

### Data Analysis

We used logistic regression to evaluate the association of income, education, and occupational status with type 2 diabetes among African American and White women and men. We examined each of these associations in a series of models: controlling for age and examination-related confounders only, controlling for each of the potential mediating variables described above, and controlling for all of the potential mediators together. We repeated the analyses controlling for self-assessed health, to assess the possibility that

diabetes-related poor health caused low SES, rather than low SES caused diabetes.

SUDAAN statistical software (version 7.5.2; Research Triangle Institute, Research Triangle Park, NC) was used to adjust for clustering and sample weights.<sup>10</sup> In models using PIR, “no information” was included as a separate category but is not included in the tables.

Several sensitivity analyses were conducted—that is, excluding proxy reports, non-standard afternoon or evening OGTT, or imputed values for BMI, and including suspected type 1 diabetic individuals in the type 2 diabetic group.

### Results

The distribution of key variables among the subjects in each of the 4 strata is shown in Table 1. As anticipated, each stratum is different. Some variables (including SES variables) varied substantially with race/ethnicity,

while body size and behavioral variables differed by sex.

### African American Women

In models adjusted for age and examination-related confounders, PIR was strongly associated with diabetes prevalence among African American women (Table 2). African American women whose family income was at least twice the poverty level had an odds ratio of 0.42 (95% confidence interval [CI]=0.25, 0.70) relative to women with family incomes below the poverty line. Those in the intermediate PIR category (PIR=1–1.999) had a marginally significant odds ratio of 0.62 (95% CI=0.36, 1.06). Addition of mediating variables increased the odds ratio for the higher income group from 0.42 to 0.53 (95% CI=0.29, 0.96) and for the near-poor group from 0.62 to 0.81 (95% CI=0.45, 1.47). The most important mediator was body size. Neither education nor Duncan socioeconomic index score was a significant predictor of diabetes prevalence among African American women.

**TABLE 3—Odds Ratios (95% Confidence Intervals) for Type 2 Diabetes Prevalence Among Non-Hispanic White Women, by Socioeconomic Status (SES): Third National Health and Nutrition Examination Survey, 1988–1994**

Control Variable	SES Variables				
	<1 <sup>a</sup>	Poverty Income Ratio			≥5
		1–1.999	2–2.999	3–4.999	
Age and confounders <sup>b</sup>	1.00	0.76 (0.42, 1.38)	0.61 (0.35, 1.05)	0.35 (0.19, 0.65)	0.25 (0.13, 0.48)***
Body size	1.00	0.93 (0.44, 1.93)	0.77 (0.39, 1.50)	0.45 (0.21, 0.95)	0.36 (0.17, 0.77)*
Physical activity	1.00	0.83 (0.46, 1.51)	0.69 (0.40, 1.20)	0.42 (0.22, 0.79)	0.33 (0.16, 0.65)*
Diet	1.00	0.74 (0.40, 1.36)	0.63 (0.36, 1.13)	0.37 (0.20, 0.68)	0.27 (0.13, 0.53)**
Cigarette smoking	1.00	0.80 (0.44, 1.46)	0.64 (0.36, 1.15)	0.36 (0.19, 0.70)	0.26 (0.13, 0.52)**
Alcohol use	1.00	0.81 (0.45, 1.47)	0.68 (0.38, 1.21)	0.41 (0.21, 0.78)	0.31 (0.15, 0.64)*
All mediators	1.00	0.97 (0.44, 2.15)	0.91 (0.45, 1.84)	0.54 (0.23, 1.26)	0.50 (0.22, 1.14)
		Years of Education			
		0–8 <sup>a</sup>	9–11	12	≥13
Age and confounders <sup>b</sup>	1.00	1.00	1.23 (0.70, 2.16)	0.88 (0.50, 1.53)	0.38 (0.21, 0.71)***
Body size	1.00	1.00	1.54 (0.64, 3.69)	1.23 (0.56, 2.73)	0.70 (0.32, 1.53)
Physical activity	1.00	1.00	1.28 (0.71, 2.33)	0.98 (0.53, 1.80)	0.50 (0.25, 1.00)*
Diet	1.00	1.00	1.21 (0.69, 2.12)	0.93 (0.54, 1.62)	0.42 (0.22, 0.79)**
Cigarette smoking	1.00	1.00	1.24 (0.71, 2.18)	0.89 (0.51, 1.54)	0.39 (0.21, 0.72)***
Alcohol use	1.00	1.00	1.18 (0.66, 2.10)	0.93 (0.53, 1.64)	0.48 (0.26, 0.87)**
All mediators	1.00	1.00	1.60 (0.62, 4.12)	1.50 (0.63, 3.53)	1.06 (0.41, 2.70)
		Duncan Socioeconomic Index Score			
		<21 <sup>a</sup>	21–32	>32	
Age and confounders <sup>b</sup>	1.00	1.00	0.70 (0.51, 0.97)	0.48 (0.29, 0.80)**	
Body size	1.00	1.00	0.81 (0.53, 1.25)	0.66 (0.40, 1.11)	
Physical activity	1.00	1.00	0.79 (0.54, 1.14)	0.56 (0.33, 0.95)	
Diet	1.00	1.00	0.69 (0.50, 0.95)	0.49 (0.29, 0.82)**	
Cigarette smoking	1.00	1.00	0.71 (0.50, 1.00)	0.48 (0.29, 0.81)**	
Alcohol use	1.00	1.00	0.76 (0.54, 1.08)	0.54 (0.33, 0.90)*	
All mediators	1.00	1.00	0.95 (0.61, 1.49)	0.77 (0.43, 1.38)	

<sup>a</sup>Reference category.

<sup>b</sup>All models are adjusted for age and the following confounders: time of day (morning, afternoon, or evening); day of the week (weekday or weekend); season of the year (June–August, December–March, or other); number of minutes between the time glucose was administered and the “2-hour” venipuncture (categorized as high or low when it deviated from 120 minutes by more than 1 standard deviation [6.575 minutes]); and reported length of fast before the oral glucose tolerance test (categorized as high [ $>13$  hours], medium, or low [ $<6$  hours for morning examinees or  $<4.5$  hours for afternoon or evening examinees]).

\*.01  $< P < .05$ ; \*\*.001  $< P < .01$ ; \*\*\* $P < .001$ .

### White Women

PIR was also the SES variable most strongly associated with diabetes among White women (Table 3). The relationship between PIR and diabetes was inverse and graded, with age-adjusted odds ratios decreasing as PIR increased, relative to women below the poverty line. Education was also inversely associated with diabetes prevalence among White women, in particular for the group with more than 12 years of education (age-adjusted odds ratio [OR] = 0.38; 95% CI = 0.21, 0.71). Duncan socioeconomic index score was also associated with diabetes prevalence.

PIR effects were attenuated but still substantial after potential mediators were adjusted for. When body size variables were added to the age-adjusted model for White women, the odds ratio for a PIR of 5 or higher increased from 0.25 to 0.36 (95% CI = 0.17, 0.77). None of the other potential mediators had a substantial impact on the association between PIR and diabetes, but when all of them were controlled for, the association between PIR and

diabetes prevalence was no longer statistically significant. The fully adjusted odds ratio for the highest income group was 0.50 (95% CI = 0.22, 1.14).

### African American Men

None of the SES variables were strongly associated with diabetes prevalence among African American men (Table 4). The higher income (PIR =  $\geq 2$ ) group had an odds ratio of 0.77 (95% CI = 0.44, 1.34) relative to those with incomes below the poverty line, and the intermediate (near-poor) income group had the lowest odds ratio for diabetes prevalence (OR = 0.61; 95% CI = 0.36, 1.04). Additional analyses were conducted to determine whether these nonlinear effects were sensitive to the categories and cutpoints chosen. When PIR was modeled in stratum-specific quintiles (data not shown), the results were similar. Education also did not yield significant associations with diabetes in African American men. Contrary to expectations, the association between occupational status and diabetes prevalence was

positive, although it did not reach statistical significance. The addition of other covariables to the models did not substantially alter any of these associations.

### White Men

The only SES variable that was significantly associated with diabetes prevalence among White men was PIR (Table 5). This association was not linear, although the highest prevalence was found in the lowest income group. As with the results for African American men, use of PIR modeled in stratum-specific quintiles produced very similar results (data not shown). In age-adjusted models, education had a weak inverse gradient with diabetes prevalence, with an odds ratio for those with more than 12 years of education of 0.60 (95% CI = 0.32, 1.13) relative to those with less than 9 years of education. As with African American men, the (nonsignificant) direction of the association between occupational status and diabetes prevalence was positive.

Most of the potentially mediating variables examined, and all of them together, ac-

**TABLE 4—Odds Ratios (95% Confidence Intervals) for Type 2 Diabetes Prevalence Among African American Men, by Socioeconomic Status (SES): Third National Health and Examination Survey, 1988–1994**

Control Variable	SES Variables		
	<1 <sup>a</sup>	Poverty Income Ratio	
		1–1.999	≥2
Age and confounders <sup>b</sup>	1.00	0.61 (0.36, 1.04)	0.77 (0.44, 1.34)
Body size	1.00	0.69 (0.44, 1.08)	0.85 (0.49, 1.46)
Physical activity	1.00	0.62 (0.36, 1.06)	0.76 (0.44, 1.32)
Diet	1.00	0.63 (0.36, 1.08)	0.83 (0.49, 1.43)
Cigarette smoking	1.00	0.62 (0.36, 1.06)	0.68 (0.37, 1.25)
Alcohol use	1.00	0.55 (0.33, 0.93)	0.64 (0.37, 1.12)
All mediators	1.00	0.62 (0.39, 0.99)	0.75 (0.44, 1.27)
		Years of Education	
	0–8 <sup>a</sup>	9–11	12
Age and confounders <sup>b</sup>	1.00	1.02 (0.46, 2.27)	0.68 (0.35, 1.30)
Body size	1.00	1.11 (0.46, 2.68)	0.77 (0.41, 1.44)
Physical activity	1.00	1.01 (0.45, 2.26)	0.68 (0.34, 1.33)
Diet	1.00	0.95 (0.41, 2.17)	0.72 (0.36, 1.42)
Cigarette smoking	1.00	1.00 (0.46, 2.18)	0.61 (0.32, 1.18)
Alcohol use	1.00	0.96 (0.42, 2.22)	0.59 (0.30, 1.15)
All mediators	1.00	0.98 (0.35, 2.70)	0.68 (0.32, 1.43)
		Duncan Socioeconomic Index Score	
	<21 <sup>a</sup>	21–32	>32
Age and confounders <sup>b</sup>	1.00	1.02 (0.62, 1.68)	1.94 (0.80, 4.69)
Body size	1.00	0.96 (0.57, 1.60)	2.14 (0.81, 5.62)
Physical activity	1.00	1.01 (0.62, 1.65)	2.04 (0.88, 4.70)
Diet	1.00	1.13 (0.67, 1.90)	2.01 (0.80, 5.06)
Cigarette smoking	1.00	1.02 (0.62, 1.67)	1.76 (0.71, 4.41)
Alcohol use	1.00	1.07 (0.62, 1.86)	1.98 (0.78, 5.01)
All mediators	1.00	1.06 (0.57, 1.96)	2.08 (0.77, 5.64)

<sup>a</sup>Reference category.

<sup>b</sup>All models are adjusted for age and the following confounders: time of day (morning, afternoon, or evening); day of the week (weekday or weekend); season of the year (June–August, December–March, or other); number of minutes between the time glucose was administered and the “2-hour” venipuncture (categorized as high or low when it deviated from 120 minutes by more than 1 standard deviation [6.575 minutes]); and reported length of fast before the oral glucose tolerance test (categorized as high [ $>13$  hours], medium, or low [ $<6$  hours for morning examinees or  $<4.5$  hours for afternoon or evening examinees]).

tually strengthened the association between PIR and diabetes among White men. The slight inverse gradient between diabetes prevalence and education, on the other hand, was eliminated after all the potential mediators were adjusted for, although no one variable had a particularly strong impact. The unexpected direct (but nonsignificant) association between occupational status and diabetes prevalence was not diminished by the addition of covariables.

### Self-Assessed Health

When the analyses were repeated with a variable for self-assessed health, to control for the possibility that poor health had led to low SES, the results were essentially unchanged (data not shown). PIR was still more strongly and consistently associated with diabetes prevalence than either education or occupational status.

### Sensitivity Analyses

There were no meaningful differences in the results when data provided by proxies, sub-

jects who received nonstandard OGTTs, or imputed body size variables were removed from the data set, or when subjects with suspected type 1 diabetes were included with type 2 diabetic subjects.

## Discussion

These analyses provide strong, consistent evidence that SES is inversely associated with type 2 diabetes in both African American and White women. They do not provide such evidence for African American or White men. Income as a percentage of the poverty line was the SES variable most consistently associated with diabetes prevalence. It was strongly and negatively associated with diabetes prevalence in both African American and White women in NHANES III. This association was only partially mediated by variables usually regarded as modifiable personal health behaviors or characteristics. There was also a trend toward a negative association between PIR and diabetes prevalence among both African Ameri-

can and White men, although for African American men this did not attain statistical significance. The relationships of diabetes prevalence with education and occupation were much less consistent.

The finding of socioeconomic inequalities in diabetes prevalence by many researchers in different populations<sup>11–15</sup> suggests that this phenomenon is unlikely to be a chance finding or the result of confounding by some variable coincidentally associated with SES in a specific population. One possible explanation for this association is that poor health results in low SES, rather than the reverse. While there is evidence that health status affects income and wealth,<sup>16</sup> research directed at this issue has indicated that these “social selection” effects do not explain a substantial part of the SES association with health.<sup>17–19</sup> When we repeated our analyses, controlling for self-assessed health status, PIR continued to be more strongly associated with diabetes prevalence than other SES variables.

Income, particularly modeled as PIR, may be a more sensitive indicator of current adult SES than either education, which is usually

**TABLE 5—Odds Ratios (95% Confidence Intervals) for Type 2 Diabetes Prevalence Among Non-Hispanic White Men, by Socioeconomic Status (SES): Third National Health and Examination Survey, 1988–1994**

Control Variable	SES Variables				
	<1 <sup>a</sup>	Poverty Income Ratio			≥5
		1–1.999	2–2.999	3–4.999	
Age and confounders <sup>b</sup>	1.00	0.44 (0.17, 1.11)	0.70 (0.30, 1.61)	0.39 (0.16, 0.92)	0.68 (0.26, 1.77)*
Body size	1.00	0.27 (0.11, 0.64)	0.50 (0.23, 1.10)	0.31 (0.15, 0.67)	0.61 (0.27, 1.38)***
Physical activity	1.00	0.48 (0.19, 1.20)	0.78 (0.34, 1.81)	0.47 (0.20, 1.12)	0.85 (0.33, 2.15)
Diet	1.00	0.41 (0.16, 1.03)	0.74 (0.33, 1.63)	0.40 (0.18, 0.92)	0.72 (0.29, 1.82)**
Cigarette smoking	1.00	0.39 (0.16, 0.98)	0.65 (0.29, 1.45)	0.34 (0.15, 0.76)	0.59 (0.24, 1.47)**
Alcohol use	1.00	0.37 (0.15, 0.90)	0.71 (0.31, 1.66)	0.40 (0.17, 0.96)	0.76 (0.25, 2.27)**
All mediators	1.00	0.24 (0.11, 0.55)	0.64 (0.32, 1.28)	0.38 (0.20, 0.75)	0.81 (0.34, 1.92)***
		Years of Education			
		0–8 <sup>a</sup>	9–11	12	≥13
Age and confounders <sup>b</sup>	1.00	1.00	0.93 (0.47, 1.85)	0.86 (0.50, 1.50)	0.60 (0.32, 1.13)
Body size	1.00	1.00	1.03 (0.51, 2.07)	0.86 (0.45, 1.65)	0.72 (0.38, 1.36)
Physical activity	1.00	1.00	0.98 (0.52, 1.87)	0.92 (0.55, 1.54)	0.71 (0.39, 1.27)
Diet	1.00	1.00	1.05 (0.51, 2.14)	0.96 (0.51, 1.82)	0.72 (0.35, 1.47)
Cigarette smoking	1.00	1.00	0.92 (0.47, 1.79)	0.83 (0.50, 1.39)	0.56 (0.30, 1.05)
Alcohol use	1.00	1.00	1.05 (0.51, 2.16)	0.99 (0.55, 1.80)	0.73 (0.36, 1.49)
All mediators	1.00	1.00	1.29 (0.61, 2.70)	1.11 (0.53, 2.36)	1.04 (0.48, 2.26)
		Duncan Socioeconomic Index Score			
		<21 <sup>a</sup>	21–32	>32	
Age and confounders <sup>b</sup>	1.00	1.00	1.14 (0.67, 1.95)	1.14 (0.63, 2.07)	
Body size	1.00	1.00	1.06 (0.65, 1.74)	1.27 (0.72, 2.25)	
Physical activity	1.00	1.00	1.12 (0.66, 1.89)	1.26 (0.72, 2.23)	
Diet	1.00	1.00	1.09 (0.62, 1.94)	1.18 (0.62, 2.26)	
Cigarette smoking	1.00	1.00	1.22 (0.72, 2.08)	1.18 (0.64, 2.17)	
Alcohol use	1.00	1.00	1.19 (0.70, 2.02)	1.33 (0.70, 2.53)	
All mediators	1.00	1.00	1.07 (0.62, 1.88)	1.64 (0.83, 3.23)	

<sup>a</sup>Reference category.

<sup>b</sup>All models are adjusted for age and the following confounders: time of day (morning, afternoon, or evening); day of the week (weekday or weekend); season of the year (June–August, December–March, or other); number of minutes between the time glucose was administered and the “2-hour” venipuncture (categorized as high or low when it deviated from 120 minutes by more than 1 standard deviation [6.575 minutes]); and reported length of fast before the oral glucose tolerance test (categorized as high [ $>13$  hours], medium, or low [ $<6$  hours for morning examinees or  $<4.5$  hours for afternoon or evening examinees]).

\*.01  $< P < .05$ ; \*\*.001  $< P < .01$ ; \*\*\* $P < .001$ .

completed by early adulthood, or usual occupation, which may be misleading or uninformative, especially for women and retired people. Income is not only a direct (albeit seriously incomplete)<sup>20</sup> measure of economic resources but also a primary determinant of social prestige in the United States.<sup>21</sup> The fact that years of education, while strongly associated with diabetes prevalence among White women, was not a significant predictor of diabetes prevalence among African American women may reflect several factors.<sup>22</sup> Systematic racial inequalities in education (the majority of African Americans in this study resided in the South and would have attended segregated schools with greatly inferior resources) and in the economic and occupational returns to education could have limited the protective effect of education for African American women. The study also had less statistical power to detect an effect among African American women because of the smaller numbers with post–high school education.

Socioeconomic inequalities in health have been attributed to a number of different mech-

anisms, including unhealthy behaviors, inadequate access to health care, nutritional inadequacies and other inequalities in material circumstances, and psychologic stress.<sup>23,24</sup> Most of these mechanisms are at least plausible intermediate risk factors for type 2 diabetes.

The data available for this study permitted direct examination of only a few of the possible mechanisms by which SES could affect type 2 diabetes. Adult body size was examined as a potential mediator, as were measures of physical activity, dietary caloric and fat intakes, and use of alcohol and tobacco. These variables, which are most often seen as the appropriate targets for health promotion efforts aimed at individual risk behaviors, generally did not explain the major portion of the effects associated with SES. Body size variables were more powerful mediators than any of the other variables examined. Difficulties in the measurement of behavioral variables may explain why these risk factors do not appear to explain socioeconomic inequalities in diabetes prevalence, but it is likely that there are other, unmeasured mediating factors as well.

Unequal access to health care, although clearly implicated in some health inequalities, is not likely to be an important factor in the socioeconomic inequalities in the prevalence (as opposed to the course) of diabetes. Whereas some studies have indicated the potential for primary prevention of diabetes, there is little evidence to date that medical care has been effective in preventing the onset of type 2 diabetes.<sup>25–27</sup> Medical care does play an important role in controlling diabetes and in the complications, quality of life, and mortality associated with the disease.<sup>28</sup>

SES may affect diabetes by its influence on prenatal and perinatal factors. A growing body of work, especially that of Barker and colleagues, has used both animal studies and epidemiologic research to suggest that diabetes is the result of developmental problems related to maternal nutrition during (and perhaps before) pregnancy.<sup>29</sup> This thesis, however, remains controversial. Attempts by other investigators to replicate these findings have had inconsistent results, and some reviewers have raised serious methodologic concerns.<sup>30</sup>

It has been argued that the relationship between SES and type 2 diabetes is mediated by the physiologic response to chronic psychologic stress, although epidemiologic evidence that directly links stress with diabetes prevalence has not yet been produced. Laboratory studies have shown that hyperglycemic conditions similar to type 2 diabetes can be induced by stress in normoglycemic animals<sup>31</sup> and that stress can impair glucose tolerance in normoglycemic humans.<sup>32</sup> There is evidence from human studies that stress can have a negative impact on glycemic control in established diabetes.<sup>33–36</sup>

Another possible link between low SES and type 2 diabetes is depression, which is associated with both.<sup>37–40</sup> Depression is also much more prevalent among women than men,<sup>41</sup> which could explain the finding that SES is strongly associated with type 2 diabetes among women but not men.

There are a number of limitations of this study that should be noted. Some important elements of SES were not examined. Childhood SES, economic assets (including home ownership), and neighborhood variables were not examined.

Other methodologic problems exist. Despite extensive efforts to maximize response rates to all elements of the surveys, substantial nonresponse did occur; however, there is no evidence that nonresponse induced bias in our data.

Recent studies suggest that, among African Americans, type 2 diabetes may not be uncommon among those whose diabetes was diagnosed in young adulthood.<sup>42</sup> By excluding cases with onset before 40 years of age and with insulin use within 1 year of diagnosis, this study may have excluded some type 2 cases. However, the small number of such cases did not have any important effect on the results.

Despite the abundant evidence for socioeconomic inequalities in many health outcomes, efforts to identify specific pathways have, as in this study, rarely succeeded in explaining most of these effects. It is possible that the pathways by which SES affects the development of diabetes are so complex and redundant that efforts to identify a single or primary mechanism are not likely either to succeed or to offer effective strategies to control the increasing prevalence of diabetes.<sup>43</sup> If so, efforts to reduce socioeconomic inequalities in diabetes must address the “upstream” social, political, and economic policies that produce and structure those inequalities and not only the “downstream” individual behaviors that result from them.<sup>44</sup> □

## Contributors

J.M. Robbins planned the study, analyzed the data, and wrote the paper. V. Vaccarino, H. Zhang, and S. V.

Kasl supervised the study design and data analysis, provided feedback in the interpretation of the data, critically revised the paper, and gave final approval of the manuscript. H. Zhang also provided statistical expertise.

## Acknowledgments

This research was supported by US Public Health Service grant 5-T32-MH 1435-22 from the National Institute of Mental Health.

## References

1. Knowler WC, McCance DR, Nagi DK, Pettitt DJ. Epidemiological studies of the causes of non-insulin-dependent diabetes mellitus. In: Leslie RDG, ed. *Causes of Diabetes: Genetic and Environmental Factors*. Chichester, England: John Wiley & Sons; 1993:187–218.
2. West K. *Epidemiology of Diabetes and Its Vascular Lesions*. New York, NY: Elsevier; 1978.
3. Gaillard TR, Schuster DP, Bossetti BM, Green PA, Osei K. Do sociodemographics and economic status predict risks for type II diabetes in African Americans? *Diabetes Educ*. 1997;23:294–300.
4. National Center for Health Statistics. Plan and operation of the Third National Health and Nutrition Examination Survey, 1988–1994. *Vital Health Stat 1*. 1994;No. 32.
5. National Center for Health Statistics. Analytic and reporting guidelines: the Third National Health and Nutrition Examination Survey, 1988–1994. In: National Center for Health Statistics. *NHANES III Reference Manuals and Reports*. Hyattsville, Md: Centers for Disease Control and Prevention; 1996. CD-ROM No. 6-0178 (1096).
6. National Center for Health Statistics. National Health and Nutrition Examination Survey III interviewer’s manual. In: National Center for Health Statistics. *NHANES III Reference Manuals and Reports*. Hyattsville, Md: Centers for Disease Control and Prevention; 1996. CD-ROM No. 6-0178 (1096).
7. Stevens G, Cho JH. Socioeconomic indexes and the new 1980 census occupational classification scheme. *Soc Sci Res*. 1985;14:142–168.
8. Gunter EW, Lewis BG, Koncikowski SM. Laboratory procedures used for the Third National Health and Nutrition Examination Survey (NHANES III), 1988–1994. In: National Center for Health Statistics. *NHANES III Reference Manuals and Reports*. Hyattsville, Md: Centers for Disease Control and Prevention; 1996. CD-ROM No. 6-0178 (1096).
9. Ainsworth BE, Haskell WL, Leon AS, et al. Compendium of physical activities: classification of energy costs of human physical activities. *Med Sci Sports Exerc*. 1993;25:71–80.
10. Shah BV, Barnwell BG, Bieler GS. *SUDAAN User’s Manual, Release 7.5*. Research Triangle Park, NC: Research Triangle Institute; 1997.
11. Stern MP, Rosenthal M, Haffner SM, Hazuda HP, Franco LJ. Sex differences in the effects of sociocultural status on diabetes and cardiovascular risk factors in Mexican Americans. *Am J Epidemiol*. 1984;120:834–851.
12. Leonetti DL, Tsunehara CH, Wahl PW, Fujimoto WY. Educational attainment and the risk of

non-insulin-dependent diabetes or coronary heart disease in Japanese-American men. *Ethn Dis*. 1992;2:326–336.

13. Marmot MG, Davey Smith G, Stansfeld S, et al. Health inequalities among British civil servants: the Whitehall II study. *Lancet*. 1991;337:1387–1393.
14. Mackenbach JP, Looman CW, van der Meer JBW. Differences in the misreporting of chronic conditions, by level of education: the effect on inequalities in prevalence rates. *Am J Public Health*. 1996;86:706–711.
15. Resnick HE, Valsania P, Halter JB, Lin X. Differential effects of BMI on diabetes risk among Black and White Americans. *Diabetes Care*. 1998;21:1828–1835.
16. Smith JP. Healthy bodies and thick wallets: the dual relation between health and economic status. *J Econ Perspect*. 1999;13:145–166.
17. Bartley M, Plewis I. Does health-selective mobility account for socioeconomic differences in health? Evidence from England and Wales, 1971 to 1991. *J Health Soc Behav*. 1997;38:376–386.
18. Marmot M, Ryff CD, Bumpass LL, Shipley M, Marks N. Social inequalities in health: next questions and converging evidence. *Soc Sci Med*. 1997;44:901–910.
19. Williams DR. Socioeconomic differentials in health: a review and redirection. *Soc Psychol Q*. 1990;53:81–99.
20. Krieger N, Williams DR, Moss NE. Measuring social class in US public health research: concepts, methodologies, and guidelines. *Annu Rev Public Health*. 1997;18:341–378.
21. Liberatos P, Link BG, Kelsey JL. The measurement of social class in epidemiology. *Epidemiol Rev*. 1988;10:87–121.
22. Farley R. *Blacks and Whites: Narrowing the Gap?* Cambridge, Mass: Harvard University Press; 1984.
23. Feinstein JS. The relationship between socioeconomic status and health: a review of the literature. *Milbank Q*. 1993;71:279–322.
24. MacIntyre S. The Black Report and beyond: what are the issues? *Soc Sci Med*. 1997;44:723–745.
25. Simmons D, Voyle J, Swinburn B, O’Dea K. Community-based approaches for the primary prevention of non-insulin-dependent diabetes mellitus. *Diabet Med*. 1997;14:519–526.
26. Melander A. Pharmacological intervention: the antidiabetic approach. *Eur J Clin Invest*. 1998;28(suppl 2):23–25.
27. Goldberg RB. Prevention of type 2 diabetes. *Med Clin North Am*. 1998;82:805–821.
28. Labarthe DR. *Epidemiology and Prevention of Cardiovascular Diseases: A Global Challenge*. Gaithersburg, Md: Aspen Publications; 1998.
29. Barker DJP. *Mothers, Babies, and Disease in Later Life*. London, England: BMJ Publishing Group; 1994.
30. Joseph KS, Kramer MS. Review of the evidence on fetal and early childhood antecedents of adult chronic disease. *Epidemiol Rev*. 1996;18:158–174.
31. Surwit RS, Williams PG. Animal models provide insight into psychosomatic factors in diabetes. *Psychosom Med*. 1996;58:582–589.

32. Wing RR, Epstein LH, Blair E, Nowalk MP. Psychologic stress and blood glucose levels in nondiabetic subjects. *Psychosom Med.* 1985;47:558–564.
33. Aikens JE, Mayes R. Elevated glycosylated albumin in NIDDM is a function of recent everyday environmental stress. *Diabetes Care.* 1997;20:1111–1113.
34. Goetsch VL, Wiebe DJ, Veltum LG, Van Dorsten B. Stress and blood glucose in type II diabetes mellitus. *Behav Res Ther.* 1990;28:531–537.
35. Goetsch VL, Van Dorsten B, Pbert LA, Ullrich IH, Yeater RA. Acute effects of laboratory stress on blood glucose in noninsulin-dependent diabetes. *Psychosom Med.* 1993;55:492–496.
36. Wales JK. Does psychological stress cause diabetes? *Diabet Med.* 1995;12:109–112.
37. Gavard JA, Lustman PJ, Clouse RE. Prevalence of depression in adults with diabetes. *Diabetes Care.* 1993;16:1167–1178.
38. Eaton WW, Armenian H, Gallo J, Pratt L, Ford DE. Depression and risk for onset of type II diabetes. *Diabetes Care.* 1996;19:1097–1102.
39. Gazmararian JA, James SA, Lepkowski JM. Depression in Black and White women: the role of marriage and socioeconomic status. *Ann Epidemiol.* 1995;5:455–463.
40. Lynch JW, Kaplan GA, Shema SJ. Cumulative impact of sustained economic hardship on physical, cognitive, psychological, and social functioning. *N Engl J Med.* 1997;337:1889–1895.
41. Merikangas KR, Weissman MM, Pauls DL. Genetic factors in the sex ratio of major depression. *Psychol Med.* 1985;15:63–69.
42. Boyle JP, Engelgau MM, Thompson TJ, et al. Estimating prevalence of type 1 and type 2 diabetes in a population of African Americans with diabetes mellitus. *Am J Epidemiol.* 1999;149:55–63.
43. Link BG, Phelan J. Social conditions as fundamental causes of disease. *J Health Soc Behav.* 1995;Special no.:80–94.
44. McKinlay JB, Marceau LD. Upstream healthy public policy: lessons from the battle of tobacco. *Int J Health Serv.* 2000;30:49–69.