# Life-Course Socioeconomic Position and Incidence of Diabetes Mellitus Among Blacks and Whites: The Alameda County Study, 1965–1999

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Diabetes mellitus is a major cause of morbidity and mortality in the United States.[1,2] Type 2 diabetes disproportionately affects Hispanics, as well as non-Hispanic Black Americans, American Indians/Alaska Natives, and some Asian/Pacific Islander groups. In the United States, members of racial and ethnic minority groups are almost twice as likely to develop or have type 2 diabetes than are non-Hispanic Whites.[2–5] Significant racial and ethnic differences also exist in the rates of diabetes-related preventive services, quality of care, and disease outcomes.[6–10]

Researchers have attempted to determine why, relative to Whites, members of racial and ethnic minority groups are disproportionately affected by diabetes. For example, compared with White Americans, Black Americans are presumed to have stronger genetic[5,11] or physiological[11–13] susceptibility to diabetes, or greater frequency or intensity of known diabetes risk factors, such as obesity, physical inactivity, and hypertension.[14–17]

Black Americans also are more likely than are White Americans to occupy lower socioeconomic positions.[18] Low socioeconomic position (SEP) across the life course is known to influence the prevalence[19–24] and incidence[3,19,25–30] of type 2 diabetes. The risk of diabetes also is greater for people who are obese,[3,17,31] physically inactive,[3,32] or have hypertension,[33,34] all of which are conditions more common among people with lower SEP.[16,35–37]

Several studies have focused on the extent to which socioeconomic factors, body composition (i.e., weight, height, body mass index, and waist circumference), and behaviors explain the excess risk of diabetes attributed to race.[4,12,19,30] For example, 2 separate studies, one with data from the Health and Retirement Study[19] and the other with data from the Atherosclerosis Risk in Communities Study,[30] used race to predict diabetes incidence. Attempting to separate the direct and indirect effects of race on

*Objectives.* We examined associations between several life-course socioeconomic position (SEP) measures (childhood SEP, education, income, occupation) and diabetes incidence from 1965 to 1999 in a sample of 5422 diabetes-free Black and White participants in the Alameda County Study.

*Methods.* Race-specific Cox proportional hazard models estimated diabetes risk associated with each SEP measure. Demographic confounders (age, gender, marital status) and potential pathway components (physical inactivity, body composition, smoking, alcohol consumption, hypertension, depression, access to health care) were included as covariates.

*Results.* Diabetes incidence was twice as high for Blacks as for Whites. Diabetes risk factors independently increased risk, but effect sizes were greater among Whites. Low childhood SEP elevated risk for both racial groups. Protective effects were suggested for low education and blue-collar occupation among Blacks, but these factors increased risk for Whites. Income was protective for Whites but not Blacks. Covariate adjustment had negligible effects on associations between each SEP measure and diabetes incidence for both racial groups.

*Conclusions.* These findings suggest an important role for life-course SEP measures in determining risk of diabetes, regardless of race and after adjustment for factors that may confound or mediate these associations. (*Am J Public Health.* 2010;100:137–145. doi:10.2105/AJPH.2008.133892)

diabetes,[38] these studies assessed, via statistical adjustment, which socioeconomic measures and diabetes-related risk factors, when adjusted, could account for the excess risk among Black participants relative to White participants.[19,30] Adjustment for education lessened the effect of Black race on diabetes incidence in the Atherosclerosis Risk in Communities Study.[30] In the Health and Retirement Study, excess risk attributed to Black race was not explained by early-life socioeconomic disadvantage, but it was reduced after adjustment for education and later-life economic resources.[19] The validity of this analytic approach has been challenged, however, because the socioeconomic measures used were assumed to have the same meaning across all racial/ethnic groups, a questionable assumption[38] in the United States, especially in 1965.

We sought to explore the predictive effects of several life-course socioeconomic factors on the incidence of diabetes among both Black and White Americans. We examined demographic confounders (age, gender, marital status) and diabetes risk factors (obesity, large waist circumference, physical inactivity, high blood pressure, depression, access to health care) as possible mediators of the observed associations between SEP and incident diabetes (i.e., the development of new cases of diabetes over time).

## METHODS

For our analyses, we used data from the Alameda County Study, a population-based, longitudinal investigation of the determinants of health and physical functioning and associated risk factors. A random, stratified, household sampling design was used to recruit a closed sample of 6928 noninstitutionalized adults aged 17 to 94 years (20.3% non-White) who resided in Alameda County, California, in 1965. All household residents who were ever married or 20 years or older were eligible to participate, regardless of race or ethnicity.[39]

Participants completed comprehensive, mailed questionnaires at each of 5 study waves: 1965 (baseline), 1974, 1983 (only 50% of sample received the questionnaire), 1994, and 1999. The style, length, and wording of the questions and the format of the responses were consistent across study waves. All data were self-reported. Follow-up with participants was conducted regardless of migration or disability status. Response rates at each wave ranged between 85% and 95% of eligible respondents.[39–41]

Of 6928 participants (86% of those eligible) at baseline, we excluded those who reported a race/ethnicity other than "White" or "Negro" (n=491; 7.1%) or had missing data in 1965 for model covariates (n=764; 11.0%), prevalent diabetes (n=157; 2.3%), inconsistent dates of diagnosis (n=89; 1.3%), or unknown diabetes status (n=5; 0.07%). Excluded respondents were more likely to be Black, female, older, obese, physically inactive, of lower socioeconomic means, and without health insurance. Therefore, the ability of these factors to predict or explain any excess risk of diabetes may be limited. The final sample included 5422 participants (12% Black).

## Measures

*Diabetes status.* At each study wave, 2 questions determined self-reported diabetes status: "Have you had diabetes during the past 12 months [yes/no]?" and "When did it start [year]?" Incident cases were events reported at wave $t$ but not at wave $t-1$ and whose year of diagnosis was between wave $t-1$ and wave $t$. Time to event was measured as the difference between diagnosis year and baseline. Cumulative incidence was the summed total of new cases arising between 1965 and 1999.

*Race.* Racial group membership was assessed at baseline (1965) by the question, "What is your race?" The original "White" and "Negro" response categories were reclassified as non-Hispanic White (White) and non-Hispanic Black (Black) for these analyses.

*Socioeconomic factors.* Childhood SEP was defined by the occupation (nonmanual vs manual) of the participant's father or, when the father's occupation was not available, by his education (6.3% of participants). Childhood SEP was dichotomized as low (manual occupation or formal education ≤12 years) or high

**TABLE 1—Baseline Distribution of Sample Characteristics, by Racial Group: Alameda County Study, 1965–1999**

| | Blacks | Whites | P |
|---|---|---|---|
| Total, no. | 648 | 4774 | |
| Age, y, mean (SD) | 42.6 (14.0) | 43.4 (16.1) | .23 |
| Gender, % | | | .85 |
| Men | 46.3 | 46.7 | |
| Women | 53.7 | 53.3 | |
| Marital status, % | | | <.001[a] |
| Married | 67.0 | 76.2 | |
| Unmarried | 33.0 | 23.8 | |
| Height, inches, mean (SD) | 66.5 | 66.6 | .90 |
| Childhood SEP,[b] % | | | <.001[a] |
| Low | 71.9 | 49.0 | |
| High | 28.1 | 51.0 | |
| Education, y, mean (SD) | 10.4 (3.2) | 12.3 (3.2) | <.001[c] |
| Education, y, % | | | <.001[a] |
| ≤12 | 78.7 | 61.2 | |
| >12 | 21.3 | 38.8 | |
| Household income, 1999 $, mean (SD) | 9 857.6 (2.1) | 15 787.9 (2.0) | <.001[c] |
| Occupation, % | | | <.001[a] |
| White collar | 20.1 | 42.4 | |
| Blue collar | 54.2 | 24.1 | |
| Health insurance, % | | | <.001[a] |
| Yes | 71.0 | 88.4 | |
| No | 29.0 | 11.6 | |
| Regular access to doctor/clinic, % | | | .005[a] |
| Yes | 73.9 | 78.7 | |
| No | 26.1 | 21.3 | |
| High blood pressure, % | | | <.001[a] |
| Yes | 16.4 | 8.9 | |
| No | 83.6 | 91.1 | |
| Depression, % | | | .02[a] |
| Yes | 17.0 | 13.6 | |
| No | 83.0 | 86.4 | |
| BMI category,[d] % | | | <.001[e] |
| Obese | 11.6 | 4.6 | |
| Overweight | 37.2 | 25.9 | |
| Normal/underweight | 51.2 | 69.5 | |
| BMI, kg/m$^2$, mean (SD) | 25.1 (3.9) | 23.5 (3.5) | <.001[c] |
| Waist circumference, % | | | .002[a] |
| Large[f] | 8.3 | 5.4 | |
| Not large (normal) | 91.7 | 94.6 | |
| Waist circumference, inches, mean (SD) | 31.5 (4.8) | 30.8 (5.0) | .01[c] |
| Physical activity, % | | | <.001[e] |
| Inactive/low activity | 40.4 | 29.0 | |
| Moderate activity | 41.1 | 45.8 | |
| High activity | 18.5 | 25.2 | |

*Continued*

## TABLE 1—Continued

| | | |
|---|---|---|
| Smoking status, % | | | .02[e] |
| Never smoker | 35.6 | 38.5 |
| Former smoker | 13.6 | 16.7 |
| Current smoker | 50.8 | 44.8 |
| Alcohol consumption, % | | | <.001[e] |
| Abstention | 32.1 | 17.2 |
| 1–45 drinks/mo | 55.9 | 66.8 |
| ≥46 drinks/mo | 12.0 | 16.0 |

*Note.* BMI = body mass index; SEP = socioeconomic position.

[a]By $\chi^2$ test for proportional difference in distribution of covariate category by racial group.

[b]Childhood SEP was based on a respondent's father's occupation (or education when occupation data were not available [6.5% of total]), as follows: low = manual (blue-collar) occupation or 12 years or fewer of education; high = white-collar occupation or more than 12 years of education [reference]).

[c]By *t* test for comparison of continuous variable means by race.

[d]BMI categories were as follows: obese = 30 kg/m² or more; overweight = 25.0–29.9 kg/m²; normal and underweight = 24.9 kg/m² or lower.

[e]By $\chi^2$ test for trend across covariate categories.

[f]Large waist circumference was defined as more than 34.6 inches for women and more than 40.2 inches for men.

(nonmanual occupation or >12 years of education). Analyses adjusted for baseline height (in inches). Components of adult height have been used as markers of malnutrition,[42,43] risk-conferring fetal insults,[44,45] and other childhood socioeconomic exposures[42,44,46] not captured by parental SEP measures.

At each study wave, household income data were collected through use of delimited categories. For each wave, a multiple imputation procedure[47] accounted for missing data and assigned a continuous income value. A detailed description of this imputation method has been reported previously.[26] The imputed, continuous, household income variable was standardized to 1999 dollars to permit direct comparison across study waves, adjusted for household size, and log transformed to normalize the distribution for analysis. Descriptive statistics employed a categorical income variable (low, moderate, high) created at each wave using tertiles of each race-specific imputed income distribution.

Completed years of education were assessed at each wave and, on the basis of the baseline distribution for Whites and Blacks combined, categorized as 12 years or fewer or as more than 12 years. Self-reported occupation (current, most recent, or if respondent was retired, primary lifetime occupation) was assessed with US census criteria and categorized as white collar, blue collar, "keep house," or "other occupation." The "other" category included the unemployed, students, and unclassifiable participants. In this report, results are limited to blue-collar and white-collar occupation.

*Covariates.* Demographic risk factors included age, gender, and marital status (single, married, and separated, divorced, or widowed). Access to health care was measured with 2 dichotomous (yes/no) variables: possessing health insurance and having a "regular" doctor or health clinic.

Smoking status was defined as never, former, or current smoker. A score combining alcohol type (wine, liquor, beer), frequency (never, less than once a week, once or twice a week, more than twice a week), and intake at each sitting (none, 1–2 drinks, 3–4 drinks, ≥5 drinks) assessed alcohol use. The score was split into 3 monthly consumption categories: abstention (0 drinks), light to moderate (1–45 drinks), and heavy (≥46 drinks). These categories predicted mortality in prior studies.[48,49] Involvement in physical activity (none or low, moderate, and high activity) was measured with data on the frequency and type of 4 activities: physical exercise, long walks, swimming, and taking part in active sports. These components and scale have been used previously and were associated with all-cause mortality.[50] Self-reported height and weight data were used to create a continuous body mass index (BMI) measure (i.e., weight in kilograms divided by height in meters squared), which was collapsed into 3 categories: obese (≥30 kg/m²), overweight (25.0–29.9 kg/m²), and normal or underweight (≤24.9 kg/m²).[51] Self-reported waist circumference (in inches) was recorded at baseline only.

High blood pressure was determined by the question, "Have you had high blood pressure during the past 12 months?" Depression was identified by a score of 5 or more on the Alameda County Depression Scale,[39] a valid and reliable 18-item scale used to indicate significant depressive symptomatology in other studies.[52,53]

### Statistical Analyses

Differences in the distribution of model covariates by race were assessed by the $\chi^2$ test, Cochran–Armitage trend, and the 2-sided Student *t* test. Diabetes incidence proportions (i.e., percentages of a given population that developed the disease over the 34-year study period) and densities (i.e., new cases per 1000 person-years at risk) were calculated for all covariates by race. Cox proportional hazard regression models[54] estimated hazard ratios and 95% confidence intervals for associations between incident diabetes and each socioeconomic measure in pooled and race-stratified models. Subsequent analyses controlled for effects of baseline covariates on diabetes risk. We tested and met Cox model sensitivity and assumptions using Kaplan–Meier curves and SEP–time interactions.

Participants who dropped out between 2 study waves were censored at that interval's midpoint. Participants who died in the course of the study (n=2337; 13.6% Black) were censored in their year of death. Interactions between race and model covariates were tested and observed for education and obesity. All tests of significance were 2 tailed. Analyses were performed with SAS version 9.1 (SAS Institute Inc, Cary, NC).

### RESULTS

Of 5422 study participants at baseline, 262 (4.8%) reported incident diabetes over the 34-year study period. Of 648 Black participants, 7.9% (n=51) developed diabetes, compared with 4.4% (n=211) of White participants (incidence density was 4.2 for Blacks and 2.0 for Whites).

Table 1 summarizes the baseline distribution of sample characteristics by race. Blacks were

more likely than were Whites to report known diabetes risk factors, such as obesity, large waist circumference, physical inactivity, and high blood pressure (all $P<.05$, by $\chi^2$ and $t$ test for difference by race). Blacks were significantly more likely than were Whites to be of lower SEP ($P<.001$ for all socioeconomic measures, by $\chi^2$ or $t$ test for difference by race).

The race-specific distribution of diabetes incidence proportion and density for each covariate is shown in Table 2. For most covariates, incidence among Blacks was at least 1.5 times greater than incidence among Whites. Variations existed, especially with socioeconomic factors. Incidence was greater for participants with low childhood SEP than for those with high childhood SEP, although the difference was significant only for Whites. Incidence did not differ by income category for either race. Regarding education and occupation, incidence was higher among Whites with lower SEP than among Whites with higher SEP. By contrast, Blacks with low education or blue-collar occupation were less likely to report new diabetes than were their high-SEP counterparts. Whites with health insurance, or with a regular doctor or clinic, were more likely to report diabetes than were Whites with no access to care. The reverse trend was observed with Blacks.

Hazard ratios and 95% confidence intervals for unadjusted, race-stratified associations between baseline covariates and diabetes incidence are presented in Table 3. Among White participants, diabetes incidence was significantly associated with low childhood SEP, low education (i.e., ≤12 years), and low income, as well as with high blood pressure, excess body mass, and former or current smoking status (hazard ratio [HR] ranged from 1.6–6.4; 95% confidence interval [CI] =1.1, 9.3).

Similarly, for Black participants, data suggested that increased diabetes risk was associated with low childhood SEP, no access to health care, high blood pressure, excess body mass, physical inactivity, former or current smoking status, and heavy drinking; however, low education and blue-collar occupation were protective against diabetes (low education: HR=0.5; 95% CI=0.3, 1.0; blue-collar occupation: HR=0.7; 95% CI=0.4, 1.4). For Black participants, confidence intervals for all associations, except obesity, were imprecise

**TABLE 2—Incidence Proportion and Crude Incidence Density (Incident Cases per 1000 Person-Years at Risk) of Type 2 Diabetes Mellitus Associated With Baseline Characteristics, by Racial Group: Alameda County Study, 1965–1999**

| | Blacks (n = 648) | | | Whites (n = 4774) | | |
|---|---|---|---|---|---|---|
| Variable Category | Total No. of Incident Cases | % With Diabetes | Incidence Density | Total No. of Incident Cases | % With Diabetes | Incidence Density |
| Total population | 51 | 7.9 | 4.2 | 211 | 4.4 | 2.0 |
| Age, y | | | | | | |
| <40 | 24 | 8.7 | 4.8 | 105 | 4.9 | 2.0 |
| ≥40 | 27 | 7.2 | 3.8 | 106 | 4.0 | 2.0 |
| Gender | | | | | | |
| Women | 29 | 8.3 | 4.4 | 108 | 4.2 | 1.9 |
| Men | 22 | 7.3 | 4.0 | 103 | 4.6 | 2.1 |
| Marital status | | | | | | |
| Married | 34 | 7.8 | 4.1 | 166 | 4.6 | 1.9 |
| Unmarried | 17 | 7.9 | 4.3 | 45 | 4.0 | 2.0 |
| Height | | | | | | |
| Below mean | 25 | 7.3 | 3.8 | 105 | 4.4 | 2.0 |
| Above mean | 26 | 8.5 | 4.6 | 106 | 4.5 | 1.9 |
| Childhood SEP[a] | | | | | | |
| Low | 39 | 8.4 | 4.4 | 133 | 5.7[b] | 2.6 |
| High | 12 | 6.6 | 3.5 | 78 | 3.2 | 1.5 |
| Education, y | | | | | | |
| ≤12[c] | 34 | 6.7[b] | 3.6 | 143 | 4.9[b] | 2.4 |
| >12 | 17 | 12.3 | 6.6 | 68 | 3.7 | 1.5 |
| Income tertile | | | | | | |
| Low | 18 | 8.3 | 4.5 | 82 | 5.2 | 2.4 |
| Moderate | 15 | 7.0 | 3.5 | 64 | 4.0 | 1.8 |
| High | 18 | 8.3 | 4.7 | 65 | 4.1 | 1.7 |
| Occupation | | | | | | |
| Blue collar | 28 | 8.0 | 4.2 | 56 | 4.9 | 2.4 |
| White collar | 14 | 10.8 | 5.9 | 93 | 4.6 | 2.0 |
| Health insurance | | | | | | |
| No | 16 | 8.5 | 5.0 | 14 | 2.5[b] | 1.3 |
| Yes | 35 | 7.6 | 3.9 | 197 | 4.7 | 2.0 |
| Regular health provider | | | | | | |
| No | 16 | 9.5 | 5.7 | 35 | 3.5 | 1.6 |
| Yes | 35 | 7.3 | 3.7 | 176 | 4.7 | 2.1 |
| Depression | | | | | | |
| Yes | 9 | 8.2 | 4.6 | 29 | 4.5 | 2.3 |
| No | 42 | 7.8 | 4.1 | 182 | 4.4 | 1.9 |
| High blood pressure | | | | | | |
| Yes | 10 | 9.4 | 5.4 | 26 | 6.1 | 3.7 |
| No | 41 | 7.6 | 4.0 | 185 | 4.3 | 1.8 |
| BMI category[d] | | | | | | |
| Obese[c] | 10 | 13.3[e] | 6.9 | 36 | 16.6[e] | 8.3 |
| Overweight | 20 | 8.3 | 4.3 | 68 | 5.5 | 2.5 |
| Normal/underweight | 21 | 6.3 | 3.4 | 107 | 3.2 | 1.4 |

*Continued*

**TABLE 2—Continued**

| | | | | | | |
|---|---|---|---|---|---|---|
| Waist circumference | | | | | | |
| Large[f] | 7 | 13.0 | 7.4 | 29 | 11.3[b] | 6.9 |
| Normal | 44 | 7.4 | 3.9 | 182 | 4.0 | 1.8 |
| Physical activity level | | | | | | |
| Inactive/low | 22 | 8.4 | 4.8 | 57 | 4.1 | 2.1 |
| Moderate | 22 | 8.3 | 4.3 | 102 | 4.7 | 2.0 |
| High | 7 | 5.8 | 2.9 | 52 | 4.3 | 1.7 |
| Smoking status | | | | | | |
| Current smoker | 30 | 9.1 | 5.0 | 106 | 5.0[e] | 2.2 |
| Former smoker | 6 | 6.8 | 4.2 | 45 | 5.7 | 2.4 |
| Never smoked | 15 | 6.5 | 3.2 | 60 | 3.3 | 1.4 |
| Drinking | | | | | | |
| Abstention | 16 | 7.7 | 4.3 | 31 | 3.8 | 1.9 |
| 1–45 drinks/mo | 28 | 7.7 | 4.0 | 147 | 4.6 | 2.0 |
| ≥46 drinks/mo | 7 | 9.0 | 4.6 | 33 | 4.3 | 1.9 |

*Note.* BMI = body mass index; SEP = socioeconomic position.
[a]Childhood SEP was based on a respondent's father's occupation (or education when occupation data were not available [6.5% of total]), as follows: low = manual (blue-collar) occupation or 12 years or fewer of education; high = white-collar occupation or more than 12 years of education [reference]).
[b]$P < .05$, by the $\chi^2$ test for proportional difference in distribution of covariate category by racial group.
[c]$P < .05$, for interaction between covariate category and racial group.
[d]BMI categories were as follows: obese = 30 kg/m$^2$ or more; overweight = 25.0–29.9 kg/m$^2$; normal and underweight = 24.9 kg/m$^2$ or lower.
[e]$P < .05$, by the $\chi^2$ test for trend across covariate categories within racial group.
[f]Large waist circumference was defined as more than 34.6 inches for women and more than 40.2 inches for men.

because of the small sample size. The hazard ratio associated with obesity was considerably stronger for White than for Black participants (6.4 vs 2.1, respectively).

Table 4 displays associations between each SEP measure and diabetes incidence by race in unadjusted and adjusted models. For each measure, lower SEP was associated with elevated risk among White participants, although confidence intervals for blue-collar occupation included the null (HRs and 95% CIs for demographic-adjusted models were as follows: childhood SEP: HR=1.9; 95% CI=1.4, 2.5; low education: HR=1.7; 95% CI=1.3, 2.4; income: HR=0.7; 95% CI=0.6, 0.9; blue-collar occupation: HR=1.3; 95% CI=0.9, 1.8). Adjustment for potential pathway components did not attenuate effect sizes associated with childhood SEP or income; however, the effect size for education was reduced and the association with blue-collar occupation was eliminated.

Among Black participants, in demographic-adjusted models, low childhood SEP elevated diabetes risk (HR=1.3; 95% CI=0.7, 2.6),

whereas increasing income had no effect (HR=1.0; 95% CI=0.7, 1.4). Conversely, both low education and blue-collar occupation suggested a protective effect compared with high education and white-collar occupation (low education: HR=0.5; 95% CI=0.2, 0.9; blue-collar occupation: HR=0.7; 95% CI=0.4, 1.4). Adjustment for potential pathway components did not attenuate the effect sizes observed in demographic-adjusted models, although CIs were imprecise for all associations in the adjusted models.

## DISCUSSION

To our knowledge, our study is the first to explore the predictive effects of several life-course socioeconomic factors on the incidence of diabetes among both Black and White Americans. Black participants were more than twice as likely as were White participants to develop type 2 diabetes over the 34-year study period. Blacks also reported such diabetes risk factors as obesity, physical inactivity, and high blood pressure more frequently than did Whites. These factors

were independently associated with increased risk for both racial groups.

The contribution of various socioeconomic measures to diabetes incidence differed by race in these data. Low childhood SEP was associated with increased risk of type 2 diabetes, regardless of race. Income was protective for Whites but was not related to incidence among Blacks. Low education and blue-collar occupation were protective for Blacks but increased the risk for Whites. Effect sizes were more robust and CIs were more precise for Whites. Adjustment for demographic confounders and potential components of the causal pathways between SEP and diabetes, such as obesity, physical inactivity, and high blood pressure, did not meaningfully alter effect sizes or CIs for either racial group.

### Strengths and Limitations

Several limitations require consideration. Most significant was the use of self-reported data, which may have produced misclassification of outcome or exposure status. Given the study design, diagnostic confirmation of diabetes status was not possible; however, prior studies have shown that self-reported disease status compares well with clinically diagnosed diabetes.[55,56] Whether this holds equally for Blacks and Whites is uncertain.

The type of diabetes (type 1 or type 2) could not be verified in these data. Participants who reported diabetes after 1965 were counted as cases, regardless of age at diagnosis. Type 2 diabetes accounts for 90% to 95% of cases diagnosed after age 20 years.[57] The race-specific distribution of SEP and other covariates did not differ by age at diagnosis, although Whites accounted for most cases diagnosed before age 40 years. Associations between SEP and diabetes risk did not differ by age for either racial group (results not shown). Therefore, misclassification of diabetes type would lead to minimal bias in case ascertainment.

Measurement error caused by time-related changes in exposure status over the 34-year study also could have affected results. The small sample of Black participants precluded use of time-dependent analyses, although measures of early and later-life SEP were used. Given the time-dependent nature of most covariates, use of only 1 time measure could lead to misclassification. Differential

TABLE 3—Unadjusted Hazard Ratios (HRs) for 34-Year Incidence of Type 2 Diabetes Associated With Baseline Characteristics, by Racial Group: Alameda County Study, 1965–1999

| | Blacks, HR (95% CI) | Whites, HR (95% CI) |
|---|---|---|
| Racial group (White = reference) | 2.3 (1.7, 3.1) | 1.0 |
| Age, y (continuous) | 1.0 (1.0, 1.0) | 1.0 (1.0, 1.0) |
| Gender | | |
| Women | 1.1 (0.6, 1.9) | 0.9 (0.7, 1.1) |
| Men (Ref) | 1.0 | 1.0 |
| Marital status | | |
| Unmarried | 1.1 (0.6, 1.9) | 1.1 (0.8, 1.5) |
| Married (Ref) | 1.0 | 1.0 |
| Childhood SEP[a] | | |
| Low | 1.3 (0.7, 2.5) | 1.9 (1.4, 2.5) |
| High (Ref) | 1.0 | 1.0 |
| Height, inches (continuous) | 1.0 (0.9, 1.1) | 1.0 (1.0, 1.0) |
| Education, y (continuous) | 1.0 (0.9, 1.1) | 0.9 (0.9, 1.0) |
| ≤12 | 0.5 (0.3, 1.0) | 1.7 (1.3, 2.3) |
| >12 (Ref) | 1.0 | 1.0 |
| Income, 1999 $ (continuous) | 1.0 (0.7, 1.4) | 0.8 (0.6, 0.9) |
| Occupation | | |
| Blue collar | 0.7 (0.4, 1.4) | 1.3 (0.9, 1.8) |
| White collar (Ref) | 1.0 | 1.0 |
| Health insurance | | |
| No | 1.3 (0.7, 2.4) | 0.7 (0.4, 1.1) |
| Yes (Ref) | 1.0 | 1.0 |
| Regular health provider | | |
| No | 1.6 (0.9, 2.8) | 0.8 (0.5, 1.1) |
| Yes (Ref) | 1.0 | 1.0 |
| Depression | | |
| Yes | 1.1 (0.5, 2.3) | 1.3 (0.8, 1.9) |
| No (Ref) | 1.0 | 1.0 |
| High blood pressure | | |
| Yes | 1.4 (0.7, 2.9) | 2.3 (1.5, 3.5) |
| No (Ref) | 1.0 | 1.0 |
| BMI[b] (continuous) | 1.0 (1.0, 1.1) | 1.1 (1.1, 1.2) |
| Obese | 2.1 (1.0, 4.4) | 6.4 (4.4, 9.3) |
| Overweight | 1.3 (0.7, 2.3) | 1.9 (1.4, 2.5) |
| Normal/underweight (Ref) | 1.0 | 1.0 |
| Waist circumference (continuous) | 1.0 (1.0, 1.0) | 1.0 (1.0, 1.0) |
| Large[c] | 2.0 (0.9, 4.5) | 4.5 (3.0, 6.7) |
| Normal (Ref) | 1.0 | 1.0 |
| Physical activity level | | |
| Inactive/low activity | 1.8 (0.8, 4.2) | 1.3 (0.9, 2.0) |
| Moderate activity | 1.6 (0.7, 3.8) | 1.2 (0.8, 1.7) |
| High activity (Ref) | 1.0 | 1.0 |
| Smoking status | | |
| Current smoker | 1.6 (0.9, 3.1) | 1.6 (1.1, 2.2) |
| Former smoker | 1.4 (0.5, 3.6) | 1.7 (1.1, 2.5) |

*Continued*

measurement error or imprecise measurement of SEP and other factors by race also could have biased results.[58]

Survival bias also may have influenced the results. Participants who developed diabetes between study waves may have died or dropped out before being counted as cases. Approximately 43% of the original Black participants died or were lost to follow-up. Blacks who left the study were younger, healthier, and of lower SEP than were those who remained. Consequently, the number of cases observed among Blacks may underestimate the true incidence. The ability of SEP or other factors to predict incidence among Blacks also may be limited.

Finally, the childhood SEP (low vs high), education (≤12 vs >12 years), and occupation (blue-collar vs white-collar) variables were dichotomized to preserve statistical power. Given the interrelated nature of these socioeconomic measures, dichotomization may limit their interpretability[59] through loss of information or underestimation of variability within and between groups.[60] Future studies should maximize sample size to allow for enhanced measurement and analysis of socioeconomic factors.

This study had several strengths. First, data were collected on 5 occasions over a 34-year period. Second, longitudinal data allowed investigation of incident diabetes. Third, the data permitted simultaneous investigation of many potential confounders and pathway components connecting SEP to diabetes incidence. Finally, no other studies have described the association between multiple life-course socioeconomic measures and diabetes incidence stratified by race.

### Race, Socioeconomic Position, and Diabetes Risk

These results support findings from other studies showing a greater frequency of diabetes risk factors[14–17] and incidence[4,12,19,30] among Blacks than among Whites. Many diabetes risk factors, such as obesity, physical inactivity, and hypertension, are patterned by SEP.[16,35–37] Low SEP is associated with incident diabetes.[3,19,25–30] In this study, many Blacks reported lower SEP, which likely contributed to the associations between SEP and diabetes risk factors and incidence within this group.

**TABLE 3—Continued**

| | | |
|---|---|---|
| Never smoked (Ref) | 1.0 | 1.0 |
| Drinking | | |
| Abstained from drinking | 1.1 (0.6, 2.0) | 1.0 (0.7, 1.5) |
| 1–45 drinks/mo (Ref) | 1.0 | 1.0 |
| >46 drinks/mo | 1.2 (0.5, 2.7) | 1.0 (0.7, 1.4) |

*Note.* CI = confidence interval; BMI = body mass index; SEP = socioeconomic position. For Blacks, n = 648; for Whites, n = 4774.
[a]Childhood SEP was based on a respondent's father's occupation (or education when occupation data were not available [6.5% of total]), as follows: low = manual (blue-collar) occupation or 12 years or fewer of education; high = white-collar occupation or more than 12 years of education [reference]).
[b]BMI categories were as follows: obese = 30 kg/m$^2$ or higher; overweight = 25.0–29.9 kg/m$^2$; normal and underweight = 24.9 kg/m$^2$ or lower.
[c]Large waist circumference was defined as more than 34.6 inches for women and more than 40.2 inches for men.

Discrimination against Black Americans likely contributes to the association between SEP and diabetes by intensifying the impact of low SEP on racial health inequities.[61] In the United States, membership in a non-White racial/ethnic groups historically has provided the impetus for unequal distribution of resources and opportunities by the dominant (White) group.[62–64] Institutional and other forms of discrimination increase physical and mental stress, hinder social mobility, perpetuate the segregation of communities, and limit purchasing power for health-related goods and services,[63,64] all of which plausibly influence diabetes risk. Whether the impact of discrimination on diabetes incidence varies by SEP has not been assessed. Comprehensive investigation of the role of discrimination in the development of diabetes was not possible with these data, but it is an important area for future research.

Complex relationships between SEP and diabetes incidence emerged for each racial group in this study. Low childhood SEP increased risk among Blacks and Whites. Higher income and education and white-collar occupation protected Whites from diabetes, but they showed either a null or a negative association for Blacks.

The relationship between childhood SEP and diabetes or diabetes-related conditions has been assessed in few studies.[9,20,25,26] For example, childhood SEP, measured by parental occupation, had no effect on prevalent metabolic syndrome in a study of Black adults in Pitt County, North Carolina.[65] By contrast, low childhood SEP modestly increased diabetes risk among 100 330 women in the Nurse's Health Study after control for race/ethnicity.[26] Our study, which, to our knowledge, is the first to investigate the race-specific effect of low childhood SEP on diabetes risk, demonstrates a strong association between childhood disadvantage and incident diabetes, regardless of race.

The reasons for the divergent risk patterns for education, occupation, and income by race in these data are unclear. The protective effects of blue-collar occupation and low education could originate from reduced socioeconomic variability within the sample. For each SEP measure, Blacks were concentrated at the lower end of the spectrum. The unequal distribution of socioeconomic resources among Blacks compared with Whites could contribute to an assessment of SEP and its influence on disease incidence by race that is inaccurate or differential, or both.[58,59]

A particular social position may not bestow the same amount or type of resources, opportunities, or prestige for Black Americans as for White Americans;[66,67] this would be especially true in 1965, the year the study began. Furthermore, common measures of SEP, such as education, income, and occupation, often are not comparable across racial groups,[68] a difference that could be exacerbated by the use of dichotomous measures of SEP.[60] For our study, small sample size also reduced the predictive power of each SEP measure, resulting in smaller HRs and wider CIs.

Finally, selection bias also could influence the protective effects of low education and blue-collar occupation. Black participants who died or were lost to follow-up were more likely to have lower education or to be blue-collar workers than were those who remained in the study (results not shown). Consequently, the remaining low-SEP participants were likely healthier and at lower risk of diabetes. Blue-collar occupation and low education may be surrogates for unmeasured socioeconomic or other factors that protect against incident diabetes. These or other unmeasured factors could influence the association between SEP and diabetes incidence, but could also lead to differential dropout.[69] These selection biases, however, are difficult to distinguish from competing risks (J. Kaufman, PhD, Department of Epidemiology, McGill University, written communication, June 2008), which also could contribute to the unexpected protective effect of low education and blue-collar occupation on diabetes for Blacks in this study. The potential explanations for the protective effects of blue-collar occupation and low education on diabetes risk described here require further exploration.

Among all participants, the effects of different socioeconomic measures on diabetes incidence were not noticeably attenuated after adjustment for demographic confounders or other covariates. The limited ability of BMI, waist circumference, or physical inactivity to account for the excess risk was unexpected, given the distributions of these factors in both groups and their independent effects on disease incidence. Equally surprising was the increased risk associated with access to health care among Whites. These results may reflect imprecise covariate assessment, differential measurement error or disease detection by race, or other bias. Furthermore, these data did not include measures of factors such as insulin resistance, dietary intake, family history, or neighborhood characteristics that also could act as mechanisms linking low SEP and diabetes incidence.

## Conclusions

Findings from this study underscore the importance of life-course SEP measures in determining the risk of diabetes in adulthood, regardless of race and after adjustment for factors that may confound or mediate these associations. The growing gap between wealthy and poor Americans, coupled with persistent individual and community-level SEP disparities by race, likely will lead to increasing rates of diabetes among people with lower socioeconomic means,

TABLE 4—Hazard Ratios (HRs) for 34-Year Incidence of Type 2 Diabetes Mellitus Associated With Life-Course Socioeconomic Factors, by Racial Group: Alameda County Study, 1965–1999

| Model | Childhood SEP,[a] HR (95% CI) | Education,[b] HR (95% CI) | Income,[c] HR (95% CI) | Occupation,[d] HR (95% CI) |
|---|---|---|---|---|
| | | Blacks | | |
| Model 1 | 1.3 (0.7, 2.5) | 0.5 (0.3, 1.0) | 1.0 (0.7, 1.4) | 0.7 (0.4, 1.4) |
| Model 2 | 1.3 (0.7, 2.6) | 0.5 (0.2, 0.9) | 1.0 (0.7, 1.4) | 0.7 (0.4, 1.4) |
| Model 3 | 1.3 (0.7, 2.5) | 0.5 (0.2, 0.9) | 0.9 (0.6, 1.4) | 0.8 (0.4, 1.6) |
| Model 4 | 1.3 (0.7, 2.6) | 0.5 (0.2, 1.0) | 0.9 (0.6, 1.4) | 0.7 (0.3, 1.3) |
| Model 5 | 1.3 (0.7, 2.6) | 0.5 (0.2, 0.9) | 0.9 (0.6, 1.4) | 0.7 (0.3, 1.5) |
| Model 6 | 1.3 (0.7, 2.6) | 0.5 (0.2, 0.9) | 1.0 (0.6, 1.4) | 0.7 (0.3, 1.3) |
| Model 7 | 1.3 (0.7, 2.6) | 0.5 (0.2, 0.8) | 1.0 (0.7, 1.5) | 0.6 (0.3, 1.3) |
| Model 8 | 1.3 (0.7, 2.5) | 0.5 (0.2, 0.9) | 1.0 (0.7, 1.5) | 0.7 (0.3, 1.3) |
| Model 9 | 1.4 (0.7, 2.7) | 0.5 (0.2, 0.9) | 0.9 (0.6, 1.4) | 0.7 (0.3, 1.4) |
| | | Whites | | |
| Model 1 | 1.9 (1.4, 2.5) | 1.7 (1.3, 2.3) | 0.8 (0.6, 0.9) | 1.3 (0.9, 1.8) |
| Model 2 | 1.9 (1.4, 2.5) | 1.7 (1.3, 2.4) | 0.7 (0.6, 0.9) | 1.3 (0.9, 1.8) |
| Model 3 | 1.7 (1.3, 2.3) | 1.5 (1.1, 2.0) | 0.8 (0.7, 1.0) | 0.9 (0.6, 1.4) |
| Model 4 | 1.7 (1.3, 2.3) | 1.6 (1.2, 2.3) | 0.7 (0.6, 0.9) | 1.2 (0.8, 1.6) |
| Model 5 | 1.7 (1.2, 2.2) | 1.4 (1.0, 2.0) | 0.8 (0.6, 1.0) | 0.9 (0.6, 1.3) |
| Model 6 | 1.7 (1.3, 2.3) | 1.6 (1.2, 2.1) | 0.8 (0.7, 1.0) | 1.2 (0.8, 1.7) |
| Model 7 | 1.8 (1.4, 2.4) | 1.6 (1.2, 2.2) | 0.8 (0.6, 0.9) | 1.2 (0.8, 1.7) |
| Model 8 | 1.8 (1.4, 2.4) | 1.7 (1.3, 2.3) | 0.8 (0.7, 1.0) | 1.2 (0.9, 1.8) |
| Model 9 | 1.6 (1.2, 2.1) | 1.3 (0.9, 1.8) | 0.9 (0.7, 1.1) | 0.9 (0.6, 1.3) |

*Note.* CI = confidence interval. All covariates were measured at baseline. For Blacks, n = 648; for Whites, n = 4774. Models are as follows: model 1, unadjusted; model 2 adjusted for age, gender, and marital status; model 3, adjusted for age, gender, marital status, and childhood SEP measures (parental occupation or education, own height and education); model 4 adjusted for age, gender, marital status, and adult SEP measures (income, occupation); model 5 adjusted for age, gender, marital status, childhood SEP measures, adult SEP measures, and health insurance status; model 6 adjusted for age, gender, marital status, body mass index (continuous), and waist circumference (continuous); model 7 adjusted for age, gender, marital status, physical activity, alcohol use, and smoking; model 8 adjusted for age, gender, marital status, regular access to a medical doctor or clinic, depression, and high blood pressure; model 9 adjusted for all covariates.
[a]Dichotomous variable. Childhood SEP was based on a respondent's father's occupation (or education when occupation data were not available [6.5% of total]), as follows: low = manual (blue-collar) occupation or 12 years or fewer of education; high = white-collar occupation or more than 12 years of education [reference]).
[b]Dichotomous variable. Twelve years or fewer vs more than 12 years (reference).
[c]Continuous variable.
[d]Dichotomous variable. Blue-collar occupation vs white-collar occupation (reference).

especially those from non-White communities. Therefore, efforts to eliminate racial and socio-economic inequities must be enhanced and sustained to reduce the burden of diabetes and other health conditions linked to social disadvantage across the life course. ■

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## Contributors
S. C. Maty originated the study, performed data analysis, and wrote the article. S. A. James and G. A. Kaplan provided assistance with concept development, study design, interpretation of results, and article preparation.

## Human Participant Protection
This study was approved by the institutional review board of the University of Michigan, Ann Arbor.

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