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Lifecourse socioeconomic position and the 34-year incidence (1965–1999) of diabetes mellitus among black and white participants in the Alameda County Study

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Abstract

Objective—This study examined associations between several lifecourse socioeconomic position (SEP) measures (childhood SEP, education, income, occupation) and diabetes incidence from 1965–1999 in a sample of 5,422 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, health care access) were included as covariates.

Results—Diabetes incidence was 2-fold greater for blacks than 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 lifecourse SEP measures in determining risk of diabetes, regardless of race, and net of factors that may confound or mediate these associations.

Diabetes mellitus is a major cause of morbidity and mortality in the United States (U.S.).[1,2] Type 2 diabetes disproportionately affects Hispanics/Latinos, as well as non-Hispanic black Americans, American Indian/Alaskan Natives, and some Asian/Pacific Islander groups. In the U.S., members of racial and ethnic minority groups are almost twice as likely to develop or have type 2 diabetes compared to non-Hispanic whites.[2–5] Significant racial and ethnic

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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 manuscript preparation.

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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 to white Americans, black Americans are presumed to have stronger genetic[5,11] or physiologic[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 to occupy lower socioeconomic positions than white Americans.[18] Low socioeconomic position (SEP) across the lifecourse 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 persons who are obese,[3,17,31] physically inactive,[3,32] or have hypertension[33,34]; all conditions more common among persons with lower SEP.[16,35–37]

The extent to which socioeconomic factors, body composition, and behaviors explain the excess risk of diabetes attributed to race has been the focus of several studies[4,12,19,30] For example, two separate studies, with data from the Health and Retirement Study (HRS)[19] and the Atherosclerosis Risk in Communities Study (ARIC),[30] used race to predict diabetes incidence. Attempting to separate the direct and indirect effects of race on diabetes,[38] these studies assessed, via statistical adjustment, which socioeconomic measures and diabetes-related risk factors attenuated the excess risk of diabetes observed in black relative to white participants.[19,30] Adjustment for education lessened the effect of black race on diabetes incidence in the ARIC study.[30] In the HRS, excess risk attributed to black race was not explained by early-life socioeconomic disadvantage, but was reduced after adjustment for education and later-life economic resources.[19] The validity of this analytic approach has been challenged, however, as the socioeconomic measures used are assumed to have the same meaning across all racial/ethnic groups, which likely was not the case[38] in the U.S. in 1965.

This study is the first to explore the predictive effects of several lifecourse socioeconomic factors on the incidence of diabetes stratified by racial group. Demographic confounders (age, gender, marital status) and diabetes risk factors (obesity, large waist circumference, physical inactivity, high blood pressure, depression, access to health care) were examined as possible mediators of the observed associations between SEP and incident diabetes.

MATERIALS AND METHODS

Study Population

These analyses 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 6,928 non-institutionalized adults aged 17–94 years (20.3% non-white) who resided in Alameda County, California in 1965. All household residents who were ever-married or at-least 20 years of age were eligible to participate, regardless of race or ethnicity.[39]

Participants completed comprehensive, mailed questionnaires at each of five study waves: 1965 (baseline), 1974, 1983 (50% sample), 1994, and 1999. Question style, length, wording and response formats were consistent across study waves. All data were self-reported. Participants were followed regardless of migration or disability status. Response rates at each wave ranged between 85 and 95 percent of eligible respondents.[39–41]

Of 6,928 participants (86% of eligible) at baseline, we excluded those who reported a race/ethnicity other than “white” or “negro” (n=491, 8.3%), had missing data in 1965 for model covariates (n=764, 11.0%), and those with prevalent diabetes (n=157, 2.3%), inconsistent dates of diagnosis (89, 1.3%), or whose diabetes status was unknown (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 5,422 participants (12% black).

Measures

Diabetes Status—At each study wave, two questions determined self-reported diabetes status: ‘have you had any of these conditions <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 happened 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 participants’ fathers’ occupation (non-manual vs. manual) or education, when occupation was not available (6.3 percent of observations). Childhood SEP was dichotomized as low (manual occupation or formal education ≤12 years) or high (non-manual occupation or >12 years of education). Analyses adjusted for baseline height (inches). Components of adult height have been used as markers of malnutrition,[42,43] risky 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 using 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, based upon the baseline distribution for whites and blacks combined, categorized as ≤12 and >12 years. Self-reported current, most recent, or, if retired, primary lifetime occupation was assessed using U.S. census criteria, and categorized as white-collar, blue-collar, keep house, or other occupation. The ‘other’ category included unemployed, students, and unclassifiable participants. Results presented 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 using two dichotomous (yes/no) variables: possessing health insurance and having a ‘regular’ doctor or health clinic.

Smoking status was defined as never, former or current. A score combining alcohol type (wine, liquor, beer), frequency (never, less than once a week, 1–2 times, >2 times per week) and intake at each sitting (never, 1–2 drinks at one sitting, 3–4, ≥ 5 drinks) assessed alcohol

use. The score was split into three monthly consumption categories: abstain (0 drinks), light to moderate (1–45), and heavy (46+ drinks). These categories predicted mortality in prior studies.[48,49] Involvement in physical activity (no or low, moderate, and high activity) was measured using data on the frequency and type of four activities: physical exercise, long walks, swimming, or 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 (weight/height^2 in kilograms(kg)/meters(m)^2), which was collapsed into three categories: obese (BMI ≥30 kg/m^2), overweight (BMI 25–29.9 kg/m^2), and normal/underweight (BMI ≤24.9 kg/m^2).[51] Self-reported waist circumference (inches) was recorded at baseline only.

High blood pressure status was measured by the question, “Have you had any of these conditions <high blood pressure> during the past 12 months?” Depression was identified by a score of five 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

Chi-square, Cochran–Armitage trend, and 2-sided Student t-tests assessed differences in the distribution of model covariates by race. Diabetes incidence proportions and densities (new cases per 1,000 person-years at risk) were calculated for all covariates by race. Cox proportional hazard regression models[54] estimated hazard ratios and 95 percent 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. Cox model sensitivity and assumptions were tested and met using Kaplan–Meier curves and SEP–time interactions.

Participants who dropped out between two study waves were censored at the interval midpoint. Participants who died through 1999 (n=2,337, 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 two-tailed. Analyses were performed using Statistical Analysis System software, Version 9.1 (SAS Institute, Inc., Cary, North Carolina).

RESULTS

Of 5,422 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 to 4.4% (n=211) of white participants (incidence density = 4.2 (blacks), 2.0 (whites)).

Table 1 summarizes the baseline distribution of sample characteristics by race. Blacks were more likely than whites to report known diabetes risk factors, such as obesity, large waist circumference, physical inactivity, and high blood pressure (X^2 and t-tests for difference by race: all $p<0.05$). Compared to whites, blacks significantly were more likely to be of lower SEP (X^2 or t-tests for difference by race: $p<0.0001$ for all socioeconomic measures).

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 exist, especially with socioeconomic factors. Incidence was greater for participants with low childhood SEP than those with high childhood SEP, although the difference was significant only for whites. Incidence did not differ by income category for either race. For education and occupation, higher incidence was found among whites with lower SEP compared to higher SEP. In contrast, blacks with low education or blue-collar occupation were less likely to report new diabetes compared to

their high SEP counterparts. The difference for occupation was not significant for either race. Whites with health insurance, or a regular doctor or clinic, were more likely to report diabetes compared to whites with no access to care. The reverse trend was observed in blacks.

Hazard ratios (HR) and 95% confidence intervals (CI) 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, education (≤12 years versus >12), and income, as well as high blood pressure, excess body mass, and former or current smoking status (HR range 1.6–6.4 and 95% CI range 1.1–9.3).

Similarly, increased diabetes risk estimates were suggested 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 among black study participants. 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). Confidence intervals for all associations, except obesity, among blacks were imprecise and likely due to small sample size. Hazard ratios for diabetes incidence associated with obesity significantly differed by racial group.

Table 4 displays associations between each SEP measure and diabetes incidence by race in unadjusted and adjusted models. Lower SEP, regardless of measure, was associated with elevated risk among white participants, although confidence intervals for blue-collar occupation included the null (demographic-adjusted models: Childhood SEP HR=1.9, 95% CI=1.4, 2.5; low education (≤12 years) 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, yet reduced the effect of education and removed any association with blue-collar occupation.

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 to high education and white-collar occupation (low education (≤12 years) 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 confidence intervals were imprecise for all associations in the adjusted models.

DISCUSSION

This is the first study to investigate the effects of multiple lifecourse socioeconomic indicators on diabetes incidence for black and white Americans. Black participants were more than twice as likely as white participants to develop type 2 diabetes over the 34-year study period. Blacks also reported diabetes risk factors, such as obesity, physical inactivity and high blood pressure, more frequently than 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 not related to incidence among blacks. Low education and blue-collar occupation were protective for blacks, but increased risk for whites. Effect sizes and confidence intervals were more robust 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 confidence intervals 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, self-reported disease status compares well to 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 90–95 percent of cases diagnosed after age 20.[57] The race-specific distribution of SEP and other covariates did not differ by age at diagnosis, although whites accounted for most cases under age 40. 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 due to 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 utilized. Given the time-dependent nature of most covariates, use of only one time measure could lead to misclassification. Differential measurement error or imprecise measurement of SEP and other factors by race also could have biased results.[58]

Survival bias also likely influenced the results. Participants who developed diabetes between study waves may have died or dropped-out before being counted as cases. Approximately 43% of original black participants died or were lost to follow-up. Blacks who left the study were younger, healthier and of lower SEP compared to 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 in 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] via 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 five 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 lifecourse socioeconomic measures and diabetes incidence stratified by race.

Race, SEP and Diabetes Risk

These results support findings from other studies showing greater frequency of diabetes risk factors[14–17] and incidence[4,12,19,30] among blacks compared to 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 these data, many blacks reported

lower SEP, which likely contributed to the associations between SEP and diabetes risk factors and incidence within this group.

Discrimination likely contributes to the association between SEP and diabetes by intensifying the impact of low SEP on racial health inequities.[65] In the U.S., membership in a non-white racial/ethnic group historically has provided the impetus for unequal distribution of resources and opportunities by the dominant (white) group.[66,67,68] Institutional and other forms of discrimination increase physical and mental stress, hinder social mobility, perpetuate segregation of communities, and limit purchasing power for health-related goods and services[67,68]; all characteristics that 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 in these data, but 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 in blacks and whites. Higher income and education, and white-collar occupation protected whites from diabetes, but showed either a null or 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.[61] In contrast, low childhood SEP modestly increased diabetes risk among 100,330 women from the Nurse's Health Study after controlling for race/ethnicity.[26] The current study is the first to investigate the race-specific effect of low childhood SEP on incident diabetes, demonstrating a strong association with childhood disadvantage, 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 to whites could contribute to inaccurate and/or differential assessment of SEP and its influence on disease incidence by race.[58,59]

A particular social position may not bestow the same amount or type of resources, opportunities or prestige for blacks compared to whites of similar social standing,[62,63] especially in 1965. Furthermore, common measures of SEP, like education, income, and occupation, often are not comparable across racial groups[64]; a difference that could be exacerbated by the use of dichotomous measures of SEP.[60] Small sample size also reduced the predictive power of each SEP measure, resulting in smaller hazard ratios and wider confidence intervals.

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 be blue-collar workers compared to those who remained in the study (results not shown). Consequently, the remaining low SEP participants were probably 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 also lead to differential drop-out.[65] These selection biases, however, are difficult to distinguish from competing risks (J. Kaufman, PhD, 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 above require further exploration in more detailed studies.

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.

Conclusion

Findings from this study underscore the importance of lifecourse SEP measures in determining the risk of diabetes in adulthood, regardless of race, and net of 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 in persons of lower socioeconomic means, especially those from non-white communities. Therefore, efforts to eliminate racial and socioeconomic inequities must be enhanced and sustained in order to reduce the burden of diabetes and other health conditions linked to social disadvantage across the lifecourse.

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REFERENCES

1. Gregg EW, Gu Q, Cheng YJ, et al. Mortality trends in men and women with diabetes, 1971–2000. Ann Intern Med. 2007; 147:149–155. [PubMed: 17576993]
2. Cowie CC, Rust KF, Byrd-Holt DD, et al. Prevalence of diabetes and impaired fasting glucose in adults in the U.S. population. Diabetes Care. 2006; 29:1263–1268. [PubMed: 16732006]
3. Geiss LS, Pan L, Caldwell B, et al. Changes in incidence of diabetes in U.S. adults, 1997–2003. Am J Prev Med. 2006; 30:371–377. [PubMed: 16627124]
4. Shai I, Jiang R, Manson JE, et al. Ethnicity, obesity, and risk of type 2 diabetes in women: a 20-year follow-up study. Diabetes Care. 2006; 29:1585–1590. [PubMed: 16801583]
5. Egede LE, Dagogo-Jack S. Epidemiology of type 2 diabetes: focus on ethnic minorities. Med Clin North Am. 2005; 89:949–975. [PubMed: 16129107]
6. Kirk JK, D'Agostino RB Jr, Bell RA, et al. Disparities in HbA1c levels between African-American and non-Hispanic white adults with diabetes: a meta-analysis. Diabetes Care. 2006; 29:2130–2136. [PubMed: 16936167]
7. Brown AF, Gregg EW, Stevens MR, et al. Race, ethnicity, socioeconomic position, and quality of care for adults with diabetes enrolled in managed care: the Translating Research Into Action for Diabetes (TRIAD) study. Diabetes Care. 2005; 28:2864–2870. [PubMed: 16306546]
8. Lanting LC, Joung IMA, Mackenbach JP, et al. Ethnic differences in mortality, end-stage complications, and quality of care among diabetic patients. Diabetes Care. 2005; 28:2280–2288. [PubMed: 16123507]
9. Marshall MC Jr. Diabetes in African Americans. Postgrad Med J. 2005; 81:734–740. [PubMed: 16344294]

10. Harris MI. Racial and ethnic differences in health care access and health outcomes for adults with Type 2 Diabetes. Diabetes Care. 2001; 24:454–459. [PubMed: 11289467]
11. Abate N, Chandalia M. The impact of ethnicity on type 2 diabetes. J Diabetes Complications. 2003; 17:39–58. [PubMed: 12505756]
12. Carnethon MR, Palaniappan LP, Burchfiel CM, et al. Serum insulin, obesity, and the incidence of type 2 diabetes in black and white adults: the Atherosclerosis Risk in Communities Study: 1987–1998. Diabetes Care. 2002; 25:1358–1364. [PubMed: 12145235]
13. Harris MI, Cowie CC, Gu K, et al. Higher fasting insulin but lower fasting C-peptide levels in African Americans in the US population. Diabetes Metab Res Rev. 2002; 18:149–155. [PubMed: 11994907]
14. Kant A, Graubard B, Kumanyika S. Trends in black-white differentials in dietary intakes of U.S. adults, 1971–2002. Am J Prev Med. 2007; 32:264–272. [PubMed: 17383557]
15. Kurian AK, Cardarelli KM. Racial and ethnic differences in cardiovascular disease risk factors: a systematic review. Ethn Dis. 2007; 17:143–152. [PubMed: 17274224]
16. Marshall SJ, Jones DA, Ainsworth BE, et al. Race/ethnicity, social class, and leisure-time physical inactivity. Med Sci Sports Exerc. 2007; 39:44–51. [PubMed: 17218883]
17. Narayan KM, Boyle JP, Thompson TJ, et al. Effect of BMI on lifetime risk for diabetes in the U.S. Diabetes Care. 2007; 30:1562–1566. [PubMed: 17372155]
18. Woolf SH, Johnson RE, Geiger HJ. The rising prevalence of severe poverty in America. A growing threat to public health. Am J Prev Med. 2006; 31:332–341. [PubMed: 16979459]
19. Wray LA, Alwin DF, McCammon RJ, et al. Social status, risky health behaviors, and diabetes in middle-aged and older adults. J Gerontol B Psychol Sci Soc Sci. 2006; 61:S290–S298. [PubMed: 17114308]
20. Best LE, Hayward MD, Hidajat MM. Life course pathways to adult-onset diabetes. Soc Biol. 2005; 52:94–111. [PubMed: 17619606]
21. Dalstra JA, Kunst AE, Borrell C, et al. Socioeconomic differences in the prevalence of common chronic diseases: an overview of eight European countries. Int J Epidemiol. 2005; 34:316–326. [PubMed: 15737978]
22. Robbins JM, Vaccarino V, Zhang H, et al. Socioeconomic status and type 2 diabetes in African Americans and non-Hispanic white women and men: evidence from the Third National Health and Nutrition Examination Survey. Am J Public Health. 2001; 91:76–83. [PubMed: 11189829]
23. Evans JMM, Newton RW, Ruta DA, et al. Socio-economic status, obesity and prevalence of Type 1 and Type 2 diabetes mellitus. Diabet Med. 2000; 17:478–480. [PubMed: 10975218]
24. Signorello LB, Schlundt DG, Cohen SS, Steinwandel MD, Buchowski MS, McLaughlin JK, Hargreaves MK, Blot WJ. Comparing diabetes prevalence between African Americans and Whites of similar socioeconomic status. Am J Public Health. 2007; 97:2260–2267. [PubMed: 17971557]
25. Maty SC, Lynch JW, Raghunathan TE, et al. Childhood socioeconomic position, gender, adult body mass index and the 34-year incidence of type 2 diabetes mellitus in the Alameda County Study. Am J Public Health. 2008; 98:1486–1494. [PubMed: 18556612]
26. Lidfeldt J, Li TY, Hu FB, et al. A prospective study of childhood and adult socioeconomic status and incidence of type 2 diabetes in women. Am J Epidemiol. 2007; 165:882–889. [PubMed: 17284723]
27. Maty SC, Everson-Rose SA, Haan MN, Raghunathan T, Kaplan GA. Education, income and occupation and the 34-year incidence of type 2 diabetes mellitus in the Alameda County Study. Int J Epidemiol. 2005; 34:1274–1281. [PubMed: 16120636]
28. Robbins JM, Vaccarino V, Zhang H, et al. Socioeconomic status and diagnosed diabetes incidence. Diabetes Res Clin Pract. 2005; 68:230–236. [PubMed: 15936465]
29. Kumari M, Head J, Marmot M. Prospective study of social and other risk factors for incidence of type 2 diabetes in the Whitehall II study. Arch Intern Med. 2004; 164:1873–1880. [PubMed: 15451762]
30. Brancati FL, Kao WHL, Folsom AR, et al. Incident type 2 diabetes mellitus in African American and white adults – The Atherosclerosis Risk in Communities Study. JAMA. 2000; 283:2253–2259. [PubMed: 10807384]

31. Hart CL, Hole DJ, Lawlor DA, et al. How many cases of type 2 diabetes mellitus are due to being overweight in middle age? Evidence from the Midspan prospective cohort studies using mention of diabetes mellitus on hospital discharge or death records. Diabet Med. 2007; 24:73–80. [PubMed: 17227327]
32. Sigal RJ, Kenny GP, Wasserman DH, et al. Physical activity/exercise and Type 2 Diabetes. A consensus statement from the American Diabetes Association. Diabetes Care. 2006; 29:1433–1438. [PubMed: 16732040]
33. D'Agostino R Jr, Hamman RF, Karter AJ, et al. Cardiovascular disease risk factors predict the development of type 2 diabetes: The Insulin Resistance Atherosclerosis Study. Diabetes Care. 2004; 27:2234–2240. [PubMed: 15333490]
34. Gress TW, Nieto FJ, Shahar E, et al. Hypertension and antihypertensive therapy as risk factors for type 2 diabetes mellitus. Atherosclerosis Risk in Communities Study. N Engl J Med. 2000; 342:905–912. [PubMed: 10738048]
35. Harper S, Lynch J. Trends in socioeconomic inequalities in adult health behaviors among U.S. states, 1990–2004. Public Health Rep. 2007; 122:177–189. [PubMed: 17357360]
36. Kanjilal S, Gregg SW, Cheng YJ, et al. Socioeconomic status and trends in disparities in 4 major risk factors for cardiovascular disease among US Adults, 1971–2002. Arch Intern Med. 2006; 166:2348–2355. [PubMed: 17130388]
37. Power C, Graham H, Due P, et al. The contribution of childhood and adult socioeconomic position to adult obesity and smoking behavior: an international comparison. Int J Epidemiol. 2005; 34:335–344. [PubMed: 15659473]
38. Kaufman JS, Cooper RS. Commentary: Considerations for use of racial/ethnic classification in etiologic research. Am J Epidemiol. 2001; 154:291–298. [PubMed: 11495850]
39. Berkman, LF.; Breslow, L. Health and Ways of Living: the Alameda County Study. New York, NY: Oxford University Press; 1983.
40. Kaplan, GA. Health and aging in the Alameda County Study. In: Schaie, KW.; Blazer, DG.; House, JSK., editors. Aging, Health Behaviors, and Health Outcomes. Hillsdale, NJ: Lawrence Erlbaum Associates; 1992. p. 69-88.
41. Hochstim, JR. Health and ways of living. In: Kessler, II.; Levin, ML., editors. The Community as an Epidemiologic Laboratory. Baltimore, MD: Johns Hopkins Press; 1970. p. 149-175.
42. Wadsworth MEJ, Hardy RJ, Paul AA, et al. Leg and trunk length at 43 years in relations to childhood health, diet and family circumstances; evidence from the 1946 national birth cohort. Int J Epidemiol. 2002; 31:383–390. [PubMed: 11980800]
43. Gunnell DJ, Smith GD, Frankel SJ, et al. Socio-economic and dietary influences on leg length and trunk length in childhood: a reanalysis of the Carnegie (Boyd Orr) survey of diet and health in prewar Britain (1937–39). Paediatr Perinat Epidemiol. 1998; 12 suppl 1:96–113. [PubMed: 9690276]
44. Li L, Manor O, Power C. Early environment and child-to-adult growth trajectories in the 1958 British birth cohort. Am J Clin Nutr. 2004; 80:185–192. [PubMed: 15213047]
45. Sorensen HT, Sabroe S, Rothman KJ, et al. Birth weight and length as predictor for adult height. Am J Epidemiol. 1999; 149:726–729. [PubMed: 10206622]
46. Barros AJD, Victoria CG, Horta BL, et al. Effects of socioeconomic change from birth to early adulthood on height and overweight. Int J Epidemiol. 2006; 35:1233–1238. [PubMed: 16926211]
47. Raghunathan TE, Lepkowski JM, Van Hoewyk J, et al. A multivariate technique for multiply imputing missing values using a sequence of regression models. Surv Methodol. 2001; 27:83–95.
48. Everson SA, Roberts RE, Goldberg DE, et al. Depressive symptoms and increased risk of stroke mortality over a 29-year period. Arch Intern Med. 1998; 158:1133–1138. [PubMed: 9605786]
49. Kaplan GA, Seeman TE, Cohen RD, et al. Mortality among the elderly in the Alameda County Study: behavioral and demographic risk factors. Am J Public Health. 1987; 77:307–312. [PubMed: 3812836]
50. Kaplan GA, Strawbridge WJ, Cohen RD, et al. Natural history of leisure-time physical activity and its correlates: Associations with mortality from all causes and cardiovascular disease over 28 years. Am J Epidemiol. 1996; 144:793–797. [PubMed: 8857828]

51. National Institute of Health/National Heart, Lung, and Blood Institute. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults - the evidence report. Obes Res. 1998; 6 suppl 2:51S–209S. [PubMed: 9813653]
52. Roberts RE, Kaplan GA, Camacho TC. Psychological distress and mortality: evidence from the Alameda County Study. Soc Sci Med. 1990; 31:527–536. [PubMed: 2218634]
53. Kaplan GA, Roberts RE, Camacho TC, et al. Psychosocial predictors of depression: prospective evidence from the Human Population Laboratory Studies. Am J Epidemiol. 1987; 125:206–220. [PubMed: 3812429]
54. Cox, DR.; Oakes, D. Analysis of Survival Data. New York, NY: Chapman & Hall; 1984.
55. Goldman N, Lin IF, Weinstein M, et al. Evaluating the quality of self reports of hypertension and diabetes. J Clin Epidemiol. 2003; 56:148–154. [PubMed: 12654409]
56. Martin LM, Leff M, Calonge N, et al. Validation of self reported chronic conditions and health services in a managed care population. Am J Prev Med. 2000; 18:215–218. [PubMed: 10722987]
57. Centers for Disease Control and Prevention. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention; 2005. National diabetes fact sheet: general information and national estimates on diabetes in the United States, 2005. Available at: http://www.cdc.gov/diabetes/pubs/factsheet05.htm
58. Kaufman JS, Cooper RS, McGee DL. Socioeconomic status and health in blacks and whites: the problem of residual confounding and the resiliency of race. Epidemiology. 1997; 8:621–628. [PubMed: 9345660]
59. Braveman PA, Cubbin C, Egerter S, et al. Socioeconomic status in health research: one size does not fit all. JAMA. 2005; 294:2879–2888. [PubMed: 16352796]
60. Altman DG, Patrick Royston P. The cost of dichotomizing continuous variables. BMJ. 2006; 332:1080. [PubMed: 16675816]
61. Lucove JC, Kaufman JS, James SA. Association between adult and childhood socioeconomic status and prevalence of the metabolic syndrome in African Americans: the Pitt County Study. Am J Public Health. 2007; 97:234–236. [PubMed: 17194854]
62. Adler N, Singh-Manoux A, Schwartz J, Stewart J, Matthews K, Marmot MG. Social status and health: A comparison of British civil servants in Whitehall-II with European- and African-Americans in CARDIA. Soc Sci Med. 2008; 66:1034–1045. [PubMed: 18180089]
63. Kahn JR, Fazio EM. Economic status over the life course and racial disparities in health. J Gerontol B Psychol Sci Soc Sci. 2005; 60(Spec No. 2):76–84. [PubMed: 16251596]
64. Williams DR, Collins C. US Socioeconomic and racial differences in health: Patterns and explanations. Annu Rev Sociol. 1995; 21:349–386.
65. Hernan MA, Hernandez-Diaz S, Robins JM. A structural approach to selection bias. Epidemiology. 2004; 15:615–625. [PubMed: 15308962]
66. Williams DR. Race, socioeconomic status, and health. The added effects of racism and discrimination. Ann NY Acad Sci. 1999; 896:173–188. [PubMed: 10681897]
67. Shavers VL, Shavers BS. Racism and health inequity among Americans. J Natl Med Assoc. 2006; 98:386–396. [PubMed: 16573303]
68. Williams DR, Jackson PB. Social sources of racial disparities in health. Health Aff (Millwood). 2005; 24:325–334. [PubMed: 15757915]
69. Jones CP. Invited commentary: “race,” racism, and the practice of epidemiology. Am J Epidemiol. 2001; 154:299–304. [PubMed: 11495851]

TABLE 1

Baseline distribution of sample characteristics by racial group (black (n=648), white (n=4774)) in the Alameda County Study (1965–1999)

Variable	Category	Racial Group		p-value
		Blacks (%)	Whites (%)	
Age (years)	Mean (SD)	42.6 (14.0)	43.4 (16.1)	0.23
Gender	Men	46.3	46.7	0.85
	Women	53.7	53.3	
Marital	Married	67.0	76.2	<0.0001*
Status	Unmarried	33.0	23.8	
Height (inches)	Mean (SD)	66.5	66.6	0.90
Childhood SEP†	Low	71.9	49.0	<0.0001*
	High	28.1	51.0	
Education (years)	Mean (SD)	10.4 (3.2)	12.3 (3.2)	<0.0001**
Education	≤ 12 Years	78.7	61.2	<0.0001*
	>12 Years	21.3	38.8	
Household Income (1999 dollars)	Mean (SD)	9857.6 (2.1)	15787.9 (2.0)	<0.0001**
Occupation	White-Collar	20.1	42.4	<0.0001*
	Blue-Collar	54.2	24.1	
Health Insurance	Yes	71.0	88.4	<0.0001*
	No	29.0	11.6	
Regular Access to	Yes	73.9	78.7	0.005*
MD/Clinic	No	26.1	21.3	
High Blood	Yes	16.4	8.9	<0.0001*
Pressure	No	83.6	91.1	
Depression	Yes	17.0	13.6	0.02*
	No	83.0	86.4	
Body Mass Index	Obese	11.6	4.6	<0.0001***
(BMI) (kg/m^2)	Overweight	37.2	25.9	
Category‡	Normal	51.2	69.5	
BMI (kg/m^2)	Mean (SD)	25.1 (3.9)	23.5 (3.5)	<0.0001**
Waist§	Large	8.3	5.4	0.002*
Circumference	Not Large (normal)	91.7	94.6	
Waist Circumf. (in)	Mean (SD)	31.5 (4.8)	30.8 (5.0)	0.01**
Physical Activity	Inactive/Low Activity	40.4	29.0	<0.0001***
	Moderate Activity	41.1	45.8	
	High Activity	18.5	25.2	
Smoking Status	Never Smoker	35.6	38.5	0.02***

Variable	Category	Racial Group		p-value
		Blacks (%)	Whites (%)	
	Former Smoker	13.6	16.7	
	Current Smoker	50.8	44.8	
Alcohol	Abstain	32.1	17.2	<0.0001***
Consumption	1–45 Drinks per Month	55.9	66.8	
	46+ Drinks per Month	12.0	16.0	

* p-value ≤ 0.05 for X^2 test for proportional difference in distribution of covariate category by racial group

** p-value for T-test for comparison of continuous variable means by race

*** p-value ≤ 0.05 for X^2 test for trend across covariate categories

† Childhood SEP is based on respondents' fathers' occupation (or education when occupation data not available (6.5% of total)): Low = manual (blue-collar) occupation or education ≤12 years; High = white-collar occupation or ≥12 years of education (referent)

‡ Obese = Body Mass Index (BMI) ≥30 kg/m^2; Overweight = BMI 25–29.9 kg/m^2; Normal/Underweight = BMI ≤24.9 kg/m^2

§ Large Waist Circumference = >34.6in for women and >40.2in for men

TABLE 2

Incidence proportion and crude incidence density (incident cases per 1,000 person-years at risk) of type 2 diabetes mellitus over 34-years (1965–1999) associated with baseline characteristics by racial group (black (n=648), white (n=4774)) in the Alameda County Study

Variable Category	BLACK			WHITE		
	Total Incident Cases	Percent of Category with Diabetes	Incidence Density	Total Incident Cases	Percent of Category with Diabetes	Incidence Density
Total Population	51	7.9	4.2	211	4.4	2.0
Age <40 years	24	8.7	4.8	105	4.9	2.0
Age ≥40 years	27	7.2	3.8	106	4.0	2.0
Female	29	8.3	4.4	108	4.2	1.9
Male	22	7.3	4.0	103	4.6	2.1
Married	34	7.8	4.1	166	4.6	1.9
Unmarried	17	7.9	4.3	45	4.0	2.0
Below Mean Height	25	7.3	3.8	105	4.4	2.0
Above Mean Height	26	8.5	4.6	106	4.5	1.9
Low Childhood SEP[†]	39	8.4	4.4	133	5.7 *	2.6
High Childhood SEP	12	6.6	3.5	78	3.2	1.5
Education ≤12 years ***	34	6.7 *	3.6	143	4.9 *	2.4
Education >12 years	17	12.3	6.6	68	3.7	1.5
Low Income Tertile	18	8.3	4.5	82	5.2	2.4
Moderate Income Tertile	15	7.0	3.5	64	4.0	1.8
High Income Tertile	18	8.3	4.7	65	4.1	1.7
Blue-Collar Occupation	28	8.0	4.2	56	4.9	2.4
White-Collar Occupation	14	10.8	5.9	93	4.6	2.0
NO Health Insurance	16	8.5	5.0	14	2.5 *	1.3
YES Health Insurance	35	7.6	3.9	197	4.7	2.0

Variable Category	BLACK			WHITE		
	Total Incident Cases	Percent of Category with Diabetes	Incidence Density	Total Incident Cases	Percent of Category with Diabetes	Incidence Density
NO Regular Health Provider	16	9.5	5.7	35	3.5	1.6
YES Regular Health Provider	35	7.3	3.7	176	4.7	2.1
YES Depression	9	8.2	4.6	29	4.5	2.3
NO Depression	42	7.8	4.1	182	4.4	1.9
YES High Blood Pressure	10	9.4	5.4	26	6.1	3.7
NO High Blood Pressure	41	7.6	4.0	185	4.3	1.8
Obese (BMI ≥30 kg/m^2) ***	10	13.3 **	6.9	36	16.6 **	8.3
Overweight (BMI 25–29.9 kg/m^2)	20	8.3	4.3	68	5.5	2.5
Normal/Underweight (BMI ≤24.9 kg/m^2)	21	6.3	3.4	107	3.2	1.4
Large Waist Circumference‡	7	13.0	7.4	29	11.3 *	6.9
Normal Waist Circumference	44	7.4	3.9	182	4.0	1.8
Inactive/Low Activity	22	8.4	4.8	57	4.1	2.1
Moderate Activity	22	8.3	4.3	102	4.7	2.0
High Activity	7	5.8	2.9	52	4.3	1.7
Current Smoker	30	9.1	5.0	106	5.0 **	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
Abstain from drinking	16	7.7	4.3	31	3.8	1.9
1–45 drinks per month	28	7.7	4.0	147	4.6	2.0
> 46 drinks per month	7	9.0	4.6	33	4.3	1.9

* p-value ≤ 0.05 for X^2 test for difference in distribution of covariate category within racial group

** p-value ≤ 0.05 for X^2 test for Trend across covariate categories within racial group

*** p-value ≤ 0.05 for interaction between covariate category and racial group

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[†]Childhood SEP is based on respondents' fathers' occupation (or education when occupation data not available (6.5% of total)): Low = manual (blue-collar) occupation or education ≤12 years; High = white-collar occupation or ≥12 years of education (referent)

[‡]Large Waist Circumference = >34.6in for women and >40.2in for men

TABLE 3

Unadjusted hazard ratios (HR) and 95% confidence intervals (CI) for the 34-year incidence of type 2 diabetes associated with baseline characteristics by racial group (black (n=648), white (n=4774)) in the Alameda County Study (1965–1999)

Variable Category	Black		White	
	HR	95% CI	HR	95% CI
Racial Group	2.3	1.7, 3.1	1.0	-
Age (years) (continuous)	1.0	1.0, 1.0	1.0	1.0, 1.0
Women	1.1	0.6, 1.9	0.9	0.7, 1.1
Men	1.0		1.0	
Unmarried	1.1	0.6, 1.9	1.1	0.8, 1.5
Married	1.0		1.0	
Low Childhood SEP*	1.3	0.7, 2.5	1.9	1.4, 2.5
High Childhood SEP (referent)	1.0		1.0	
Height (inches) (continuous)	1.0	0.9, 1.1	1.0	1.0, 1.0
Education (years) (continuous)	1.0	0.9, 1.1	0.9	0.9, 1.0
≤12 years Education	0.5	0.3, 1.0	1.7	1.3, 2.3
>12 years Education (referent)	1.0		1.0	
Income (1999 dollars) (continuous)	1.0	0.7, 1.4	0.8	0.6, 0.9
Blue Collar Occupation	0.7	0.4, 1.4	1.3	0.9, 1.8
White Collar Occupation (referent)	1.0		1.0	
No Health Insurance	1.3	0.7, 2.4	0.7	0.4, 1.1
Yes Health Insurance (referent)	1.0		1.0	
No Regular Health Provider	1.6	0.9, 2.8	0.8	0.5, 1.1
Yes Regular Health Provider (referent)	1.0		1.0	
Yes Depression	1.1	0.5, 2.3	1.3	0.8, 1.9
No Depression (referent)	1.0		1.0	
Yes High Blood Pressure	1.4	0.7, 2.9	2.3	1.5, 3.5
No High Blood Pressure (referent)	1.0		1.0	
Body Mass Index (kg/m^2) (BMI) (continuous)	1.0	1.0, 1.1	1.1	1.1, 1.2
Obese (BMI ≥30 kg/m^2)	2.1	1.0, 4.4	6.4	4.4, 9.3
Overweight (BMI 25–29.9 kg/m^2)	1.3	0.7, 2.3	1.9	1.4, 2.5
Normal/Underweight (BMI ≤24.9) (referent)	1.0		1.0	
Waist Circumference (inches) (continuous)	1.0	1.0, 1.0	1.0	1.0, 1.0
Large Waist Circumference†	2.0	0.9, 4.5	4.5	3.0, 6.7
Normal Waist Circumference (referent)	1.0		1.0	
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 (referent)	1.0		1.0	

Variable Category	Black		White	
	HR	95% CI	HR	95% CI
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
Never Smoked (referent)	1.0		1.0	
Abstain from drinking	1.1	0.6, 2.0	1.0	0.7, 1.5
1–45 drinks per month (referent)	1.0		1.0	
> 46 drinks per month	1.2	0.5, 2.7	1.0	0.7, 1.4

*Childhood SEP is based on respondents' fathers' occupation (or education when occupation data not available (6.5% of total)): Low = manual (blue-collar) occupation or education ≤12 years; High = white-collar occupation or ≥12 years of education (referent)

†Large Waist Circumference = >34.6in for women and >40.2in for men

TABLE 4

Hazard ratios (HR) and 95% confidence intervals (CI) for the 34-year incidence of type 2 diabetes mellitus associated with lifecourse socioeconomic factors by racial group (Black (n=648), White (n=4774)) in the Alameda County Study (1965–1999): All covariates measured at baseline

	Childhood SEP†		Education‡		Income§		Occupation#	
MODEL*	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI
BLACK								
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
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
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
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
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
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
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
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
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
WHITE								
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
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
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
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
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
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
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
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
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

*Model 1 is unadjusted

Model 2 is adjusted for age, gender and marital status

Model 3 is adjusted for age, gender, marital status, and childhood SEP measures (parental occupation/education, height, and own education)

Model 4 is adjusted for age, gender, marital status, and adult SEP measures (income, occupation)

Model 5 is adjusted for age, gender, marital status, childhood SEP measures, adult SEP measures and health insurance status

Model 6 is adjusted for age, gender, marital status, BMI (continuous), and waist circumference (continuous)

Model 7 is adjusted for age, gender, marital status, physical activity, alcohol use, and smoking

Model 8 is adjusted for age, gender, marital status, regular access to a medical doctor or clinic, depression, and high blood pressure

Model 9 is fully adjusted for all covariates

[†]Childhood SEP is based on respondents' fathers' occupation (or education when occupation data not available (6.5% of total)): Low = manual (blue-collar) occupation or education ≤12 years; High = white-collar occupation or ≥12 years of education (referent)

[‡]Education, ≤12 years vs. >12 years (referent)

[§]Income, continuous

[#]Occupation, Blue-collar vs. White-collar (referent)