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Education, income, occupation, and the 34-year incidence (1965–99) of Type 2 diabetes in the Alameda County Study

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Abstract

Background—Lower socioeconomic position (SEP) is related to higher prevalence of Type 2 diabetes, yet little is known about the relationship of SEP with incident diabetes.

Methods—The association between SEP, measured by self-reported education, income, and occupation, and Type 2 diabetes incidence was examined in a community sample of 6147 diabetes-free adults from Alameda County, CA. Cox proportional hazards models estimated the effect of baseline (1965) and time-dependent (value changes over time) measures of SEP on incident diabetes over a 34-year study period (1965–99). Demographic confounders (age, gender, race, and marital status) and potential components of the causal pathway (physical inactivity, smoking, alcohol consumption, body composition, hypertension, depression, and health care access) were included as fixed or time-dependent covariates.

Results—Education, income, and occupation were associated with increased diabetes risk in unadjusted models. In baseline models adjusted for demographics, respondents with <12 years of education had 50% excess risk compared with those with more education [hazard ratio (HR) = 1.5, 95% confidence interval (95% CI) 1.11–2.04], but income and occupation were no longer significantly associated with increased risk. Further adjustment minimized the significance of all associations. Time-dependent effects were consistently elevated for low education and male blue-collar occupation, but non-significant after full adjustment (HR = 1.1, 95% CI 0.79–1.47 and HR = 1.3, 95% CI 0.91–1.89, respectively).

Conclusions—Socioeconomic disadvantage, especially with low educational attainment, is a significant predictor of incident Type 2 diabetes, although associations were largely eliminated after covariate adjustment. Obesity and overweight appear to mediate these associations.

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Keywords

Socioeconomic factors; Type 2 diabetes mellitus; incidence

Type 2 diabetes mellitus imposes a major public health burden across populations. Over 150 million people suffer from the disease worldwide,[1] including more than 18 million in the US.[2] In the US, Type 2 diabetes is a major source of morbidity and mortality causing significant medical complications and resulting in over 200 000 deaths annually.[2]

The prevalence of Type 2 diabetes is strongly patterned by socioeconomic position (SEP). Persons with a lower SEP consistently have a higher prevalence of diabetes and an excess burden of morbidity and mortality compared with persons of greater SEP.[3–6] Although diabetes prevalence is rising in the overall population, it is increasing more steeply for people with lower SEP.[5] An inverse, graded association between SEP and diabetes prevalence has been found using different measures of SEP, such as education,[3,5,7–10] occupation,[3,7,10] income,[3] poverty income ratio,[7] the Green index (composite of education and occupation),[11] and ecological measures of material deprivation or poverty.[12–15]

The social determinants of diabetes incidence are similar to those for prevalent disease. Persons of lower SEP have limited income, poorer occupational opportunity, and reduced access to health care services and information: factors that contribute to both diabetes risk and complications of disease management. However, few studies have examined the impact of socioeconomic factors on diabetes incidence. Limitations of previous research include small sample sizes, short follow-up, and lack of statistical control of covariates.[16–24] Little is known about the different pathways through which socioeconomic factors may influence the occurrence of Type 2 diabetes. Many known risk factors, such as excess body weight, large waist circumference, and physical inactivity are patterned by SEP.[25–34] Whether or not these factors are components of the causal pathway between SEP and diabetes incidence has not been extensively examined.

This study used five waves of data collected over 34 years to examine the relationship between three measures of SEP (education, income, and occupation) and the incidence of Type 2 diabetes in a community sample. Three hypotheses were proposed. First, lower education, lower income, and blue-collar occupation would be significantly associated with an increased risk of developing diabetes; second, time-dependent measures of SEP would impart greater risk than baseline measures; and third, the association between SEP and incident diabetes would be explained by known diabetes risk factors or other factors that are potential components of the causal pathway.

Methods

Study population

We used data from the Alameda County Study (ACS), a population-based, longitudinal study of the predictors of health and physical functioning in a random, stratified, closed sample of 6928 non-institutionalized adults aged 17–94 years who resided in Alameda County, CA, in 1965. Comprehensive, mailed, self-administered questionnaires were distributed at each of the five study waves: 1965 (baseline), 1974, 1983, 1994, and 1999. Response rates for the five surveys were between 85 and 95% of eligible respondents.[35–37]

Of the 6928 eligible participants in 1965, we excluded those who reported having diabetes (n = 157, 2.3%) or whose diabetes status at baseline was unknown (n = 5, 0.07%). Eighty-nine (1.3%) respondents were excluded owing to inconsistencies in their reported date of diagnosis. Participants with missing data in 1965 for key variables (n = 530, 7.7%) were also removed. These respondents were more likely to be older, female, non-white, overweight or obese, physically inactive, of lower socioeconomic means, and uninsured. Consequently, any association between SEP and diabetes incidence in the final sample would probably be biased toward the null. The final sample was limited to the remaining 6147 (88.7%) individuals [53.6% female, 20.3% non-white (11.7% black, 3.9% Hispanic, and 4.8% other)].

Measures

Diabetes status was determined at each wave by self-report from two questions: 'have you had any of these conditions <diabetes> during the past 12 months?' (yes/no) and 'when did it start <year>?' Incident cases were those reported at study wave (t) that were not reported at wave ($t - 1$), and whose year of diagnosis occurred between wave (t) and wave ($t - 1$). Cumulative incidence was the total number of new cases that occurred between 1965 and 1999. Time-to-event was calculated as the difference between baseline and year of diagnosis.

SEP was measured by education, income, and occupation. Total years of education were assessed at each wave and categorized, based on the baseline distribution, as less than, equal to, or greater than 12 years.

Household income data were collected in bounded categories at each wave. A multiple imputation approach[38] using a sequential regression imputation process[39] was employed to account for missing household income data and to assign a continuous income value at each wave. Minimal variation in missing income values, between 4.4 and 7.3%, existed at each study wave. This process used data from the 1965, 1974, 1983, 1994, and 1999 Current Population Survey (CPS), a national representative sample of US households,[40] as a comparison group. Each participant was assigned an income value based on the relationship between income and several covariates (age, education, gender, race, marital status, occupation, and number of household members) present in both the ACS and CPS data. The imputation was bound within reported income categories for ACS respondents with non-missing income information. The CPS income distribution was used to create the categorical boundary for missing income data. This technique assumed data were missing at random with the joint distribution fully conditioned on all observed information. Approximations for missing income data were generated using separate regression models that created variables using non-missing or other imputed variables as covariates. The process was repeated until all imputed values converged. This imputation process has been shown to increase efficiency and provide unbiased risk estimates owing to its comprehensive use of all available data.[41]

For these analyses, the continuous imputed household income variable was standardized to 1999 dollars to allow for direct comparison across waves, adjusted for the reported number of persons in the household, and log transformed achieving normality of the distribution. Three income categories, low, moderate, and high were created using tertiles of the imputed income distribution.

Self-reported current or most recent occupation was coded using US census criteria. Retired participants were assigned their primary lifetime occupation. These data were sorted into four categories: white-collar, blue-collar, keep house, or other. The 'other' category included unemployed, students, and unclassifiable participants. Few men entered the 'keep house' category so gender-specific analyses were performed. Results are limited to white-collar and blue-collar categories.

Covariates were measured at baseline and each subsequent study wave. Demographic factors included age, gender, racial group (white/non-white), and marital status (single, married, and separated, divorced, or widowed). Remaining covariates were known diabetes risk factors or potential components of the causal pathway between SEP and diabetes incidence.

Self-reported weight and height data were used to create continuous values for body mass index (BMI) and collapsed into three groups: obese (BMI ≥30 kg/m^2), overweight (BMI 25–29.9 kg/m^2), and normal or underweight (BMI ≤ 24.9 kg/m^2).[42] Waist circumference of >880 mm for women and >1020 mm for men indicated excessive central adiposity.[42] Waist circumference was only measured at baseline. A physical activity scale was constructed using information about the frequency, type and intensity of four activities: physical exercise, long walks, swimming, or taking part in active sports, and was reduced to three categories: no or low, moderate, and high activity. These items and scale construction have been used previously and associated with all-cause mortality.[43]

Alcohol consumption was assessed by a score combining alcohol type (beer, wine, or liquor), frequency (never, less than once a week, 1–2 times per week, 2+ times per week) and intake at each sitting (never, 1–2 drinks, 3–4 drinks, 5+ drinks). The composite score created three classes of alcohol consumption: abstain (0 drinks per month), light to moderate (1–45 drinks per month), and heavy (46+ drinks per month).[44] Smoking status was defined as current, former, or never smoked.

In the US, having health insurance does not guarantee a consistent source of care; two factors that independently influence health outcomes. Therefore, access to health care was measured using two dichotomous (yes/no) variables: possessing health insurance and having a ‘regular’ doctor or health clinic. High blood pressure was assessed with the question, ‘Have you had high blood pressure during the past 12 months?’ Depression was defined as a score of five or more on a reliable and valid 18 item scale used in other analyses to indicate significant depressive symptomatology.[45–47]

Statistical analysis

Incidence density was calculated for education, income, and occupation by all covariates. Cochran–Armitage tests determined whether a monotonic trend existed for the binomial proportion of each covariate by income and education. Chi-square tests measured covariate associations with occupation.

Cox proportional hazards regression models estimated relationships between diabetes incidence and education, income, or occupation measured at baseline and as time-dependent predictors. SEP measures were not modelled simultaneously. Evidence for effect differences by demographic variables was not consistent or significant (data not shown). Therefore, adjustment of demographic variables was deemed appropriate for these analyses. Cox model sensitivity and assumptions were tested and met using Kaplan–Meier curves and SEP–time interactions. The Efron method was used for ties. All tests of significance were two-tailed.

Participants who died (n = 2611) through 1999 were censored in the year of death. Participants who dropped out amid two waves of data collection were censored at the mid-point of the interval. Analyses were performed using Statistical Analysis System, Version 8.2 software (SAS Institute, Cary, NC).

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Results

Of 6147 participants at baseline, 318 (5.2%) reported developing diabetes over the 34-year study period. Mean age at diagnosis was 58.6 years (SD = 12.2).

Table 1 summarizes the distribution and 34-year incidence rate by select characteristics at baseline. Table 2 presents trends for education and income significant across most covariates. Chi-square tests for all covariates, except moderate activity, were significant for each occupation category.

Table 3 presents model results where all variables were measured at baseline. Low or moderate education, lower income, and blue-collar occupation were associated with an increased risk of diabetes in unadjusted models. No effect was observed for women who kept house (data not shown). Adjustment for demographic confounders (model 3) attenuated the effect of low and moderate education by 43 and 21%, respectively, by 48% for log income, and by 29 and 95% for blue-collar men and women, respectively. Although the relationship between low education and incident diabetes remained significant, those with other SEP measures were no longer significant. Subsequent models (models 4 and 5) added potential components of the pathway between SEP and incident diabetes. Behavioural covariates (physical activity, alcohol use, and smoking) reduced the risk attributed to lower education, but had little effect on income and blue-collar occupation. Body composition (BMI and waist circumference) additionally weakened the effect of each SEP measure on disease incidence. The final model (model 6) included all covariates. Although the magnitude of the association between each socioeconomic measure and incident diabetes did not diminish after full adjustment, none remained statistically significant (Table 3).

Table 4 presents models where all variables except waist circumference were time-dependent. The magnitude of unadjusted associations for time-dependent education and income were smaller than baseline. The effect size for time-dependent blue-collar occupation was stronger than that seen at baseline for men, yet similar for women. Keeping house had no associated risk (data not shown). Adjustment for time-dependent covariates reduced the size and statistical significance of the association between SEP and diabetes incidence. Behaviours minimized risk for all SEP measures, especially education (model 4). Subsequent addition of waist circumference and BMI accounted for any remaining risk owing to blue-collar work in women or low education (model 5). The magnitude was reduced, but not eliminated for male blue-collar work. After full adjustment (model 6), only blue-collar occupation in men was associated with excess risk (Table 4).

Baseline and time-dependent predictors were also modelled simultaneously. Results indicate that time-dependent measures were a better fit for occupation, but not education or income (data not shown).

Discussion

SEP was a significant predictor of the 34-year incidence of Type 2 diabetes mellitus. The association occurred regardless of the SEP measure used, except for women who kept house, although statistical significance lessened after adjustment for demographic confounders and potential components of the causal pathway.

In age-adjusted baseline models, <12 years of education was associated with a 90% increased risk of diabetes compared with >12 years of education. An increase of 1 SD in log income translated to a 23% lower risk. Blue-collar occupation imparted a 42 and 55% higher risk than white-collar work for men and women, respectively. Compared with baseline, time-dependent SEP effects were relatively stronger and more robust for male blue-collar occupation, similar for female blue-collar workers, and weaker for education and income. Excess risk was largely explained by covariates known to play a role in the development of Type 2 diabetes, especially BMI.

Low education was a strong predictor of incident diabetes, although time-dependent models produced weaker effects than baseline. After 1965, only 9% of the sample added years of education, so a time-dependent measure may not be appropriate. These results suggest education may be a better measure of early life exposures.

Time-dependent income had a weaker relationship with diabetes incidence than baseline. Given that the mean age at diagnosis was 58.6 years, many cases probably occurred among retired persons. As income generally falls after retirement, this decline may explain the weakened association. Retirement income, therefore, may not be an accurate measure of economic assets.

Several factors may account for excess risk owing to time-dependent occupation relative to baseline. For example, accumulation of exposure may increase risk. Blue-collar workers are more likely to work longer hours under hazardous conditions with minimal financial compensation compared with white-collar workers. Although the proportion of blue-collar workers was similar across study waves (25–29%), students and homemakers were more likely to move into the white-collar category. If white-collar workers became healthier than the blue-collar group over time, associations between blue-collar work and diabetes incidence would increase. Alternatively, one-third of the sample was <33 years old at baseline and probably had not achieved their occupational potential. Occupation measured in middle or later adulthood may be a better measure of SEP exposure for these participants. Reverse causation also could inflate the effect of blue-collar work on diabetes incidence. Type 2 diabetes has a long pre-clinical stage so individuals may suffer symptoms limiting their job choices and earning potential prior to diagnosis. Lastly, undetected disease and related disability may affect blue-collar workers more than white-collar workers owing to occupational differences.

Limitations exist that restrict the conclusions we can draw from our analyses. Most significant is the use of self-reported data, which may lead to misclassification of exposure and disease status. In these data diabetes status could not be diagnostically confirmed. However, the use of self-reported disease status correlates well with medically diagnosed diabetes.[48–50] Diabetes type (Type 1 or Type 2) could not be definitively determined. Type 2 is predominantly diagnosed in persons >40 years. After the age of 30, only 7.4% of all cases of diabetes are due to Type 1.[51] Participants who developed diabetes after 1965 were included as cases regardless of age at diagnosis. Covariate distributions did not differ by age at diagnosis. Misclassification of Type 1 diabetes as Type 2, therefore, would lead to minimal bias in the association between SEP and incident diabetes.

Survival bias also may have affected our results. Participants who developed diabetes between study waves may have been more likely to drop out or die before being counted as incident cases compared with participants without diabetes. If those individuals were socioeconomically disadvantaged, the relationship between lower SEP and incident diabetes would be minimized. Despite selective survival or participation, the incidence rate for this cohort (2.4 per 1000 person-years) is identical to national self-reported incidence rates.[52]

Diabetes risk associated with SEP may be confounded by demographic factors. Statistical adjustment for race,[53] gender, or age is suitable when the variable is not an exposure of interest. In this study, adjustment provided an average, conservative risk estimate across demographic groups and probably controlled for unmeasured factors such as discrimination, material deprivation, and differing social roles; factors correlated with race, age, and gender and possibly associated with diabetes risk.

This study had several strengths. First, we used data collected on five occasions over a 34-year period. Second, longitudinal data permitted investigation of the predictors of incident diabetes. Most prior studies of the association between Type 2 diabetes and socioeconomic factors have used prevalent data. Third, three measures of SEP were investigated at different points in time. Finally, these data permitted simultaneous investigation of a variety of potential confounders and components of the causal pathways from SEP to incident diabetes.

Many known diabetes risk factors were associated with increased incidence in these data. These results support other study findings[54–56] and give credence to risk factor measurements. In baseline and time-dependent models, body composition attenuated risk for all SEP measures. As these factors also are patterned by SEP, the results suggest BMI and waist circumference may be components of the pathway between SEP and diabetes incidence.

These results corroborate findings from the few studies that examined the effect of socioeconomic factors on diabetes incidence.[16–24] Most prior research used education as the sole measure of SEP.[17,19,21,22,24] Regardless of methodology, length of follow-up, or ethnic group, lower educational attainment was associated with increased risk of developing diabetes, although pathways through which education may influence incidence were not always considered.[19,21,22,24] Other studies measured SEP using occupation,[20] occupational prestige,[18] neighbourhood disadvantage,[18,23] or military rank.[16] These studies, however, were limited by their brief follow-up periods, small sample sizes, minimal statistical control of covariates, consideration of only one socioeconomic predictor, and lack of investigation of pathways through which socioeconomic factors may influence the development of diabetes over time.

Our results support the conclusions that socioeconomic disadvantage, especially in educational attainment, is a significant predictor of incident Type 2 diabetes in adults. Time-dependent effects were stronger than baseline for occupation, yet less important for education or income. Adjustment for confounders and potential risk factors minimized associations between SEP and diabetes. Finally, body composition, particularly BMI, is an important component of the pathway between SEP and diabetes incidence.

KEY MESSAGES

- Socioeconomic disadvantage, especially in educational attainment, is a significant predictor of Type 2 diabetes incidence in adults.
- Adjustment for confounders and potential risk factors, primarily BMI, largely eliminated associations between SEP and diabetes incidence.
- BMI is an important component of the pathway between SEP and diabetes incidence.

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Table 1

Characteristics of the Alameda County Study (ACS) population (*n* = 6147) at baseline (1965) and crude incidence rate of Type 2 diabetes mellitus over 34 years (1965–99)

			Diabetes	
Variable	**Category**	***n* (%)**	**Cases of incident diabetes**	**Incidence rate per 1000 person-years**
Age	≤29 years	1523 (24.8)	68	1.9
	30–39 years	1285 (20.9)	96	3.1
	40–49 years	1373 (22.3)	85	2.6
	50–59 years	926 (15.1)	39	2.0
	60–69 years	595 (9.7)	23	2.3
	≥70 years	445 (7.2)	7	1.5
Gender	Women	3293 (53.6)	175	2.4
	Men	2854 (46.4)	143	2.3
Racial group	White	4898 (79.7)	218	2.0
	Non-white	1249 (20.3)	100	4.0
	Non-black	719 (11.7)	53	3.9
	Non-Hispanic	238 (3.9)	26	5.3
	Non-other	292 (4.8)	21	3.2
Marital Status	Single	623 (10.1)	22	1.6
	Married	4624 (75.2)	245	2.3
	Separated–divorced–widowed	900 (14.7)	51	3.3
Education	<12 years	2103 (34.2)	115	3.0
	12 years	1896 (30.8)	109	2.5
	>12 years	2148 (35.0)	94	1.8
Education	Low Tertile	2050 (33.3)	118	2.8
	Moderate Tertile	2046 (33.3)	110	2.4
	High Tertile	2051 (33.4)	90	1.9
Occupation: men	White-collar job	1228 (43.0)	59	2.1
	Blue-collar job	1359 (47.6)	74	2.8
	Unemployed/student/other	267 (9.4)	10	1.7
Occupation: women	White-collar job	1089 (33.1)	58	2.3
	Blue-collar job	417 (12.7)	29	3.4
	Keep house	1612 (48.9)	81	2.3
	Unemployed/student/other	175 (5.3)	7	1.7
Use of regular MD or clinic	No	1387 (22.6)	66	2.3
	Yes	4760 (77.4)	252	2.4
Health insurance	No	930 (15.1)	43	2.5
	Yes	5217 (84.9)	275	2.3
Depression	Yes	889 (14.5)	49	2.9
	No	5258 (85.5)	269	2.3
High blood pressure	Yes	597 (09.7)	41	4.1

			Diabetes	
Variable	**Category**	***n* (%)**	**Cases of incident diabetes**	**Incidence rate per 1000 person-years**
	No	5550 (90.3)	277	2.2
Weight group	Obese (BMI > 30 kg/m^2)	343 (05.6)	58	8.7
	Overweight (BMI 25–29.9)	1665 (27.1)	106	3.0
	Normal/under (BMI < 25)	4139 (67.3)	154	1.7
Waist circumference[a]	>34.6 in women/40.2 in men	354 (05.8)	42	7.2
	<34.6 in women/40.2 in men	5793 (94.2)	276	2.1
Physical activity	Inactive/low activity	1957 (31.8)	102	2.8
	Moderate activity	2739 (44.6)	150	2.4
	High activity	1451 (23.6)	66	1.9
Alcohol consumption	Abstain	1272 (20.7)	65	2.6
	1–45 drinks per month	3969 (64.6)	210	2.3
	>46 drinks per month	906 (14.7)	43	2.2
Smoking status	Never smoked	2392 (38.9)	107	2.0
	Former smoker	975 (15.9)	55	2.5
	Current smoker	2780 (45.2)	156	2.6

[a]Waist circumference measured at baseline (1965) only.

Table 2

Characteristics (%) of ACS population (n = 6147) in 1965 by education, income, and occupation

Subject characteristic	Education[a]			Income[a]			Occupation[b]			
	<12 years	12 years	>12 years	Low	Middle	High	White-collar	Blue-collar	Keep house (women)	Other
Age > 40 years	72.2	52.2	38.7	44.0	53.1	65.8	56.0	62.2	53.2	17.9
Women	54.2	57.8	49.3	56.3	52.5	51.9	47.0	23.5	100.0	39.6
Non-white racial group	31.2	16.7	12.9	32.3	17.8	10.9	11.2	33.3	18.2	23.5
Single	4.0	7.8	18.2	7.9	8.2	14.4	12.3	7.3	1.2	42.8
Separated–divorced–widowed	22.3	13.1	8.6	17.8	10.3	15.8	14.0	15.1	16.5	9.3
Obese	8.7	4.7	3.3	6.5	5.3	4.8	4.1	7.3	6.3	3.6
Overweight	33.5	25.4	22.3	26.6	27.0	27.6	26.4	36.8	19.5	19.2
Large waist	10.2	4.2	2.8	6.9	6.0	4.3	4.3	5.1	9.6	2.3
Inactive/low activity	47.6	27.9	19.9	37.0	29.4	29.1	26.0	36.4	39.1	17.7
Moderate activity	39.1	48.4	46.5	43.1	45.7	44.9	46.4	43.2	43.3	44.8
Abstain from drinking	29.5	18.1	14.4	27.0	19.8	15.3	13.6	23.7	28.9	16.3
>45 drinks/month	12.6	15.0	16.6	10.9	14.7	18.6	18.1	16.7	6.7	18.3
Former smoker	14.8	14.0	18.6	13.7	15.5	18.4	18.5	17.3	11.0	14.0
Current smoker	46.0	51.0	39.4	46.5	46.1	43.0	43.7	54.2	38.7	41.0
No ‘Regular’ MD/clinic	22.4	20.8	24.2	25.0	22.2	20.5	20.9	26.8	18.3	29.9
No health insurance	23.0	11.9	10.2	24.6	11.9	8.9	9.4	15.8	20.0	24.9
High blood pressure	14.8	7.4	6.8	11.1	8.7	9.3	8.6	10.0	12.2	5.7
Depressed affect	19.5	14.7	9.3	18.8	14.3	10.3	10.4	15.3	19.3	14.9

[a]Cochran–Armitage trend tests significant for monotonic trend across all education levels ($P < 0.001$) for each covariates except not having use of a regularMDor clinic; and at $P < 0.05$ across all income levels, except with separated/widowed/divorced, overweight, moderate activity, and current smoker.

[b]Chi-square tests for all covariates except moderate activity were significant across each occupation group at $P < 0.001$.

Table 3

Association (relative hazard) between 34-year incidence of Type 2 diabetes and education, log income, and blue-collar occupation in the ACS (1965–99): all covariates measured at baseline

	Education (vs >12 years)						Blue-collar occupation (vs white-collar)			
	<12 years		12 years		Log income (US dollars)		Men		Women	
Model	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI
1	1.89	1.44–2.49	1.47	1.12–1.94	0.76	0.65–0.89	1.42	1.01–2.00	1.55	0.99–2.42
2	1.90	1.43–2.54	1.48	1.12–1.95	0.75	0.64–0.87	1.42	1.01–2.00	1.55	0.99–2.43
3	1.51	1.11–2.04	1.38	1.04–1.82	0.87	0.74–1.02	1.30	0.91–1.85	1.03	0.65–1.66
4	1.42	1.04–1.92	1.32	0.99–1.75	0.88	0.75–1.04	1.27	0.89–1.83	1.00	0.62–1.61
5	1.24	0.91–1.69	1.29	0.97–1.72	0.92	0.78–1.08	1.17	0.81–1.69	0.85	0.52–1.38
6	1.27	0.93–1.74	1.31	0.99–1.74	0.90	0.76–1.06	1.19	0.83–1.72	0.86	0.53–1.41

Model 1 is unadjusted; Model 2 is adjusted for age; Model 3 is adjusted for demographics [age, gender, racial/ethnic group (white/non-white), and marital status (single, married, widowed-separated-divorced)]; Model 4 is adjusted for demographics, and behaviours (physical activity, smoking status, alcohol consumption); Model 5 is adjusted for demographics, behaviours, and body composition (BMI group,waist circumference); and Model 6 is adjusted for demographics, behaviours, body composition, high blood pressure, depression, health insurance, and regular access to a medical doctor or clinic.

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Table 4

Association (relative hazard) between 34-year incidence of Type 2 diabetes and education, log income, and blue-collar occupation in the ACS (1965–99): all covariates are time-dependent except age and waist circumference

	Education (vs >12 years)						Blue-collar occupation (vs white-collar)			
	<12 years		12 years		Log income (US dollars)		Men		Women	
Model	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI
1	1.72	1.31–2.25	1.29	0.98–1.69	0.82	0.71–0.93	1.63	1.16–2.31	1.53	1.01–2.32
2	1.72	1.30–2.30	1.29	0.98–1.69	0.82	0.71–0.94	1.59	1.12–2.25	1.52	1.01–2.31
3	1.37	1.02–1.85	1.22	0.93–1.60	0.91	0.79–1.05	1.47	1.03–2.11	1.10	0.71–1.70
4	1.23	0.91–1.67	1.15	0.87–1.51	0.94	0.82–1.09	1.45	1.01–2.09	1.04	0.67–1.61
5	1.04	0.76–1.42	1.09	0.83–1.44	1.00	0.87–1.15	1.29	0.90–1.86	0.86	0.55–1.35
6	1.08	0.79–1.47	1.11	0.84–1.46	0.99	0.86–1.15	1.31	0.91–1.89	0.87	0.56–1.35

Model 1 is unadjusted; Model 2 is adjusted for age at baseline; Model 3 is adjusted for demographics [age at baseline, gender, racial/ethnic group (white/nonwhite), and marital status (single, married, widowed-separated-divorced)]; Model 4 is adjusted for demographics, and behaviours (physical activity, smoking status, alcohol consumption); Model 5 is adjusted for demographics, behaviours, and body composition (BMI group, waist circumference at baseline); and Model 6 is adjusted for demographics, behaviours, body composition, high blood pressure, depression, health insurance, and regular access to a medical doctor or clinic.