Associations Between Socioeconomic Status and Major Complications in Type 1 Diabetes: The Pittsburgh Epidemiology of Diabetes Complication (EDC) Study

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PURPOSE: To understand the effect of socioeconomic status (SES) on the risk of complications in type 1 diabetes (T1D), we explored the relationship between SES and major diabetes complications in a prospective, observational T1D cohort study.

METHODS: Complete data were available for 317 T1D persons within 4 years of age 28 (ages 24–32) in the Pittsburgh Epidemiology of Diabetes Complications Study. Age 28 was selected to maximize income, education, and occupation potential and to minimize the effect of advanced diabetes complications on SES.

RESULTS: The incidences over 1 to 20 years’ follow-up of end-stage renal disease and coronary artery disease were two to three times greater for T1D individuals without, compared with those with a college degree (p < .05 for both), whereas the incidence of autonomic neuropathy was significantly greater for low-income and/or nonprofessional participants (p < .05 for both). HbA1c was inversely associated only with income level. In sex- and diabetes duration-adjusted Cox models, lower education predicted end-stage renal disease (hazard ratio [HR], 2.9; 95% confidence interval [95% CI], 1.1–7.7) and coronary artery disease (HR, 2.5, 95% CI, 1.3–4.9), whereas lower income predicted autonomic neuropathy (HR, 1.7; 95% CI, 1.0–2.9) and lower-extremity arterial disease (HR, 3.7; 95% CI, 1.1–11.9).

CONCLUSIONS: These associations, partially mediated by clinical risk factors, suggest that lower SES T1D individuals may have poorer self-management and, thus, greater complications from diabetes.

Key Words: Autonomic Neuropathy, Coronary Artery Disease, Diabetes Complications, End-Stage Renal Disease, Prospective Study, Socioeconomic Status, Type 1 Diabetes.

INTRODUCTION

Socioeconomic status (SES) is inversely associated with many chronic diseases in the general population, with disadvantaged individuals faring worse than others (1–3), and this health inequality is becoming more pronounced over time (4). For diabetes, however, researchers evaluating the relationship between SES and diabetes complications have produced varied results.

Studies in type 1 diabetes (T1D) either show an increased rate of complications in lower SES groups (5) or no effect of SES (6, 7). In particular, the extent to which SES correlates with the vascular complications of T1D, such as nephropathy and coronary artery disease (CAD), is unclear. At present, the best-available evidence suggests that lower levels of education attained are associated with heart disease, particularly in T1D women (8).

Another concern is that studies vary widely in their design (cross-sectional vs prospective) and in their definitions of diabetes and SES measures. Cross-sectional evaluation of SES associations makes it difficult to evaluate whether SES causes, or results from, advanced complications from diabetes (5, 8–10). Also, when some authors have examined the independent contribution of different SES measures, some SES measures appear to be more predictive than others depending on the outcome of interest (11). We therefore sought to evaluate the relationship(s) between three SES measures (household income, education, and
occupation) and incident diabetes complications in a cohort of childhood-onset T1D.

MATERIALS AND METHODS

Study Population

The Pittsburgh Epidemiology of Diabetes Complications (EDC) Study is a prospective study of risk factors for complications resulting from childhood-onset (age <17 years) T1D. Participants (n = 658) were either diagnosed or seen within 1 year of diagnosis at Children’s Hospital of Pittsburgh between 1950 and 1980 and placed on continuous insulin therapy at diagnosis. Since initial examination in 1986 to 1988, participants have been followed biennially by survey and by examination for the first 10 years and again at 18 years. The follow-up time for this analysis ranged from 1.0 to 19.8 years. Study protocols were approved by the University of Pittsburgh Institutional Review Board.

SES Variables

EDC study variables for SES include occupation, education level, and household income. For this analysis, each SES measure was evaluated in patients who were 28 years of age to allow for maximal representation of educational attainment, along with a reasonable establishment of relative occupational and financial standing in each participant. In addition, this age was chosen to minimize the effect of advanced diabetes complications on education, income potential, and occupation status. To obtain an “Age 28” cohort, clinical and survey data were collected from the EDC study cycle at which participants were closest to age 28, with the age range limited to ±4 years (ages 24–32). This “Age 28” data was then considered as baseline for subsequent incidence analyses. Complete clinical and SES data were available for 317 participants. The majority (78%) of those excluded from this analysis were either too old (age ≥32) at baseline examination in 1986–1988 (60%) or too young (age <24) before last follow-up (18%). The remaining (22%) EDC participants were excluded because of missing SES data.

Occupation was defined on the basis of self-reported work title and categorized according to the Hollingshead Index of social position (12). Education was defined as the self-reported highest educational level categorized as follows: some high school, high school graduate, some college, college graduate, and education beyond college graduation. All study participants had at least some high school education. Household income was self-reported by the use of categories of annual pre-tax income (in U.S. dollars) earned by each household. For this study, these categorical income measures were grouped into one of five income categories based on their Age 28 study cycle. For EDC study cycles 1–3 (1986–1992), annual household income categories were as follows: 1 = ≤$10,000, 2 = $10,001–20,000, 3 = $20,001–30,000, 4 = $30,001–40,000, and 5 = >$40,000. For EDC study cycles 4–10 (1992–2006), income categories were: 1 = ≤$20,000, 2 = $20,001–30,000, 3 = $30,001–40,000, 4 = $40,001–50,000, and 5 = >$50,000.

Complication Measures

Diabetes complications assessed in this study included coronary artery disease (CAD), end-stage renal disease (ESRD), proliferative retinopathy, lower extremity arterial disease (LEAD), and autonomic neuropathy (AN). CAD was defined as EDC physician-diagnosed angina, ischemic electrocardiographic changes, fatal or nonfatal myocardial infarction confirmed by either Q waves on electrocardiogram or hospital records, angiographic stenosis (>50% blockage), coronary artery bypass surgery, angioplasty, or CAD death.

Creatinine was assayed by the use of an Ectachem 400 Analyzer (Eastman Kodak, Rochester, NY), and serum and urinary albumin were measured by immunonephelometry (13). Albumin excretion rates were calculated by the use of urinary albumin levels from at least two validated timed sample collections. Degree of renal disease was categorized as normal (albumin excretion rates <20 µg/min), microalbuminuria (20–200 µg/min), or overt nephropathy (>200 µg/min). ESRD was defined as renal failure or transplantation.

Proliferative retinopathy was classified by the University of Wisconsin-Madison Fundus Photography Reading Center on the basis of the modified Arlie House system using stereoscopic fundus photographs (14). For participants refusing fundus photographs (n = 43), proliferative retinopathy was defined as receiving laser phototherapy for proliferative retinopathy.

Resting ankle-brachial systolic blood pressures were taken via a Doppler blood-flow detector with the patient in the supine position. The right and left tibialis posterior and dorsalis pedis pressures were compared with the arm pressure, and ankle-to-brachial index was calculated with the arm pressure measurement taken closest in time to the
ankle pressure. An ankle-to-brachial index < 0.9 on either side at rest was considered evidence of LEAD. In addition, LEAD also included a history of claudication as determined by the ROSE questionnaire (15) or self-reported history of amputation for a vascular cause. AN was defined as an R-R interval expiration-inspiration ratio < 1.1.

Clinical Measures
Height and weight were measured to determine body mass index. Blood pressure was measured with a random-zero sphygmomanometer after a 5-min rest according to the Hypertension Detection and Follow-up Program protocol (16). Hypertension was defined as systolic blood pressure ≥ 140 mm Hg, diastolic blood pressure ≥ 90 mm Hg, or antihypertensive medication use. An ever smoker was defined as having smoked ≥ 100 cigarettes in their lifetime. Depressive symptoms were assessed with the Beck Depression Inventory (17).

Fasting blood samples were taken to measure glycosylated hemoglobin and lipids. For analysis purposes, original HbA1c (EDC HbA1c) values were converted to Diabetes Complications and Control Trial-aligned HbA1c values by use of the following regression equation derived from duplicate assays: Diabetes Complications and Control Trial HbA1c = 0.14 + 0.83 (EDC HbA1c). Total cholesterol and triglycerides were measured enzymatically, and high-density lipoprotein (HDL) cholesterol was assessed after a heparin and manganese chloride precipitation method (18). Non-HDL cholesterol was calculated as the difference between total cholesterol and HDL cholesterol. Intensive insulin therapy was defined as either ≥ 3 daily insulin injections or use of an insulin pump.

Statistical Analysis
Individuals in the lowest occupation category (8X–9X) included full-time students, homemakers, retired persons, and those who were disabled. Full-time students at the Age 28 study cycle were reclassified to their occupation from the next study cycle (n = 6). Similarly, when available, homemakers at the Age 28 study cycle were reclassified as their occupation from the study cycle immediately before or after if they worked at those time-points (n = 25). No one listed their occupation as "retired" in their Age 28 study cycle. Disabled individuals (n = 2) were excluded because the disability resulted from advanced diabetes complications. Missing education (n = 10) or income (n = 17) data from the Age 28 study cycle were imputed only if the respective variables were the same in the study cycles before and after the Age 28 cycle.

All three SES variables were dichotomized for analysis. Occupation was classified as either professional (Hollingshead 1A–3C) or nonprofessional (Hollingshead 4A–7X), education as those with or without a college degree, and income was classified in two ways: 1) lowest income category versus other four categories, and 2) highest income category versus other four categories.

Associations between dichotomous SES variables and complications were analyzed by the use of the χ² or Fisher’s exact tests, as appropriate, adjusting for multiple comparisons using the Bonferroni correction. The Student’s t test (or Mann-Whitney U) was used to compare continuous variables by SES group. Spearman’s correlations were performed between each SES measure. Multivariable Cox regression analysis was used to assess the association between SES measures and time to complication development (excluding individuals with the specific complication at "age 28" baseline) to adjust for sex, diabetes duration, and univariately significant clinical measures for each complication. Because age and duration of diabetes are highly correlated in this cohort, and age was selected to be within a narrow range (24–32 years), only duration of diabetes was included in multivariable models. The proportional hazards assumption was assessed visually and verified by testing time-dependent interaction variables. A p-value < .05 was considered statistically significant. Analyses were completed with either SPSS 17.0 (SPSS, Chicago, IL) or SAS 9.2 (SAS Institute, Cary, NC).

RESULTS
Demographic and clinical characteristics stratified by socioeconomic measures for the EDC “Age 28” study population are shown in Table 1. Overall, the mean age (± SD) and diabetes duration in this cohort were 28.4 (± 1.6) and 20.1 (± 4.4) years, respectively. Age, diabetes duration, sex, and race were similar across SES groups. Married individuals tended to have a higher household income or a professional occupation. Clinically, HbA1c decreased with increasing income level (p = .01). Blood pressure did not differ by any SES groupings; however, the proportion with hypertension decreased as income increased (p = .02). Lower non-HDL cholesterol was associated with both a college degree and a professional occupation (p < .05 for both). BMI was not associated with any SES measure. Daily insulin dose at baseline was significantly lower for all high SES measures, and those with more education or better employment were also significantly more likely to be on intensive insulin therapy by age 28. Albumin excretion rates were significantly lower for all high SES measures.

The prevalences of diabetes-related complications by age 28 are also shown in Table 1. Presence of overt nephropathy did not differ by SES status. CAD and LEAD presence at age 28 was very low and did not differ by SES status, nor did presence of proliferative retinopathy (PR) or AN. Depression was more common in the lowest income group and in individuals without a college degree (p < .05 for both).

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### TABLE 1. Baseline characteristics (mean [SD] or % [n]) by SES category for the Age-28 cohort of the Pittsburgh EDC study population (n = 317)

<table>
<thead>
<tr>
<th>Income</th>
<th>Lowest</th>
<th>Middle</th>
<th>Highest</th>
<th>Education</th>
<th>&lt;College grad</th>
<th>College grad</th>
<th>Occupation</th>
<th>Nonprofessional</th>
<th>Professional</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age, yrs</td>
<td>28.2 (1.7)</td>
<td>28.5 (1.5)</td>
<td>28.4 (1.6)</td>
<td>28.3 (1.4)</td>
<td>28.4 (1.6)</td>
<td>28.4 (1.5)</td>
<td>28.4 (1.5)</td>
<td>28.4 (1.5)</td>
<td>28.4 (1.5)</td>
</tr>
<tr>
<td>Mean T1D duration, yrs</td>
<td>19.6 (4.2)</td>
<td>20.3 (4.4)</td>
<td>19.7 (4.4)</td>
<td>19.9 (4.6)</td>
<td>20.1 (4.3)</td>
<td>20.1 (4.5)</td>
<td>43.8 (77)</td>
<td>54.6 (77)</td>
<td>54.6 (77)</td>
</tr>
<tr>
<td>Female</td>
<td>54.2 (32)</td>
<td>47.2 (94)</td>
<td>47.5 (28)</td>
<td>47.2 (94)</td>
<td>47.2 (94)</td>
<td>50.8 (60)</td>
<td>43.8 (77)</td>
<td>54.6 (77)</td>
<td>54.6 (77)</td>
</tr>
<tr>
<td>Caucasian</td>
<td>96.6 (57)</td>
<td>99.5 (198)</td>
<td>98.3 (58)</td>
<td>99.0 (197)</td>
<td>98.3 (116)</td>
<td>98.9 (174)</td>
<td>98.6 (139)</td>
<td>98.6 (139)</td>
<td>98.6 (139)</td>
</tr>
<tr>
<td>Married</td>
<td>29.3 (117)</td>
<td>51.3 (102)</td>
<td>66.1 (39)*</td>
<td>49.2 (100)</td>
<td>50.5 (58)</td>
<td>42.9 (75)</td>
<td>58.9 (83)</td>
<td>58.9 (83)</td>
<td>58.9 (83)</td>
</tr>
</tbody>
</table>

**Clinical variables**

- **HbA1c (%)**
  - Lowest: 9.16 (1.60) vs 8.80 (1.60)*
  - Middle: 8.76 (1.52) vs 8.60 (1.39)
  - Highest: 8.47 (1.39) vs 8.87 (1.57)

- **Systolic BP, mmHg**
  - Lowest: 114.9 (14.8) vs 112.3 (14.3)
  - Middle: 113.6 (14.6) vs 112.3 (14.3)
  - Highest: 111.1 (13.8) vs 112.3 (14.3)

- **Diastolic BP, mmHg**
  - Lowest: 74.8 (10.5) vs 71.9 (10.2)
  - Middle: 72.4 (10.4) vs 71.9 (10.2)
  - Highest: 72.5 (9.9) vs 71.9 (10.2)

- **Hypertension, %**
  - Lowest: 20.3 (12) vs 16.1 (32)
  - Middle: 14.1 (28) vs 10.2 (12)
  - Highest: 6.8 (4) vs 4.2 (2)

- **BMI, kg/m²**
  - Lowest: 24.1 (3.3) vs 24.1 (3.2)
  - Middle: 24.3 (3.1) vs 24.1 (3.2)
  - Highest: 25.1 (3.4) vs 24.1 (3.2)

- **Insulin dose, U kg⁻¹ day⁻¹**
  - Lowest: 0.76 (0.21) vs 0.71 (0.22)*
  - Middle: 0.75 (0.22) vs 0.71 (0.22)*
  - Highest: 0.68 (0.18)* vs 0.71 (0.22)*

- **Intensive insulin therapy, %**
  - Lowest: 15.5 (9) vs 10.7 (9)
  - Middle: 15.5 (30) vs 10.7 (9)
  - Highest: 22.4 (13) vs 10.7 (9)

**Complication variables**

- **Prevalent ON**
  - Lowest: 30.5 (18) vs 26.3 (52)
  - Middle: 22.7 (45) vs 18.6 (51)
  - Highest: 18.6 (11) vs 18.6 (51)

- **Prevalent CAD**
  - Lowest: 3.4 (2) vs 2.5 (5)
  - Middle: 4.5 (9) vs 6.8 (8)
  - Highest: 3.2 (19) vs 6.8 (8)

- **Prevalent LEAD**
  - Lowest: 10.2 (66) vs 7.5 (15)
  - Middle: 6.3 (13) vs 5.9 (7)
  - Highest: 5.1 (3) vs 5.9 (7)

- **Prevalent retinopathy**
  - Lowest: 28.8 (17) vs 26.6 (53)
  - Middle: 27.3 (54) vs 31.6 (57)
  - Highest: 32.2 (19) vs 31.6 (57)

- **Prevalent AN**
  - Lowest: 29.1 (16) vs 16.8 (32)
  - Middle: 9.8 (19) vs 7.8 (9)
  - Highest: 10.3 (61)* vs 7.8 (9)

- **Ever smoke**
  - Lowest: 43.1 (25) vs 43.4 (66)
  - Middle: 48.2 (81) vs 22.9 (27)*
  - Highest: 11.9 (7) vs 44.6 (78)*

**Values are % (n) unless otherwise indicated.**

*Baseline data comes from the study cycle closest to age 28 (± 4 yrs).

**‡** Data shown as median (IQR).

†Baseline data comes from the study cycle closest to age 28 (± 4 yrs).

*‡ ≥ 0.05 for trend across income groups.

*‡ p < 0.05 based on chi² or analysis of variance, as appropriate.

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Smoking was much less common in all three high SES categories (p < .01 for all).

All three SES measures (both dichotomous and ordinal) were highly correlated with each other (all correlations p ≤ .003). Education and occupation were the most correlated (r = .54, p < .001), and education and income were the least correlated (r = .17, p = .003).

The median follow-up time for determining the incidence of each complication after age 28 varied from 8.5 to 12.3 years (range, 1.0–19.8 years for all complications). Education at age 28 was the only significant SES measure associated with developing either ESRD (p = .01) or CAD (p = .002; Table 2). Interestingly, LEAD was associated with income at age 28 (p = .04), but not with education or occupation (p = .23 and .55, respectively; Table 2). Both low-income level and nonprofessional occupation were significantly associated with AN (p = .02 and .03, respectively; Table 2). Although AN occurred more frequently in those with lower education, this finding was not significant (p = .07). None of the three SES measures was associated with developing PR in this cohort (Table 2).

Unadjusted and adjusted Cox proportional hazards model results are shown in Table 3 for incident ESRD, CAD, LEAD, and AN, because these complications were all significantly associated with at least one SES measure. T1D individuals without a college degree by age 28 were three times as likely to develop ESRD and more than twice as likely to develop CAD as those with a college degree, even after adjusting for sex and diabetes duration (hazard ratio [HR], 2.9, 95% confidence interval [95% CI], 1.1–7.7 and HR, 2.5; 95% CI, 1.3–4.9, respectively). However, these associations with education were partially mediated by other significant clinical variables and became nonsignificant for ESRD (Table 3, Model 2).

LEAD was only associated with low income in both unadjusted and in sex- and diabetes duration-adjusted Cox models; however, unlike the other complications, the association between income and LEAD persisted even after adjusting for all other significant clinical factors (Table 3, Model 2). Adjusting for sex and diabetes duration, low-income T1D individuals were also nearly two times more likely to develop AN compared with those with high income in the Age-28 cohort (HR, 1.7; 95% CI, 1.0–2.9), but this association was largely explained by adjusting for other significant clinical measures (Table 3, Model 2). Neither education nor occupation at age 28 was related to AN. PR was not associated with any SES measures in unadjusted and adjusted Cox modeling (data not shown).

**DISCUSSION**

Our data indicate that SES is a robust predictor for diabetes complications in a large cohort of childhood-onset T1D.
Education levels at baseline predicted both incident ESRD and incident CAD. The relationship with incident ESRD became attenuated for education (HR, 2.9 reduced to 2.1) after adjusting for other key potential mediators, whereas income was unaffected by such adjustment (HR, 3.3–3.8). However, income at age 28, not education, was predictive of both incident LEAD and incident AN. Adjustment for clinical risk factors for each major complication considerably attenuated the strength of these SES associations, except for that between low income and LEAD. PR was not associated with any of the SES measures.

The use of age 28 to determine SES was reported recently in the general population (Framingham Offspring Study) (19); however, this strategy is even more important in a population with childhood-onset T1D with an average diabetes duration of 20 years by age 28. Furthermore, examining multiple SES measures and multiple outcomes concurrently in a large T1D population allowed for a robust assessment of the effect of specific SES measures on specific outcomes in this population.

These results largely confirm previous findings in T1D (8, 20, 21). The authors of several studies now have shown that various SES measures are associated with poor glycemic control, even in young (age <18 years) T1D individuals (22–24). Chronic hyperglycemia is highly predictive of major diabetes complications and early mortality (25). Similarly, both low social class (determined by residence) and low education are associated with multiple cardiovascular risk factors in T1D, namely, hypertension, dyslipidemia, and smoking status (6, 26, 27).

With 10 years of follow-up, Muhlhauser et al. (21) found that low social status (a composite of baseline education and occupation levels) was significantly predictive of a composite measure of major complications (blindness, amputation, and renal replacement therapy), even after they adjusted for other known risk factors, such as HbA1c, smoking, blood pressure, cholesterol, and presence of overt nephropathy and retinopathy. This discovery suggests that although our SES associations with diabetes complications were often explained by other known risk factors, these SES associations may persist with increased incidence or longer follow-up, as seen in the persistent association between income and both ESRD and LEAD, despite adjusting for known risk factors. However, access to care varies dramatically between Germany and the United States, and the attenuation to non-significance of SES measures and incident CAD, ESRD, and AN might be real. In other words, low SES in the United States might merely reflect poor self-care, possibly attributable to inadequate access to diabetes education or inability to afford good self-care (i.e., testing costs) and not be truly predictive of future diabetes complications.

The reasons for the different SES associations with different diabetes complications are unclear. The primary role of education in predicting major cardiovascular and renal events makes logical sense, as T1D requires a large amount of self-care, and diabetes education clearly improves glycemic control (23). However, we cannot fully explain why income is the primary SES measure associated with AN and LEAD because these are also more prevalent with poor glycemic control. Likewise, PR was not associated

**TABLE 3.** Unadjusted and adjusted hazards ratios for major incident diabetes complications by SES measure using Cox proportional hazards models

<table>
<thead>
<tr>
<th>SES measure</th>
<th>Unadjusted HR</th>
<th>95% CI</th>
<th>p</th>
<th>AIC</th>
<th>Model 1* HR</th>
<th>95% CI</th>
<th>p</th>
<th>AIC</th>
<th>Model 2† HR</th>
<th>95% CI</th>
<th>p</th>
<th>AIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incident end-stage renal disease</td>
<td></td>
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<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Low income</td>
<td>3.3</td>
<td>0.8, 13.9</td>
<td>.10</td>
<td>313.0</td>
<td>3.1</td>
<td>0.7, 13.2</td>
<td>.12</td>
<td>313.9</td>
<td>3.8</td>
<td>0.8, 17.6</td>
<td>.09</td>
<td>233.7</td>
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<tr>
<td>&lt;College graduate</td>
<td>2.9</td>
<td>1.1, 7.7</td>
<td>.03</td>
<td>311.0</td>
<td>2.9</td>
<td>1.1, 7.7</td>
<td>.03</td>
<td>311.4</td>
<td>2.1</td>
<td>0.8, 5.9</td>
<td>.14</td>
<td>234.9</td>
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<tr>
<td>Nonprofessional</td>
<td>1.5</td>
<td>0.7, 3.2</td>
<td>.30</td>
<td>315.7</td>
<td>1.5</td>
<td>0.7, 3.2</td>
<td>.31</td>
<td>316.2</td>
<td>1.4</td>
<td>0.6, 3.3</td>
<td>.46</td>
<td>236.8</td>
</tr>
<tr>
<td>Incident coronary artery disease</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Low income</td>
<td>1.7</td>
<td>0.8, 3.8</td>
<td>.19</td>
<td>543.4</td>
<td>1.8</td>
<td>0.8, 3.9</td>
<td>.17</td>
<td>535.2</td>
<td>1.6</td>
<td>0.6, 4.1</td>
<td>.37</td>
<td>463.0</td>
</tr>
<tr>
<td>&lt;College graduate</td>
<td>2.4</td>
<td>1.2, 4.7</td>
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<td>538.4</td>
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AIC = Akaike’s information criterion; CI = confidence interval; HR = hazard ratio; SES = socioeconomic status.

*Model 1: Adjusted for sex and diabetes’ duration.
†Model 2: Adjusted for Model 1 variables and HbA1c, non-HDL cholesterol, hypertension status, prevalent overt nephropathy, and smoking status.
with any SES measure, perhaps because of the high overall prevalence of PR in this cohort (> 60%).

The Pittsburgh EDC study has several strengths for evaluating associations between various SES measures and diabetes complications. Specifically, the prospective design with biennial follow-up allowed us to obtain three different socioeconomic measures at or around age 28 for this cohort. This prospective nature also allowed for a median 8–12 years (depending on complication) of follow-up after age 28, longer than other SES studies in T1D (20, 21, 28). Access to concurrent demographic, clinical, and complication data for each individual at age 28 permitted adjustment for known risk factors to minimize confounding.

This study, however, also has limitations. The sample size was small for both CAD and ESRD incidences. Thus, it is possible our data failed to reveal the significance of an SES measure that indeed does contribute to incident CAD or ESRD in T1D. Although income or occupation level might have been significant with a larger sample size, the relative importance of these measures with education is unlikely to change. Also, this EDC cohort consists of individuals with long-standing diabetes (mean diabetes duration = 20 years). Therefore, these SES associations with complications might, in part, result from outdated diabetes education and care practices, and may not be generalizable to individuals recently diagnosed with T1D. In addition, whether our participants had health insurance was not determined in the first two EDC study cycles (1986–1990, n = 159). Thus, we cannot be sure that those who later developed diabetes complications in this cohort were more likely to be uninsured at their age 28 visit. However, by the third study cycle (1990–1992), > 90% of our cohort had health insurance, increasing to 95% by the 1994–1996 study cycle. Thus, it is unlikely that health insurance coverage was a major confounder in this population.

In conclusion, SES in early adulthood predicts future diabetes complications in T1D, after adjustment for sex and diabetes duration. However, much of these relationships can be explained by known risk factors for complications (i.e., metabolic control, smoking status, hypertension, dyslipidemia, or presence of subclinical renal damage), suggesting that individuals with low SES and T1D are more likely to have poorer management of their diabetes. These findings indicate that special efforts must be made to ensure that T1D individuals with low SES receive adequate diabetes education and follow-up to reduce their risk of complications.

REFERENCES


