

Published in final edited form as:

Ann Epidemiol. 2011 May ; 21(5): 374–381. doi:10.1016/j.annepidem.2011.02.007.

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

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Abstract

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 minimize the effect of advanced diabetes complications on SES.

Results—The incidences over 1–20 years follow-up of end-stage renal disease (ESRD) and coronary artery disease (CAD) were 2–3 times higher for T1D individuals without, compared to those with a college degree ($p < 0.05$ for both), while autonomic neuropathy (AN) incidence was significantly higher for low income and/or non-professional participants ($p < 0.05$ for both). HbA_{1c} was inversely associated only with income level. In sex- and diabetes duration-adjusted Cox models, lower education predicted ESRD (HR=2.9, 95% CI, 1.1–7.7) and CAD (HR=2.5, 1.3–4.9), whereas lower income predicted AN (HR=1.7, 1.0–2.9) and lower extremity arterial disease (HR=3.7, 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, more diabetes complications.

Keywords

autonomic neuropathy; coronary artery disease; diabetes complications; end-stage renal disease; prospective study; socioeconomic status; type 1 diabetes

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

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health inequality is becoming more pronounced over time.(4) In diabetes, however, studies evaluating the relationship between SES and diabetes complications have produced varied results.

Studies in type 1 diabetes 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 type 1 diabetes (T1D), such as nephropathy and coronary artery disease (CAD), is unclear. At present, the best available evidence suggests that lower education is associated with heart disease, particularly in T1D women.(8)

Another concern is that studies vary widely in their design (cross-sectional vs. prospective) and 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 diabetes complications.(5,8–10) Also, when studies examine 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 type 1 diabetes.

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) type 1 diabetes. 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 1986–1988, 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 1.0–19.8 years. Study protocols were approved by the University of Pittsburgh Institutional Review Board.

Socioeconomic status (SES) variables

EDC study variables for SES include occupation, education level, and household income. For this analysis, each SES measure was evaluated at age 28 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 (60%) of those excluded from this analysis were too old (age>32) at initial examination in 1986–1988. The majority (78%) of those excluded from this analysis were either too old (age>32) at baseline examination in 1986–88 (60%) or too young (age<24) prior to last follow-up (18%). The remaining (22%) EDC participants were excluded due to 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 using categories of annual pre-tax income (in US 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= \leq \$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= \leq \$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 electrocardiogram (ECG) changes, fatal or nonfatal myocardial infarction confirmed by either Q waves on ECG or hospital records, angiographic stenosis (\geq 50% blockage), coronary artery bypass surgery, angioplasty, or CAD death.

Creatinine was assayed using an Ectachem 400 Analyzer (Eastman Kodak, Rochester, NY), and serum and urinary albumin were measured by immunonephelometry.⁽¹³⁾ Albumin excretion rates (AER) were calculated using urinary albumin levels from at least 2 validated timed sample collections. Degree of renal disease was categorized as normal (AER $<$ 20 μ g/min), microalbuminuria (20–200 μ g/min), or overt nephropathy (ON, $>$ 200 μ g/min). ESRD was defined as renal failure or transplantation.

Proliferative retinopathy was classified by the University of Wisconsin-Madison Fundus Photography Reading Center based on 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 in the supine position with a Doppler blood-flow detector. The right and left tibialis posterior and dorsalis pedis pressures were compared with the arm pressure, and ankle-to-brachial index (ABI) was calculated using the arm pressure measurement taken closest in time to the ankle pressure. ABI $<$ 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. Autonomic neuropathy (AN) was defined as an R-R interval expiration-inspiration (E/I) 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 \geq 140 mmHg, diastolic blood pressure \geq 90 mmHg, or antihypertensive medication use. An ever smoker was defined as having smoked \geq 100 cigarettes in their lifetime. Depressive symptoms were assessed using the Beck Depression Inventory.⁽¹⁷⁾

Fasting blood samples were taken to measure glycosylated hemoglobin and lipids. For analysis purposes, original HbA₁ values were converted to Diabetes Complications and Control Trial (DCCT)-aligned HbA_{1c} values using the following regression equation derived from duplicate assays: DCCT HbA_{1c} = 0.14 + 0.83(EDC HbA₁). Total cholesterol and triglycerides were measured enzymatically, and 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, as 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 non-professional (Hollingshead 4A-7X), education as those with or without a college degree, and income was classified in two ways: 1) lowest income category vs. other 4 categories, and 2) highest income category vs. other 4 categories.

Associations between dichotomous SES variables and complications were analyzed using the χ^2 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. Since age and diabetes duration are highly correlated in this cohort, and age was selected to be within a narrow range (24–32 years), only diabetes duration was included multivariable models. The proportional hazards assumption was assessed visually and verified by testing time-dependent interaction variables. A p -value ≤ 0.05 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 (\pm SD) and diabetes duration in this cohort were 28.4 (\pm 1.6) and 20.1 (\pm 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, HbA_{1c} decreased with increasing income level ($p=0.01$). Blood pressure did not differ by any SES groupings; however, the proportion with hypertension decreased as income increased ($p=0.02$). Lower non-HDL cholesterol was associated with both a college degree and a professional occupation ($p<0.05$ for both). BMI was not associated with any SES measure. Daily insulin dose at baseline was significantly lower for all three 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 three 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 autonomic neuropathy (AN). Depression was more common in the lowest income group and in individuals without a college degree ($p < 0.05$ for both). Smoking was much less common in all three high SES categories ($p < 0.01$ for all).

All three SES measures (both dichotomous and ordinal) were highly correlated with each other (all correlations $p \leq 0.003$). Education and occupation were the most correlated ($r = 0.54$, $p < 0.001$), and education and income were the least correlated ($r = 0.17$, $p = 0.003$).

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 ($P = 0.01$) or CAD ($p = 0.002$) (Table 2). Interestingly, LEAD was only associated with income at age 28 ($p = 0.04$), but not with education or occupation ($p = 0.23$ and 0.55 , respectively) (Table 2). Both low income level and non-professional occupation were significantly associated with AN ($p = 0.02$ and 0.03 , respectively) (Table 2). Although AN occurred more frequently in those with lower education, this was not significant ($p = 0.07$). 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, as these complications were all significantly associated with at least one SES measure. Type 1 diabetic 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 (HR=2.9, 95% CI 1.1–7.7, and HR=2.5, 1.3–4.9, respectively). However, these associations with education were partially mediated by other significant clinical variables and became non-significant 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 to 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 type 1 diabetes. 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, while income was unaffected by such adjustment (HR=3.3 to 3.8). However, income at age 28, not education, was predictive of both incident LEAD and incident AN. Adjusting 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.

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 type 1 diabetes with an average diabetes duration of 20 years by age 28. Furthermore, examining multiple SES measures and multiple outcomes concurrently in a large type 1 diabetes 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 type 1 diabetes.(8,20,21) Multiple studies now have shown that various SES measures are associated with poor glycemic control, even in young (age<18 years) type 1 diabetic 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 type 1 diabetes, namely, hypertension, dyslipidemia, and smoking status.(6,26,27)

With 10 years of follow-up, Muhlhauser et al 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 adjusting for other known risk factors, such as HbA_{1c}, smoking, blood pressure, cholesterol, and presence of overt nephropathy and retinopathy.(21) This suggests that while 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 due 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 type 1 diabetes 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, as these are also more prevalent with poor glycemic control. Likewise, PR was not associated with any SES measure, perhaps due to 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 type 1 diabetes. (20,21,28) Access to concurrent demographic, clinical, and complication data for each individual at age 28 permitted adjustment for known risk factors and 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 type 1 diabetes. While 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 type 1 diabetes. 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, socioeconomic status in early adulthood predicts future diabetes complications in type 1 diabetes, 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, and/or presence of subclinical renal damage), suggesting that individuals with low SES and type 1 diabetes 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.

Acknowledgments

This work was supported by the National Institute of Diabetes and Digestive and Kidney Diseases at the National Institutes of Health (grant numbers R01-DK034818 and F30-DK082137 to A.M.S.).

Abbreviations

AER	albumin excretion rate
CAD	coronary artery disease
DBP	diastolic blood pressure
EDC	Epidemiology of Diabetes Complications
ESRD	end-stage renal disease
IIT	intensive insulin therapy
LEAD	lower extremity artery disease
HDL-c	high density lipoprotein cholesterol
ON	overt nephropathy
SBP	systolic blood pressure

REFERENCES

1. Blaxter M. Evidence on inequality in health from a national survey. *Lancet*. Jul 4; 1987 2(8549):30–3. [PubMed: 2885514]
2. Gonzalez MA, Rodriguez Artalejo F, Calero JR. Relationship between socioeconomic status and ischaemic heart disease in cohort and case-control studies: 1960–1993. *Int J Epidemiol*. Jun; 1998 27(3):350–8. [PubMed: 9698119]
3. Kaplan GA, Keil JE. Socioeconomic factors and cardiovascular disease: a review of the literature. *Circulation*. Oct; 1993 88(4 Pt 1):1973–98. [PubMed: 8403348]
4. Feldman JJ, Makuc DM, Kleinman JC, Cornoni-Huntley J. National trends in educational differentials in mortality. *Am J Epidemiol*. May; 1989 129(5):919–33. [PubMed: 2705434]
5. Lievre M, Marre M, Robert JJ, Charpentier G, Iannascoli F, Passa P. Cross-sectional study of care, socio-economic status and complications in young French patients with type 1 diabetes mellitus. *Diabetes Metab*. Feb; 2005 31(1):41–6. [PubMed: 15803112]

6. Robinson N, Edouard L, Diehl A, Fuller JH. Social class and risk factors for vascular disease in diabetes. *Diabete Metab.* Oct; 1984 10(4):245–9. [PubMed: 6391975]
7. Rossing P, Hougaard P, Parving HH. Risk factors for development of incipient and overt diabetic nephropathy in type 1 diabetic patients: a 10-year prospective observational study. *Diabetes Care.* May; 2002 25(5):859–64. [PubMed: 11978681]
8. Chaturvedi N, Stephenson JM, Fuller JH. The relationship between socioeconomic status and diabetes control and complications in the EURODIAB IDDM Complications Study. *Diabetes Care.* May; 1996 19(5):423–30. [PubMed: 8732703]
9. Matsushima M, Shimizu K, Maruyama M, Nishimura R, LaPorte RE, Tajima N. Socioeconomic and behavioural risk factors for mortality of individuals with IDDM in Japan: population-based case-control study. *Diabetes Epidemiology Research International (DERI) US-Japan Mortality Study Group. Diabetologia.* Jun; 1996 39(6):710–6. [PubMed: 8781767]
10. Forssas E, Keskimaki I, Reunanen A, Koskinen S. Widening socioeconomic mortality disparity among diabetic people in Finland. *Eur J Public Health.* Mar; 2003 13(1):38–43. [PubMed: 12678312]
11. Winkleby MA, Jatulis DE, Frank E, Fortmann SP. Socioeconomic status and health: how education, income, and occupation contribute to risk factors for cardiovascular disease. *Am J Public Health.* Jun; 1992 82(6):816–20. [PubMed: 1585961]
12. Cirino PT, Chin CE, Sevcik RA, Wolf M, Lovett M, Morris RD. Measuring socioeconomic status: reliability and preliminary validity for different approaches. *Assessment.* Jun; 2002 9(2):145–55. [PubMed: 12066829]
13. Ellis D, Coonrod BA, Dorman JS, Kelsey SF, Becker DJ, Avner ED, et al. Choice of urine sample predictive of microalbuminuria in patients with insulin-dependent diabetes mellitus. *Am J Kidney Dis.* Apr; 1989 13(4):321–8. [PubMed: 2705450]
14. Early Treatment of Diabetic Retinopathy Study Coordinating Center: Manual of Operations. University of Maryland School of Medicine; Baltimore, MD: 1980.
15. Rose GA, Blackburn H. Cardiovascular survey methods. *Monogr Ser World Health Organ.* 1968; 56:1–188.
16. The hypertension detection and follow-up program: Hypertension detection and follow-up program cooperative group. *Prev Med.* Jun; 1976 5(2):207–15. [PubMed: 935073]
17. Beck AT, Steer RA. Internal consistencies of the original and revised Beck Depression Inventory. *J Clin Psychol.* Nov; 1984 40(6):1365–7. [PubMed: 6511949]
18. Warnick GR, Albers JJ. Heparin--Mn2+ quantitation of high-density-lipoprotein cholesterol: an ultrafiltration procedure for lipemic samples. *Clin Chem.* Jun; 1978 24(6):900–4. [PubMed: 207462]
19. Loucks EB, Lynch JW, Pilote L, Fuhrer R, Almeida ND, Richard H, et al. Life-course socioeconomic position and incidence of coronary heart disease: the Framingham Offspring Study. *Am J Epidemiol.* Apr 1; 2009 169(7):829–36. [PubMed: 19179358]
20. Kovacs M, Charron-Prochownik D, Obrosky DS. A longitudinal study of biomedical and psychosocial predictors of multiple hospitalizations among young people with insulin-dependent diabetes mellitus. *Diabet Med.* Feb; 1995 12(2):142–8. [PubMed: 7743761]
21. Muhlhauser I, Overmann H, Bender R, Jorgens V, Berger M. Predictors of mortality and end-stage diabetic complications in patients with Type 1 diabetes mellitus on intensified insulin therapy. *Diabet Med.* Oct; 2000 17(10):727–34. [PubMed: 11110506]
22. Ismail IS, Nazaimoon WM, Mohamad WB, Letchuman R, Singaraveloo M, Pendek R, et al. Sociodemographic determinants of glycaemic control in young diabetic patients in peninsular Malaysia. *Diabetes Res Clin Pract.* Jan; 2000 47(1):57–69. [PubMed: 10660222]
23. Araujo MB, Mazza CS. Assessment of risk factors of poor metabolic control in type 1 diabetic children assisted in a public hospital in Argentina. *Pediatr Diabetes.* Oct; 2008 9(5):480–7. [PubMed: 18761645]
24. Carter PJ, Cutfield WS, Hofman PL, Gunn AJ, Wilson DA, Reed PW, et al. Ethnicity and social deprivation independently influence metabolic control in children with type 1 diabetes. *Diabetologia.* Oct; 2008 51(10):1835–42. [PubMed: 18679654]

25. Nathan DM, Cleary PA, Backlund JY, Genuth SM, Lachin JM, Orchard TJ, et al. Intensive diabetes treatment and cardiovascular disease in patients with type 1 diabetes. *N Engl J Med*. Dec 22; 2005 353(25):2643–53. [PubMed: 16371630]
26. Unwin N, Binns D, Elliott K, Kelly WF. The relationships between cardiovascular risk factors and socio-economic status in people with diabetes. *Diabet Med*. Jan; 1996 13(1):72–9. [PubMed: 8741816]
27. Nadas J, Putz Z, Fovenyi J, Gaal Z, Gyimesi A, Hidvegi T, et al. Cardiometabolic risk and educational level in adult patients with type 1 diabetes. *Acta Diabetol*. Jun; 2009 46(2):159–62. [PubMed: 18843447]
28. Gnani R, Petrelli A, Demaria M, Spadea T, Carta Q, Costa G. Mortality and educational level among diabetic and non-diabetic population in the Turin Longitudinal Study: a 9-year follow-up. *Int J Epidemiol*. Aug; 2004 33(4):864–71. [PubMed: 15131089]

Table 1
 Baseline^a Characteristics (mean (SD) or % (n)) by SES Category for the Age 28 Cohort of the Pittsburgh EDC Study Population (n=317)

	Income			Education			Occupation	
	Lowest Income	Middle Income	Highest Income	< College Grad	College Grad	College Grad	Non-professional	Professional
% (N)	18.6 (59)	62.8 (199)	18.6 (59)	62.8 (199)	37.2 (118)		55.5 (176)	44.5 (141)
Mean Age (yrs)	28.2 (1.7)	28.5 (1.5)	28.4 (1.6)	28.5 (1.6)	28.3 (1.4)		28.4 (1.6)	28.4 (1.5)
Mean T1D duration (yrs)	19.6 (4.2)	20.3 (4.4)	19.7 (4.4)	20.2 (4.3)	19.9 (4.6)		20.1 (4.3)	20.1 (4.5)
Female (% (n))	54.2 (32)	47.2 (94)	47.5 (28)	47.2 (94)	50.8 (60)		43.8 (77)	54.6 (77)
Caucasian (% (n))	96.6 (57)	99.5 (198)	98.3 (58)	99.0 (197)	98.3 (116)		98.9 (174)	98.6 (139)
Married (% (n))	29.3 (17)	51.3 (102)	66.1 (39)**†	49.2 (100)	50.5 (58)		42.9 (75)	58.9 (83)**
Clinical Variables								
HbA _{1c} (%)	9.16 (1.60)	8.68 (1.52)	8.47 (1.39) * †	8.80 (1.60)	8.60 (1.39)		8.87 (1.57)	8.55 (1.45)
Systolic BP (mmHg)	114.9 (14.8)	113.6 (14.6)	111.1 (13.8)	114.1 (15.8)	112.3 (12.0)		113.2 (14.3)	113.7 (14.8)
Diastolic BP (mmHg)	74.8 (10.5)	72.4 (10.4)	72.5 (9.9)	73.5 (10.4)	71.9 (10.2)		73.3 (9.8)	72.4 (11.0)
Hypertension (%)	20.3 (12)	14.1 (28)	6.8 (4) †	16.1 (32)	10.2 (12)		13.6 (24)	14.2 (20)
Total-c (mg/dL)	192.6 (57.8)	188.4 (40.7)	189.2 (45.0)	192.4 (49.8)	184.3 (35.2)		193.0 (46.7)	184.8 (42.5)
HDL-c (mg/dL)	53.2 (12.0)	53.1 (12.6)	53.3 (15.0)	52.6 (12.1)	54.1 (14.3)		52.1 (11.8)	54.4 (14.2)
Non-HDL-c (mg/dL)	139.4 (58.5)	135.4 (42.3)	135.8 (44.6)	139.8 (51.4) *	130.2 (34.6) *		140.9 (49.0) *	130.4 (41.5) *
BMI (kg/m ²)	24.1 (3.3)	24.3 (3.1)	25.1 (3.4)	24.2 (3.2)	24.7 (3.1)		24.1 (3.2)	24.8 (3.2)
Insulin dose (U kg ⁻¹ day ⁻¹)	0.76 (0.21)	0.75 (0.22)	0.68 (0.18) * †	0.76 (0.20)	0.71 (0.22) *		0.77 (0.20)	0.71 (0.22) **
Intensive Insulin Therapy (%)	15.5 (9)	15.5 (30)	22.4 (13)	12.8 (25)	23.7 (27) *		8.7 (15)	27.2 (37) **
AER (μg/min) ^b	24.4 (8.6-216)	12.8 (6.4-106)	9.9 (4.9-53.3) *	16.1 (6.8-136)	10.8 (5.6-7.0) *		18.6 (7.4-120)	10.0 (5.5-65.2) *
Serum creatinine (mg/dL) ^b	0.80 (0.6-0.9)	0.90 (0.7-1.0)	0.90 (0.8-1.0) **†	0.90 (0.7-1.0)	0.90 (0.7-1.0)		0.90 (0.7-1.0)	0.90 (0.8-1.1)
Complication Variables								
Prevalent ON (%)	30.5 (18)	22.7 (45)	18.6 (11)	26.3 (52)	18.6 (22)		25.0 (44)	21.4 (30)
Prevalent CAD (%)	3.4 (2)	4.5 (9)	3.4 (2)	2.5 (5)	6.8 (8)		2.3 (4)	6.4 (9)
Prevalent LEAD (%)	10.2 (6)	6.5 (13)	5.1 (3)	7.5 (15)	5.9 (7)		6.8 (12)	7.1 (10)

	Income			Education			Occupation	
	Lowest Income	Middle Income	Highest Income	< College Grad	College Grad	College Grad	Non-professional	Professional
Prevalent Retinopathy (%)	28.8 (17)	27.3 (54)	32.2 (19)	26.6 (53)	31.6 (37)	31.6 (37)	27.3 (48)	30.0 (42)
Prevalent AN (%)	26.8 (15)	21.8 (41)	19.0 (11)	23.0 (43)	20.9 (24)	20.9 (24)	22.4 (37)	21.9 (30)
Prevalent Depression (%) ^c	29.1 (16)	9.8 (19)	10.3 (6) ** †	16.8 (32)	7.8 (9) *	7.8 (9) *	14.1 (24)	12.4 (17)
Ever Smoker (%)	43.1 (25)	40.7 (81)	11.9 (7) ** †	43.4 (86)	22.9 (27) **	22.9 (27) **	44.6 (78)	24.8 (35) **

Abbreviations: AER, albumin excretion rate; ON, overt nephropathy; CAD, coronary artery disease; LEAD, lower extremity artery disease; AN, autonomic neuropathy

* $p \leq 0.05$;

** $p \leq 0.01$ based on χ^2 or ANOVA, as appropriate;

† $p \leq 0.05$ for trend across income groups

^a Baseline data comes from the study cycle closest to age 28 (± 4 yrs);

^b Data shown as median (IQR);

^c Beck's Depression Index Score ≥ 14

Table 2
Incidence (% (n)) of Major Diabetes Complications in Type 1 Diabetes by Baseline (Age 28) SES Categories.

	N ^a	Median FUT (yrs)	Income			Education			Occupation	
			Lowest Income	Middle Income	Highest Income	< College Grad	College Grad	Non-professional	Professional	
N			59	199	59	199	118	176	141	
End-stage renal disease	314	10.7	11.9 (7)	11.2 (22)	3.4 (2)*	13.1 (26)	4.3 (5)***	12.1 (21)	7.1 (10)	
Coronary artery disease	304	12.3	21.1 (12)	20.5 (39)	12.1 (7)	24.2 (47)	10.0 (11)***	20.9 (36)	16.7 (22)	
Lower extremity arterial disease	295	11.2	18.9 (10)	18.3 (34)	5.4 (3)**†	17.9 (33)	12.6 (14)	17.1 (28)	14.5 (19)	
Autonomic neuropathy	236	8.7	46.3 (19)	33.1 (49)	23.4 (11)**†	37.9 (55)	26.4 (24)*	39.5 (51)	26.2 (28)**	
Proliferative retinopathy	226	8.5	42.9 (18)	37.5 (54)	30.0 (12)	37.0 (54)	37.5 (30)	39.8 (51)	33.7 (33)	

Abbreviation: FUT, follow-up time

* $p \leq 0.10$;

** $p \leq 0.05$;

*** $p \leq 0.01$ based on χ^2 analysis

† $p \leq 0.05$ for trend across income groups

^aNumbers for each complication do not total 317 due to exclusion of individuals with prevalent complication at their Age 28 study cycle

Table 3

Unadjusted and Adjusted Hazards Ratios for Major Incident Diabetes Complications by SES Measure Using Cox Proportional Hazard Models.

Incident end-stage renal disease												
Unadjusted				Model 1 ^a				Model 2 ^b				
SES Measure	HR	95% CI	P	AIC	HR	95% CI	P	AIC	HR	95% CI	P	AIC
Low Income	3.3	0.8, 13.9	0.10	313.0	3.1	0.7, 13.2	0.12	313.9	3.8	0.8, 17.6	0.09	233.7
< College Graduate	2.9	1.1, 7.7	0.03	311.0	2.9	1.1, 7.7	0.03	311.4	2.1	0.8, 5.9	0.14	234.9
Non-Professional	1.5	0.7, 3.2	0.30	315.7	1.5	0.7, 3.2	0.31	316.2	1.4	0.6, 3.3	0.46	236.8
Incident coronary artery disease												
Unadjusted				Model 1 ^a				Model 2 ^b				
SES Measure	HR	95% CI	P	AIC	HR	95% CI	P	AIC	HR	95% CI	P	AIC
Low Income	1.7	0.8, 3.8	0.19	543.4	1.8	0.8, 3.9	0.17	535.2	1.6	0.6, 4.1	0.37	463.0
< College Graduate	2.4	1.2, 4.7	0.01	538.4	2.5	1.3, 4.9	0.01	529.6	2.0	1.0, 3.8	0.05	461.0
Non-Professional	1.2	0.7, 2.1	0.45	544.5	1.2	0.7, 2.1	0.46	536.5	1.2	0.7, 2.2	0.54	463.5
Incident lower extremity arterial disease												
Unadjusted				Model 1 ^d				Model 2 ^b				
SES Measure	HR	95% CI	P	AIC	HR	95% CI	P	AIC	HR	95% CI	P	AIC
Low Income	3.8	1.2, 12.2	0.03	483.2	3.7	1.1, 11.9	0.03	485.6	3.9	1.2, 13.3	0.03	460.2
< College Graduate	1.5	0.8, 2.9	0.18	488.8	1.5	0.8, 2.9	0.18	490.8	1.2	0.6, 2.4	0.53	466.4
Non-Professional	1.2	0.7, 2.1	0.59	490.3	1.2	0.7, 2.2	0.52	492.3	1.1	0.6, 2.1	0.72	466.7
Incident autonomic neuropathy												
Unadjusted				Model 1 ^a				Model 2 ^b				
SES Measure	HR	95% CI	P	AIC	HR	95% CI	P	AIC	HR	95% CI	P	AIC
Low Income	1.8	1.1, 3.1	0.02	689.2	1.7	1.0, 2.9	0.04	688.5	1.4	0.8, 2.4	0.29	646.1
< College Graduate	1.3	0.8, 2.2	0.25	693.0	1.4	0.9, 2.3	0.17	691.3	1.5	0.9, 2.5	0.16	644.9
Non-Professional	1.4	0.9, 2.1	0.20	692.8	1.5	0.9, 2.3	0.12	691.1	1.3	0.8, 2.2	0.24	645.5

^aModel 1: Adjusted for sex and diabetes duration.

^bModel 2: Adjusted for Model 1 variables and HbA_{1c}, non-HDL cholesterol, hypertension status, prevalent overt nephropathy, and smoking status