Published in final edited form as:

Ann Epidemiol. 2011 May ; 21(5): 367–373. doi:10.1016/j.annepidem.2011.02.011.

Association of Socioeconomic Status with Mortality in Type 1 Diabetes: The Pittsburgh Epidemiology of Diabetes Complications (EDC) Study

Aaron M Secrest, PhD¹, Tina Costacou, PhD¹, Bruce Gutelius, MD, MPH^{1,2}, Rachel G Miller, MS¹, Thomas J Songer, PhD¹, and Trevor J Orchard, MD, MMedSci^{1,*}

¹Department of Epidemiology, University of Pittsburgh, Pittsburgh, PA 15260, USA

²Bureau of Tobacco Control, New York City Department of Health and Mental Hygiene, New York, NY 10013, USA

Abstract

Purpose—Socioeconomic status (SES) as a risk factor for mortality in type 1 diabetes (T1D) has not been adequately studied prospectively

Methods—Complete clinical and SES (income, education, occupation) data were available for 317 T1D participants in the Pittsburgh Epidemiology of Diabetes Complications Study within 4 years of age 28 (chosen to maximize income, education, and occupational potential, and to minimize the SES effect of advanced diabetes complications). Vital status was determined as of 1/1/2008.

Results—Over a median 16 years of follow-up, 34 (10.7%) deaths occurred (SMR=4.1, 95% CI, 2.7–5.5). SMRs did not differ from the general population for those in the highest education and income groups, whereas in those with low SES, SMRs were increased. Mortality rates were 3 times lower for individuals with versus without a college degree ($p=0.004$) and nearly 4 times lower for the highest versus lower income groups ($p=0.04$). In Cox models adjusting for diabetes duration and sex, education was the only SES measure predictive of mortality (HR=3.0, 1.2–7.8), but lost significance after adjusting for HbA_{1c}, non-HDL cholesterol, hypertension, and microalbuminuria (HR=2.1, 0.8–5.6).

Conclusions—The strong association of education with mortality in T1D is partially mediated by better glycemic, lipid and blood pressure control.

Keywords

type 1 diabetes; mortality; socioeconomic status; income; education; occupation; glycemic control; diabetes self-care

© 2011 Elsevier Inc. All rights reserved.

*Corresponding author: Dr. Trevor J. Orchard, MD, 3512 Fifth Avenue, 2nd Floor, Pittsburgh, PA 15213, Phone: 412-383-1032, Fax: 412-383-1020, orchardt@edc.pitt.edu.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

Introduction

Mortality is inversely associated with socioeconomic gradients in the general population.(1–4) Disadvantaged individuals have higher rates of mortality, and data suggest this inequality is becoming more apparent over time.(5) However, only a few studies have examined the relationship between socioeconomic status (SES) and mortality in diabetes, specifically type 1 diabetes, and study designs and definitions of SES measures have varied widely.

Some studies report a clear SES gradient in mortality for those with diabetes,(6–8) while others have reported that SES plays less of a role or no role in mortality in those with diabetes compared to the general population.(9–11) The SES differences in mortality seen for individuals with diabetes appear to be largely the result of cardiovascular-related deaths. A disproportionate number of these cardiovascular deaths have occurred among individuals with type 1 diabetes.(7,10) Also, noted associations between SES and mortality in the literature differ by SES measures, and studies separating diabetes by type report a stronger association of SES with type 1 diabetes.(7,8)

Given these diverse data in the literature, we thus examined the relationship between SES and all-cause mortality using a large prospective cohort of childhood-onset type 1 diabetes. Three different SES measures were captured at or near age 28, including household income, education, and occupation, and were used to evaluate this relationship. We also sought to identify which SES measures were most strongly associated with all-cause mortality in type 1 diabetes.

Methods

Study Population

Participants in the Pittsburgh Epidemiology of Diabetes Complications (EDC) Study, an ongoing prospective investigation of patients with childhood-onset (age <17 years) type 1 diabetes were included in this analysis. EDC participants ($n=658$) were either diagnosed or seen within 1 year of diagnosis at Children's Hospital of Pittsburgh between 1950 and 1980 and were on insulin therapy at initial discharge. Participants have been followed biennially by survey since the baseline examination in 1986–1988, with study examinations occurring biennially for the first 10 years and again at 18 years. Data through the 18-year follow-up (2004–2006) were available for analysis (mean follow-up age and diabetes duration 42.2 ± 5.3 years and 34.0 ± 5.2 years, respectively). The study protocol was approved by the University of Pittsburgh Institutional Review Board.

Socioeconomic Status Variables

Socioeconomic status (SES) variables in the EDC Study include household income, education level, and occupation, all of which have been collected in the study since the baseline exam. To allow for maximal educational attainment, along with the establishment of relative occupational and financial standing, each of these SES measures was assessed as close to age 28 as possible. This age was also selected to minimize the effect of advanced diabetes complications on education, income potential, and occupation status. To obtain this “Age 28” cohort, clinical and survey data were obtained from the EDC Study cycle at which participants were closest to age 28 with the age range limited to ± 4 years (24–32 years old). Each person's “Age 28” study cycle was then considered as the baseline point for all further analyses. Complete clinical and SES data were available for 317 participants. The majority (78%) of EDC participants excluded from this analysis was either over age 32 at baseline examination in 1986–88 (60%) or not examined between ages 24 and 32 (18%). The remaining 22% were excluded due to incomplete SES data. No major differences were noted for those not analyzed, apart from the obligatory age and diabetes duration differences.

Education was defined by the self-report of highest educational attainment and categorized in five groups: some high school, high school graduate, some college, college graduate, and education beyond college graduation. All EDC participants had at least some high school education. Occupation was defined by self-reported work title and categorized according to the Hollingshead Index of social position.⁽¹²⁾ The lowest Hollingshead Index occupation category (8X–9X) consists of full-time students, homemakers, retired persons, and disabled persons. Full-time students at the Age 28 study cycle were reclassified based on their occupation from the next study cycle ($n=6$). Likewise, women listed as homemakers at the Age 28 study cycle were reclassified as their occupation from the study cycle immediately before or after, if they reported working in either of these cycles ($n=25$). Disabled individuals were excluded ($n=2$), as the disability resulted from advanced diabetes complications.

Household income was defined by self-report and based on the combined annual pre-tax income (in US dollars) for the entire household. However, for this study, relative income levels were standardized by assigning participants to 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= $\geq$ \$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= $\geq$ \$50,000. Education ($n=10$) or income ($n=17$) data were imputed if these data points were missing from the “Age 28” study cycle but were reported as the same in the study cycles before and after the “Age 28” study cycle.

Mortality Data

Vital status was determined as of January 1, 2008. For every deceased person, death certificates were obtained and as many of the following as possible: 1) medical records surrounding the death; 2) autopsy/coroner’s reports; and 3) interview with next-of-kin regarding the circumstances surrounding the death. Underlying cause of death was determined by a Mortality Classification Committee of at least two physician epidemiologists using all available data based on standardized procedures.⁽¹³⁾

Clinical Data

Blood pressure was measured after a 5-min rest using a random-zero sphygmomanometer based on the Hypertension Detection and Follow-up Program protocol.⁽¹⁴⁾ Hypertension was defined as a systolic blood pressure ≥ 140 mmHg or diastolic blood pressure ≥ 90 mmHg or antihypertensive medication use. An ever smoker was defined as a person who had smoked ≥ 100 cigarettes over their lifetime. Body mass index (BMI, in kg/m^2) was determined using height and weight measures, and waist and hip circumferences were measured at least twice and averaged to calculate waist-to-hip ratio (WHR). Intensive insulin therapy was defined as either ≥ 3 daily insulin injections or use of an insulin pump.

Fasting blood samples were obtained to determine glycosylated hemoglobin, lipids, and serum creatinine. Stable glycosylated hemoglobin (HbA_{1c}) was measured by ion-exchange chromatography (Isolab, Akron, OH) and by automated HPLC (Diamat, BioRad, Hercules, CA). Measurements obtained using the two methods are almost identical ($r=0.95$). For consistency in analysis, original HbA_1 values were converted to Diabetes Complications and Control Trial (DCCT)-aligned HbA_{1c} values using the following regression equation derived from duplicate assays: $\text{DCCT HbA}_{1c} = 0.14 + 0.83(\text{EDC HbA}_1)$. HDL cholesterol was quantified after a heparin and manganese chloride precipitation method,⁽¹⁵⁾ and total cholesterol and triglycerides were measured enzymatically. Non-HDL cholesterol was defined as total cholesterol minus HDL cholesterol. Serum and urinary albumin were

determined by immunonephelometry,(16) and creatinine was assayed with an Ectachem 400 Analyzer (Eastman Kodak, Rochester, NY).

Statistical Analysis

All three SES measures were dichotomized for analysis based on inspection of each variable and consideration of sample size. Income was grouped as: highest income category vs. 4 lower categories. Education was classified as persons with or without a college degree. Occupation was categorized as either professional (Hollingshead 1A–3C) or non-professional (Hollingshead 4A–7X). For all analyses, each participant's "Age 28" study cycle was considered as baseline.

Associations between dichotomous SES variables and mortality were analyzed using the χ^2 or Fisher's exact tests, as appropriate based on expected counts. The Student's *t* test (normally distributed) or the Mann-Whitney U test (non-normally distributed) was used to compare continuous variables by SES group. Non-normally distributed variables are presented as median (IQR). Spearman's correlations were performed between each SES measure.

General population life tables from Allegheny County, Pennsylvania were used to estimate expected mortality by the person-years method. Age-, sex-, and race-adjusted standardized mortality ratios (SMRs) were calculated as the observed divided by the expected number of deaths in each age, sex, and race category.

The association between SES measures and mortality was assessed using Cox proportional hazards models to adjust for sex, diabetes duration and other measures significantly associated with mortality in this type 1 diabetes cohort. As age and diabetes duration are highly correlated in this cohort, and since age was selected to be within a narrow range (24–32 years), only duration was made available to multivariable models. The proportional hazards assumption was assessed visually and verified by testing time-dependent interaction variables. Statistical significance was established at p -value ≤ 0.05 . All analyses were performed using SAS 9.2 (SAS Institute, Cary, NC).

Results

Over a median follow-up time of 16.3 years (range 2.0–21.7 years), there were 34 (10.7%) deaths in our "age 28" cohort of childhood-onset type 1 diabetes as of January 1, 2008. No differences by sex, age, race, or diabetes duration were seen at the "age 28" baseline examination (Table 1). Of the SES measures, income and education level were associated with mortality in type 1 diabetes; occupation was not. The highest income group comprised 20.1% of the surviving cohort, but only 5.9% of deaths ($p=0.04$). Similarly, 39.9% of those still alive had a college degree, compared to 14.7% of the deceased ($p=0.004$). Very few baseline clinical measures (HDL-cholesterol, BMI, daily insulin dose, intensive insulin therapy use) did not differ by subsequent vital status. All other baseline clinical measures were significantly worse for those who died (Table 1).

Those who died during follow-up had significantly higher proportions of baseline overt nephropathy ($p<0.001$), proliferative retinopathy ($p=0.003$), and autonomic neuropathy ($p=0.002$) compared to the surviving population. However, no differences were seen in the baseline prevalences of CAD ($p>0.9$), depression ($p=0.28$), or smoking ($p=0.28$).

All three SES variables (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 ($r = 0.17$, $p = 0.003$). Figure 1 presents the mortality by dichotomous SES categories.

No clear patterns were seen in primary cause of death by any SES measure (Supplemental Table 1). More than 85% of the deaths in this cohort were diabetes-related, with the majority of these deaths resulting from chronic diabetes complications (e.g., cardiovascular disease, renal disease, infection).

We then calculated the standardized mortality ratios (SMRs) for this cohort compared to a local age-, sex- and race-matched general population (Allegheny County) (Table 2 and Figure 2). Over a median 16 years of follow-up, overall mortality in this cohort was 4 times higher than seen in the local general population (SMR=4.1, 95% CI 2.7–5.5). Stratifying this cohort by SES measures shows a dramatic difference in SMRs (Figure 2). Individuals in the highest income or education group had mortality rates no different from the age-, sex-, and race-matched general population, whereas the mortality rates for the lower income or education T1D population were approximately five times higher than the general population. This difference was not as evident for baseline occupation level.

In Cox proportional hazards models, the only SES measure that significantly predicted mortality univariately was education, wherein type 1 diabetic individuals without a college degree were three times more likely to die than those with a college degree (HR=3.0, 95% CI 1.2–7.8). This association remained significant after adjusting for sex and diabetes duration (Table 3). However, adjusting for other significant clinical risk factors (i.e., baseline HbA_{1c}, non-HDL cholesterol, hypertension status and presence of microalbuminuria, all of which have previously been shown to predict mortality in T1D), the association between education and mortality was diminished and became no longer significant (HR=2.1, 0.8–5.6). While income level did not reach statistical significance in Cox modelling, the low income group HR was 3.2 (95% CI 0.8–13.5), and the association was not substantively diminished with adjustment for other risk factors. Occupation was not significantly associated with mortality in either unadjusted or adjusted Cox models. Based on AIC, all of the SES models were comparable to one another.

Discussion

These data indicate that all-cause mortality rates in type 1 diabetes are significantly associated with education and income levels attained by early adulthood. Low income and low education groups had significantly higher rates of all-cause mortality compared to the local general population, whereas high income and education groups did not significantly differ from the general population in their mortality rates. Cox proportional hazards modelling revealed that education and income were similarly associated with mortality. This relationship became attenuated for education (HR=3.0 reduced to 2.1) after adjusting for other key potential mediators, while the relationship between mortality and income was largely unaffected by such adjustment (HR=3.2 to 3.0).

The strategy of identifying SES measures at a single point in time in young adulthood has been reported recently in the general population (Framingham Offspring Study).⁽¹⁷⁾ 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 the time they reach age 28. Given the very low levels of prevalent major complications ($n=3$ for ESRD and $n=4$ for hard CAD events), we believe that analysis using SES from this age 28 cohort most adequately captured the educational attainment and earning potential for this cohort prior to the adverse influence of complications on SES.

Clear evidence exists that low SES, both at and after onset of type 1 diabetes is associated with a number of risk factors: poor glycemic control,(18–20) smoking,(21,22) dyslipidemia, (21,23), hypertension,(22,24) and multiple hospitalizations.(25) All of these have been associated with increased mortality risk in type 1 diabetes. It is unclear, however, whether SES is an independent predictor of mortality or whether it merely reflects these other measures which drive mortality. Our data suggest that the effect of educational attainment on mortality in type 1 diabetes is reduced after accounting for other risk factors, as seen in other reports.(24,26) Income, however, appears to be a stronger predictor of mortality, as its effect was not diminished with similar adjustment.

Gnavi et al. examined the role of SES (education level) in a 9-year follow-up of the Turin Longitudinal Study.(10) In type 1 diabetes, individuals with only a primary school education or no formal education were 3–4 times as likely to die during follow-up as those who graduated high school (HR for T1D males=3.1, 95% CI 1.6–6.1, HR for T1D females= 4.4, 1.6–12.3), after adjusting for age and neighborhood. Similarly, in our 16-year follow-up, we found that T1D individuals with less education were 3 times as likely to die as their more highly educated counterparts, after adjusting for diabetes duration and sex. While previous SES studies in T1D have reported overall and sex-specific standardized mortality ratios (SMRs),(7,10) our study is the first to report SMRs in T1D stratified by different SES measures, with dramatic results.

The strengths and weaknesses of this study should be noted. We did rely on SES measures from a single point in time for analysis, and repeated measures have been shown to provide a more accurate SES picture in the general population.(3,4) However, a key objective of this study was to assess SES prior to advanced diabetes complications adversely affecting SES. So, while repeated measures at a later date may have improved our assessment of actual SES, they also would have made it more difficult to interpret the actual predictive value of SES before the development of advanced complications.

With 16 years of follow-up, this is the longest prospective study in type 1 diabetes to address the role of SES in early T1D mortality. Despite this, the sample size for our study is relatively small ($n=317$) with only 10.7% dying during follow-up. This is due to the fact that our cohort was young at baseline (~age 28), and our oldest person at follow-up was only 53 years-old. However, we still found strong associations with SES and mortality in type 1 diabetes. These associations would likely persist with increased follow-up; however, it could be argued that the SES effect would be stronger at younger ages, based on the phenomenon recently termed “metabolic memory”, and seen in the Diabetes Control and Complications Trial (DCCT) and other observational studies.(27,28) Metabolic memory represents the notion that early chronic exposure to hyperglycemia leads to irreversible microvascular changes occurring prior to modern advances in diabetes management. We hypothesize that the SES effect on access to diabetes care led to more irreversible microvascular damage in the low SES groups in this cohort.

The income variable was based on household income and could not readily be adjusted for household size. As such, the actual income available to the T1D individual might be over-estimated. We were also unable to adjust for inflation due to the nature of the original income variable (categorical ranges of income), and could only standardize the income levels nearly midway through the study cycles. Adjusting for inflation, however, would not substantively change the results, as the dichotomized income comparisons were between the highest income group and all others.

Finally, the Hollingshead Index is not thought by all to be the best SES measure for occupation.(12) We dichotomized occupation (professional vs. non-professional) based on

the Hollingshead Index and found little association (Figure 1) with mortality, an association which has been reported in both general and diabetes populations worldwide.(1,3,24) However, with longer follow-up, the small differences apparent in Figures 1 and 2 for occupation may reach statistical significance.

In conclusion, baseline education level at age 28 strongly predicted all-cause mortality in our type 1 diabetes population, suggesting the vital importance of education in diabetes self-care. This association was weakened by adjusting for HbA_{1c}, non-HDL cholesterol, hypertension, and presence of microalbuminuria, indicating that the relationship between education and mortality is partially mediated by these other risk factors. However, a larger type 1 diabetes prospective study in Germany showed that the effect of SES persists despite adjustment for known mortality risk factors.(29). Consequently, the effect of SES in type 1 diabetes requires additional prospective research, as this population is already at very high risk of early mortality. Regardless, these data indicate the need for improvements in diabetes care and education, especially in those from lower socioeconomic backgrounds.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Abbreviations

AER	albumin excretion rate
AN	autonomic neuropathy
BMI	body mass index
BP	blood pressure
CAD	coronary artery disease
EDC	Epidemiology of Diabetes Complications
HbA_{1c}	glycosylated hemoglobin
HDL-c	high-density lipoprotein cholesterol
MA	microalbuminuria
ON	overt nephropathy
WHR	waist-to-hip ratio

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.).

References

1. Marmot MG, McDowall ME. Mortality decline and widening social inequalities. *Lancet*. 1986; 2(8501):274–276. [PubMed: 2874291]
2. Kunst AE, Mackenbach JP. The size of mortality differences associated with educational level in nine industrialized countries. *Am J Public Health*. 1994; 84(6):932–937. [PubMed: 8203689]
3. Smith GD, Hart C, Blane D, Gillis C, Hawthorne V. Lifetime socioeconomic position and mortality: prospective observational study. *BMJ*. 1997; 314(7080):547–552. [PubMed: 9055712]
4. Pensola TH, Martikainen P. Cumulative social class and mortality from various causes of adult men. *J Epidemiol Community Health*. 2003; 57(9):745–751. [PubMed: 12933784]

5. Feldman JJ, Makuc DM, Kleinman JC, Cornoni-Huntley J. National trends in educational differentials in mortality. *Am J Epidemiol.* 1989; 129(5):919–933. [PubMed: 2705434]
6. 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.* 1996; 39(6):710–716. [PubMed: 8781767]
7. Roper NA, Bilous RW, Kelly WF, Unwin NC, Connolly VM. Excess mortality in a population with diabetes and the impact of material deprivation: longitudinal, population based study. *BMJ.* 2001; 322(7299):1389–1393. [PubMed: 11397742]
8. Forssas E, Arffman M, Koskinen S, Reunanen A, Keskimaki I. Socioeconomic differences in mortality among diabetic people in Finland. *Scand J Public Health.* 2010; 38(7):691–698. [PubMed: 20651001]
9. Forssas EH, Keskimaki IT, Reunanen AR, Koskinen SV. Coronary heart disease among diabetic and nondiabetic people - socioeconomic differences in incidence, prognosis and mortality. *J Diabetes Complications.* 2008; 22(1):10–17. [PubMed: 18191072]
10. 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.* 2004; 33(4):864–871. [PubMed: 15131089]
11. Koskinen SV, Martelin TP, Valkonen T. Socioeconomic differences in mortality among diabetic people in Finland: five year follow up. *BMJ.* 1996; 313(7063):975–978. [PubMed: 8892417]
12. Cirino PT, Chin CE, Sevcik RA, Wolf M, Lovett M, Morris RD. Measuring socioeconomic status: reliability and preliminary validity for different approaches. *Assessment.* 2002; 9(2):145–155. [PubMed: 12066829]
13. International evaluation of cause-specific mortality and IDDM. *Diabetes Epidemiology Research International Mortality Study Group. Diabetes Care.* 1991; 14(1):55–60. [PubMed: 1991436]
14. The hypertension detection and follow-up program: Hypertension detection and follow-up program cooperative group. *Prev Med.* 1976; 5(2):207–215. [PubMed: 935073]
15. Warnick GR, Albers JJ. Heparin--Mn²⁺ quantitation of high-density-lipoprotein cholesterol: an ultrafiltration procedure for lipemic samples. *Clin Chem.* 1978; 24(6):900–904. [PubMed: 207462]
16. 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.* 1989; 13(4):321–328. [PubMed: 2705450]
17. 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.* 2009; 169(7):829–836. [PubMed: 19179358]
18. 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.* 2000; 47(1):57–69. [PubMed: 10660222]
19. 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.* 2008; 9(5):480–487. [PubMed: 18761645]
20. Hassan K, Loar R, Anderson BJ, Heptulla RA. The role of socioeconomic status, depression, quality of life, and glycemic control in type 1 diabetes mellitus. *J Pediatr.* 2006; 149(4):526–531. [PubMed: 17011326]
21. Unwin N, Binns D, Elliott K, Kelly WF. The relationships between cardiovascular risk factors and socio-economic status in people with diabetes. *Diabet Med.* 1996; 13(1):72–79. [PubMed: 8741816]
22. 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.* 2009; 46(2):159–162. [PubMed: 18843447]
23. Robinson N, Edouard L, Diehl A, Fuller JH. Social class and risk factors for vascular disease in diabetes. *Diabete Metab.* 1984; 10(4):245–249. [PubMed: 6391975]
24. Chaturvedi N, Jarrett J, Shipley MJ, Fuller JH. Socioeconomic gradient in morbidity and mortality in people with diabetes: cohort study findings from the Whitehall Study and the WHO

- Multinational Study of Vascular Disease in Diabetes. *BMJ*. 1998; 316(7125):100–105. [PubMed: 9462313]
25. 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*. 1995; 12(2):142–148. [PubMed: 7743761]
26. Chaturvedi N, Stephenson JM, Fuller JH. The relationship between socioeconomic status and diabetes control and complications in the EURODIAB IDDM Complications Study. *Diabetes Care*. 1996; 19(5):423–430. [PubMed: 8732703]
27. LeRoith D, Fonseca V, Vinik A. Metabolic memory in diabetes--focus on insulin. *Diabetes Metab Res Rev*. 2005; 21(2):85–90. [PubMed: 15619272]
28. White NH, Sun W, Cleary PA, Danis RP, Davis MD, Hainsworth DP, et al. Prolonged effect of intensive therapy on the risk of retinopathy complications in patients with type 1 diabetes mellitus: 10 years after the Diabetes Control and Complications Trial. *Arch Ophthalmol*. 2008; 126(12):1707–1715. [PubMed: 19064853]
29. 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*. 2000; 17(10):727–734. [PubMed: 11110506]

Figure 1.
All-cause mortality by income, education, and occupation levels at age 28 in type 1 diabetes.

Figure 2. Standardized mortality ratios (with 95% confidence intervals) by SES status in the Age 28 cohort of the Pittsburgh EDC Study.

Table 1

Demographic and socioeconomic characteristics (mean $\pm$ SD or % (n)) of the “Age 28 cohort” in the Pittsburgh EDC Study by vital status (n=317).

	Vital Status as of 1/1/2008	
	Alive n=283	Deceased n=34
Age Range (yrs)	24.2–32.0	25.3–31.8
Mean Age (yrs)	28.4 $\pm$ 1.6	28.9 $\pm$ 1.6
Mean diabetes duration (yrs)	20.0 $\pm$ 4.4	20.7 $\pm$ 4.2
Female (% (n))	49.1 (139)	44.1 (15)
Caucasian (% (n))	98.6 (279)	100.0 (34)
Follow-up Time (yrs)^a	16.5 (12.4–20.1)	13.9 (8.9–16.8) **
SES Measures		
Income		
Group 1 (Lowest)	19.1 (54)	14.7 (5)
Group 2	22.3 (63)	26.5 (9)
Group 3	23.3 (66)	35.3 (12)
Group 4	15.2 (43)	17.6 (6)
Group 5 (Highest)	20.1 (57)	5.9 (2)
Education **		
Some High School	1.4 (4)	8.8 (3)
High School Graduate	24.7 (70)	29.4 (10)
Some College	33.9 (96)	47.1 (16)
College Graduate	30.7 (87)	14.7 (5)
> College Graduate	9.2 (26)	0.0 (0)
Occupation		
Group 5X–7X (Lowest)	31.8 (90)	35.3 (12)
Group 4A–4D	22.3 (63)	32.4 (11)
Group 3A–3C	19.4 (55)	17.6 (6)
Group 1A–2C (Highest)	26.5 (75)	14.7 (5)
Clinical Variables		
HbA1c (%)	8.64 $\pm$ 1.47	9.44 $\pm$ 1.79 **
Systolic BP (mmHg)	112.0 $\pm$ 13.1	125.0 $\pm$ 19.6 **
Diastolic BP (mmHg)	72.1 $\pm$ 9.9	79.5 $\pm$ 12.0 **
Hypertension (%)	10.2 (29)	44.1 (15) **
HDL-c (mg/dL)	53.4 $\pm$ 13.3	51.2 $\pm$ 10.1
Non-HDL-c (mg/dL)	132.0 $\pm$ 43.2	171.4 $\pm$ 53.9 **
BMI (kg/m ²)	24.5 $\pm$ 3.2	23.6 $\pm$ 2.6
WHR	0.83 $\pm$ 0.09	0.87 $\pm$ 0.07 *

Vital Status as of 1/1/2008		
	Alive <i>n</i> =283	Deceased <i>n</i> =34
Insulin dose (U/kg/day)	0.73 ± 0.21	0.81 ± 0.23
Intensive Insulin Therapy (%)	18.2 (50)	5.9 (2)
AER (μg/min) ^b	11.8 (6.1–51.2)	649 (53.1–1625) **
Serum creatinine (mg/dL) ^b	0.90 (0.7–1.0)	1.00 (0.8–1.3) **
Complication Variables		
Prevalent MA (%)	44.5 (126)	88.2 (30) **
Prevalent ON (%)	18.1 (51)	67.6 (23) **
Prevalent CAD (%)	4.2 (12)	2.9 (1)
Prevalent Retinopathy (%)	25.9 (73)	50.0 (17) **
Prevalent AN (%)	19.8 (54)	44.8 (13) **
Prevalent Depression (%) ^c	14.2 (39)	6.3 (2)
Ever Smoker (%)	34.8 (98)	44.1 (15)

* $p \leq 0.05$;

** $p \leq 0.01$

^a Each individual's "age 28" study cycle was considered as baseline.

^b Data shown as median (IQR);

^c Beck's Depression Index Score ≥ 14

Abbreviations: HbA1c, glycosylated hemoglobin; BP, blood pressure; HDL-c, high-density lipoprotein cholesterol; BMI, body mass index; WHR, waist-to-hip ratio; AER, albumin excretion rate; MA, microalbuminuria; ON, overt nephropathy; CAD, coronary artery disease; AN, autonomic neuropathy

Table 2

Standardized mortality ratios (adjusted for age, sex, and race) by socioeconomic status in the Age 28 cohort of the Pittsburgh EDC Study.

	Deaths	Median FUT (yrs)	Total FUT (person-yrs)	SMR	95% CI
Overall	34	16.3	4903.3	4.1	(2.7, 5.5)
Income					
All Others	32	16.6	4019.0	4.7	(3.1, 6.3)
Highest	2	15.6	884.3	1.4	(0.0, 3.3)
Education					
<College Graduate	29	17.1	1781.4	5.3	(3.4, 7.3)
≥College Graduate	5	15.3	3121.9	1.8	(0.2, 3.3)
Occupation					
Non-Professional	23	16.4	2141.6	4.7	(2.8, 6.7)
Professional	11	15.9	2761.7	3.2	(1.3, 5.1)

Abbreviations: FUT, follow-up time (person-yrs), SMR, standardized mortality ratio

Table 3

Unadjusted and adjusted hazards ratios for all-cause mortality by socioeconomic status measure based on Cox proportional hazards models.

SES Measure	Unadjusted				Model 1 ^a				Model 2 ^b			
	HR	95% CI	<i>p</i>	AIC	HR	95% CI	<i>p</i>	AIC	HR	95% CI	<i>p</i>	AIC
Low Income^c	3.2	0.8–13.5	0.11	358.7	3.1	0.7–13.1	0.12	359.9	3.0	0.7–13.2	0.14	326.8
< College Graduate^c	3.0	1.2–7.8	0.02	355.7	3.0	1.2–7.8	0.02	356.8	2.1	0.8–5.6	0.12	326.9
Non-Professional^c	1.6	0.8–3.2	0.22	360.7	1.6	0.8–3.3	0.21	361.7	1.7	0.8–3.5	0.19	327.9

^aModel 1: Adjusted for sex and diabetes duration.

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

^cReference groups: Low Income (vs. Highest Income); <College Graduate (vs. College Graduate); Non-professional (vs. Professional).

Abbreviations: HR, hazard ratio; AIC, Akaike's Information Criterion