SHORT REPORT

Childhood socioeconomic circumstances predict specific causes of death in adulthood: the Glasgow student cohort study

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J Epidemiol Community Health 2006;**60**:527–529. doi: 10.1136/jech.2005.044727

Socioeconomic circumstances occurring throughout the life course are important predictors of adult health outcomes.[1–3] In a recent review we found some evidence that, compared with studies using contemporaneous measures of childhood exposures, those studies relying on recalled childhood information in adulthood tend to report less consistent or weaker associations with adult mortality.[2] A new analysis of the Kuopio study using historical records of childhood socioeconomic circumstances finds higher mortality risk among those who experienced worse circumstances in childhood[4] that was not found when only recalled childhood socioeconomic position (SEP) was available.[5] Thus, using contemporaneously collected measures of childhood SEP seems to provide better estimates of the true association with later disease. Furthermore, the effect of childhood socioeconomic circumstances varies according to specific causes of death, which may suggest specific mechanisms or time periods of susceptibility. However, few current cohorts have both contemporaneous measures of early life SEP and sufficient number of deaths to carry out such detailed analysis. We have previously reported a higher risk of cardiovascular disease (CVD) mortality with lower childhood SEP in the Glasgow student cohort study.[6] However, the number of deaths did not allow for analysis of more specific causes of death. As we now have longer follow up and more events we have investigated the association between childhood SEP and specific causes of death.

METHODS AND RESULTS

Detailed information on the Glasgow alumni cohort study is available elsewhere.[7] Briefly, students attending Glasgow University between 1948 and 1968 were invited to participate in a health examination carried out by physicians. Information on sociodemographic characteristics, medical history, and health behaviours was obtained and father's main occupation was also recorded. A total of 11 755 men, representing about 50% of the complete male student population, participated in the study. Since 1998, 85% of the male cohort (n = 9986) has been successfully traced through the National Health Service Central Register, which provides continuous updates of the date and cause of death for members of the cohort. Students aged 30 years or more at entry (n = 382), those with missing date of emigration (n = 4), those with missing information on father's occupation (n = 366), and those with missing confounders (n = 514) were excluded from this analysis, yielding a total sample of 8720 men, among whom there were 1349 deaths up to August 2005.

Childhood SEP was assigned by coding father's occupation into social class, a five point scale from I (professional jobs) to V (unskilled manual jobs), using the registrar general's classification. Age (years), height (m), body mass index (BMI) (kg/m^2), blood pressure (mm Hg), and current smoking habit (yes/no) were recorded at the university health examination. ICD9 and ICD10 codes were used to group cause specific mortality into CVD (ICD9: 390-459; ICD10:I00-I99, G45); coronary heart disease (CHD) (ICD9: 410-414, 429.2; ICD10: I20-I25, I51.6); stroke (ICD9: 430-438; ICD10: I60-I69, G45); all cancers (ICD9: 140-208; ICD10: C00-C97); lung cancer (ICD9: 162; ICD10: C34); stomach cancer (ICD9: 231; ICD10: C16), prostate cancer (ICD9: 185; ICD10: C61), colon cancer (ICD9: 153; ICD10: C18); respiratory disease (ICD9: 46-51; ICD10: J00-J99) and, external causes of death, which included accidents, suicide and violence (ICD9: 800-999, E800-E999; ICD10: S00-T98, V01-Y89).

Cox proportional hazards models were used to estimate the risk of overall and cause specific mortality associated with early life SEP adjusting for examination date and height, BMI, systolic blood pressure, and smoking measured at university.

Students whose fathers worked in lower occupational classes were slightly older than their more affluent peers (0.10 (95% confidence interval (CI) 0.04 to 0.17) increase in years of age for each occupational class, trend p = 0.001). Height was inversely related to father's occupational class (−0.57 (95% CI −0.71 to −0.42) cm change for each occupational class, trend p<0.001) but there were no differences in BMI (0.005 (95% CI −0.046 to 0.056) increase in kg/m^2 for each occupational class, trend p = 0.84), systolic (−0.21 (95% CI −0.51 to 0.09) change in mm Hg for each occupational class, trend p = 0.17) or diastolic (−0.02 (95% CI −0.21 to 0.18) change in mm Hg for each occupational class, trend p = 0.87) blood pressure.

We found evidence that childhood SEP was inversely related to all cause mortality as well as to specific causes of death (table 1), CVD, CHD, and stroke mortality, although for the latter the trend did not reach conventional levels of statistical significance. Overall cancer mortality was not related to father's occupational class whereas lung cancer risk was higher in all other father's social class, compared with those whose fathers had professional and managerial occupations. The largest per-social class group hazard rate was for stomach cancer, although this was based on only 18 events and was estimated imprecisely. Respiratory disease mortality was also inversely related to father's SEP, but there was no association with external causes of death.

DISCUSSION

Lower early life SEP, as indexed by father's occupational class, was related to higher mortality risk from all causes, CVD, CHD, stroke, lung and stomach cancer, and respiratory disease. The magnitude of the associations with both CHD and stroke mortality were closely similar, although confidence intervals around the estimate for stroke were wide because of the small number of cases. No association was

Table 1 Student socioeconomic position at university (1948–1968) and cause specific mortality. Glasgow alumni cohort study, 1948–1968

	Father's occupational socioeconomic position Hazard ratio (HR) (95% confidence interval (CI))						
Cause of death (number of deaths)	I (ref) n = 1740	II n = 3132	III n = 3207	IV n = 511	V n = 130	HR per unit increase SEP	Trend p
All causes (n = 1349)							
examination date*	1.00	1.16 (1.00, 1.35)	1.23 (1.05, 1.43)	1.32 (1.02, 1.70)	1.63 (1.08, 2.46)	1.10 (1.04, 1.17)	<0.001
+ early adulthood risk factors†	1.00	1.15 (0.99, 1.34)	1.25 (1.07, 1.46)	1.35 (1.05, 1.75)	1.59 (1.05, 2.40)	1.11 (1.05, 1.18)	0.001
CVD (n = 524)							
examination date*	1.00	1.41 (1.09, 1.84)	1.58 (1.22, 2.06)	1.74 (1.16, 2.62)	2.30 (1.22, 4.32)	1.20 (1.10, 1.32)	<0.001
+ early adulthood risk factors†	1.00	1.38 (1.06, 1.80)	1.59 (1.23, 2.07)	1.78 (1.18, 2.67)	2.11 (1.12, 3.98)	1.21 (1.10, 1.32)	<.0001
CHD (n = 357)							
examination date*	1.00	1.36 (0.99, 1.87)	1.63 (1.19, 2.24)	1.78 (1.09, 2.90)	2.43 (1.15, 5.11)	1.23 (1.10, 1.38)	<0.001
+ early adulthood risk factors†	1.00	1.33 (0.96, 1.83)	1.63 (1.19, 2.24)	1.81 (1.11, 2.96)	2.19 (1.04, 4.62)	1.23 (1.10, 1.38)	<0.001
Stroke (n = 107)							
examination date*	1.00	1.31 (0.74, 2.31)	1.48 (0.84, 2.61)	1.73 (0.72, 4.17)	2.02 (0.47, 8.73)	1.19 (0.97, 1.46)	0.102
+ early adulthood risk factors†	1.00	1.29 (0.73, 2.28)	1.48 (0.84, 2.60)	1.73 (0.72, 4.18)	1.92 (0.44, 8.36)	1.19 (0.96, 1.46)	0.106
Cancer (n = 474)							
examination date*	1.00	1.08 (0.84, 1.38)	0.99 (0.77, 1.28)	1.27 (0.85, 1.92)	0.83 (0.34, 2.04)	1.01 (0.91, 1.12)	0.855
+ early adulthood risk factors†	1.00	1.08 (0.84, 1.38)	1.02 (0.79, 1.31)	1.31 (0.87, 1.98)	0.85 (0.34, 2.08)	1.02 (0.92, 1.13)	0.678
Lung cancer (n = 86)							
examination date*	1.00	2.40 (1.16, 4.97)	2.25 (1.08, 4.69)	1.84 (0.57, 5.99)	1.82 (0.23, 14.34)	1.18 (0.94, 1.49)	0.164
+ early adulthood risk factors†	1.00	2.35 (1.13, 4.85)	2.38 (1.14, 4.96)	2.00 (0.61, 6.49)	1.76 (0.22, 13.91)	1.21 (0.96, 1.53)	0.105
Stomach cancer (n = 18)‡							
examination date*	1.00	3.43 (0.41, 28.48)	5.94 (0.76, 46.38)	3.22 (0.20, 51.47)		1.57 (0.91, 2.71)	0.104
+ early adulthood risk factors†	1.00	3.43 (0.41, 28.52)	5.53 (0.71, 43.29)	2.93 (0.18, 47.04)		1.51 (0.87, 2.60)	0.140
Prostate cancer (n = 49)‡							
examination date*	1.00	1.15 (0.54, 2.46)	0.91 (0.41, 2.02)	1.35 (0.42, 4.31)		1.00 (0.72, 1.38)	0.996
+ early adulthood risk factors†	1.00	1.16 (0.54, 2.47)	0.90 (0.40, 2.01)	1.35 (0.42, 4.30)		1.00 (0.72, 1.38)	0.982
Colon cancer (n = 48)‡							
examination date*	1.00	1.09 (0.52, 2.25)	0.64 (0.28, 1.46)	1.17 (0.37, 3.68)		0.89 (0.64, 1.23)	0.478
+ early adulthood risk factors†	1.00	1.09 (0.53, 2.27)	0.67 (0.30, 1.53)	1.24 (0.39, 3.92)		0.91 (0.65, 1.26)	0.571
Respiratory diseases (n = 84)							
examination date*	1.00	0.99 (0.52, 1.87)	1.35 (0.74, 2.49)	1.66 (0.64, 4.28)	3.33 (0.96, 11.51)	1.26 (1.00, 1.60)	0.048
+ early adulthood risk factors†	1.00	0.96 (0.51, 1.82)	1.36 (0.74, 2.50)	1.69 (0.66, 4.37)	2.97 (0.85, 10.32)	1.26 (1.00, 1.59)	0.048
External causes (n = 76)§							
examination date*	1.00	1.44 (0.74, 2.81)	1.48 (0.76, 2.87)		0.23 (0.03, 1.79)	0.98 (0.75, 1.27)	0.861
+ early adulthood risk factors†	1.00	1.49 (0.76, 2.90)	1.57 (0.81, 3.06)		1.31 (0.17, 10.07)	1.01 (0.78, 1.31)	0.949

*Adjusted for examination date. †Additionally adjusted for height, BMI, systolic blood pressure, and smoking. ‡There were no deaths in social class V and the analysis for this outcome was carried out grouping social classes IV and V. §There were no deaths in social class IV and the analysis for this outcome was carried out grouping social classes IV and V.

found with the other specific causes of death investigated. These results, with respect to all causes, overall CVD and overall cancer, extend an earlier report from this cohort.[6] In this study we were able to assess a greater number of outcomes.

While overall cancer mortality was not associated with father's SEP there was an indication that the risk of stomach cancer mortality was higher. This is a cohort of university students that experienced better socioeconomic resources than the general population, and thus the overall risk of stomach cancer was low resulting in imprecise estimation of the effect. Stomach cancer has been linked to exposure to *Helicobacter pylorii*,[8] which is in general acquired in childhood. In previous studies, the risk of stomach cancer was higher among those with higher number of siblings,[9] a proxy for deprivation during early life.

It is interesting to compare the social patterning of lung cancer with that of respiratory diseases in this cohort. The participants to the Glasgow alumni study were young adults during the period when smoking was common and its harmful effects largely unknown, a fact reflected in high mortality attributable to lung cancer across all social class groups, although the evidence was stronger for those of intermediate social classes—II and III, (prevalence of current smoking at university was 21.1% in class I, 38.4% in class II, 33.6% in class III, 5.4% in class IV, and 1.6% in class V). On the other hand, mortality from respiratory diseases was notably higher among students with fathers holding lower social class jobs. The increasing risk of respiratory mortality with worsening early life socioeconomic circumstances was more evident than that for lung cancer mortality, suggesting that socially patterned exposures, other than smoking in young adulthood, are likely to contribute to the association between childhood socioeconomic circumstances and adult respiratory mortality in this cohort. These exposures could include childhood infections, indoor and outdoor pollution, and housing conditions such as damp or mould and poor nutrition.

Finally, childhood SEP was not associated with external causes of death, suggesting that external causes of death that are socially patterned are more likely to be related to adult than childhood SEP, as found in another Scottish study.[10]

Members of this cohort were students at Glasgow university between 1948 and 1968 and are therefore a socioeconomically selected sample.[7] Although this may limit the generalisability of these results, the associations reported here are less likely to be confounded by adult socioeconomic circumstances and socially patterned risk factors. Thus, the relative homogeneity of adult socioeconomic circumstances becomes a strength when evaluating the association between childhood SEP and mortality. However, as the number of students with fathers in class IV and V is low we lack precision in the estimates for more rare causes of death. In addition, and as with all cohort studies, the people included in the analysis represent only a proportion of the potential original cohort because of non-participation, loss to follow up, and missing data. This would only exaggerate the association between SEP and mortality if people who were not included in the study over-represented those with high SEP and high mortality risk, or with low SEP and low mortality risk. The reverse is more likely to be the case (that is, those participants from poor backgrounds would tend to have more favourable risk profiles than the potential sample of people from poor backgrounds) and thus the associations we report will be under-estimates of the underlying associations. Given these potential limitations, the findings reported here robustly attest to the powerful influence of early life socioeconomic circumstances on later health.

What this paper adds

We have shown that early life socioeconomic position determines higher risk of specific causes of death in adulthood. Assessing and comparing the magnitude and pattern of the associations is useful in pointing to exposures that might underlie these associations.

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Funding: the authors acknowledge the financial support of the Stroke Association; Chest, Heart and Stroke Scotland; the National Health Service Research and Development Cardiovascular Disease Programme; and the World Cancer Research Fund. BG is funded by the Medical Research Council through Research Fellowship in Health of the Public. GDS holds a Robert Wood Johnson Foundation Investigators Award in Health Policy Research. PM is supported by a career scientist award funded by the Research and Development Office for Health and Personal Social Services in Northern Ireland. The Centre for Public Health Research (Massey University, Wellington, New Zealand) is supported by a Programme Grant from the Health Research Council of New Zealand. The authors' work was independent of the funding sources.

Competing interests: none declared.

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Accepted for publication 5 January 2006

REFERENCES

1 **Davey Smith G**. *Health inequalities: lifecourse approaches*. Bristol: The Policy Press, 2003.
2 **Galobardes B**, Lynch JW, Davey Smith G. Childhood socioeconomic circumstances and cause-specific mortality in adulthood: systematic review and interpretation. *Epidemiol Rev* 2004;**26**:7–21.
3 **Galobardes B**, Davey Smith G, Lynch JW. Systematic review of the childhood socioeconomic circumstances on risk of cardiovascular disease in adulthood. *Ann Epidemiol* 2006;**16**:91–104.
4 **Kauhanen L**, Lakka HM, Lynch JW, *et al*. Social disadvantages in childhood and risk of all-cause death and cardiovascular disease in later life: a comparison of historical and retrospective childhood information. *Int J Epidemiol* (in press).
5 **Lynch JW**, Kaplan GA, Cohen RD, *et al*. Childhood and adult socioeconomic status as predictors of mortality in Finland. *Lancet* 1994;**343**:524–7.
6 **Davey Smith G**, McCarron P, Okasha M, *et al*. Social circumstances in childhood and cardiovascular disease mortality: prospective observational study of Glasgow University students. *J Epidemiol Community Health* 2001;**55**:340–1.
7 **McCarron P**, Davey Smith G, Okasha M, *et al*. Life course exposure and later disease: a follow-up study based on medical examinations carried out in Glasgow University (1948–68). *Public Health* 1999;**113**:265–71.
8 **Stemmermann GN**, Fenoglio-Preiser C. Gastric carcinoma distal to the cardia: a review of the epidemiological pathology of the precusors to a preventable cancer. *Pathology* 2002;**34**:494–503.
9 **Hart CL**, Davey Smith G. Relation between number of siblings and adult mortality and stroke risk: 25 year follow up of men in the collaborative study. *J Epidemiol Community Health* 2003;**57**:385–91.
10 **Davey Smith G**, Hart C, Blane D, *et al*. Adverse socioeconomic conditions in childhood and cause specific adult mortality: prospective observational study. *BMJ* 1998;**316**:1631–5.