American Journal of Epidemiology
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Vol. 164, No. 9
DOI: 10.1093/aje/kwj319
Advance Access publication September 20, 2006

Original Contribution

Association of Childhood Socioeconomic Position with Cause-specific Mortality in a Prospective Record Linkage Study of 1,839,384 Individuals

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Received for publication November 14, 2005; accepted for publication March 10, 2006.

Previous studies have lacked sufficient power to assess associations between early-life socioeconomic position and adult cause-specific mortality. The authors examined associations of parental social class at age 0–16 years with mortality among 1,824,064 Swedes born in 1944–1960. Females and males from manual compared with nonmanual childhood social classes were more likely to die from smoking-related cancers, stomach cancer, respiratory disease, cardiovascular disease, and diabetes. Males from manual compared with nonmanual social classes were more likely to die from unintentional injury, homicide, and alcoholic cirrhosis. The association with stomach cancer was little affected by adjustment for parental later-life and own adult social class or education. For other outcomes, educational attainment resulted in greater attenuation of associations than did adjustment for adult social class. Early-life social class was not related to suicide or to melanoma, colon, breast, brain, or lymphatic cancers or to leukemia. With the exception of stomach cancer, caused by *Helicobacter pylori* infection acquired in childhood, poorer social class in early life was associated with diseases largely caused by behavioral risk factors such as smoking, physical inactivity, and an unhealthy diet. Educational attainment may be important in reducing the health inequalities associated with early-life disadvantage.

cohort studies; medical record linkage; mortality; social class; Sweden

Abbreviations: CI, confidence interval; SEP, socioeconomic position.

Evidence exists that childhood socioeconomic position (SEP), independently of adult SEP, is associated with premature all-cause and cardiovascular disease mortality (1–4). However, few studies have had sufficient power to assess these associations with a range of cause-specific mortality outcomes (1). In the largest known study to date (N = 58,751) conducted in an unselected population by using record linkage from census data in Norway, adverse childhood SEP was associated with increased risk of large bowel cancer, coronary heart disease, and stroke (5, 6). Even with this study size, some estimates were imprecise because of small numbers of events. Determining the importance of childhood SEP for a range of outcomes is important for understanding disease etiology and the mechanisms that might link childhood SEP to adult mortality.

A further limitation of previous studies is that many were conducted among populations born before 1930 (1). Studying individuals born in more recent decades is important because the effects of childhood SEP on health outcomes vary over time (7). Children in the lowest socioeconomic groups born in more recent decades, and in particular after World War II, will have experienced better standards of living than those born earlier (8–10). If observed associations in previous studies largely represent the effects of

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extreme adverse circumstances, then one might expect a weaker, or no, association between childhood SEP and health outcomes in studies of populations born more recently. Here, we present the largest known study to date that examines the association of early-life SEP, based on parents' occupation, with death from a range of causes in an unselected population.

MATERIALS AND METHODS

The study cohort consisted of all individuals born in Sweden between 1944 and 1960 who were still alive and living in Sweden at the start of the follow-up period in 1970 ($N =$ 1,845,716). Date of birth and sex of the index participants, together with their parents' unique identity numbers, were extracted from the Swedish Multi-Generation Register. A linkage was made between these records and the index participants' death records (date of death and *International Classification of Diseases*, Seventh, Eighth, Ninth, or Tenth Revision codes) contained in the Swedish Cause of Death Register for deaths up to December 31, 2001, and to SEP data from the Swedish 1960, 1970, 1980, and 1990 Population and Housing Census databases.

Measures of social class and demographic variables

Occupational social class of the head of the household in which each cohort member resided was obtained from the four Population and Housing Censuses (1960, 1970, 1980, 1990). Head of household social class is determined by Statistics Sweden mainly from adult household members' occupational codes and according to an order of predominance established by this agency. Our main exposure was early-life SEP obtained from the 1960 Population and Housing Census, when participants were aged 0–16 years. We adjusted for later-life occupation-based social class of the head of household to determine the extent to which any effects of childhood social class were mediated via its association with social class in later life. This was a time-updated variable, derived by splitting the data set into time periods. Thus, for deaths occurring in 1970–1979, data from the 1970 census were used to adjust for later-life social class; for 1980–1989, data from the 1980 census were used; and, after 1990, data from the 1990 census were used. Of 66,558 deaths, 12,646 (19 percent) occurred during 1970–1979, 16,640 (25 percent) during 1980–1989, and 37,272 (56 percent) after 1990.

Occupational social class was classified into five categories: manual, nonmanual, self-employed, unemployed/unclassified, or student. We used height during participants' early adulthood, which is strongly determined by childhood socioeconomic circumstances (11) and was measured on a subsample of 462,186 males at the conscription board examination, to collapse these five categories into a binary variable (manual vs. nonmanual social class). Mean height of those whose parents were categorized as self-employed (179.5 cm (standard deviation, 6.4)) and those who were students (179.0 cm (standard deviation, 6.4)) was similar to the mean height of those whose parents were categorized as nonmanual (179.7 cm (standard deviation, 6.4)). Mean height of those whose parents were categorized as unemployed (178.0 cm (standard deviation, 6.5)) was similar to that of those whose parents were categorized as manual (178.2 cm (standard deviation, 6.5)), but, for both of these groups, height distributions were different from those for participants' parents who were self-employed, were students, or had nonmanual occupations (all $p < 0.001$). On the basis of these between-group comparisons of mean height, we defined early-life SEP as a binary variable: manual social class (including those with manual occupations or who were unemployed) versus nonmanual social class (including those with nonmanual occupations, who were self-employed, or who were students). In a sensitivity analysis, we excluded participants whose parents were unemployed, self-employed, or students; the results did not differ substantively from the results presented here.

A participant's birth date, sex, parents' age at the time of the participant's birth, mother's marital status at the time of the birth (married, cohabiting, unmarried, widowed, divorced), and each parent's educational attainment (<9 years, 9–10 years, completed secondary education, completed higher education) were obtained from the Multi-Generation Register and/or the 1970 Population and Housing Census data. We were unable to adjust for participants' educational attainment in the main analyses since this information was not available in the 1970 or 1980 Population and Housing Census. We did, however, examine the effect of adjusting for the educational attainment of participants still alive in 1990: this factor was divided into the same categories as those used for parents' educational attainment.

Outcome variables

The outcome variables were all-cause and cause-specific death, defined as the underlying cause of death recorded on the death certificate. The Web table shows the causes of death for which we examined associations, together with the *International Classification of Diseases*, Seventh, Eighth, Ninth, and Tenth Revision codes used to define these causes and the numbers of participants who died from each cause over the follow-up period. (This supplementary table, referred to ''Web table'' in the text, is posted on the website of the Human Genome Epidemiology Network (http://www.cdc.gov/genomics/hugenet/reviews.htm) as well as on the *Journal*'s website (http://aje.oupjournals.org/).) We decided a priori to assess associations with causes of death that resulted in at least 600 deaths in the follow-up period. Therefore, we examined all causes of death that contributed at least 0.9 percent of the total. We also decided a priori to examine relations with all stroke subtypes to determine whether we could confirm the suggestion of a stronger effect of childhood SEP on hemorrhagic compared with ischemic strokes (1, 12–14). We decided a priori to examine associations with aircraft accidents to further validate our social class categorization. This is the one outcome for which higher death rates are expected for those from nonmanual compared with manual social classes. Given that participants were born between 1944 and 1960 and that the follow-up period during which we collected mortality data was

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1970–2001, the deaths presented here represent those occurring for participants in the age range 10–57 years, although more than 80 percent of deaths occurred between 1980 and 2001, when participants were aged 20–57 years.

Data analysis

Of 1,845,716 eligible participants, 21,652 (1 percent) had missing data on social class at one or more time points and were excluded from the analysis cohort. Thus, the cohort consisted of 1,824,064 participants, among whom there were 66,558 deaths. We decided a priori to begin the follow-up period on January 1, 1970 (10 years after early-life SEP was ascertained) so that associations would not be due to reverse causality, that is, a childhood terminal illness resulting in premature death and a movement downward in social class of their parents. Data were analyzed by using Cox proportional hazards regression models with participants' age as the time axis so that all analyses were controlled for participants' age. Analyses were censored at the earlier of participants' emigration date, death date, or December 31, 2001. In multivariable analyses, we first adjusted for sex, then for parental characteristics (parental age at the time of the participant's birth, maternal marital status, and parental education) that might additionally confound the association. In the final models, we adjusted for time-updated later-life social class to determine whether this variable mediated any associations with early-life SEP. To ensure maximum adjustment for any effect of later-life social class, we retained this variable in the original five categories (manual, nonmanual, self-employed, unemployed, student) and entered it as four indicator variables.

To determine whether participants' own educational attainment (available in the 1990 census only) mediated the effect of early-life SEP on mortality, we fitted similar Cox models with age as the time axis but date of entry (start of follow-up) as January 1, 1990. These analyses could include only those participants who survived until this date. We used the strategy described above to adjust for confounders and later-life social class and finally adjusted for participants' educational attainment to examine whether doing so mediated the associations with early-life SEP. To examine whether the effects of early-life SEP varied by sex, we estimated all associations for males and females separately, and we derived Wald p values for the interaction of early-life SEP with sex. All analyses were conducted with Stata version 9.0 software (Stata Corporation, College Station, Texas).

RESULTS

Fifty-one percent of the participants were males. Of the 1,824,064 participants included in the analyses, 930,273 (51 percent) were in manual social classes in childhood. Of those who remained alive at the time of the 1990 census, 62 percent were in the same social class as that of their parents in the 1960 census, 28 percent had moved up from manual to nonmanual social classes, and 10 percent had moved down from nonmanual to manual social classes. Over the follow-up period, 66,558 deaths occurred (mortality rate = 12.5 per 10,000 person-years, 95 percent confidence interval (CI): 12.4, 12.6). Of these deaths, 43,149 occurred in males (mortality rate = 15.8 per 10,000 person-years, 95 percent CI: 15.7, 16.0) and 23,409 in females (mortality rate = 9.0 per 10,000 person-years, 95 percent CI: 8.9, 9.1). Injury or poisoning (37 percent), cancers (26 percent), and circulatory disorders (16 percent) were the most common main causes of death (Web table).

Table 1 shows age-adjusted associations of manual versus nonmanual childhood social class with all-cause and cause-specific mortality for males and females separately. Participants whose parents were in manual social classes in childhood had increased rates of all-cause, cancer, endocrine, mental disease, nervous system disease, circulatory system, respiratory system, digestive system, and injury and poisoning mortality. Higher cancer mortality rates among those from manual social classes in early life were due to stomach, liver, pancreatic, and lung cancers. There was no evidence of associations of early-life social class with colon, breast, brain, or lymphatic cancers or with melanoma or leukemia. Rates of coronary heart disease and all forms of stroke mortality were higher for those from manual compared with nonmanual early-life social classes: the magnitude of associations of early-life social class with hemorrhagic stroke was similar to that with ischemic stroke for both sexes (p for difference > 0.6).

The association between early-life social class and all-cause mortality was greater for males than females, with this difference being driven by a greater impact of social class on mortality from injury and poisoning (external causes), mental disorders, and alcoholic cirrhosis in males compared with females. For other causes of death, there was little evidence of sex differences in the magnitudes of associations. The stronger association with mental disorders in males compared with females did not appear to be driven by a stronger association with number of deaths due to alcohol dependence, which was the same for both sexes. As anticipated, death in aircraft accidents was the only outcome that occurred more often among those from a nonmanual compared with manual early-life social class. This outcome was also the only one, under the broad injury and poisoning category, for which there was no strong evidence of a sex difference.

Table 2 shows multivariable associations of early-life SEP with outcomes for which there were no sex differences (and also all-cause mortality) and for which there was evidence of an association in the simple age-adjusted analyses shown in table 1. These analyses used data on 1,722,739 participants for whom information on all covariates was complete. Adjustment for parental characteristics (in addition to sex and age) had little effect on the associations, except for those with subarachnoid hemorrhage (attenuating from 1.32 to 1.18) and diseases of the digestive system (attenuating from 1.51 to 1.42). Further adjustment for later-life social class resulted in some attenuation of all associations. In the fully adjusted models, mortality rates for stomach, liver, and lung cancer; diabetes; coronary heart disease; intracerebral hemorrhage; diseases of the respiratory system; and diseases of the digestive system all

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TABLE 1. Association of early-life socioeconomic position with all-cause and cause-specific mortality among 937,071 Swedish males and 886,993 Swedish females born in 1944–1960

Cause of mortality	Age-adjusted HR* of mortality comparing manual with nonmanual social class in early life				
	Males ($n = 937,071$)		Females ($n = 886,993$)		p for sex difference
	HR	95% CI*	HR	95% CI	
All-cause mortality	1.31	1.29, 1.34	1.18	1.15, 1.21	<0.001
All cancers	1.09	1.04, 1.14	1.10	1.06, 1.14	0.7
Stomach	1.30	1.06, 1.60	1.32	1.05, 1.66	0.9
Colon	0.96	0.80, 1.16	0.97	0.80, 1.18	0.7
Liver	1.23	1.02, 1.48	1.39	1.12, 1.72	0.4
Pancreatic	1.17	0.96, 1.41	1.32	1.07, 1.63	0.4
Lung	1.44	1.28, 1.64	1.40	1.24, 1.57	0.7
Melanoma	1.04	0.87, 1.26	0.95	0.77, 1.16	0.5
Breast			1.06	0.95, 1.17	
Brain	0.99	0.87, 1.13	0.98	0.84, 1.14	0.9
Lymphatic	1.19	1.01, 1.39	0.98	0.79, 1.22	0.2
Leukemia	0.97	0.83, 1.14	1.04	0.87, 1.25	0.5
Endocrine disorders	1.32	1.19, 1.47	1.45	1.24, 1.70	0.3
Diabetes	1.48	1.29, 1.70	1.59	1.32, 1.92	0.5
Mental disorders	1.88	1.71, 2.06	1.41	1.18, 1.69	0.005
Alcohol dependence	1.98	1.77, 2.20	1.79	1.41, 2.27	0.4
Diseases of the nervous system	1.14	1.02, 1.27	1.21	1.05, 1.40	0.6
Diseases of the circulatory system	1.43	1.37, 1.50	1.39	1.29, 1.49	0.5
Coronary heart disease	1.56	1.46, 1.66	1.46	1.28, 1.67	0.4
Stroke	1.42	1.28, 1.58	1.32	1.16, 1.49	0.4
Subarachnoid hemorrhage	1.39	1.17, 1.66	1.27	1.07, 1.49	0.4
Intracerebral hemorrhage	1.49	1.20, 1.84	1.52	1.14, 2.02	0.9
Ischemic stroke	1.73	1.30, 2.31	1.31	0.90, 1.90	0.3
Stroke—not specified	1.23	0.97, 1.55	1.16	0.84, 1.60	0.8
Diseases of the respiratory system	1.50	1.32, 1.70	1.70	1.46, 1.97	0.2
Pneumonia or infection	1.70	1.43, 2.02	1.48	1.18, 1.84	0.4
Diseases of the digestive system	1.54	1.40, 1.70	1.43	1.24, 1.65	0.4
Alcoholic cirrhosis	1.67	1.44, 1.94	1.10	0.84, 1.42	0.006
Symptoms, signs, and ill defined	1.20	1.01, 1.43	0.94	0.70, 1.25	0.1
Injury and poisoning (external causes)	1.32	1.28, 1.36	1.09	1.04, 1.15	<0.001
Unintentional injuries	1.39	1.33, 1.46	1.11	1.01, 1.21	<0.001
Road traffic accidents	1.36	1.28, 1.45	1.15	1.03, 1.29	0.01
Boating accidents	1.31	1.09, 1.57	0.85	0.59, 1.23	0.03
Aircraft accidents	0.53	0.40, 0.72	0.42	0.17, 1.02	0.6
Falls	1.70	1.45, 1.99	0.98	0.67, 1.43	0.007
Poisoning	1.90	1.65, 2.19	1.28	0.98, 1.66	0.005
Suicide	1.15	1.09, 1.20	1.03	0.96, 1.11	0.02
Homicide	2.11	1.73, 2.56	1.40	1.08, 1.82	0.01

* HR, hazard ratio; CI, confidence interval.

remained greater for those from manual compared with nonmanual social classes in early life.

Table 3 shows adjusted associations of early-life social class with mortality from all causes, mental disorders, alcoholic cirrhosis, and external causes of death, separately for females and males. Males from manual social classes had higher mortality rates from alcoholic cirrhosis, mental disorders, unintentional injuries (in particular, falls and

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TABLE 2. Multivariable association of early-life socioeconomic position with all-cause and cause-specific mortality among 1,722,739 Swedish study participants born in 1944–1960 with complete data on all covariates

Cause of mortality	Adjusted HR* of mortality comparing manual with nonmanual social class in early life					
	Model 1†		Model 2‡		Model 3§	
	HR	95% CI*	HR	95% CI	HR	95% CI
All-cause mortality	1.26	1.23, 1.29	1.21	1.19, 1.24	1.15	1.13, 1.17
All cancers	1.09	1.05, 1.14	1.07	1.03, 1.11	1.05	1.01, 1.09
Stomach	1.32	1.10, 1.59	1.32	1.10, 1.58	1.26	1.05, 1.52
Liver	1.30	1.11, 1.53	1.28	1.08, 1.50	1.23	1.04, 1.45
Pancreatic	1.23	1.05, 1.45	1.20	1.02, 1.40	1.17	1.00, 1.37
Lung	1.42	1.29, 1.56	1.40	1.28, 1.54	1.32	1.20, 1.45
Endocrine disorders	1.36	1.22, 1.51	1.30	1.17, 1.45	1.24	1.11, 1.39
Diabetes	1.52	1.32, 1.75	1.52	1.32, 1.74	1.41	1.22, 1.62
Diseases of the nervous system	1.17	1.05, 1.31	1.17	1.05, 1.31	1.13	1.01, 1.26
Diseases of the circulatory system	1.42	1.36, 1.48	1.39	1.33, 1.45	1.28	1.23, 1.34
Coronary heart disease	1.54	1.45, 1.64	1.52	1.42, 1.62	1.39	1.30, 1.48
Stroke	1.38	1.26, 1.52	1.29	1.17, 1.42	1.22	1.11, 1.34
Subarachnoid hemorrhage	1.32	1.14, 1.53	1.18	1.02, 1.37	1.13	0.97, 1.31
Intracerebral hemorrhage	1.49	1.26, 1.77	1.44	1.18, 1.77	1.32	1.08, 1.63
Ischemic stroke	1.57	1.21, 2.04	1.56	1.20, 2.01	1.47	1.13, 1.91
Stroke—not specified	1.21	0.97, 1.51	1.16	0.93, 1.44	1.10	0.88, 1.37
Diseases of the respiratory system	1.58	1.39, 1.80	1.63	1.45, 1.84	1.51	1.33, 1.70
Pneumonia or infection	1.62	1.36, 1.92	1.61	1.36, 1.91	1.47	1.24, 1.75
Diseases of the digestive system	1.51	1.37, 1.66	1.42	1.29, 1.56	1.31	1.19, 1.44

* HR, hazard ratio; CI, confidence interval.
† Model 1: adjusted for age at censoring and sex.
‡ Model 2: same as model 1 plus paternal and maternal age at child's birth, paternal and maternal educational attainment, and maternal marital status at child's birth.
§ Model 3: same as model 2 plus time-updated later-life social class.

unintentional poisoning), and homicide compared with those for males from nonmanual social classes, even after adjustment for parental characteristics and later-life social class. For females, early-life social class had little or no association with these outcomes in adjusted models. In these adjusted models, there was no association of early-life social class with suicide for males or females.

Table 4 shows multivariable associations between early-life social class and mortality after 1990, after which participants' educational attainment was recorded. These analyses included 1,596,396 individuals who were still alive and living in Sweden in 1990 and whose data were complete (30,770 deaths). The estimates of effect for all outcomes in the first three models are of a similar size to those in equivalent models presented in table 2 (with follow-up beginning in 1970). Other than for stomach cancer, the attenuation following adjustment for education (model 4) was considerably greater than that following adjustment for later-life social class. After we adjusted for later-life education and social class, as well as parental characteristics, individuals from manual compared with nonmanual social classes in early life still had increased rates of death from stomach cancer, lung cancer, diabetes, coronary heart disease, stroke, diseases of the respiratory system, and diseases of the digestive system.

When we examined the effects of early-life social class on those outcomes associated with only those males in this subgroup who were still alive at 1990, we found a similar, stronger effect of adjusting for later-life educational attainment than for later-life social class. For example, among males followed after 1990, the hazard ratio for alcoholic cirrhosis after adjustment for age and parental characteristics comparing manual with nonmanual childhood social class was 1.53 (95 percent CI: 1.25, 1.87). With further adjustment for later-life social class, this hazard ratio attenuated to 1.40 (95 percent CI: 1.15, 1.71), whereas, with further adjustment for educational attainment, it attenuated to 1.14 (95 percent CI: 0.93, 1.40). With adjustment for all covariates, the association was 1.11 (95 percent CI: 0.91, 1.37). Similar results for all unintentional injuries were 1.36 (95 percent CI: 1.24, 1.49), 1.27 (95 percent CI: 1.16, 1.40), 1.17 (95 percent CI: 1.06, 1.29), and 1.15 (95 percent CI: 1.05, 1.26), respectively, and for homicide were 2.35

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TABLE 3. Adjusted associations of early-life SEP* with all-cause, alcohol-related, and external causes of mortality among 882,211 Swedish males and 840,528 Swedish females born in 1944–1960 with complete data on all covariates

Cause of mortality	Age-, parental-, and own adult SEP-adjusted† HR* of mortality comparing manual with nonmanual social class in early life				*p* for sex difference
	Males ($n = 882,211$)		Females ($n = 840,528$)		
	HR	95% CI*	HR	95% CI	
All-cause mortality	1.19	1.16, 1.22	1.10	1.07, 1.13	<0.001
Alcoholic cirrhosis	1.42	1.19, 1.71	0.94	0.69, 1.29	0.02
Mental disorders	1.54	1.37, 1.72	1.14	0.92, 1.42	0.007
Alcohol dependence	1.59	1.39, 1.82	1.41	1.06, 1.88	0.3
Injury and poisoning (external causes)	1.16	1.10, 1.20	0.99	0.92, 1.06	<0.001
Unintentional injuries	1.23	1.15, 1.31	1.04	0.92, 1.17	<0.001
Road traffic accidents	1.22	1.11, 1.34	1.11	0.94, 1.30	0.07
Boating accidents	1.01	0.78, 1.31	0.93	0.62, 1.40	0.2
Aircraft accidents	0.62	0.41, 0.94	0.40	0.10, 1.52	0.5
Falls	1.60	1.30, 1.98	0.89	0.55, 1.43	0.02
Poisoning	1.74	1.43, 2.12	1.01	0.72, 1.41	0.003
Suicide	1.03	0.96, 1.10	0.92	0.83, 1.02	0.02
Homicide	2.48	1.85, 3.32	1.62	1.09, 2.40	0.12

* SEP, socioeconomic position; HR, hazard ratio; CI, confidence interval.
† Adjusted for age, sex, paternal and maternal age at child's birth, paternal and maternal educational attainment, maternal marital status at child's birth, and time-updated later-life social class.

(95 percent CI: 1.53, 3.61), 2.24 (95 percent CI: 1.46, 3.45), 1.93 (95 percent CI: 1.26, 2.96), and 1.93 (95 percent CI: 1.26, 2.96), respectively.

DISCUSSION

In this large cohort study, we found that, for both females and males, rates of death from stomach, liver, and lung cancer; diabetes; coronary heart disease; stroke; diseases of the respiratory system; and diseases of the digestive system were higher among those from manual compared with nonmanual social classes in early life. The association with stomach cancer was not affected by adjustment for available covariates. For other outcomes, adjusting for educational attainment resulted in greater attenuation of associations than adjustment for later-life social class, but important associations remained after adjustment for age, parental characteristics, own education, and later-life social class. The association of early-life SEP with increased all-cause mortality is often regarded as commonplace, but our findings are notable. They show that, for populations born after World War II (between 1944 and 1966), simply knowing the occupation of subjects' parents in their early life could predict who would be most likely to die from different causes during the first 50 years of life.

There were important sex differences in the associations of early-life social class with some causes of death. Males from manual social classes in childhood had greater rates of death from nervous system conditions, alcoholic cirrhosis, most forms of unintentional injury (in particular, falls and poisoning), and homicide compared with males in nonmanual social classes; for females, there were only weak or no associations of early-life SEP with these outcomes. The effect on these associations of adjusting for educational attainment was greater than the effect of adjusting for later-life social class. These outcomes are more common in general among males, and the sex difference may be driven by differentials in the type of risk-taking behavior among those from lower SEP backgrounds between females and males. Males from lower socioeconomic groups will be more likely than females from these groups to behave in ways that would increase their risk of injury and homicide.

An important strength of this study is its very large size, which enabled us to examine the association of early-life SEP with a wide range of cause-specific mortality outcomes. We also had sufficient power to detect sex differences. Since this was a record linkage study, it is representative of all Swedes who were born between 1944 and 1960 and were alive and still living in Sweden in 1970. We began follow-up in 1970 and therefore did not include deaths occurring less than 10 years after assessment of early-life social class; thus, reverse causality is unlikely to explain our findings. Other studies have tried to compare the magnitude of associations between early-life and later-life SEP, but doing so presents methodological difficulties because the association of later-life SEP with mortality will always be more prone to reverse causality than that of early-life SEP with later mortality. We split the follow-up period into 10-year intervals and adjusted for time-updated later-life SEP; therefore, we

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TABLE 4. Multivariable association of early-life socioeconomic position with all-cause and cause-specific mortality among 1,596,396 ($n = 30,770$ total deaths) Swedes born in 1944–1960 with complete data on all covariates*

Cause of mortality	Adjusted HR† of mortality comparing manual with nonmanual social class in early life									
	Model 1‡		Model 2§		Model 3¶		Model 4#		Model 5**	
	HR	95% CI†	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI
All-cause mortality	1.27	1.24, 1.30	1.23	1.21, 1.26	1.17	1.15, 1.19	1.10	1.08, 1.13	1.08	1.05, 1.10
All cancers	1.09	1.06, 1.12	1.08	1.05, 1.11	1.06	1.03, 1.09	1.03	1.00, 1.08	1.03	0.99, 1.07
Stomach	1.36	1.12, 1.67	1.34	1.10, 1.63	1.27	1.04, 1.55	1.31	1.07, 1.59	1.29	1.06, 1.57
Liver	1.27	1.07, 1.51	1.25	1.05, 1.49	1.21	1.02, 1.44	1.10	0.92, 1.33	1.10	0.92, 1.33
Pancreatic	1.22	1.02, 1.45	1.22	1.02, 1.45	1.17	0.98, 1.39	1.09	0.92, 1.29	1.09	0.92, 1.29
Lung	1.42	1.28, 1.58	1.39	1.25, 1.54	1.32	1.19, 1.47	1.24	1.12, 1.37	1.22	1.10, 1.35
Endocrine disorders	1.36	1.20, 1.54	1.30	1.14, 1.48	1.25	1.10, 1.42	1.15	1.01, 1.31	1.13	0.99, 1.29
Diabetes	1.65	1.40, 1.94	1.64	1.39, 1.93	1.55	1.32, 1.82	1.35	1.15, 1.59	1.31	1.11, 1.55
Diseases of the nervous system	1.24	1.05, 1.46	1.24	1.05, 1.46	1.17	0.99, 1.38	1.00	0.85, 1.12	0.97	0.85, 1.12
Diseases of the circulatory system	1.44	1.37, 1.52	1.42	1.35, 1.49	1.30	1.23, 1.37	1.22	1.16, 1.28	1.18	1.12, 1.25
Coronary heart disease	1.53	1.42, 1.65	1.52	1.41, 1.63	1.39	1.29, 1.49	1.31	1.22, 1.40	1.27	1.18, 1.36
Stroke	1.41	1.27, 1.57	1.32	1.19, 1.47	1.23	1.11, 1.37	1.19	1.07, 1.33	1.16	1.04, 1.30
Subarachnoid hemorrhage	1.42	1.18, 1.71	1.30	1.08, 1.57	1.20	1.00, 1.45	1.15	0.96, 1.37	1.12	0.94, 1.34
Intracerebral hemorrhage	1.60	1.28, 2.00	1.57	1.26, 1.96	1.41	1.13, 1.76	1.24	0.99, 1.57	1.24	0.99, 1.57
Ischemic stroke	1.52	1.14, 2.03	1.49	1.12, 1.99	1.43	1.07, 1.91	1.33	1.00, 1.78	1.33	0.99, 1.78
Stroke—not specified	1.11	0.87, 1.42	1.10	0.86, 1.41	1.05	0.82, 1.35	0.97	0.76, 1.25	0.96	0.75, 1.24
Diseases of the respiratory system	1.65	1.42, 1.92	1.61	1.38, 1.87	1.55	1.33, 1.80	1.31	1.13, 1.52	1.26	1.08, 1.46
Pneumonia or infection	1.64	1.33, 2.02	1.64	1.33, 2.02	1.52	1.23, 1.88	1.27	1.03, 1.59	1.20	0.97, 1.50
Diseases of the digestive system	1.55	1.40, 1.72	1.46	1.31, 1.62	1.35	1.22, 1.50	1.19	1.07, 1.33	1.16	1.04, 1.30

* Based on only those deaths occurring after 1990 and adjusted (where shown) for educational attainment of the participant in 1990.
† HR, hazard ratio; CI, confidence interval.
‡ Model 1: adjusted for age at censoring and sex.
§ Model 2: same as model 1 plus paternal and maternal age at child's birth, paternal and maternal educational attainment, and maternal marital status at child's birth.
¶ Model 3: same as model 2 plus later-life social class based on head of household occupation according to the 1990 Population and Housing Census.
# Model 4: same as model 2 plus educational attainment of participant obtained from the 1990 Population and Housing Census.
** Model 5: same as model 2 plus both later-life social class and educational attainment obtained from the 1990 Population and Housing Census.

always adjusted for later-life SEP assessed within 10 years of death. Because there was a greater possibility of reverse causality with the measure of later-life compared with early-life SEP, we did not attempt to compare the magnitudes of the associations between these two exposures. Nonetheless, our results demonstrate that the effect of early-life SEP on many cause-specific mortality outcomes is independent of later-life SEP and educational attainment.

The association of early-life SEP with increased stomach cancer mortality is most likely mediated via *Helicobacter pylori* infection, which is usually acquired in childhood, is associated with childhood poverty, and is a pathologic agent for stomach cancer (15). It is notable that adjustment for later-life social class or educational attainment had very little effect on this association, supporting the assumption that it was driven by an early-life insult with long-lasting and irreversible effects.

The other cause-specific mortality outcomes that were more common in those from manual social classes are known to be importantly affected by behavioral risk factors. Thus, diabetes is strongly influenced by dietary factors and physical inactivity (16, 17), respiratory deaths and lung cancer are strongly related to smoking (18), and liver and pancreatic cancer are related to smoking and alcohol consumption (19, 20). Similarly, among males, unintentional injury and homicide are often preceded by binge drinking. By contrast, there is no strong evidence for such behavioral risk factors in the etiology of those outcomes not related to childhood SEP—melanoma, breast cancer, brain cancer, and leukemia. Behavioral risk factors are often adopted in childhood or adolescence and, together with their associated metabolic and vascular abnormalities, have been found to explain (mediate) at least some of the association between childhood SEP and cardiovascular disease (21). Thus, our results provide some indirect evidence that early-life SEP is associated with increased mortality risk because of a relation with behavioral risk factors. Since this was a record linkage study, we did not have information on behavioral risk factors and therefore could not directly assess their role as potential mediating factors.

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Our finding that educational attainment had a stronger mediating effect than later-life social class is consistent with findings from a cohort of individuals born in Aberdeen, Scotland, in the 1950s, in which adjustment for educational attainment resulted in marked attenuation of the association between father's social class at birth and adult smoking, binge alcohol drinking, and obesity (22). In that study, adjustment for adult social class and income had a much weaker attenuating effect. Adjustment for educational attainment in that cohort also had a marked attenuating effect on the association of childhood social class with coronary heart disease and stroke (23). These findings suggest that educational attainment specifically, more so than material resources, mediates the association of early-life SEP with adult health outcomes. Sociocultural characteristics of those individuals with higher education, such as self-confidence and ability to access and understand health promotional materials, may be relevant. Moreover, an individual's behavior with respect to smoking, alcohol consumption, diet, and physical activity is likely to be influenced by his or her peers, and educational experiences may determine one's peers at sensitive periods in the life course (late adolescence and early adulthood).

We found no evidence that the effect of early-life SEP was stronger for hemorrhagic compared with ischemic stroke, and we found similar attenuation of these associations with adjustment for later-life social class and education. Thus, we did not find support for the suggestion that childhood infection/diarrhea may play a specific role in the etiology of hemorrhagic stroke (1). However, it should be noted that the main risk factors for cerebral hemorrhage are likely to vary with birth cohort and age. Three earlier studies suggesting a stronger association of early-life SEP with cerebral hemorrhage than with cerebral infarct were conducted in populations born at a time and/or in a country where infant mortality from diarrhea and dehydration was considerably greater than in Sweden during 1944–1960 (12–14).

In conclusion, we found that females and males from manual compared with nonmanual social class backgrounds in early life are more likely to die from a range of disease outcomes—smoking-related cancers, respiratory disease, cardiovascular disease, diabetes—known to be associated with behavioral risk factors. In addition, males from manual compared with nonmanual social class backgrounds are more likely to die from unintentional injury, homicide, and alcoholic cirrhosis. Educational attainment resulted in marked attenuation of these associations, with later-life social class having a less marked attenuating effect. Much of the association between early-life SEP and adult disease may be mediated via behavioral risk factors that can be modified by improved education. Thus, reducing socioeconomic inequalities in health requires inputs in early life and in particular policies that improve the educational achievements of those from the lowest socioeconomic groups.

ACKNOWLEDGMENTS

Conflict of interest: none declared.

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