International Journal of Epidemiology, 2019, 1701–1709
doi: 10.1093/ije/dyz042
Advance Access Publication Date: 30 March 2019
Original article

Miscellaneous

Life-course socioeconomic differences and social mobility in preventable and non-preventable mortality: a study of Swedish twins

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Editorial decision 21 February 2019; Accepted 11 March 2019

Abstract

Background: Despite advances in life expectancy, low socioeconomic status is associated with a shorter lifespan. This study was conducted to investigate socioeconomic differences in mortality by comparing preventable with non-preventable causes of death in 39 506 participants from the Swedish Twin Registry born before 1935.

Methods: Childhood social class, own education, own social class and social mobility were used as separate indicators of socioeconomic status. These data were linked to the Swedish Cause of Death Register. Cause of death was categorized as preventable or non-preventable mortality according to indicators presented in the Avoidable Mortality in the European Union (AMIEHS) atlas. Using Cox proportional hazard models, we tested the association between the socioeconomic measures and all-cause mortality, preventable mortality and non-preventable mortality. Additional co-twin control analyses indicated whether the associations reflected genetic confounding.

Results: The social gradient for mortality was most prominent for the adult socioeconomic measures. There was a social gradient in both preventable mortality and non-preventable mortality, but with an indication of a moderately stronger effect in preventable causes of death. In analyses of social mobility, those who experienced life-time low socioeconomic status (SES) or downward social mobility had an increased mortality risk compared with those with life-time high SES and upward social mobility. Adjustments for genetic confounding did not change the observed associations for education, social class or social mobility and mortality. In the co-twin control analyses of reared-apart twins, the association between childhood social class and mortality

weakened, indicating possible genetic influences on this association.
Conclusions: Our results indicate that there is an association between low adult socioeconomic status and increased mortality independent of genetic endowment. Thus, we do not find support for indirect social selection as the basis for mortality inequalities in Sweden

Key words: Mortality, socioeconomic status, social mobility, social gradient, social selection, co-twin control

Key Messages

- Mortality followed a social gradient with higher mortality for lower socioeconomic groups, independent of preventability, in a large Swedish twin study linked to register-based mortality data.
- There was a similar gradient in both preventable and non-preventable mortality, with an indication of a moderately stronger effect in preventable causes of death, and the adult socioeconomic indicator was more important compared with the childhood measure.
- The impact of socioeconomic factors was stronger in premature mortality (<70 years of age), but the social gradient was present also in late-life mortality.
- Familial confounding could not explain the observed associations between adult socioeconomic circumstances and mortality inequalities, indicating that socioeconomic status in itself may have an effect on mortality.

Introduction

Social stratification in relation to health can be manifested through socioeconomic inequalities in mortality, and the literature consistently shows that low socioeconomic status (SES) is associated with a shorter lifespan.[1–5] Despite this, the underlying pathways are still ambiguous.[6] Perhaps surprisingly, socioeconomic inequalities in mortality are manifested to a similar degree in modern welfare states, such as Sweden, where the societal resources are more equally distributed, than in countries with less generous welfare systems. This incongruity has been described as the Nordic paradox.[7] It has been hypothesized that this paradox may be driven in egalitarian societies by increased selection into socioeconomic groups due to individual characteristics, such as genetics, rather than social background. This could, in turn, lead to greater health inequalities caused by the aggregation of favourable and non-favourable individual characteristics within different socioeconomic groups.[8] That is, as more equal societies provide greater social opportunities for the general population, they may also create a population that is increasingly stratified by ill health, social mobility and genetic endowment. Early health status may directly determine adult SES and health, through a direct health selection.[9] However, there are also plausible indirect selection mechanisms where not health itself but underlying causes, such as personal charactersitics, influence both adult SES and later-life health status.[10] Nonetheless, little is known about how unmeasured factors related to genetic influences and selection mechanisms may affect mortality inequalities.

Genetic influences on socioeconomic inequalities in mortality and longevity have been investigated without consistent results. Familial or genetic influences can be investigated using different methodological approaches, for example based on adoption studies or by using sibling or twin designs. Earlier studies have found that the negative association between SES and mortality remained after taking familial factors into account,[11] that the association was attenuated,[12] or that shared familial factors seemed to explain the association.[13]

As socioeconomically privileged groups have access to more tangible and intangible resources that can be used to prevent poor health, it has been suggested that socioeconomic inequalities in mortality risk should be greater for preventable than for non-preventable causes of death.[14] Preventable causes of death include causes of death that can potentially be avoided through health-care interventions, behavioural changes and injury prevention.[15] This hypothesis is supported by several studies, where a steeper social gradient for preventable than for non-preventable causes of death has been observed.[14,15] However, less is known about the impact of genetic influences and health selection on the observed socioeconomic differences in preventable and non-preventable causes of death. Likewise it

is not clear whether the influences of SES in childhood and mid-life remain into very late life.

The aim of this study was to examine socioeconomic differences in mortality and causes of death in Sweden in a genetically informative design using twins. The specific research questions were as follows. (1) Does the magnitude of socioeconomic differences in mortality in Sweden differ between preventable and non-preventable causes of death? (2) Is the effect of socioeconomic factors different on mid- and late-life mortality? (3) Do the socioeconomic differences in mortality remain when adjusting for genetic confounding?

Methods

Sample and study design

This cohort study included participants in the Swedish Twin Registry (STR) born before 1935 ($n = 39\ 506$). The study sample was retrieved from three STR cohorts: the Old cohort of like-sexed pairs born 1886–1925, the Middle cohort of like-sexed pairs born 1926–1958, and Screening Across the Lifespan Twin study (SALT), of like- and unlike-sexed pairs born 1886–1958.[16,17] Participants in these three cohorts entered the STR at different times. The final sample with complete data on vital status and migration consisted of 36 248 individuals (Supplementary information S1, available as Supplementary data at *IJE* online) were linked through their personal identification number to the Swedish Cause of Death Register from 1960 to December 31, 2014.

Measures

All socioeconomic measures were self-reported, with varying coverage dependent on cohort: childhood social class $n = 19\ 116$, educational attainment $n = 27\ 466$ and occupational class $n = 22\ 725$. A sub-sample of twins that participated in the Swedish Adoption/Twin Study of Aging (SATSA) [19] contributed with information on rearing status that enabled co-twin control analyses on childhood social class. The reared-apart twins were separated before the age 11 [mean age 2.80 standard deviation (SD) 3.08]. Childhood social class was categorized according to the three-level social class classification commonly used during the first half of the 20th century in Sweden, where 3 is the lowest and 1 is the highest.[18] Parental data from the Old cohort was pre-coded into this classification and therefore we harmonized parental occupation from the other STR samples (SALT and SATSA) to this classification. In the statistical analyses, childhood social class was dichotomized by merging levels 1 and 2 due to the small sample size in the co-twin control based solely on the reared-apart twins. Own occupation was coded in accordance with the Swedish socio-economic classification (SEI)[19] and then categorized into a five-level scale following Bukodi *et al.*[20]: (1) unskilled manual employees, (2) skilled manual workers, lower non-manual employees, farmers, (3) self-employed (not including professionals), (4) intermediate non-manuals and (5) higher non-manuals (including professionals). Data on educational attainment was classified using the International Standard Classification of Education (ISCED 2011) into a four-point scale (1= Primary, 2= Lower secondary education, 3= Upper secondary education, 4= Post-secondary non-tertiary, Short-cycle tertiary education, Bachelor's degree, Master's degree or Doctoral degree). Additionally, a four-level social mobility measure was created (High-High, High-Low, Low-Low and Low-High) based on childhood social class as the class of origin and own educational attainment as the adult social location. STR cohort, sex and zygosity were included as covariates.

Causes of death were coded by International Classification of Diseases (ICD) codes (ICD 7 for 1958–1968, ICD 8 for 1969–1986, ICD 9 for 1987–1996 and ICD 10 since 1997) in the Cause of Death Register. Preventable mortality was categorized according to indicators presented by Avoidable Mortality in the European Union (AMIEHS).[21] We included all 45 indicators of potentially preventable conditions presented in the electronic atlas. In addition to this classification, we included causes of death due to injuries.[15] The classification by preventability is presented in Supplementary information S2, available as Supplementary data at *IJE* online. Causes of death not classified as preventable were deemed non-preventable.

Statistical analyses

Person-years of follow-up were calculated from the respective entry dates in the three STR cohorts (Supplementary information S1, available as Supplementary data at *IJE* online) until date of death or censoring due to emigration or end of study on 31 December 2014. Age-standardized mortality rates were estimated as number of deaths over total person-years, using direct standardization with internal age distribution as standard. Cox proportional hazards models were used to estimate hazard ratios (HRs) with 95% confidence intervals (CIs) of the associations of socioeconomic indicators with total mortality, preventable mortality and non-preventable mortality. The four socioeconomic indicators (childhood social class, education, occupation and social mobility) were evaluated separately in the analyses. When analysing preventable mortality, non-preventable causes of death were censored,

and vice versa. Age was used as the time scale in all Cox regression models, hence adjusting the HRs for age. In a first step, the models were applied to the total sample using the twins as individuals, adjusting the precision of estimates (i.e. standard errors and CIs) for dependence between twins in pairs using robust variances including twin-pair identifier variable as a cluster term. The models were additionally adjusted for STR cohort (entry date) and sex.

In a second step to control for familial confounding, a co-twin control analysis was performed by using twin pairs discordant on both the SES indicator (education, occupation, childhood social class and social mobility) and the mortality classification. The co-twin control analysis of childhood social class was performed on the SATSA twin pairs who had been reared apart. Lastly, the models were stratified on zygosity [dizygotic (DZ) and monozygotic (MZ) twins] to separate the effects of possible familial and genetic confounding, respectively. An attenuation in DZ twin pairs would indicate familial confounding, and further attenuation in MZ twin pairs would indicate genetic confounding.

To investigate differences in mid- and late-life mortality, we used age 70 as the threshold for premature mortality based on OECD health statistics.[22] Follow-up time was split at age 70, and separate effects of SES were estimated before and after age 70 using interaction models. Sex differences were investigated by stratifying on sex.

Models only included observations with complete information on all the covariates. The analysis was performed using STATA IC version 14.2.[22]

The study was approved by the regional ethical review board in Stockholm, Sweden.

Results

Descriptive statistics

Participant characteristics are presented in Table 1, including data on number of deaths and mortality rates by preventable and non-preventable causes. The total number of deaths was 29 283, meaning that 81% of the total population was deceased at the end of follow-up. Out of all deaths, 56% were classified as preventable. Mortality rates were higher among males and those with an earlier entry date. Mortality type did not differ as a function of zygosity. Mortality rates were higher in the lower levels of all the socioeconomic indicators.

To validate the self-reported childhood socioeconomic measure we computed intra-pair correlations (Supplementary information S3, available as Supplementary data at *IJE* online). As twins reared together have a shared childhood environment, the intra-pair correlation can be considered an indicator of the reliability of retrospective self-reported measures. The substantial correlation of childhood social class for the reared-together twins (0.90) indicates acceptable reliability of the measure.

Mortality by socioeconomic indicator

We found a social gradient in all-cause mortality with higher mortality in lower levels of each socioeconomic indicator (Supplementary information S4, available as Supplementary data at *IJE* online). Results from models estimating the effect of the socioeconomic measures on preventable and non-preventable mortality are presented in Table 2. There was a similar gradient in both, with an indication of a moderately stronger effect in preventable causes of death. The social gradient in mortality appeared to be somewhat stronger for the adult socioeconomic measures (own education and occupation) than for childhood social class. However, the confidence intervals overlap and possible differences between the different indicators should not be over-interpreted.

When further adjusting for familial and genetic confounding in co-twin analyses, the association between education and social class and all-cause mortality was comparable to the analyses using the total sample. For preventable mortality, the co-twin control entailed a slight attenuation of the associations, but only for the lowest levels of education and social class as well as for childhood social class. For non-preventable mortality, the association was strengthened but the precision was reduced. The association with childhood social class was attenuated in the co-twin control, indicating that the association may partly reflect familial confounding. To differentiate between genetic and familial confounding, we performed the co-twin control analyses on MZ and DZ twin pairs separately. The association was further reduced in the MZ analyses, indicating genetic confounding, but precision was low. No meaningful differences were observed dependent on zygosity for the adult SES measures, indicating that there was little familial confounding, and no genetic confounding.

Mortality by social mobility

In analyses of social mobility (Table 2), the highest mortality was among those with life-time low SES (Low-Low group), as well as for the group with downward social mobility (High-Low). The association between social mobility patterns and mortality was more pronounced for preventable mortality. Similar HRs were observed in the total sample and in the co-twin control analyses.

Mortality before and after 70 years of age

Before age 70, the social gradient in preventable mortality was slightly stronger than for non-preventable

Table 1. Sample characteristics and standardized mortality rates by mortality status

				All-cause mortality		Preventable mortality		Non-preventable mortality	
	n	%	Person-years	Deaths (*n*)	Rate	Deaths (*n*)	Rate	Deaths (*n*)	Rate
	36 248		1 121 001.5	29 283	54.69	16 345	30.389	12 938	24.30
Sex									
Male	16 508	45.5	484 868.95	13 888	67.45	7847	37.80	6041	29.69
Female	19 740	54.5	636 132.56	15 395	47.14	8498	25.97	6897	21.17
Zygosity									
Monozygotic	9788	27.0	311 768.03	8283	54.49	4895	32.30	3388	22.19
Dizygotic	17 874	49.3	566 665.77	15 325	56.15	9242	33.80	6083	22.35
Unknown	2066	5.7	62 933.418	1609	62.44	690	30.72	919	31.72
Dizygotic opposite sex	6520	18.0	179 634.29	4066	48.54	1518	17.85	2548	30.68
STR cohort (entry date)									
Old cohort (1960)	21 450	58.2	646 628.93	20 788	58.30	12 685	35.65	8103	22.65
Middle cohort (1973)	12 289	33.9	451 366.94	6247	42.35	2868	19.00	3379	23.35
SALT (1998)	2509	6.9	23 005.637	2248	49.65	792	16.97	1456	32.68
Education (ISCED)									
1 (Primary)	18 602	51.3	582 032.34	15 630	54.85	9455	32.94	6175	21.91
2 (Lower secondary)	3454	9.5	119 559.96	2660	45.52	1517	25.75	1143	19.77
3 (Upper secondary)	2517	6.9	90 891.674	1387	40.69	781	22.45	606	18.23
4 (University)	1791	4.9	69 388.35	841	34.69	448	17.78	393	16.92
Missing	9884	27.3	259 129.19	8765	66.39	4144	31.42	4621	34.97
Occupation (SEI)									
1 (Unskilled employees)	8036	22.2	262 383.68	6440	53.95	3960	32.51	2480	21.44
2 (Lower non-manuals, Skilled employees)	8954	24.7	299 273.13	6877	51.94	4122	30.49	2755	21.45
3 (Self-employed)	828	2.3	29 763.661	576	43.09	333	24.15	246	18.94
4 (Intermediate non-manuals)	2500	6.9	89 065.081	1608	42.67	929	24.35	679	18.32
5 (Higher non-manuals)	1421	3.9	51 599.318	853	44.61	466	23.83	387	20.78
Missing	14 509	40.0	388 916.64	12 926	61.40	6535	31.44	6391	29.95
Childhood social class									
1 (Low)	8399	23.2	274 955.06	7482	56.63	4547	34.12	2935	22.51
2 (High)	10 167	28.0	340 918.58	8773	51.28	5229	30.41	3544	20.87
Missing	17 682	48.8	505 127.87	13 028	56.14	6569	27.99	6459	28.15
Childhood social class (twins reared apart)									
1 (Low)	388	37.4	11 543.532	370	62.04	248	41.53	122	20.52
2 (High)	649	62.6	22 000.194	573	51.92	335	30.45	238	21.47
Social mobility									
High-High	2557	7.1	97 954.212	1803	40.84	1015	22.91	788	17.93
High-Low	5342	14.7	175 399.99	4854	52.68	2957	31.87	1897	22.26
Low-Low	5567	15.4	180 563.05	5120	56.73	3156	34.47	1964	18.94
Low-High	1210	3.3	46 587.682	861	44.48	498	25.53	363	18.94
Missing	21 572	59.5	620 496.57	16 645	57.63	8719	30.05	7926	27.59

mortality (Supplementary information S5, available as Supplementary data at *IJE* online). The largest effects were observed among those with the lowest educational level. Similar differences in mortality were also observed by social class (Supplementary information S5, available as Supplementary data at *IJE* online). Above age 70, the social gradient appeared to be weaker than before age 70, although still present in both preventable and non-preventable mortality. Higher mortality was observed for the life-time low and downward social mobility groups in both mid- and late-life, although slightly attenuated after age 70. After adjusting for familial factors through co-twin control, the association with mortality remained for education, social class and social mobility. The association between childhood social class and mortality was stronger for premature mortality. In the co-twin control, the

Table 2. Hazard ratios in preventable mortality and non-preventable mortality by educational attainment, social class, childhood social class and social mobility. Showing results from standard cohort analysis and co-twin control analysis, all models adjusted for STR cohort and sex

	Preventable mortality								Non-preventable mortality							
	Cohort analysis		Co-twin control analysis						Cohort analysis		Co-twin control analysis					
			All twins		MZ		DZ				All twins		MZ		DZ	
	HR	CI (95%)	HR	CI (95%)	HR	CI (95%)	HR	CI (95%)	HR	CI (95%)	HR	CI (95%)	HR	CI (95%)	HR	CI (95%)
Education (ISCED)																
1 (Low)	2.02	(1.83, 2.22)	1.97	(1.49, 2.40)	1.93	(1.05, 3.54)	2.00	(1.46, 3.74)	1.56	(1.41, 1.72)	1.63	(1.24, 2.14)	2.52	(1.45, 4.36)	1.44	(1.04, 1.99)
2	1.51	(1.36, 1.68)	1.35	(1.01, 1.79)	1.13	(0.60, 2.14)	1.45	(1.05, 2.01)	1.32	(1.18, 1.47)	1.41	(1.06, 1.88)	1.91	(1.06, 3.45)	1.27	(0.90, 1.78)
3	1.53	(1.36, 1.71)	1.70	(1.28, 2.27)	1.25	(0.69, 2.27)	2.02	(1.45, 2.82)	1.37	(1.21, 1.55)	1.36	(1.03, 1.78)	1.85	(1.12, 3.06)	1.22	(0.87, 1.71)
4 (High)	1		1		1		1		1		1		1		1	
Social class (SEI)																
1 (Low)	1.67	(1.52, 1.85)	1.46	(1.13, 1.88)	1.44	(0.88, 2.36)	1.51	(1.12, 2.04)	1.25	(1.12, 1.40)	1.51	(1.14, 2.01)	1.29	(0.76, 2.21)	1.62	(1.15, 2.29)
2	1.39	(1.26, 1.53)	1.34	(1.06, 1.71)	1.45	(0.91, 2.31)	1.33	(1.00, 1.76)	1.13	(1.02, 1.26)	1.45	(1.11, 1.89)	1.40	(0.85, 2.31)	1.50	(1.09, 2.06)
3	1.05	(0.91, 1.21)	1.01	(0.72, 1.42)	0.73	(0.35, 1.49)	1.07	(0.72, 1.60)	0.92	(0.79, 1.08)	1.05	(0.72, 1.54)	0.82	(0.40, 1.67)	1.14	(0.72, 1.82)
4	1.12	(1.00, 1.25)	1.01	(0.78, 1.29)	0.97	(0.60, 1.56)	1.06	(0.79, 1.43)	0.96	(0.85, 1.09)	1.20	(0.91, 1.60)	1.24	(0.72, 2.13)	1.18	(0.84, 1.65)
5 (High)	1		1		1		1		1		1		1		1	
Childhood social class[a]																
1 (Low)	1.43	(1.21, 1.69)	1.18	(0.77, 1.81)	1.08	(0.49, 2.37)	1.33	(0.79, 2.26)	1.00	(0.80, 1.24)	1.58	(0.89, 2.81)	1.67	(0.61, 4.59)	1.54	(0.76, 3.09)
2 (High	1		1		1		1		1		1		1		1	
Social mobility																
High-High	1		1		1		1		1		1		1		1	
High-Low	1.43	(1.33, 1.54)	1.54	(1.21, 1.96)	1.89	(1.21, 2.96)	1.39	(1.04, 1.87)	1.21	(1.12, 1.32)	1.50	(1.13, 1.99)	1.36	(0.81, 2.26)	1.59	(1.12, 2.24)
Low-Low	1.55	(1.44, 1.67)	1.49	(1.13, 1.97)	1.90	(1.13, 3.18)	1.35	(0.97, 1.88)	1.29	(1.19, 1.40)	1.34	(0.95, 1.88)	1.27	(0.70, 2.32)	1.37	(0.91, 2.08)
Low-High	1.08	(0.97, 1.20)	1.17	(0.83, 1.65)	0.98	(0.52, 1.85)	1.25	(0.82, 1.89)	1.03	(0.91, 1.17)	1.14	(0.74, 1.74)	1.01	(0.48, 2.12)	1.21	(0.72, 2.04)

[a]Analyses on parental social class was performed on a subsample of reared apart twins.

association remained in mortality before age 70 but not in mortality after age 70.

Sex differences

When the models were additionally stratified by sex, no major differences could be observed between men and women (Supplementary information S6, available as Supplementary data at *IJE* online). A slightly elevated mortality for low childhood social class in preventable mortality was only observed for women.

Discussion

In this study of 36 245 twins with information linked to national register-based mortality data, we found a social gradient in mortality, with increased mortality risks among lower socioeconomic groups, regardless of which socioeconomic indicator was tested. The social gradient was evident for both preventable and non-preventable mortality although the association seemed more pronounced for preventable than for non-preventable causes of death. Adult socioeconomic indicators were more important than childhood socioeconomic circumstances. We did not find that familial or genetic confounding could explain the observed associations between adult socioeconomic circumstances and mortality inequalities, indicating that SES in itself may have an effect on mortality. Consistent with previous research[15] there appeared to be a weaker social gradient after age 70 although it was clearly present in both premature and in late-life mortality. We also found that the difference in mortality risk between preventable and non-preventable causes of death was seemingly greater in premature mortality compared with when mortality at all ages was investigated.

Our results clearly show that there are socioeconomic inequalities in mortality in Sweden, despite a system that aims to decrease inequalities. In terms of the Nordic paradox, this study demonstrates that the socioeconomic inequalities in mortality in Sweden cannot solely be attributed to individual characteristics such as genetics or shared familial factors. Specifically, we were not able to observe any familial explanations for the observed association between mortality and the adult socioeconomic indicators (education and occupation) or the two social mobility categories with low adult SES. Further, we could not observe that familial confounding differed between preventable and non-preventable mortality. In contrast, the association between childhood social class and mortality inequalities may be attributed to genetic and familial factors. However, the co-twin analyses based on reared-apart twins were hampered by small sample size, and the low precision allows for interpretations both over and below the 0 within the confidence interval. Our findings are in line with both a Danish[23] and a Swedish twin study[11] on the influence of educational attainment on all-cause mortality, extending those findings to preventable and non-preventable mortality. Yet, the findings are inconsistent with previous studies on the reared-apart sample[24] which did not find any associations between occupation, education and a measure of chronic illness. Given the strong associations between chronic illnesses and mortality, these findings are incongruent with the findings from our study. However, the differences might be due to the smaller sample size in the previous study. Moreover, the previous study encompassed much fewer individuals of old age and, consequently, fewer individuals with chronic conditions. Further, they did find modest familial influences on the association with self-rated health. We found no indication of familial or genetic influence on the association between the adult socioeconomic indicators and mortality. This illustrates the complexity of the relationship between SES and health, where the importance of both the socioeconomic indicators and the different aspects of health varies depending on the measures used, the method, and the characteristics of the study population. Although adult social class and education were associated with mortality even in the co-twin analyses, this was not the case for childhood social class. Early socioeconomic influences observed at population level seem instead to be a consequence of familial influences beyond the socioeconmic circmstances, indicating an indirect selection driven by personal attributes. This supports previous findings of no effect of childhood social class on mortality inequalities,[25] and was further supported by the social mobility results. The groups with life-time low SES and downward social mobility had the greatest disadvantage, which highlights the importance of a life-course perspective on the importance of socioeconomic[26] and lifestyle-factors[27] on mortality. This also suggests that socioeconomic disadvantages in childhood can be compensated for by upward social mobility. It is particularly interesting that mortality inequalities for the downward social mobility group remained after adjustments for genetic confounding, considering that their co-twin by default had a life-time high socioeconomic trajectory. The increased mortality for the downward social mobility group could therefore not be attributed to childhood social class or to familial influences. It is possible that early health differences within the twin pair led to separate socioeconomic trajectories through direct health selection.

The main strength of this study is the large data set with genetically informed data that was linked to national register-based mortality data. Still, our study has several potential limitations. First of all, we made assumptions

based on a life-course follow-up of socioeconomic factors and mortality, however, we were not able to account for unobserved health differences early in life. Mortality selection may have taken place before the study sample entered the STR. In particular, preventable causes of death may have occurred at younger ages, which may in part account for the weak association observed between childhood social class and preventable mortality. Another limitation is that the available socioeconomic measures were from different sources and that most were also self-reported. The childhood social class measure was also less precise compared with the adult social-class measures. This could additionally have contributed to the weak effects of childhood socioeconomic circumstances. There may also be specific social circumstances in Sweden that make these results difficult to generalize to other populations. Nevertheless, the birth cohorts that constitute our sample grew up in a society characterized by less equality and wealth than contemporary Sweden. These cohorts also preceded the large educational reforms that came about in Sweden in the 1960's and 1970's[28] that made higher education more accessible for groups without financial resources. Our classification of preventable and non-preventable causes of death used a broad definition of preventable mortality in order to include causes of death that were not only related to health care interventions, but also capture preventability related to health behaviours and injuries. Plug et al.[29] found that mortality inequalities were more prominent in preventable causes of death related to health care compared with other types of preventable causes of death such as those related to risky health behaviours. It is therefore possible that we could have observed a larger difference between preventable and non-preventable mortality by only including health care related preventable causes of death. There is also the possibility of differential misclassification of causes of death. This may in particular be a concern for death occurring in late and very late life when older individuals often have multiple diseases, and we only used the main causes of death. Such misclassification would have resulted in a bias toward the null. We did not include any covariates related to health beaviours such as smoking, alcohol consumption or physical activity. Instead, by dividing cause-specific mortality into preventable and non-preventable mortality, these factors were already recognized.

In conclusion, separating causes of death based on preventability provided further insights into the sources of mortality inequalities. Additional analyses adjusting for possible familial confounding did not explain the observed associations, which further confirmed the relationship between low socioeconomic status in itself and increased mortality. The combined results demonstrated the importance of the adult socioeconomic influences on mortality inequalities. Our results indicate the importance of directed efforts to reduce the impact of socioeconomic inequalities on health and mortality. Moreover, our results show that the substantial socioeconomic inequalities in mortality in Sweden, a country characterized by strong egalitarian ambitions, cannot be attributed to indirect social selection based on individual characteristics but instead reflects a direct selection predicated on health. These findings re-ignite the notion of the Nordic paradox. If social selection can't explain the relatively high rates of mortality inequalities in Sweden, then what can?

Supplementary Data

Supplementary data are available at *IJE* online.

Funding

This work was supported by Swedish Research Council: Grant (2013–08689), and the Swedish Research Council for Health, Working Life, and Welfare (FORTE): Grant 2013–02292.

Acknowledgments

We acknowledge The Swedish Twin Registry for access to data. The Swedish Twin Registry is managed by Karolinska Institutet and receives funding through the Swedish Research Council under the grant no 2017–00641.

Conflict of interest: None declared.

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