RESEARCH REPORT

Childhood and adulthood socioeconomic position across 20 causes of death: a prospective cohort study of 800 000 Norwegian men and women

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Objective: To assess the impact of childhood and adulthood socioeconomic position (SEP) across 20 causes of death in a large population-wide sample of Norwegian men and women.
Methods: Census data on parental occupational class from 1960 and data from the tax register on household income in 1990 were linked to the death register for 1990–2001, and 20 causes of death were studied. Relative indices of inequalities were computed. Norwegians in the age group 0–20 years in 1960 and still alive in 1990 were followed for deaths in 1990 to 2001. This follow up involved 795 324 individuals (78%) and 20 887 deaths.
Main results: In men most support for an effect of childhood socioeconomic position was found for stomach cancer, lung cancer, coronary heart disease, ''other violent death'', and all causes of death. In women similar effects were found for lung cancer, cervical cancer, coronary heart disease, chronic obstructive pulmonary disease, and all causes of death.
Conclusions: The effect of childhood socioeconomic position relative to adulthood varies by cause of death. Although there are some exceptions, the patterns in men and women are generally similar.

Adverse social circumstances in childhood have been related to an increased risk of death in adulthood in several studies.[1] Evidence points to a reasonably consistent picture of the contribution of childhood socioeconomic conditions to adult mortality risk. These effects are particularly strong for stomach cancer and haemorrhagic stroke. For coronary heart disease and chronic obstructive pulmonary disease, poor socioeconomic conditions over a lifetime seem to increase the risk in a cumulative manner. For smoking related and external causes of death, the relative effects of childhood and adult influences seem to be more context-specific and to vary between populations.

Most of these studies have looked at the issue in men and for a restricted range of causes, predominantly cardiovascular outcomes. When women have been included in the studies few outcomes have been studied, owing to the small number of deaths. We know little about whether childhood socioeconomic position (SEP) influences the cause-specific mortality pattern in adult life similarly for men and women. There are several reasons why the social environment in childhood may affect men and women differently. For example, there may be sex differences in the social patterning of risk, such as health related behaviour.[2–8] Also, socioeconomic influences on factors unique to women, such as those related to reproduction, may generate differences.

Power *et al* recently investigated this issue among mothers of the 1958 cohort in Britain.[9] They examined eight causes within a total of 3132 deaths. Effects of childhood SEP on mortality were generally smaller than had been seen for men, perhaps because of the older average age at death in women.[10 11] Large studies need to be conducted in order to generate more robust evidence. In this study we took advantage of a large nationwide sample of men and women from a register linkage to compare the influence of childhood and adulthood SEP across a wide range of causes of death to see whether the pattern varied by sex and to extend current evidence to outcomes not previously studied.

METHODS

The study included some of the 20 most common causes of death during the years 1990 to 2001. All death certificates were registered with Statistics Norway, without any missing cases. Deaths were coded according to the *International Classification of Diseases* (ICD): ICD-9 until 1996 and ICD-10 onwards (codes in ascending order): stomach cancer (ICD-9: 151, ICD-10: C16), large bowel and rectal cancer (ICD-9: 153–154, ICD-10: C18–C21), pancreatic cancer (ICD-9: 157, ICD-10: C25), lung cancer (ICD-9: 162, ICD-10: C34), breast cancer (ICD-9: 174, ICD-10: C50), prostate cancer (ICD-9: 185, ICD-10: C61), malignant melanoma (ICD-9: 172, ICD-10: C43), cervical cancer (ICD-9: 180, ICD-10: C53), ovarian cancer (ICD-9: 183, ICD-10: C56), diabetes (ICD-9: 2500–2509 and ICD-10: E100–E149), psychiatric causes (ICD-9: 290, 292–302, 304, 306–319 and ICD-10: F00–F09, F11–F69), alcohol related diseases (ICD-9: 291, 303, 305, 571 and ICD-10: F10, K70, Y91), coronary heart disease (ICD-9: 410–414 and ICD-10: I20–I25), haemorrhagic stroke (ICD-9: 430, 431 and ICD-10: I60, I61), thrombotic stroke (ICD-9: 290.4, 433, 434, 435 and ICD-10: I63, I67.8), aortic aneurysm (ICD-9: 441 and ICD-10: I710–I719), chronic obstructive pulmonary disease (ICD-9: 490–496 and ICD-10: J40–J47), asthma (ICD-9: 493 and ICD-10:J450–J459), suicide (ICD-9: E950–E959 and ICD-10: X60–X84), and other violent deaths (ICD-9: 800–999 and ICD-10: V01–X59, Y85–Y86).

The population included all Norwegian citizens in the age group 0 to 20 years in 1960 who lived in households with fewer than 13 people, lived in private households together with both parents (94.7%), and who had survived until 1990 at the time when adult SEP was recorded, in all 1 025 573 individuals (table 1). In the regression analyses, those with fathers working as farmers (17.9%) and those with missing data on occupation in 1960 or income in 1990 were excluded from the analysis. This study population included 74.3% of all 0 to 20 year olds in

Abbreviations: RII, relative index of inequality; SEP, socioeconomic position

Table 1 Percentage distribution (%) and age adjusted all cause mortality rates, 1990 to 2001 (per 10 000 person/years), according to father's occupational class in 1960 and household income in 1990 for the included and excluded individuals

	Men			Women		
Variable	**Total**	**Number of deaths (%)**	**Rate**	**Total**	**Number of deaths (%)**	**Rate**
Childhood, 1960:						
Upper non-manual	31 884	798 (6.0)	22.6	29 714	507 (6.1)	15.6
Lower non-manual	103 315	2691 (19.3)	26.3	96 160	1530 (19.6)	16.4
Skilled manual	147 754	4781 (27.7)	30.5	135 920	2499 (27.7)	17.8
Unskilled manual	146 966	5332 (27.5)	33.2	135 058	2764 (27.5)	19.2
*Missing occupation**	*6774*	*265 (1.3)*	*33.3*	*8184*	*177 (1.7)*	*19.0*
Farmers	*97 517*	*3 035 (18.3)*	*25.7*	*86 327*	*1560 (17.6)*	*15.7*
Adulthood, 1990:						
Highest quartile	139 133	3346 (26.0)	20.1	109 438	1802 (22.3)	13.1
Middle high quartile	133 679	3284 (25.0)	22.2	114 869	1783 (23.4)	14.2
Middle low quartile	127 997	3182 (24.0)	24.3	120 512	1765 (24.5)	15.0
Lowest quartile†	118 190	4652 (22.1)	40.2	130 308	2617 (26.5)	22.5
Missing	*15 211*	*2427 (2.9)*	*156.7*	*16 236*	*1070 (3.3)*	*68.7*
Total	534 210	16 891 (100.0)	29.2	491 363	9037 (100.0)	17.4

*Farmers and those missing data on occupation in 1960 and income in 1990 were excluded from analysis.
†Lowest corresponds to fewer than 117 300 Norwegian krone (NOK), middle low 167 300 NOK, middle high 220 450 NOK, and highest 9 864 500 NOK.

1960 who had survived until 1990. Deaths were followed over the period 3 November 1990 to 31 December 2001, at a time when the cohort would have reached the age of 41 to 61 years. In the 1960 census, children who in official registers were recorded as living in their parental household could be linked to their parents' occupational class. An upper cut off at 20 years was used because only a few had married (0.3%) and would then be registered with a household other than their parental one.[12]

Occupational class in 1960 was divided into four categories: upper non-manual, lower non-manual, skilled manual, and unskilled manual. Farmers (18%) and those with missing occupation (1.5%) were excluded from the analysis. The income measure used in the 1990 census was earned gross income, which is the same as pensionable income and included income from work inside and outside ordinary jobs and income from self employment, deflated to 1990 Norwegian kroner (NOK), and divided by the square root of household members (consumer units). Zero or negative income was treated as missing, affecting 3%. Income was divided into four groups defined by the quartiles.

The relation between mortality and a childhood or adult SEP indicator was summarised by means of the relative index of inequality (RII). This index measures the size of differences in mortality across all SEP levels.[13] The RIIs were fitted by Cox's proportional models, in which each socioeconomic category was given an SEP score, indicating the proportion of the population having a higher SEP level. The SEP score was determined by taking half the proportion of people with the same SEP level plus the proportion with a higher level. In this population the scores for the 1960 SEP measure were: upper non-manual 0.037, lower non-manual 0.195, skilled manual 0.487, and unskilled manual 0.829. For 1990 the scores were: highest income 0.125, second highest 0.375, third highest 0.625, and lowest income 0.875. The SEP score was then treated as a continuous variable in the regression models, and the exponentiated β coefficient associated with the SEP score yields the RII. The RII can be interpreted as comparing the hypothetically worst off with the best off person in the SEP hierarchy. The Cox proportional hazard regression was considered more appropriate than logistic regression because the study involved follow up of deaths in a period of 11 years. However, as the risk of death in the population is rather low for this age group, choosing any of these models is likely to have given similar results.

RESULTS

In all, 55.2% of the cohort grew up with manual working fathers (table 1). Those with farmers as fathers had similar mortality to those with lower non-manual in men and upper non-manual in women. Those with missing information on father's occupational class (1.5%) had increased mortality. The 3% with missing income in 1990 had greatly increased mortality, particularly the men.

There were 13 588 deaths among men and 7299 deaths among women in the follow up period (table 2). The pattern of the relative contribution of childhood and adulthood social conditions and size of effect varied with respect to the cause of death. For unadjusted total mortality, the RII of father's occupational class was 1.48 in men and 1.30 in women.

The effects presented take all categories of the social measures into account and represent the increased risk in the hypothetically most disadvantaged over the least disadvantaged group. In men there were particularly strong effects of childhood circumstances for stomach cancer, lung cancer, psychiatric causes, alcohol related causes, coronary heart disease, chronic obstructive pulmonary disease (COPD), ''other non-violent'' causes, and all causes of death. In men, there was an effect of adult social position for lung cancer, prostate cancer, psychiatric causes, alcohol related causes, coronary heart disease, COPD, asthma, diabetes, suicide, ''other violent'', and all causes of death.

In women the pattern appeared to be similar to men for most causes of death. There were fewer deaths in women, and hence the confidence intervals were wider. The point estimates differed somewhat in terms of size of effect, but the relative importance of childhood and adulthood SEP were to a large extent similar to that of men for most causes. In women there was an effect of childhood SEP for lung cancer, cervical cancer, coronary heart disease, COPD, suicide, and all causes. For suicide there was a protective effect of disadvantage in childhood. Stomach cancer had smaller and insignificant effect sizes in women although the relative importance was similar to men. Cervical cancer was related to disadvantage in both childhood and adulthood. There were similar patterns and effect sizes in men and women for psychiatric causes; those

Table 2 Age adjusted relative indices of inequality in 1960* and 1990 for cause-specific mortality in 1990 to 2001 in men and women and both sexes, and interaction between sexes for the effect of 1960

	Men			Women				
Cause of death	Deaths (n)	1960 RII	1990 RII	Deaths (n)	1960 RII	1990 RII	Both sexes adjusted for age and sex, 1960 RII	p Value†
Stomach cancer	206	2.86 (1.70 to 4.83)	1.56 (0.93 to 2.60)	111	1.61 (0.81 to 3.19)	1.05 (0.52 to 2.10)	2.34 (1.54 to 3.54)	0.161
Cancer large bowel and rectum	499	1.03 (0.75 to 1.42)	1.30 (0.93 to 1.80)	406	1.30 (0.94 to 1.91)	1.39 (0.97 to 1.99)	1.16 (0.92 to 1.47)	0.306
Cancer pancreas	208	1.27 (0.77 to 2.08)	1.17 (0.70 to 1.98)	128	1.67 (0.88 to 3.15)	1.22 (0.65 to 2.31)	1.41 (0.95 to 2.08)	0.492
Lung cancer	839	1.78 (1.39 to 2.29)	2.30 (1.78 to 2.97)	545	1.74 (1.28 to 2.37)	1.43 (1.05 to 1.96)	1.77 (1.46 to 2.15)	0.837
Breast cancer	–	–	–	1137	0.91 (0.74 to 1.12)	1.20 (0.97 to 1.50)	–	–
Cervical cancer	–	–	–	263	2.04 (1.30 to 3.20)	2.13 (1.32 to 3.44)	–	–
Ovarian cancer	–	–	–	365	1.04 (0.72 to 1.51)	1.21 (0.83 to 1.77)	–	–
Prostate cancer	100	1.18 (0.58 to 2.40)	2.38 (1.17 to 4.83)	–	–	–		–
Malignant melanoma	267	0.82 (0.53 to 1.26)	0.80 (0.51 to 1.26)	144	1.09 (0.60 to 1.98)	1.19 (0.65 to 2.19)	0.90 (0.64 to 1.28)	0.449
Psychiatric causes	471	1.61 (1.15 to 2.25)	22.20 (12.98 to 37.97)	90	1.93 (0.89 to 4.16)	10.95 (2.22 to 54.08)	1.66 (1.22 to 2.25)	0.675
Alcohol related	945	1.90 (1.50 to 2.42)	10.41 (7.73 to 14.02)	248	1.49 (0.95 to 2.36)	12.42 (6.82 to 22.66)	1.81 (1.47 to 2.23)	0.362
CHD	2431	2.11 (1.81 to 2.44)	2.96 (2.54 to 3.45)	402	2.57 (1.78 to 3.72)	3.56 (2.40 to 5.27)	2.17 (1.89 to 2.49)	0.331
Thrombotic stroke	73	0.72 (0.31 to 1.65)	1.25 (0.52 to 3.03)	25	1.63 (0.39 to 6.92)	1.84 (0.43 to 7.92)	0.89 (0.43 to 1.82)	0.340
Haemorrhagic stroke	318	1.39 (0.93 to 2.09)	3.14 (2.05 to 4.81)	250	1.69 (1.07 to 2.67)	2.90 (1.81 to 4.65)	1.51 (1.12 to 2.05)	0.537
Aortic aneurysm	108	1.21 (0.61 to 2.40)	1.91 (0.94 to 3.88)	15	–	–	–	–
COPD	154	2.18 (1.21 to 3.95)	5.58 (2.82 to 11.04)	163	2.99 (1.67 to 5.37)	9.37 (4.72 to 18.60)	2.56 (1.69 to 3.89)	0.449
Asthma	44	1.37 (0.46 to 4.04)	5.52 (1.60 to 19.03)	64	2.11 (0.85 to 5.26)	8.94 (3.06 to 26.10)	1.75 (0.87 to 3.52)	0.496
Diabetes	245	1.45 (0.91 to 2.29)	7.72 (4.53 to 13.13)	92	1.23 (0.58 to 2.59)	6.44 (2.64 to 15.69)	1.38 (0.93 to 2.04)	0.727
Suicide	1044	1.01 (0.81 to 1.27)	2.14 (1.69 to 2.71)	402	0.64 (0.45 to 0.91)	2.29 (1.54 to 3.40)	0.89 (0.74 to 1.07)	0.037
Other violent	1800	1.51 (1.28 to 1.79)	2.76 (2.29 to 3.32)	493	0.83 (0.61 to 1.14)	2.37 (1.65 to 3.38)	1.32 (1.14 to 1.54)	0.002
All causes	13 588	1.48 (1.39 to 1.57)	2.67 (2.50 to 2.86)	7299	1.30 (1.19 to 1.41)	2.20 (2.01 to 2.41)	1.41 (1.34 to 1.48)	0.021

Individuals aged 41 to 61 years at end of follow up.
*Farmers and missing occupation excluded from all analyses.
†Interaction between sexes for the effect of SEP 1960.
CHD, coronary heart disease; COPD, chronic obstructive pulmonary disease; RII, relative indices of inequality.

Table 3 Age adjusted relative indices of inequality in 1960 and 1990 for cause specific mortality 1990–2001adjusted for the other year's socioeconomic position and interaction in men and women

	Men			Women			Both sexes adjusted for age and sex,
Cause of death	1960 adjusted	1990 adjusted	Interaction*	1960 adjusted	1990 adjusted	Interaction	1960 RII
Stomach cancer	2.76 (1.60 to 4.75)	1.37 (0.82 to 2.30)	0.945	1.63 (0.80 to 3.30)	0.99 (0.49 to 2.00)	0.445	2.28 (1.49 to 3.51)
Cancer large bowel and rectum	0.96 (0.69 to 1.33)	1.30 (0.94 to 1.82)	0.102	1.27 (0.88 to 1.82)	1.36 (0.95 to 1.94)	0.418	1.08 (0.85 to 1.39)
Cancer pancreas	1.34 (0.79 to 2.25)	1.13 (0.67 to 1.91)	0.391	1.60 (0.83 to 3.07)	1.17 (0.62 to 2.22)	0.178	1.43 (0.95 to 2.15)
Lung cancer	1.53 (1.17 to 2.00)	2.17 (1.68 to 2.82)	0.814	1.73 (1.25 to 2.39)	1.36 (0.99 to 1.86)	0.986	1.62 (1.32 to 1.99)
Breast cancer	–	–	–	0.90 (0.72 to 1.11)	1.22 (0.98 to 1.51)	0.025	–
Cervical cancer	–	–	–	1.77 (1.09 to 2.87)	2.01 (1.24 to 3.25)	0.153	–
Ovarian cancer	–	–	–	1.03 (0.71 to 1.51)	1.21 (0.83 to 1.76)	0.101	–
Prostate cancer	1.04 (0.50 to 2.14)	2.37 (1.16 to 4.83)	0.460	–	–	–	–
Malignant melanoma	0.82 (0.53 to 1.29)	0.82 (0.52 to 1.30)	0.145	1.20 (0.65 to 2.21)	1.17 (0.63 to 2.16)	0.983	0.94 (0.65 to 1.34)
Psychiatric causes	1.10 (0.70 to 1.72)	21.94 (12.79 to 37.65)	0.949	1.71 (0.44 to 6.66)	10.28 (2.06 to 51.32)	0.251	1.15 (0.79 to 1.76)
Alcohol related	1.16 (0.87 to 1.54)	10.23 (7.58 to 13.81)	0.531	1.43 (0.70 to 2.08)	12.24 (6.70 to 22.37)	0.950	1.16 (0.90 to 1.50)
CHD	1.86 (1.59 to 2.18)	2.74 (2.35 to 3.19)	0.650	2.54 (1.69 to 3.83)	3.28 (2.21 to 4.86)	0.482	1.94 (1.67 to 2.25)
Thrombotic stroke	0.70 (0.29 to 1.69)	1.32 (0.54 to 3.21)	0.267	1.34 (0.31 to 5.85)	1.78 (0.41 to 7.76)	0.137	0.83 (0.39 to 1.76)
Haemorrhagic stroke	1.31 (0.84 to 2.02)	3.04 (1.97 to 4.67)	0.296	1.35 (0.84 to 2.17)	2.81 (1.75 to 4.52)	0.641	1.33 (0.96 to 1.83)
Aortic aneurysm	1.02 (0.50 to 2.10)	1.90 (0.93 to 3.89)	0.715	–	–		0.92 (0.47 to 1.81)
COPD	1.76 (0.87 to 3.56)	5.21 (2.62 to 10.39)	0.013	2.35 (1.18 to 4.68)	8.75 (4.39 to 17.44)	0.154	2.03 (1.24 to 3.32)
Asthma	0.95 (0.28 to 3.25)	5.55 (1.59 to 19.34)	0.128	1.33 (0.47 to 3.79)	8.73 (2.98 to 25.59)	0.110	1.14 (0.51 to 2.53)
Diabetes	1.00 (0.59 to 1.68)	7.72 (4.52 to 13.19)	0.391	1.31 (0.55 to 3.09)	6.28 (2.56 to 15.37)	0.833	1.07 (0.69 to 1.67)
Suicide	0.87 (0.69 to 1.11)	2.18 (1.72 to 2.77)	0.007	0.63 (0.42 to 0.92)	2.41 (1.62 to 3.59)	0.204	0.80 (0.65 to 0.98)
Other violent	1.30 (1.07 to 1.57)	2.66 (2.21 to 3.21)	0.854	0.81 (0.57 to 1.16)	2.42 (1.69 to 3.47)	0.274	1.17 (0.99 to 1.38)
All causes	1.25 (1.17 to 1.34)	2.60 (2.43 to 2.78)	0.078	1.20 (1.10 to 1.31)	2.16 (1.98 to 2.36)	0.170	1.23 (1.17 to 1.30)

Individuals aged 41 to 61 years at end of follow up.
*Interaction between 1960 and 1990 socioeconomic position. Farmers and missing occupation excluded from all analyses.
CHD, coronary heart disease; COPD, chronic obstructive pulmonary disease; RII, relative indices of inequality.

being disadvantaged in childhood had increased mortality risk compared with the advantaged, while the disadvantaged in adulthood had a 10- to 20-fold increased mortality risk. Coronary heart disease had similar associations with childhood and adulthood social circumstances, but the effect estimate was somewhat greater in women at both time points. The pattern was similar in men and women for COPD, with a greater effect of adulthood social position. For suicide in women, a disadvantaged background was protective, but this effect was reversed for adult social position. In men there was no association with childhood social position, while advantaged social position in adulthood was protective. For other violent causes, disadvantaged adult social position increased the risk in both men and women, while men with a disadvantaged childhood background had an increased risk. When both sexes were analysed together, an effect of childhood SEP was found for stomach cancer, lung cancer, psychiatric causes, alcohol related causes, coronary heart disease, haemorrhagic stroke, COPD, ''other violent causes'', and all causes of death. There were no significant sex differences in the effect of childhood SEP except for suicide, violent causes, and all causes of death.

In table 3, childhood and adulthood SEP were mutually adjusted. The relative importance of childhood and adulthood SEP remained similar for some causes of death. However, for psychiatric causes the childhood effect was explained by adult SEP, and was not significant in the adjusted model. This was seen in both men and women. In men, the same was observed for alcohol related deaths and COPD. For adult SEP the effect estimates were similar in the unadjusted and adjusted models except for haemorrhagic stroke in men. For all causes of death, the adjusted effect of childhood fell to 1.23 in men and 1.19 in women, but was still significant. When both sexes were analysed together there were significant effects of childhood for stomach cancer, lung cancer, coronary heart disease, COPD, and all causes of death.

DISCUSSION

Childhood SEP was related to various causes of death in both men and women. In men we found most support for an effect of childhood SEP on stomach cancer, lung cancer, coronary heart disease, ''other violent causes'', and all causes of death. In women similar effects were found for lung cancer, cervical cancer, coronary heart disease, chronic obstructive pulmonary disease, and all causes of death. The pattern for the effect of childhood and adult social position was similar in both sexes for most causes except for suicide, where women appeared to experience a protective effect of disadvantage in childhood.

This large study covers the whole Norwegian population and follows up deaths in late adulthood. It adds comparative data on women, which are to a large extent lacking in earlier studies. It also provides a much more detailed account of particular causes of death than previous studies. Data on childhood and adulthood SEP were taken from registries and collected independently from the outcome measure and should thus not be prone to recall bias favouring one time point.

The low correlation between parental occupational class and adulthood household income is probably a reflection of a high degree of income mobility in Norway in the late 20th century among people from working class backgrounds, and that low income is related to social changes in adulthood such as marital status and participation in the labour market.[14] Occupational class was not available in the1990 census, and we would expect a larger correlation had occupation been used at both time points. We hold that both measures reflect key dimensions of social stratification that were relevant in Norway in the period studied. During the 1980s income inequality increased in Norway. As household income may fluctuate through the life course, we think it is a valid indicator of SEP at the stage of the life course when it is measured.[15] Education is related to early life exposures, particularly in adolescence and early adulthood.[16] For this reason we chose not to use education as an indicator of adult socioeconomic position.

Farmers were deliberately excluded from the analysis because their appropriate position in any socioeconomic hierarchy is less clear. We checked whether including them between manual and non-manual social class gave different results. This changed the effect estimates slightly for some causes, but the relative importance of childhood and adulthood social position measures and the concordance between men and women did not change. Household income was recorded in 1990 at the time when follow up of deaths started. This could result in reverse causality, as sick people would have a lower income.[17] We investigated whether there was any change in effect when changing the year of starting the follow up of deaths to 1992 or 1994 rather than 1990. This produced similar estimates. Furthermore, there would be more deaths at the end of the follow up period, at a time when participants were older, and any effect of reverse causality would play a greater role for deaths in the beginning of this period when there were fewer deaths. Thus we do not think reverse causality has seriously biased the results.

The relative contribution of childhood and adulthood socioeconomic circumstances to the risk of various outcomes of death may to some extent be historically contingent and reflect biological, behavioural, and development processing operating over the life course of individuals born in the 1940s and 1950s.[18] The cause-specific pattern was strikingly similar between men and women, with a statistically significant interaction value for suicide, other violent causes, and all causes only. In a recent study of childhood socioeconomic status, women born in highly educated families had increased risk compared with men, independent of adulthood.[19] The investigators suggest several explanations, all of which may have a different social pattern determined by sex: downward mobility, not meeting high demands set by highly educated parents, psychological distress, and mental disorders.

Important risk factors such as smoking differ in their social patterning between countries and time periods. Smoking related causes such as lung cancer and COPD had similar effects of childhood and adulthood in both sexes. In recent decades smoking prevalence has declined but less so among socially disadvantaged people.[20] This decline took place earlier in men than in women in Norway. Results from our study indicate that even if these declines have taken place, the relative importance of childhood and adulthood and the social patterning appear similar between the sexes.

The results of our study are to a large extent in accordance with the other studies that have addressed this issue. For all causes of death, increased risk of childhood disadvantage has been found in most other studies.[1] In the Collaborative Study, men whose fathers had manual occupations or those who had more siblings had a higher mortality from stomach cancer.[21 22] Effects of childhood conditions have also been found for lung cancer[21–23] and respiratory disease, but there has been no consistent evidence for prostate cancer or malignant melanoma. In the Oslo Mortality Study a strong effect of childhood conditions was found for psychiatric causes, and for alcohol related causes effects have mostly been found in northern Europe.[3 23] In a recent cohort study in Norway of deaths in early adulthood of cohorts born between 1955 and 1970, an independent effect of childhood SEP was found for cardiovascular causes.[24] For coronary heart disease, childhood circumstances have been implicated in most studies. There is limited evidence on cervical cancer, ovarian cancer, pancreatic cancer, aortic aneurysm, asthma, or diabetes.

What this paper adds

- Various studies have looked at the impact of childhood and adulthood socioeconomic position and adult health, but the evidence across causes of death and sex is limited.
- This study provides extended evidence on a number of causes of death not previously studied.
- The cause-specific pattern is similar in both sexes for most causes of death.

Policy implications

- Policies addressing health inequalities for specific outcomes should consider the relative impact of social inequalities in childhood and adulthood.
- In order to fully acknowledge the impact that social inequalities may have on health throughout life epidemiological studies need to consider the cause-specific pattern.

The major discrepancy between this study and previous work[1] is with respect to haemorrhagic stroke, where earlier studies have found a marked influence of childhood SEP or other indicators of childhood influences, such as the number of siblings.[21] We also analysed haemorrhagic stroke without subarachnoid haemorrhage. When both childhood and adulthood were included in the model, the estimate for childhood was 1.38 (0.74 to 2.58) in men and 0.69 (0.30 to 1.55) in women. Recently evidence has emerged that severe diarrhoea and dehydration in childhood may result in higher blood pressure in later life.[25 26] It is hypothesised that the ability to respond to an acute episode of severe diarrhoea or dehydration in infancy by retaining sodium would have short term survival advantages when challenged by a repeat attack of the condition, but in the long term could lead to raised blood pressure. The current cohort was born at a time when infant mortality had already declined markedly, when it may not have experienced a high prevalence of the socially patterned experience of severe diarrhoea and dehydration, and thus childhood influences on haemorrhagic stroke may be less marked. Furthermore, at the relatively young age at death of this cohort many of the haemorrhagic strokes would be subarachnoid haemorrhages, which have a different aetiology from intracerebral haemorrhages occurring in later life.

In this large study of the relative importance of childhood and adulthood SEP across a broad array of causes, findings from earlier studies on the cause-specific pattern are largely confirmed and seem to apply to both men and women. With some notable exceptions, such as suicide and other violent causes—where the social patterning may differ according to sex—it appears that the cause-specific pattern seems to be similar, although the effect estimates varied somewhat.

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