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Association of socioeconomic position with smoking and mortality: the contribution of early life circumstances in the 1946 birth cohort

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ABSTRACT

Background A large part of the socioeconomic mortality gradient can be statistically accounted for by social patterning of adult health behaviours. However, this statistical explanation does not consider the early life origins of unhealthy behaviours and increased mortality risk.

Methods Analysis is based on 2132 members of the MRC National Survey of Health and Development with mortality follow-up and complete data. Smoking behaviour was summarised by pack-years of exposure. Socioeconomic circumstances were measured in childhood (father's social class (age 4), maternal education (age 6)) and age 26 (education attainment, home ownership, head of household social class). We estimated the direct effect of early circumstances, the indirect effect through smoking and the independent direct effect of smoking on inequality in all-cause mortality from age 26 to 66.

Results Mortality risk was higher in those with lower socioeconomic position at age 26, with a sex-adjusted HR (relative index of inequality) of 1.97 (95% CI 1.18 to 3.28). Smoking and early life socioeconomic indicators together explained 74% of the socioeconomic gradient in mortality (the gradient). Early life circumstances explained 47% of the gradient, 23.5% directly and 23.0% indirectly through smoking. The explanatory power of smoking behaviour for the gradient was reduced from 50.8% to 28% when early life circumstances were added to the model.

Conclusions Early life socioeconomic circumstances contributed importantly to social inequality in adult mortality. Our life-course model focusing on smoking provides evidence that social inequalities in health will persist unless prevention strategies tackle the intergenerational transmission of disadvantage and risk.

INTRODUCTION

Adverse behavioural factors such as smoking and poor diet tend to be more prevalent among individuals in lower socioeconomic positions (SEP).¹⁻⁸ The extent to which such behaviours explain the persistent inverse social gradient in mortality is a fundamental health policy question.⁹ An analysis of mortality in the Whitehall II cohort that utilised repeated measures of key risk behaviours over 24 years of follow-up showed that a large part of the socioeconomic mortality gradient could be explained in this way.¹⁰ The analysis advanced the methodology by building a more realistic time-dependent model of changing adult health behaviours and their health effects, thereby increasing

substantially the explained proportion of the inequality in death rates compared with previous studies that were based only on the social pattern of behaviour at baseline.¹¹⁻¹⁵

As the article and accompanying editorial noted,^{9 10} socioeconomic inequalities in health are not reducible to health behaviours. The underlying reasons for the common social pattern in health behaviour—the causes of the causes—need to be investigated so that health inequities can be understood with a view to their reduction.¹⁶ The present analysis thus examines the childhood and early adult origins of the mortality gradient observed in the MRC National Survey of Health and Development (NSHD),^{17 18} a British birth cohort study that has followed study members from birth, so far to age 66 years.

Previous studies have examined the childhood and adult origins of the mortality gradient at the same time in order to understand its causes and to provide evidence for preventive strategies within a life-course framework.^{19 20} Starting in utero, each stage of life may contribute to individual levels of risk and to the social gradient in risk. The 'independent' effects of risk factor levels including SEP at different life-course stages or summed exposures across the life course have been estimated.^{17 21-25} Other studies have gone beyond these conceptually cross-sectional approaches to estimate indirect as well as direct contributions of risk factors, thereby seeking to identify causal pathways, using techniques such as simultaneous multivariate and structural equation modelling.^{26 27} To our knowledge, ours is the first paper to focus on midlife smoking as the proximal mediating factor, quantifying both direct and indirect effects of early life-course circumstances on smoking pack-years and the social gradient in mortality between ages 26 and 66.

METHODS

The NSHD's study design, population and sampling method have been described previously.^{28 29} Briefly, 5362 individuals born in England, Wales and Scotland during 1 week in March 1946 were enrolled in the study. Since 1971, 4461 study members have been flagged for death notification on the National Health Service Central Registry.²⁸

Measures of early life origins were selected a priori based on Kuh *et al.*¹⁸ In childhood, they included maternal education and father's social class when study members were 4 years old, based on the Registrar General's Social Classification (RGSC). In early adulthood, they included study

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member educational attainment and home ownership by 26 years.¹⁸ Head of household's social class (RGSC at age 26) was derived from study member and spousal social class, and uses the class of the resident male in the house, if present, or female's class if not. The relative index of inequality (RII) based on RGSC age 26 was used in the Cox regression analysis of mortality.

Data for smoking over the life course, up to maximum age 64, were collected at successive sweeps of the cohort and summarised in pack-years.³⁰ A pack-year is equal to smoking one pack of 20 cigarettes each day for 1 year. Age of initiation was set at the mean (age 16) for those with missing data. The association between early life origins and smoking cessation by age 36 was explored through logistic regression. Among individuals with a history of smoking, ex-smokers were compared with current smokers to determine the odds of continued smoking at age 36.

Cox's proportional hazards regression was used to study the relationship between life-course SEP and mortality, mediated by smoking behaviour (pack-years). Failure event was set as mortality; entry into the study was set to month 312 (age 26) and follow-up was until death, emigration or month 800 (age 66). The sex-adjusted HR for mortality based on the RII constituted the base model that was further adjusted for individual and combined covariates as follows (those with missing covariates were excluded):

- ▶ Base model: RII (age 26)+sex
- ▶ Model A: Base+early childhood SEP indicators
- ▶ Model B: Base+early adult SEP indicators
- ▶ Model C: Base+pack-years smoked
- ▶ Model D: Base+early childhood and early adult SEP indicators
- ▶ Model E: Base+early childhood and early adult SEP indicators+pack-years smoked

The percentage reduction in the coefficient for RII was used to calculate the mediation proportion (attenuation) for each adjustment using the formula

$$(\beta_{\text{RII-base}} - \beta_{\text{RII-model}}) / (\beta_{\text{RII-base}}) \times 100\%.$$

The bootstrap method with 2000 re-samplings was used to calculate 95% CIs for the table 3 attenuation models.

RESULTS

Of the 5362 study members, 2132 were eligible for analysis after excluding observations with missing covariates ($n=2508$, smoking pack-years was the single largest contributor; $n=943$) or death, emigration or study withdrawal prior to age 26 ($n=722$). Those excluded were compared with those included in the analysis. There were at most small differences in sociodemographic and behavioural variables between the two groups (data not shown).

Childhood circumstances predicted adult smoking habits at age 36 (table 1): individuals with fathers in a manual social class had higher odds of continued smoking compared with non-manual. Those whose mothers had only primary education were more likely to continue smoking compared with those whose mothers had secondary level education. Smoking increased the mortality risk by 2% per pack-year smoked (table 2). Compared with those owning/buying their home at age 26, those who rented had an increased mortality risk.

The separate contribution of each covariate to the social gradient in mortality was estimated using the RII for RGSC at age

Table 1 ORs for continued smoking by age 36 for indicators of childhood socioeconomic position (age 4) obtained by logistic regression (current smoker vs ex-smoker and never smoker), 1768 study members

	OR, sex adjusted (95% CI)
Father's social class	
Non-manual	1
Manual	1.61 (1.32 to 1.95)
Father's education	
Secondary level	1
Primary only	1.25 (1.01 to 1.53)
Mother's education	
Secondary level	1
Primary only	1.62 (1.29 to 2.03)

26 (table 2). Smoking (pack-years) accounted for 51% of the social gradient in mortality in the cohort based on social class at age 26.

Direct and indirect pathways to mortality

Childhood and early adult SEP indicators were added to the base model by life stage (table 3). Early childhood indicators combined (model A) accounted for 18% of the mortality gradient. Early adult indicators (model B) accounted for about double the proportion (35%) of the mortality gradient. The combination of early childhood and early adult indicators attenuated the mortality gradient by 47% (model D), a proportion approaching that attributable to smoking (51%, model C). Considering smoking history together with early life origins in the full model (model E), the proportion of the gradient explained is 74%.

The attenuating effects in models C, D and E were decomposed to quantify the life-course pathway model (figure 1). The direct smoking effect (28% of inequality in mortality explained) is derived from the difference between the full model E (74% explained) and the early life-course model D, which omits smoking (47% explained). Comparing model C with the direct smoking effect provides the indirect effect of early life mediated by smoking (51–28%). To account for the direct effect of early life origins, the indirect effect through smoking is taken from the total early life effect (23.5% explained).

DISCUSSION

This study highlights the major role of early life circumstances as causes of inequalities in health. It emphasises how important it is, in the context of the policy debate, to recognise the inter-generational transmission of risk and the accumulation of disadvantages that can occur during childhood.³¹ First, the combination of material and psychosocial conditions in childhood was almost as powerful as smoking in accounting for the inequality in adult mortality experienced in this cohort. Second, the effects of early life circumstances were seen to extend far into adult life and to influence mortality risk by shaping continued exposure to smoking and by implication other risk behaviours that were omitted so as to facilitate analytic clarity. Smoking history was in substantial part attributable to early life circumstances, characterised by father's occupational class and mother's level of education.

There is considerable evidence for the operation of pathways that connect early life disadvantage with poor health and

Table 2 HRs for risk factors and effect of adjustment for single risk factors on the relative index of inequality for mortality by head of household social class at age 26 obtained using Cox's proportional hazards regression

Adjustment variables	All-cause mortality		
	HR for risk factor, sex adjusted (95% CI)	Relative index of inequality (95% CI)	% Change in relative index of inequality
Sex (base model)	0.84 (0.64 to 1.12)	1.97 (1.18 to 3.28)	Baseline
Early life indicators (age 4)			
Father's social class	1.09 (0.80 to 1.50)	1.86 (1.08 to 3.21)	-8.3
Mother's education	1.30 (0.87 to 1.94)	1.77 (1.04 to 3.02)	-15.8
Early adulthood indicators (age 26)			
Educational qualifications	1.20 (0.88 to 1.64)	1.71 (0.97 to 3.01)	-21.2
Home ownership	1.44 (1.08 to 1.94)	1.74 (1.04 to 2.93)	-18
Cigarette smoking (pack-years)	1.02 (1.02 to 1.03)	1.40 (0.82 to 2.37)	-50.8

Based on 195 deaths, 2132 study members, and mortality between ages 26 and 66.

increased mortality risk in adulthood.³² These pathways involve combinations of early exposure to socioeconomic, behavioural and biological factors that affect adult mortality risk in ways specific to age, place of residence and calendar time. There may be critical events, such as poor in utero growth leading to low birth weight³³; accumulation of risk, such as that due to constrained upward social mobility²⁴; chains of risk involving a sequence of adverse exposures, such as may be initiated by poor housing conditions in childhood;³⁴ or a complex interaction between these pathways, that lead to an increase in adult mortality risk.

Both childhood and adult socioeconomic circumstances have been shown to influence adult smoking behaviour. Lower childhood SEP is associated with an increased risk of smoking initiation, progression to regular smoking and a reduced likelihood of cessation. Similar results were found when analysis used adult SEP.³⁵ Manual social class in childhood decreased the likelihood of adult smoking cessation.⁶ Current smoking (vs never and ex-smoking), after adjusting for adult social class in some populations, is associated with childhood social class in women.⁶ Childhood social class and nicotine dependence both influence adult smoking behaviour.⁴

Our analysis shows empirically that an explanation of health inequality wholly reliant on adult health behaviours is incomplete. Time-dependent analysis of changing risk behaviours raises the proportion of inequality explainable by more

accurately specifying exposure.¹⁰ We have extended this logic by extending the time at risk back to early childhood to include some of the 'causes of the causes'—in this case, the inequalities in early socioeconomic and cultural advantage that produce the social gradient in mortality, in part through their influence on patterns of adult health behaviours. A key finding is that markers of early life increase the explanatory power of the model for the mortality gradient by approaching 50% over the substantial proportion explained by smoking behaviour on its own.

Our model utilises pack-years as the measure of life-course exposure to tobacco smoke. In consequence, the analysis does not account for passive exposure. The summary pack-years measure further relies on some assumptions. Among these, it is assumed that reporting bias concerning smoking status is similar by social class across rounds of data collection and that the social gradients in age at initiation and cessation do not distort the findings from the analytical models. The social gradient in smoking cessation in the NSHD is greater than the gradient in smoking uptake.^{6 36} Thus, the same level of exposure based on the pack-years measure may represent a somewhat different health effect across social strata.

As indicated above, the statistical model for the mortality gradient focuses on the intermediate role of smoking. Other health behaviours, environmental exposures and biological factors have been omitted. If the model included additional adult health behaviours such as dietary pattern, the independent effect of early life would likely diminish because the model would more fully represent the causal relationships that link childhood deprivation to adult health behaviours and social disparities in adult mortality.^{7 37} The association of manual father's social class with mortality was not significant in this analysis. In contrast, there was an inverse association in the cohort when participants with missing data were included in the Cox model as a separate group.³⁰ If the effect of early life has been underestimated in the present analysis, it may be that the estimate of the explanatory power of early life circumstances is also biased downwards.

The extent of inequality in mortality is similar in Great Britain and France, but smoking and other social patterns of health behaviour account for much less of the social gradient in France.³⁸ Whatever other adult risk factors underlie the health inequalities, the primary cause in France is likely to be found in the social patterning of childhood advantage, as in Great Britain. Effective policy to reduce health inequality in different societies may therefore depend on population-specific variations

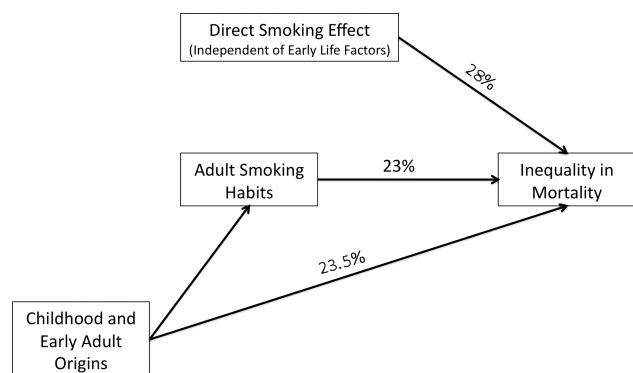


Figure 1 Life-course pathway from early life origins to inequality in mortality mediated by smoking. Figures are percentage of inequality in mortality accounted for in the cohort. Adult smoking habits account for 51% of inequality in mortality, of which 45% (23%/51%) is attributable to childhood and early adult origins.

Table 3 Effect of adjustment for early childhood and early adult socioeconomic indicators and health behaviours on the relative index of inequality (head of household social class at age 26) for mortality

	All-cause mortality	
	Adjusted relative index of inequality (95% CI)	% Change in relative index of inequality (95% CI)
Adjustments		
Sex (base model)	1.97 (1.18 to 3.28)	Baseline
Early childhood variables		
Sex+father's social class+mother's education (model A)	1.74 (1.00 to 3.04)	-18.1 (-88 to 17)
Early adult variables		
Sex+highest educational attainment+home ownership (model B)	1.54 (0.87 to 2.75)	-35.8 (-229 to 0.5)
Cigarette smoking		
Sex+pack years smoked (model C)	1.40 (0.82 to 2.37)	-50.8 (-188 to -23)
Early life-course variables		
Sex+early childhood+early adult variables (model D)	1.44 (0.79 to 2.62)	-46.5 (-181 to 1.5)
Early life-course and smoking behaviour		
Sex+early childhood+early adult variables+smoking (model E)	1.19 (0.65 to 2.18)	-74.3 (-251 to -20)

Based on 195 deaths, 2132 study members, and mortality between ages 26 and 66.

in measures along with similar life-course perspectives on the causes of health inequalities.^{30 39}

What is already known on this subject

- ▶ Beliefs about the causes of social inequalities in health are central to the debate about what the appropriate policy response should be.
- ▶ Adult health behaviours provide a largely complete *statistical* explanation for inequalities in mortality when the analytical model incorporates time-varying measures of exposure.
- ▶ In the Whitehall II study, 72% of the social gradient in all-cause mortality could be explained by smoking behaviour, alcohol consumption, dietary indicators and physical activity, allowing for their variation over 24 years of follow-up.

What this study adds

- ▶ Differences in childhood and early adult circumstances are the underlying causes of social inequalities in both adult health behaviours and mortality.
- ▶ Policies focused only on adult health behaviours do not address the socially patterned causes of these behaviours or the independent role played by these causes in social inequalities in health.

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Competing interests None.

Ethics approval The study received Multi-Centre Research Ethics Committee approval, and informed consent was given by cohort participants.

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