Family Socioeconomic Position at Birth and Future Cardiovascular Disease Risk: Findings From the Aberdeen Children of the 1950s Cohort Study

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Objectives. We assessed the association of father's social class, recorded at the time of birth, with coronary heart disease and stroke in a British cohort of 11 106 individuals born in the 1950s.

Methods. Survival analysis was used to relate social class at birth to the occurrence of either fatal or nonfatal coronary heart disease or stroke.

Results. Rates of coronary heart disease and stroke increased across the social class distribution from highest to lowest, and patterns of association were similar for the 2 outcomes. The gender-adjusted hazard ratio of experiencing either coronary heart disease or stroke comparing the manual and nonmanual social class categories was 1.52 (95% confidence interval [CI] = 1.14, 2.02). This ratio fell to 1.41 (95% CI = 1.05, 1.88) after adjustment for indicators of intrauterine and childhood growth. Further adjustment for educational attainment reduced the ratio to 1.28 (95% CI = 0.94, 1.75).

Conclusions. We found that social class at birth was associated with risk of fatal and nonfatal cardiovascular disease among individuals born in the 1950s, a period of relative prosperity and after the introduction of the welfare state in Britain. This relation appeared to be mediated in part through educational attainment. (*Am J Public Health.* 2006;96:1271–1277. doi:10.2105/AJPH.2005.066290)

Several studies conducted in different populations have shown associations between adverse childhood socioeconomic position (SEP) and coronary heart disease (CHD) and stroke that are independent of adult SEP.[1–14] However, in many of these studies, childhood SEP has been retrospectively reported in adulthood,[3–6,8,11] and most of the studies have included participants born in the 1930s or earlier.[2,4–7,11] Accuracy of adult recall of childhood SEP is unlikely to be affected by CHD or stroke status; thus, any misclassification would be nondifferential and might actually lead to results underestimating the true association.[15]

It is important to study individuals born in recent decades because the effects of childhood SEP on health outcomes vary over time.[16] Children born in recent decades into families of low socioeconomic status, particularly those born after World War II in nations with publicly funded health and welfare systems (e.g., Britain), are likely to have experienced better standards of living than those born in earlier years. Evidence of this trend is provided by infant mortality rates, which are sensitive to socioeconomic circumstances. For example, between 1901 and 1905 infant mortality was 150 per 1000 live births in England and Wales and 130 in Scotland; by the period 1941 through 1945, these rates had decreased to 60 and 80 per 1000, respectively, and a marked decline over the subsequent decade resulted in rates of 30 and 37 per 1000 in the early to mid-1950s.[17]

Because of the introduction of welfare reforms throughout Britain in the late 1940s, together with improvements in the British and world economies beginning in the mid-1950s, those born in the most deprived groups in the 1950s are likely to have experienced better conditions than those born in the most deprived groups in the 1930s and earlier.[18–20]

If the associations found in previous studies largely represent the effects of extremely adverse socioeconomic circumstances such as those that gave rise to the high levels of infant mortality observed in the early 1900s, then, given that these conditions (and the associated infant mortality) have improved over time, one might expect only weak or no associations between childhood SEP and cardiovascular disease risk in studies of populations born in the 1950s or later. If this were the case, it would suggest that the varying socioeconomic conditions experienced by contemporary children might not have important effects on their future cardiovascular disease risks.

Only 4 studies have examined the association of childhood SEP with adult cardiovascular disease outcomes among individuals born in the latter half of the 20th century, and all of these investigations have revealed inverse associations.[9,10,13,21] These studies have predominantly involved only men, and all have assessed cardiovascular disease mortality. Determining whether childhood SEP affects nonfatal CHD and stroke events is also important because it would suggest an effect on the atherosclerotic process and not simply the acute event, as well as the existence of socioeconomic inequalities in cardiovascular disease morbidity rather than simply premature mortality. We assessed the association of childhood SEP, recorded at the time of birth, with fatal and nonfatal CHD and stroke in a large cohort of women and men born in Scotland in the 1950s.

METHODS

Study Population

We used data from the Aberdeen Children of the 1950s cohort study. Described in detail elsewhere,[22] the cohort comprises participants

in the Aberdeen Child Development Survey,[23] which collected data on the parental and childhood characteristics of 14 938 children enrolled in Aberdeen (Scotland) primary schools in 1962.[23] Comprehensive perinatal information on the 12 150 of these children born in Aberdeen, including the course of their mother's pregnancy and their physical characteristics at birth, was abstracted from the Aberdeen Maternity Neonatal Databank (AMND).[23] These 12 150 individuals born in Aberdeen between 1950 and 1956 are the members of the Aberdeen Children of the 1950s cohort.[22]

Assessment of Exposures, Outcomes, and Covariates

We used the occupational social class of participants' fathers at the time of their birth as our primary measure of childhood SEP. We obtained information on father's occupation from the AMND and grouped occupations into the following categories[24]: professional (I), managerial (II), nonmanual/skilled nonmanual (III nonmanual), manual/skilled manual (III manual), semiskilled (IV), and unskilled manual (V). These categories can be collapsed into 2 groups (nonmanual [I–III nonmanual] and manual [III manual–V]).

A single measure of SEP is unlikely to encompass the entire spectrum of childhood social circumstances,[25,26] but no other direct measures of individual-level SEP were available in this cohort. Other studies have used proxy indicators of SEP, such as maternal age at birth, gravidity, birth outside of marriage, and maternal height (an indicator of the mother's SEP across her life course), to examine variations in health outcomes according to SEP.[25,26] We had information on these characteristics from the AMND and therefore undertook secondary analyses to assess the associations of these proxy measurements with CHD and stroke outcomes.

In 1999, we began tracing study members through Scotland's General Register Office; 97% have been successfully traced.[22] Traced participants have been linked to the Scottish Morbidity Register, which provides information, including diagnoses coded according to the *International Classification of Diseases, Ninth* and *Tenth Revisions* (*ICD-9* and *ICD-10*),[27,28] for all admissions to hospitals in Scotland. A recent audit demonstrated an accuracy rate for Scottish Morbidity Register data of greater than 90%.[29] We defined participants as case patients if they had a primary or secondary (i.e., comorbidity) diagnosis of CHD or stroke. Inclusion of secondary diagnoses ensured that anyone with documented evidence of CHD or stroke was included as a case patient. Thirty-three (10%) of the CHD cases and 21 (19%) of the stroke cases involved secondary (comorbid) diagnoses.

Participants also have been linked to the National Health Service Central Register, which provides detailed death certificate information. We defined participants as dying from an outcome of interest if this outcome appeared as an underlying cause (the final pathological event that results in death) or contributory cause (any event that may be in the pathway leading to the final event that results in death) on their death certificate. Only 3 CHD deaths were listed as contributory; all stroke deaths were listed as underlying. When we repeated all of the analyses with participants whose outcome was based on a secondary hospital diagnosis or a contributory (but not underlying) cause of death treated as non–case patients or excluded from the analyses, the results did not differ from those presented here. The *ICD* codes used to define CHD were 410 through 414 (*ICD-9*), 429.2 (*ICD–9*), I20 through I25 (*ICD-10*), and I51.6 (*ICD–10*), and those used to define stroke were 430 through 438 (*ICD–9*), I60 through I69 (*ICD–10*), and G45 (*ICD–10*).

We assessed whether any of the associations between social class at birth and CHD or stroke were mediated via the effects of intrauterine or postnatal growth retardation or educational attainment. Data on birthweight and gestational age were abstracted from the AMND.[22] We estimated participants' intrauterine growth rates by calculating gender and gestational age (in weeks) standardized *z* (standard deviation) scores. Height and weight at entry into school were measured directly (at a mean age of 5 years),[22] and age- and gender-standardized *z* scores, based on 3-month age categories, were derived for height and weight.

Between 2000 and 2002, a survey was mailed to 11 282 surviving cohort members, and 7183 (63.7%) responded.[30] Responders were more likely than nonresponders to be female, to have been members of more affluent families in childhood, and to have had higher cognitive function scores as children.[30] Data on educational attainment were obtained from these questionnaire responses. Although these data were collected in 2000, after most of the CHD and stroke events in the sample had occurred, most of the participants had completed their formal education before their mid-20s and, therefore, before the occurrence of any outcome events.

The questionnaire also asked about adult income and occupation and behaviors such as smoking and alcohol consumption. This information referred to participants' status and behavior after occurrence of the outcome events. Thus, outcome events were likely to have affected these characteristics, and, as a result, it was not appropriate to assess the effects of these characteristics on the associations examined here.

Statistical Analyses

We used Cox proportional hazards regression models, with participants' age as the time axis, in conducting our analyses. Because Scottish Morbidity Register records of hospital admissions were available only for 1981 onward, the follow-up period began on January 1, 1981. Participants were omitted from the analyses if they had died (n=116), emigrated anywhere outside Scotland (n=927), or experienced a nonfatal stroke or CHD event (n=1) before January 1, 1981. Contributions to risk were censored at the earliest of the following: (1) first episode of the outcome of interest (if individuals had repeated hospital admissions or experienced a fatal event after an earlier admission, they were excluded after the first event), (2) emigration date (including emigration to England or Wales), (3) death from a cause other than the outcome of interest, or (4) December 31, 2003.

We were unable to obtain data on hospital admissions occurring in England and Wales; as a result, in the main survival analyses, individuals who migrated to England and Wales were considered to be no longer at risk from their date of relocation and were censored (as just described) at this date. We considered the emigration date for these individuals to

be their first posting date with a general practitioner from England or Wales (the date they first appeared on health authority lists as being registered with a general practitioner). These data were likely to have overestimated time at risk, given that most individuals do not register with a new general practitioner immediately on moving. To determine the impact of this overestimation on our results, we undertook sensitivity analyses in which we repeated the Cox proportional hazards models with the date of censoring for those who had moved to England or Wales moved back in time by 6 months, 1 year, and 5 years.

Only 701 individuals had migrated to England or Wales during the follow-up period. Although there was no statistical evidence that the social class distribution among this group differed from that of the overall cohort (P=.4), it appeared to include more individuals in social classes I and II (13.6% vs 9.6%) and slightly fewer in social class V (15.6% vs 16.2%). However, when these 701 individuals were excluded from the analyses, the results did not differ from those presented here. We assessed proportionality assumptions by inspecting cumulative incident plots and testing for evidence of statistical interactions with the time scale (age in years) of the models. There was no evidence in any model of violation of the proportionality assumption.

Small amounts of data were missing for gestational age and childhood height and weight (Table 1). In addition, the fathers of 5.6% of the participants were classified in the unemployed/unknown/disabled/dead category and therefore could not be assigned to an occupational social class. Because information on educational attainment was obtained from the survey conducted in 2000, there was a substantial percentage (42%) of missing data for this variable (Table 1). We used multiple multivariate imputation,[31] including all other covariates, the log of survival time, and the censoring indicator, to impute a distribution of missing values for these variables. We used the switching regression feature of Stata version 9.0 (Stata Corp, College Station, Tex), as described by Royston,[31] and carried out 20 cycles of regression switching, generating 5 imputation data sets.

To examine whether there was any selection bias resulting from missing data, we also conducted all analyses on the subsample of participants with complete data (n=6248; 50%). The results from these complete data subset analyses were essentially the same as those presented here but were less precisely estimated.

Within the cohort as a whole, there were 9422 families; 5048 (41.5%) of the participants had at least 1 other sibling in the cohort. Robust standard errors, taking into account possible nonindependence between siblings in the cohort, were used to estimate significance values and 95% confidence intervals (CIs). Stata version 9.0 was used in conducting all analyses (Stata Corp, College Station, Tex).

TABLE 1—Baseline Characteristics of Participants in the Aberdeen Children of the 1950s Cohort

Characteristic	Sample (n = 12 150)
Female, no. (%)	5 868 (48.3)
Father's occupational social class category at birth, no. (%)	
I/II	1 163 (9.6)
III nonmanual	1 335 (11.0)
III manual	5 319 (43.8)
IV	1 689 (13.9)
V	1 963 (16.2)
Unemployed/unknown/disabled/dead	681 (5.6)
Mother's no. of previous pregnancies, no. (%)	
1	3 991 (32.8)
2	3 505 (28.9)
3	2 202 (18.1)
4	1 208 (9.9)
≥5	1 243 (10.2)
Missing	1 (<1.0)
Maternal height, cm, no. (%)	
≤152	3 101 (25.5)
155	1 911 (15.7)
157	2 169 (17.9)
160	1 777 (14.6)
162	1 489 (12.3)
≥165	1 703 (14.0)
Maternal age at birth, y, no. (%)	
15–19	567 (4.6)
20–24	3 798 (31.3)
25–29	3 777 (31.1)
30–34	2 546 (21.0)
35–39	1 108 (9.1)
≥40	354 (2.9)
Birth outside marriage, no. (%)	555 (4.6)
Gestational age, wk, no. (%)	
<37	760 (7.0)
37–40	7 806 (71.7)
>40	2 323 (21.3)
Missing	1 262 (10.0)
Birthweight	
No. with data	12 128
Mean, kg (SD)	3.3 (0.5)
Height at primary school entry	
No. with data	11 719
Mean, cm (SD)	108.7 (10.4)
Weight at primary school entry	
No. with data	11 639
Mean, kg (SD)	18.9 (2.9)
Educational attainment, no. (%)	
No formal qualifications	1 624 (22.9)
School leaving certificate	173 (2.4)
Certificate of secondary education	165 (2.3)
Ordinary-level qualifications	1 684 (23.8)
Advanced-level qualifications	926 (13.1)
Higher national certificate	1 138 (16.1)
University degree	1 374 (19.4)
Missing	5 067 (41.7)

RESULTS

Table 1 shows the baseline characteristics of the cohort. At the start of the follow-up period (1981), 11 106 members of the cohort were alive and believed to be residing in Scotland. Over the course of the follow-up period, they contributed 245 000 person-years of risk. Among these participants, there were 302 (53 fatal) cases of CHD, 109 (4 fatal) cases of stroke, and 397 (57 fatal) cases of either CHD or stroke. (Numbers are per-individual figures, and thus, 14 individuals who experienced both a CHD and a stroke event contributed only once to the analyses in which CHD and stroke were combined. These individuals were censored at the time at which the first CHD or stroke event occurred.) Table 2 shows rates of CHD and

TABLE 2—Rates of Coronary Heart Disease (CHD) and Stroke Among Participants in the Aberdeen Children of the 1950s Cohort (n = 11 106)

	Women		Men		Total	
	No.	Rate per 10 000 Person-Years (95% CI)	No.	Rate per 10 000 Person-Years (95% CI)	No.	Rate per 10 000 Person-Years (95% CI)
CHD	69	5.7 (4.5, 7.3)	233	18.7 (16.5, 21.3)	302	12.3 (11.0, 13.8)
Stroke	51	4.2 (3.2, 5.6)	58	4.6 (3.6, 6.0)	109	4.4 (3.7, 5.4)
Either CHD or stroke	117	9.8 (8.1, 11.7)	280	22.5 (20.1, 25.3)	397	16.3 (14.7, 17.9)

Note. CI = confidence interval.

stroke. As one would expect, rates of CHD were higher among men than women, but rates of stroke were similar in the 2 groups.[32] The associations of social class with both CHD and stroke were the same among women and men, and, therefore, we present results for the groups combined (all *P* values for the interaction terms between gender and social class at birth were greater than .3).

Table 3 shows rates of CHD and stroke, together with data on other characteristics across categories of social class at birth, among the 11 106 individuals who were included from the start of the follow-up period. Rates of CHD, stroke, and both outcomes combined increased across the social class distribution from least to most advantaged. There was statistical evidence of linear associations for all of the outcomes across the social class categories and no strong statistical evidence of nonlinear associations (all *P*s > .1). The directions and magnitudes of associations were similar for CHD and stroke, and we combine these 2 outcomes in further discussions of the multivariate analyses.

Table 3 also presents CHD and stroke rates for those whose fathers were unemployed, disabled, deceased, or whose status was unknown, at the time of their birth. Rates in this group were generally similar to rates in the III manual and IV social class categories and lower than those in social class V. Because this was a heterogeneous group that could not be classified according to occupation, all further analyses involving these individuals were computed with a distribution of imputed values. The tests for trend reported in Table 2 excluded those whose fathers were in the unemployed/unknown/disabled/deceased category.

Birthweight, childhood weight, and childhood height all decreased across the social class distribution from highest to lowest, and the proportion of infants who were born prematurely (before 37 weeks of gestation) increased linearly from the lowest to highest social classes (at birth). There were also strong graded associations between occupational social class and all of the maternal characteristics assessed.

Table 4 shows the multivariable associations of social class at birth with CHD and stroke combined. There was some attenuation of the linear inverse association between social class and CHD or stroke after adjustment for indicators of intrauterine and postnatal growth (models 2, 3, and 5). Adjustment for gestational age alone had little

TABLE 3—Rates of Coronary Heart Disease (CHD) and Stroke and Distributions of Other Characteristics, by Social Class at Birth: Participants in the Aberdeen Children of the 1950s Cohort

	Social Class Category at Birth						
	I/II (n = 1004)	III Nonmanual (n = 1207)	III Manual (n = 4882)	IV (n = 1567)	V (n = 1840)	Unemployed (n = 605)	*P*[a]
CHD rate per 10 000 PY (95% CI)	8.3 (5.4, 13.8)	9.8 (6.8, 14.8)	11.0 (9.2, 13.3)	11.5 (8.5, 16.0)	20.5 (16.5, 25.8)	12.0 (7.5, 20.4)	<.001
Stroke rate per 10 000 PY (95% CI)	2.3 (1.0, 6.9)	3.0 (1.5, 6.8)	4.2 (3.1, 5.6)	3.4 (2.0, 5.6)	7.8 (5.6, 11.3)	5.2 (2.5, 12.5)	.001
Combined CHD/stroke rate per 10 000 PY (95% CI)	10.2 (6.7, 16.4)	12.5 (9.0, 17.8)	14.9 (12.7, 17.5)	14.9 (11.4, 19.9)	27.0 (22.4, 32.9)	15.7 (10.3, 24.1)	<.001
Gravidity ≥ 5, no. (%)	51 (5.1)	69 (5.7)	432 (8.9)	183 (11.7)	341 (18.5)	78 (12.9)	<.001
Maternal height ≤ 152 cm, no. (%)	119 (11.9)	236 (19.6)	1251 (25.6)	481 (30.7)	601 (32.7)	192 (31.7)	<.001
Maternal age at birth 15–19 y, no. (%)	7 (0.7)	33 (2.7)	216 (4.4)	71 (4.5)	98 (5.3)	82 (13.6)	<.001
Birth outside marriage, no. (%)	1 (0.1)	2 (0.2)	17 (0.4)	10 (0.6)	21 (1.1)	448 (74.1)	<.001
Gestational age < 37 wk, no. (%)[b]	40 (4.2)	77 (6.8)	283 (6.4)	121 (8.7)	128 (8.1)	38 (8.7)	<.001
Birthweight z score, mean (SD)	0.18 (0.98)	0.08 (0.94)	−0.01 (0.98)	−0.02 (1.02)	−0.08 (1.01)	−0.13 (1.07)	<.001
Weight at school entry z score, mean (SD)	0.19 (0.86)	0.07 (0.93)	−0.02 (0.88)	−0.06 (0.70)	−0.11 (0.82)	−0.04 (0.93)	<.001
Height at school entry z score, mean (SD)	0.37 (0.94)	0.20 (0.92)	0.01 (0.97)	−0.13 (0.86)	−0.25 (0.89)	−0.18 (1.0)	<.001
University degree, no. (%)[b]	335 (43.4)	216 (27.1)	475 (16.1)	83 (9.1)	68 (6.9)	52 (17.1)	<.001

Note. PY = person-years; CI = confidence interval.
[a]Trend tests across occupational social class categories, not including unemployed category.
[b]Number and percentage of those with complete data. For gestational age, the numbers in each social class category with complete data were as follows: I/II, 952; III nonmanual, 1134; III manual, 4455; IV, 1399; V, 1582; and unemployed, 438. The corresponding numbers for university degree were 707, 796, 2960, 913, 987, and 304.

TABLE 4—Multivariate Associations of Social Class at Birth With Coronary Heart Disease or Stroke: Participants in the Aberdeen Children of the 1950s Cohort (n = 11 106)

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
I/II	1.00	1.00	1.00	1.00	1.00	1.00
III nonmanual	1.22 (0.71, 2.09)	1.21 (0.71, 2.08)	1.19 (0.70, 2.05)	1.17 (0.68, 2.03)	1.19 (0.70, 2.05)	1.15 (0.67, 2.00)
III manual	1.45 (0.93, 2.27)	1.42 (0.91, 2.22)	1.38 (0.88, 2.16)	1.33 (0.83, 2.13)	1.36 (0.87, 2.14)	1.27 (0.79, 2.04)
IV	1.43 (0.88, 2.36)	1.39 (0.85, 2.29)	1.33 (0.81, 2.20)	1.23 (0.72, 2.11)	1.32 (0.80, 2.18)	1.16 (0.68, 1.99)
V	2.60 (1.64, 4.11)	2.50 (1.58, 3.96)	2.37 (1.49, 3.77)	2.20 (1.33, 3.66)	2.33 (1.47, 3.71)	2.04 (1.23, 3.35)
P trend	<.001	<.001	<.001	<.001	<.001	<.001
Manual vs nonmanual	1.52 (1.14, 2.02)	1.48 (1.11, 1.98)	1.42 (1.07, 1.90)	1.35 (1.00, 1.84)	1.41 (1.05, 1.88)	1.28 (0.94, 1.75)

Note. Because the time scale in the Cox proportional hazard models was age, all results are age adjusted. Model 1 was adjusted for gender only. Model 2 was adjusted for gender and birthweight, standardized for gestational age *z* score. Model 3 was adjusted for gender and height and weight at entry into primary school. Model 4 was adjusted for gender and educational attainment. Model 5 was adjusted for all covariates other than educational attainment (i.e., birthweight and heath and weight at entry into primary school). Model 6 was adjusted for all covariates, including educational attainment (i.e., birthweight, heath and weight at entry into primary school, and educational attainment).

impact on the association between childhood social class and CHD or stroke (data not shown). Greater attenuation occurred with adjustment for educational attainment (model 4), although rates among individuals born into social class V remained more than twice as high as those of individuals born into social class I or II even after adjustment for educational attainment.

As with the unadjusted association, although there was strong statistical evidence for a trend across all social class groups, the risk of CHD or stroke appeared particularly marked among those born into the lowest (V) group. With full adjustment for all potential mediating factors, the hazard ratio in this group remained twice that of those born into social class I or II (model 6). Most of the events in the Table 4 data were nonfatal. When we restricted our analyses to such cases, they did not differ from those presented in Table 4, and examination of point estimates suggested that the association between social class at birth and CHD or stroke was similar for fatal and nonfatal cases.

There was no association between being born to a teenage mother (all *P*s>.9) or being born out of marriage (all *P*s>.4) and CHD or stroke outcomes in either unadjusted or adjusted analyses. Both maternal height and gravidity were linearly associated with CHD and stroke outcomes, with risks being greatest among those whose mothers were shorter and who had had a greater number of previous pregnancies (all *P*s<.001). Associations of social class at birth and maternal height and gravidity with CHD and stroke were independent of each other and of intrauterine and postnatal growth indicators.

A model that simultaneously included social class at birth, gravidity, and maternal height in addition to birthweight and childhood weight and height showed hazard ratios of 1.35 (95% CI=1.01, 1.80) for the comparison between manual and nonmanual social classes, 1.53 (95% CI=1.15, 2.03) for the comparison between participants whose mothers had had 5 or more previous pregnancies and all other participants, and 1.29 (95% CI=1.03, 1.62) for the comparison between those whose mothers' height was 152 cm (60 in) or less and all others. When educational attainment was added to the model, the corresponding hazard ratios were 1.25 (95% CI=0.92, 1.70), 1.46 (95% CI=1.09, 1.95), and 1.27 (95% CI=1.02, 1.59).

Those who responded to the adulthood questionnaire were asked about the occupation of their fathers in childhood. In the case of the associations examined here, these retrospective reports of childhood social class showed remarkably similar associations with the data collected at birth. For example, when adult reports of childhood social class were used, the hazard ratio of either a CHD or a stroke event in the manual compared with nonmanual groups was 1.59 (95% CI=1.09, 2.32) among the 6271 participants with these data; the same hazard ratio with the data collected at birth was 1.52 (95% CI=1.02, 2.26) in this subgroup.

DISCUSSION

Our results showed that social class at the time of birth was associated with fatal and nonfatal CHD and stroke risk among both women and men born in Scotland in the 1950s, such that those from lower social class backgrounds were at increased risk. It is remarkable that, simply on the basis of knowledge of the occupation of participants' fathers at the time of their birth, one could have predicted who would be most likely to suffer from CHD or a stroke 50 years later. This association was mediated, in part, through educational attainment.

A potential strength of this study was the use of a measure in which childhood SEP was assessed at the time rather than retrospectively reported in adulthood. A systematic review conducted by Galobardes et al.[33] showed some evidence that studies involving retrospective reports produce weaker inverse SEP–cardiovascular disease associations than studies involving prospective data, but our direct comparison of adult retrospective social class data and prospective childhood social class data suggested that the association was the same with these 2 types of data.

A weakness of our study is that we were able to include only 1 direct measure of childhood SEP that was unlikely to encompass the entire spectrum of childhood social circumstances and their effects on adult cardiovascular disease risk. Thus, our results may have underestimated the true magnitude

of the association between childhood SEP and CHD or stroke. There is some evidence of this possibility in the independent associations of maternal gravidity, height, and occupational social class with cardiovascular disease outcomes. However, there are difficulties involved in interpreting the meaning of the associations of these proxy indicators of SEP with disease outcomes.[25,26]

Only individuals who were alive in 1962 (the time of the childhood survey) were included in this cohort. It is unlikely that any deaths occurring before entry into primary school were because of cardiovascular disease, however, and therefore the cohort should be representative of those at risk of cardiovascular disease in later life and not prone to survivor bias. We had educational attainment data on only 58% of the original cohort but used multiple imputations to deal with these missing data.

Our findings are consistent with studies showing similar associations in populations largely born in the first half of the 20th century[2,4–7,11] as well as studies of individuals born in more recent decades.[1,9,10,12,13,21] Taken together, these results suggest that the association between childhood SEP and future cardiovascular disease risk is not restricted to groups experiencing extremely adverse socioeconomic circumstances, such as those that were associated with high infant mortality rates in the early part of the 20th century. This persistent effect of childhood SEP on cardiovascular disease occurs against a backdrop of marked declines in the overall mortality and incidence of both CHD and strokes over the past century.[34]

These decreasing trends have been attributed in part to improvements in primary and secondary prevention. However, downward trends in blood pressure, a major risk factor for CHD and stroke, cannot be fully explained by treatment; these trends are observed in all age groups, including the very young, among whom antihypertensive medications are rarely used.[35] Childhood social class is independently (of adult SEP) related to hormone replacement therapy and rates of hysterectomy,[36,37] suggesting that cultures and behaviors related to early-life SEP have long-term influences on health service use. It is possible that efforts focusing on primary and secondary prevention of CHD are similarly related to childhood SEP.

There was marked attenuation of the association of childhood SEP with CHD and stroke after adjustment for educational attainment. Because educational attainment influences future employment opportunities and income, our results might indicate that, in this cohort, much of the effect of childhood SEP was mediated through adult SEP. In an earlier article, we showed that adjustment for educational attainment completely attenuated any association between social class at birth and CHD behavioral risk factors but that adjustment for occupational social class in adulthood and income did not result in such marked attenuation, suggesting that educational attainment in itself, rather than material resources, mediated the association of early-life SEP with adult CHD risk factors.[38]

One difficulty with relating early-life risk factors to future adult disease risk is that this can never be accomplished with contemporary children, who, by definition, have not experienced adult diseases. Comparisons between studies conducted among populations born in industrialized countries in the 1930s and earlier[2,4–7,11] and this study of individuals born in the 1950s indicate no apparent marked weakening of magnitudes or differences in direction of the association between childhood SEP and future cardiovascular disease risk.

In general, living conditions in the United Kingdom improved considerably during the period spanning the 1930s through the 1950s, but they were still worse in the 1950s than they are now. Thus, the continued improvements in living conditions that took place during the second half of the 20th century might result in contemporary socioeconomic differentials having effects on cardiovascular disease risk that are less marked than those found in this study population. However, the marked increase in risk among individuals from the least affluent group in the present study suggests the need to ensure improved conditions for those born into the lowest end of the SEP spectrum. ■

About the Authors

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Contributors

D.A. Lawlor developed the study, contributed to revitalizing the cohort, undertook the statistical analysis, wrote the first draft of the article, and coordinated the writing of the article. G. Ronalds contributed to the statistical analysis protocol and to the writing of the article. S. Macintyre contributed to revitalizing the cohort and to the intellectual development and writing of the article. H. Clark managed the data collection and merging of data sets and contributed to the writing of the article. D.A. Leon contributed to developing the study, coordinated the revitalization of the cohort, and contributed to the writing of the article.

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Human Participant Protection

This study was approved by the Scottish multicenter research ethics committee, local study site research ethics committees, and the Scottish Privacy Advisory Committee. Questionnaires were sent to surviving participants via statutory bodies (without any of the researchers having access to personal details). Participants who responded to the questionnaire provided written consent for their questionnaire data to be used.

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