Association Between Childhood Socioeconomic Status and Coronary Heart Disease Risk Among Postmenopausal Women: Findings From the British Women's Heart and Health Study

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Objectives. We assessed the association between childhood socioeconomic status (SES) and coronary heart disease among postmenopausal women.

Methods. We conducted a cross-sectional analysis of 3444 women aged 60 to 79 years.

Results. There was an independent linear association between childhood and adult SES and coronary heart disease. The association between childhood SES and coronary heart disease was attenuated when we adjusted for insulin resistance syndrome, adult smoking, physical activity, biomarkers of childhood nutrition, and passive smoking.

Conclusions. The association between adverse childhood SES and coronary heart disease is in part mediated through insulin resistance, which may be influenced by poor childhood nutrition, and in part through the association between childhood SES and adult behavioral risk factors. (*Am J Public Health.* 2004; 94:1386–1392)

Most,[1–11] but not all,[12] studies that have assessed the association between childhood SES and adult coronary heart disease have found that adverse childhood SES is associated with increased risk of coronary heart disease and that this association is independent of adult SES. To develop effective policy interventions that abolish the link between childhood poverty and coronary heart disease, it is necessary to understand the causal pathways that link them. Adverse childhood SES may result in increased risk for coronary heart disease via an influence on known behavioral risk factors, such as smoking and poor diet. Childhood poverty may be associated with poor nutrition during the intrauterine period or childhood, which in turn may program insulin resistance and thus increase risk for coronary heart disease.[13,14] Finally, childhood poverty may lead to psychological distress, via a programming effect on cortisol secretion, and consequently increase risk for coronary heart disease.[15,16] These mechanisms are not necessarily mutually exclusive.

We assessed the association between childhood SES and coronary heart disease among a large cohort of postmenopausal women. We also assessed the role of components of the insulin resistance syndrome, adult behavioral risk factors, adult biomarkers of specific childhood exposures, and indicators of psychological distress in these associations.

METHODS

We used data from the British Women's Heart and Health Study; details about the participants and the measurements have been published elsewhere.[17,18] Women aged 60 to 79 years were randomly selected from general practitioner lists in 23 British towns. A total of 4286 women (60% of those invited) participated, and baseline data (self-reported questionnaire, research nurse interview, physical examination, and primary care medical record review) were collected between April 1999 and March 2001.

Prevalence of coronary heart disease was defined as any participant who had a primary care medical record of myocardial infarction or angina or who reported ever being diagnosed by a doctor with either of these conditions.[18] Details about the longest-held occupation of the participant's father and husband and her own longest-held occupation were obtained. Adult SES was derived from the longest-held occupation of the participant's husband for married women and from her own for single women; childhood SES was derived from the longest-held occupation of the participant's father. SES was categorized into 1 of 6 social classes on the basis of the Registrar General's occupational classification: professional (social class I), managerial and technical (social class II), nonmanual—skilled (social class III-NM), manual—skilled (social class III-M), partly skilled (social class IV), and unskilled (social class V). These social classes also are grouped into 2 broad categories of manual and nonmanual social classes.[19] Other indicators of childhood SES included self-reported childhood household amenities (e.g., house with a bathroom, house with a hot-water supply) and family access to a car. Other indicators of adult SES included housing status (i.e., own, rent, live in pubic housing, or live with family), car ownership, and pension arrangements.

Details about measurements of the components of insulin resistance syndrome have been reported elsewhere.[17,20] Leg length is a useful biomarker of prepubertal childhood exposures, because it reflects infant diet, childhood nutrition, and childhood infection.[21] Forced expiratory volume in 1 second (FEV_1) also is a biomarker of early-life environmental exposures, including intrauterine exposures, infant respiratory infections, childhood nutrition, and passive smoking.[22,23] Standing height and seated height were measured, and leg length was calculated as the total height minus "trunk" length, with trunk length defined as the seated height minus the height of the stool on which the individual was sitting (407mm).[20] FEV_1 was assessed with a digital-meter vitalograph (machine that measures indicators of lung function including FEV_1).

Participants were categorized as having never smoked, being an ex-smoker, or being a current smoker at 1 of 4 levels: 1 to 9, 10

to 19, 20 to 29, or 30 or more cigarettes per day. Participants were asked to indicate their usual duration of several types of activity in hours per week,[24] and they were categorized into 1 of 3 categories: less than 1 hour (inactive), 1 to 2 hours, or more than 2 hours per week of either moderate or vigorous physical activity.

Three indicators of psychological distress were used in our study: the Euroquol mood question,[25] history of a clinical diagnosis of depression, and current use of anxiolytic, hypnotic, or antidepressant medications. Participants whose response to the Euroquol mood question was that they were "today feeling either moderately or extremely anxious and/or depressed" were coded as currently anxious or depressed, and those who indicated that they had ever been diagnosed by a doctor with depression were coded as having a history of depression. Participants brought all of their medications to the research nurse interview, and a full treatment history was recorded. Medications were coded in accordance with the British National Formulary[26]; anxiolytics and hypnotics were any medication in section 4.1, and antidepressants were any medication in section 4.3.

Statistical Analysis

Age-adjusted prevalences and means, together with 95% confidence intervals (CI), of coronary heart disease and coronary heart disease risk factors are shown across the 6 social classes (Tables 1 and 2). The possibility of an interaction between childhood and adult SES was assessed with the likelihood ratio test. Multiple logistic regression was used to assess the association between childhood SES and prevalence of coronary heart disease and to assess the effect on this association after we adjusted for potential explanatory factors on the causal pathways: components of insulin resistance syndrome, behavioral risk factors, biomarkers of childhood nutrition, infection and passive smoking, and indicators of psychological distress. A decrease in the association of at least 10% indicated that these factors had an important role in the association. Robust standard errors (standard errors estimated using the variability of the data rather than model-based estimates of standard errors) were used to estimate 95% confidence intervals in all the models; all analyses were performed with Stata, Version 8.0, software (Stata Corp, College Station, Tex).

RESULTS

Complete data on both childhood and adulthood SES were available for 3444 (80%) of the women, 518 of whom had coronary heart disease, a prevalence of 15% (95% CI=13.8, 16.3). Only women who had complete data on SES at both times in the life course were included in our analyses. Compared with women who did not have complete SES data, those who did have complete data were slightly younger (68.8 years vs 69.2 years, $P=0.05$), had a lower prevalence of coronary heart disease (15% vs 19.5%, $P=0.004$), had smaller waist-hip ratios (0.818 vs 0.824, $P=0.02$), and were less likely to be current smokers (10.4% vs 16.8%, $P<0.001$). For all other risk factors, there were no substantial differences between participants who had complete SES data and those who did not have data ($P>0.2$ for all).

In a previous study, we showed that there was very little extreme mobility across social classes from childhood to adulthood; for example, only 24 of the 3444 women moved from Social Class I during childhood to Social Class V during adulthood, and just 4 moved from Social Class V during childhood to Social Class I during adulthood.[17] However, when broad categories were considered, there was a modest amount of upward mobility, with one third of the women moving from manual social classes during childhood to nonmanual social classes during adulthood. In total, 60% of the women remained in the same broad category of social class (44% of sample stayed in manual classes at both stages of the life course, and 16% stayed in nonmanual classes at both stages), and just 7% moved down from nonmanual classes during childhood to manual classes during adulthood.

The distributions for insulin resistance and the components of insulin resistance syndrome have been published elsewhere.[17] Linear associations between childhood and adult SES (each independent of social class at the other time in the life course) were found with insulin resistance, obesity, and adverse lipid profiles.[17] Table 1 shows the age-adjusted distributions of coronary heart disease, behavioral coronary heart disease risk factors, biomarkers of childhood exposures, and indicators of psychological distress by childhood social class. There were linear associations between childhood SES and coronary heart disease, current smoking, and physical inactivity, and those who were from the most adverse social classes during childhood had the worst outcomes.

Childhood SES was linearly associated with leg length and lower FEV_1; women who were from lower social classes as children had shorter legs and lower FEV_1 as adults. Among a subgroup of women (n=1605) who were lifelong nonsmokers and were either single or married to men who were lifelong nonsmokers, the linear association between childhood SES and FEV_1 remained: age- and adult-SES-adjusted difference per increase in 1 social class category among this subgroup was -0.05 (95% CI=-0.07, -0.03; $P<0.001$). There was a weak linear association between childhood SES and current anxiety or depression, and childhood SES was not associated with a history of clinical depression or with anxiolytic and antidepressant medication. Each increase in social class category was associated with a 14% increase in coronary heart disease after we adjusted for age and adult indicators of SES (social class, housing status, car ownership, and pension arrangements). When differences between broad categories of childhood SES were considered, the age- and adult-SES-adjusted odds ratio (95% confidence interval) for coronary heart disease among women who were in manual social classes compared with those who were in nonmanual social classes was 1.36 (95% CI=1.11, 1.69; $P<0.001$).

Adult SES was linearly associated with coronary heart disease, leg length, FEV_1, smoking, and indicators of psychological distress, and these associations were independent of childhood SES (Table 2). Just 162 of the women (4.7%) were single and were therefore classified by their own occupation. The age- and childhood-SES-adjusted association between adult SES among single women did not differ from that of women who were classified by their husband's occupation. The odds ratio for an increase in 1

TABLE 1—Age-Adjusted Means or Prevalences (95% Confidence Intervals) of Coronary Heart Disease and Coronary Heart Disease Risk Factors, by Childhood Social Class

	Registrar General's Occupational Classifications							
	Professional (I) (n = 110)	Managerial and Technical (II) (n = 308)	Nonmanual–Skilled (III-NM) (n = 398)	Manual–Skilled (III-M) (n = 1149)	Partly Skilled (IV) (n = 1056)	Unskilled (V) (n = 423)	Age-Adjusted Difference[a]	Age- and Adult-SES-Adjusted Difference[a]
Coronary heart disease, %	9.4	12.9	12.3	14.0	16.5	18.5	1.15	1.14
	(5.3, 16.3)	(9.6, 17.1)	(9.4, 15.9)	(12.1, 16.2)	(14.4, 18.9)	(14.4, 18.9)	(1.06, 1.24)	(1.04, 1.20)
Ever smoked, %	40.0	39.5	39.9	42.1	45.1	44.9	1.07	1.03
	(31.3, 49.4)	(34.2, 45.1)	(35.2, 44.8)	(39.3, 45.0)	(42.1, 48.1)	(40.2, 49.7)	(1.01, 1.13)	(0.97, 1.10)
Current smoker, %	5.3	7.6	7.8	9.0	12.8	11.8	1.20	1.17
	(2.4, 11.3)	(5.2, 11.2)	(5.6, 10.9)	(7.5, 10.8)	(10.9, 14.9)	(9.1, 15.2)	(1.09, 1.32)	(1.07, 1.29)
Physically inactive, %	11.7	14.4	14.0	15.2	23.1	18.0	1.17	1.15
	(6.9, 19.3)	(10.8, 18.9)	(10.9, 18.9)	(13.1, 17.5)	(20.5, 25.9)	(14.4, 22.1)	(1.08, 1.26)	(1.06, 1.25)
Leg length, mm	767.8	768.2	766.5	757.4	753.1	756.7	–3.68	–3.14
	(760.2, 775.3)	(763.6, 772.7)	(762.4, 770.5)	(755.0, 759.8)	(750.6, 759.8)	(752.7, 760.7)	(–4.80, –2.57)	(–4.30, –1.98)
FEV_1, l	2.12	2.12	2.09	1.98	1.94	1.94	–0.05	–0.03
	(2.03, 2.21)	(2.07, 2.18)	(2.05, 2.14)	(1.95, 2.01)	(1.91, 1.97)	(1.89, 1.99)	(–0.06, –0.04)	(–0.05, –0.02)
Anxious or depressed	21.8	22.5	21.3	22.7	25.6	28.1	1.09	1.04
	(15.1, 30.5)	(18.1, 27.5)	(17.6, 25.6)	(20.4, 25.2)	(23.1, 28.4)	(24.0, 32.6)	(1.02, 1.16)	(0.98, 1.11)
History of clinical depression	17.1	14.5	15.7	16.2	16.4	15.7	1.01	0.97
	(11.2, 25.3)	(11.0, 18.9)	(12.4, 19.6)	(14.2, 18.5)	(14.2, 18.7)	(12.5, 19.5)	(0.94, 1.09)	(0.91, 1.05)
Use of anxiolytic, hypnotic, or antidepressant	9.9	8.4	11.0	10.0	9.5	10.3	1.01	1.00
	(5.6, 17.1)	(5.8, 12.1)	(8.3, 14.5)	(8.4, 11.9)	(7.9, 11.4)	(7.8, 13.6)	(0.92, 1.10)	(0.92, 1.10)

Note. FEV_1 = forced expiratory volume in 1 second; SES = socioeconomic status.
[a]Difference = age (and adult social class, housing status, car ownership, and pension arrangements) adjusted regression coefficient per increase in social class category for continuous variables and odds ratio per increase in social class category for dichotomous variables.

category of adult social class for single women was 1.16 (95% CI=0.79, 1.72), and for women who were classified by their husband's occupation, the odds ratio was 1.17 (95% CI=1.09, 1.25).

The magnitude of the association between childhood SES and leg length was stronger than that between adult SES and leg length, which is consistent with the hypothesis that adult leg length is a useful biomarker of childhood nutrition and other adverse childhood exposures.[21] Among the subgroup of women who had little or no lifelong exposure to tobacco, the age- and childhood-SES-adjusted difference in FEV_1 per increase of 1 adult social class category was −0.02 (95% CI=-0.04, -0.01; P=0.004). This is lower than that between childhood SES and FEV_1 among this subgroup, which again supports the hypothesis that this is a useful biomarker of early-life exposures. The magnitudes of the associations between adult SES and smoking and physical activity were weaker than those between childhood SES and these same risk factors, whereas the associations between adult SES and indicators of psychological distress were stronger than those between childhood SES and these indicators. None of the differences in the magnitudes between childhood and adulthood SES among any of these associations were statistically significant at the conventional 5% level (P>0.25 for all).

There was no strong evidence of any interactions between childhood and adult SES among the associations with coronary heart disease and coronary heart disease risk factors (P>0.2 for all). There was a cumulative effect of SES across the life course such that the age-adjusted odds ratio for prevalence of coronary heart disease and most risk factors was greatest among women in manual social classes at both stages of the life course compared with those in nonmanual social classes at both stages, and it was intermediate among those who were in manual social classes at just 1 point in the life course (Table 3). Women who were upwardly mobile from manual social classes during childhood to nonmanual social classes during adulthood remained at increased risk for coronary heart disease, diabetes, components of the insulin resistance syndrome, smoking, and physical inactivity compared with women who were in nonmanual social classes at both stages of the life course. Women who were upwardly mobile were no more likely to be psychologically distressed than those who were in nonmanual social classes at both stages of the life course. Women who were downwardly mobile, from nonmanual to manual social classes, were at a particularly high risk compared with those who were in nonmanual social classes at both stages of the life course for prevalence of coronary heart disease, most coronary heart disease risk factors, and indicators of psychological distress. However, the numbers of women in this category were small, and the estimates were imprecise.

TABLE 2—Age-Adjusted Means or Prevalences (95% Confidence Intervals) of Coronary Heart Disease and Coronary Heart Disease Risk Factors, by Adult Social Class

	Registrar General's Occupational Classifications							
	Professional (I) (n = 305)	Managerial and Technical (II) (n = 769)	Nonmanual–Skilled (III-NM) (n = 612)	Manual–Skilled (III-M) (n = 954)	Partly Skilled (IV) (n = 481)	Unskilled (V) (n = 323)	Age-Adjusted Difference[a]	Age- and Childhood-SES-Adjusted Difference[a]
Coronary heart disease, %	10.4	11.0	12.9	17.4	20.3	16.6	1.18	1.17
	(7.4, 14.3)	(9.0, 13.4)	(10.5, 15.8)	(15.1, 20.0)	(16.9, 24.1)	(12.9, 21.0)	(1.11, 1.27)	(1.09, 1.26)
Ever smoked, %	36.2	41.0	42.6	44.5	42.8	48.9	1.08	1.07
	(31.0, 41.7)	(37.6, 44.5)	(38.7, 46.5)	(41.4, 47.7)	(38.5, 47.3)	(43.5, 54.4)	(1.03, 1.13)	(1.02, 1.12)
Current smoker, %	6.7	9.3	9.1	11.8	10.8	11.2	1.10	1.06
	(4.4, 10.1)	(7.5, 11.6)	(7.1, 11.6)	(9.9, 14.0)	(8.3, 13.9)	(8.2, 15.1)	(1.01, 1.18)	(0.98, 1.15)
Physically inactive, %	5.8	7.4	8.1	7.8	10.5	11.2	1.12	1.09
	(3.7, 9.0)	(5.7, 9.5)	(6.1, 11.6)	(6.2, 9.7)	(8.0, 13.9)	(8.2, 15.1)	(1.05, 1.19)	(1.02, 1.16)
Leg length, mm	761.1	761.1	759.4	755.8	752.9	756.2	–2.48	–1.78
	(758.2, 764.0)	(758.2, 764.0)	(756.1, 762.7)	(753.1, 758.4)	(749.1, 756.7)	(751.7, 760.8)	(–3.44, –1.51)	(–2.77, –0.78)
FEV_1, l	2.13	2.08	1.97	1.96	1.88	1.94	–0.05	–0.04
	(2.03, 2.19)	(2.04, 2.11)	(1.93, 2.01)	(1.93, 1.99)	(1.84, 1.93)	(1.89, 2.00)	(–0.06, –0.04)	(–0.05, –0.02)
Anxious or depressed	22.0	20.0	22.2	24.9	28.5	29.7	1.12	1.11
	(17.7, 27.0)	(17.5, 23.1)	(19.1, 25.7)	(22.3, 27.8)	(24.6, 32.7)	(25.0, 34.9)	(1.06, 1.18)	(1.05, 1.17)
History of clinical depression	18.0	12.4	12.8	18.6	16.9	20.0	1.10	1.10
	(14.1, 22.7)	(10.2, 14.9)	(10.4, 15.7)	(16.3, 21.2)	(13.8, 20.5)	(16.0, 24.7)	(1.03, 1.17)	(1.03, 1.17)
Use of anxiolytic, hypnotic, or antidepressant	8.5	10.1	8.9	11.2	8.3	11.1	1.03	1.02
	(5.8, 12.2)	(8.1, 12.4)	(6.9, 11.5)	(9.3, 13.3)	(6.1, 11.1)	(8.1, 15.0)	(0.95, 1.11)	(0.95, 1.11)

Note. FEV_1 = forced expiratory volume in 1 second; SES = socioeconomic status.
[a]Difference = age (and childhood social class, household bathroom, household hot-water supply, family access to a car) adjusted regression coefficient per increase in social class category for continuous variables and odds ratio per increase in social class category for dichotomous variables.

Table 4 shows the associations between childhood SES and prevalence of coronary heart disease, and it shows the effects on these associations after we adjusted for components of insulin resistance syndrome, behavioral risk factors, biomarkers of adverse childhood exposures, and indicators of psychological distress. Compared with women who were in nonmanual social classes during childhood, women who were in manual social classes had an age- and adult-SES-adjusted odds ratio for coronary heart disease of 1.35 (95% CI=1.08, 1.67). This was attenuated by 38% when we adjusted for components of insulin resistance syndrome, 29% when we adjusted for adult smoking and physical activity, and 50% when we adjusted for leg length and FEV_1. Adjustment for indicators of psychological distress had no substantive effect on this association. The fully adjusted (for age and all potential explanatory factors) odds ratio was 1.13 (95% CI=0.85, 1.49). Among the subgroup of lifelong nonsmokers, the age- and adult-SES-adjusted odds ratio for coronary heart disease when we compared women in manual social classes with women in nonmanual social classes during childhood was 1.45 (95% CI=1.01, 2.06). This was reduced by 22% to 1.34 (0.93, 1.94) when we further adjusted for FEV_1 alone.

To determine whether the association between childhood SES and adult coronary heart disease was related to intrauterine exposures, we examined the association among a subgroup of women who had self-reported birthweight data (1194 [34.7%] of the 3444 women who had complete SEP data).[20] Among this subgroup, the age- and adult-SES-adjusted odds ratio for coronary heart disease when we compared those in manual social classes with those in nonmanual childhood social classes was 1.28 (0.91, 1.83). When we adjusted for birthweight, this odds ratio was not substantively altered (1.26; 95% CI=0.90, 1.81).

DISCUSSION

Our findings indicate that adverse SES during childhood is associated with increased risk for coronary heart disease among women and that infant and childhood nutrition, insulin resistance, and adult behavioral risk factors each play a part in this association. There was a cumulative effect of disadvantage across the life course such that women who were in manual social classes during childhood remained at increased risk for coronary heart disease even if they moved up into nonmanual social classes during adulthood. Furthermore, those who were in manual social classes at both stages of the life course had a particular risk for coronary heart disease.

Link Between Childhood SES and Risk for Coronary Heart Disease

We found that childhood SES was associated with smoking status and physical activity during adulthood, and we found that adjust-

TABLE 3—Odds Ratios (95% Confidence Intervals) for Coronary Heart Disease and Coronary Heart Disease Risk Factors Comparing Various SES in Both Childhood and Adulthood With Baseline of Nonmanual Social Class in Both Childhood and Adulthood

	Child: Nonmanual Adult: Nonmanual (n = 565)	Child: Nonmanual Adult: Manual (n = 251)	Child: Manual Adult: Nonmanual (n = 1121)	Child: Manual Adult: Manual (n = 1507)
CHD	1.00	1.70 (1.16, 2.61)	1.23 (0.89, 1.69)	2.01 (1.50, 2.72)
High insulin resistance (top quartile HOMA ≥ 2.45)	1.00	1.29 (0.86, 1.93)	1.33 (1.01, 1.74)	1.58 (1.22, 2.05)
Diabetes (clinical diagnosis or fasting glucose ≥ 7.8mmol/l)	1.00	1.62 (0.79, 3.35)	1.55 (0.91, 2.63)	2.41 (1.46, 3.96)
Hypertensive (blood pressure ≥ 140/90mmHg or antidepressant medication)	1.00	1.29 (0.94, 1.77)	1.28 (1.04, 1.59)	1.34 (1.09, 1.65)
Dyslipidaemia (TG ≥ 1.7mmol/l or HDLc < 1.0 mmol/l)	1.00	1.36 (0.99, 1.89)	1.34 (1.08, 1.66)	1.72 (1.40, 2.12)
Obesity (BMI > 30kg/m^2 or WHR > 0.85)	1.00	1.50 (1.10, 2.05)	1.18 (0.95, 2.03)	1.65 (1.35, 2.03)
Current smoker	1.00	1.15 (0.66, 2.00)	1.38 (0.95, 2.01)	1.75 (1.23, 2.49)
Inactive	1.00	1.67 (1.09, 2.55)	1.55 (1.14, 2.10)	1.90 (1.14, 2.54)
Short legs (lowest quartile leg length ≤ 732 mm)	1.00	1.73 (1.18, 2.51)	1.68 (1.28, 2.19)	2.38 (1.84, 3.07)
Low FEV_1 (lowest quartile FEV_1 ≤ 1.66 l)	1.00	1.17 (0.78, 1.76)	1.60 (1.22, 2.11)	2.16 (1.67, 2.81)
Anxious or depressed	1.00	1.23 (0.86, 1.75)	1.05 (0.82, 1.35)	1.44 (1.14, 1.81)
History of clinical depression	1.00	1.33 (0.89, 1.98)	0.91 (0.68, 1.22)	1.34 (1.03, 1.76)
Use of anxiolytic, hypnotic, or antidepressant	1.00	1.37 (0.85, 2.21)	1.08 (0.76, 1.53)	1.15 (0.82, 1.60)

Note. SES = socioeconomic status; CHD = coronary heart disease; HOMA = homeostasis model assessment (of insulin resistance); TG = triglycerides; HDLc = high-density lipoprotein cholesterol; BMI = body mass index; WHR = waist-to-hip ratio; FEV_1 = forced expiratory volume in 1 second.

TABLE 4—Odds Ratios (95% Confidence Intervals) for Coronary Heart Disease Comparing Childhood Manual Social Class With Nonmanual Social Class

Variables Included in Fully Adjusted Model	Number With Complete Data on All Variables Included in Fully Adjusted Model	Age- and Adult-SES-Adjusted OR (95% CI)	Fully Adjusted OR (95% CI)	Percentage Decrease in OR With Full Adjustment
Components of the insulin resistance syndrome[a]	2986	1.34 (1.03, 1.75) *P* = .02	1.21 (0.92, 1.58) *P* = .18	38
Behavioral risk factors[b]	3410	1.35 (1.08, 1.68) *P* = .005	1.25 (0.98, 1.59) *P* = .07	29
Biomarkers of early-life environmental exposures[c]	3363	1.36 (1.08, 1.68) *P* = .005	1.18 (0.92, 1.48) *P* = .30	50
Indicators of anxiety/depression[d]	3414	1.36 (1.10, 1.69) *P* < .001	1.35 (1.10, 1.68) *P* = .002	2.8
All potential explanatory factors[e]	2842	1.34 (1.03, 1.74) *P* = .02	1.13 (0.85, 1.49) *P* = .41	62

Note. OR = odds ratio; CI = confidence interval; SES = socioeconomic status (adult SES factors adjusted for adult social class, adult housing status, car ownership, and pension arrangements); HOMA = homeostasis model assessment (of insulin resistance); FEV_1 = forced expiratory volume in 1 second.
[a]High-density lipoprotein cholesterol, triglycerides, systolic blood pressure, diastolic blood pressure, waist to hip ratio, body mass index (all continuous variables), HOMA score-type 2 diabetes categories (first 5 categories quintiles of HOMA score, sixth category type 2 diabetes).
[b]Smoking status (never, ex, or current at 1 of 4 levels: 1–9, 10–19, 20–29, or ≥ 30 cigarettes per day) and physical activity (none, < 1, 1–2, ≥ 2 hours per week of moderate or vigorous physical activity).
[c]Leg length, FEV_1 (continuous variables).
[d]Currently feel moderately or extremely anxious and/or depressed, ever been diagnosed by a doctor with depression, currently uses anxiolytics, hypnotics, or antidepressants (all binary).
[e]All variables included in models a–d.

ing for these risk factors attenuated the association between childhood SES and prevalence of coronary heart disease. In other studies, inconsistent results have been found for the association between childhood SES and smoking and physical activity in later life,[27–29] although among women, more consistent results between adverse childhood SES and adverse adult behavioral risk factors have been found.[1,30] It is perhaps not surprising that some association should exist. Parental SES will influence parental behaviors, and these behaviors are likely to influence children's smoking and physical activity levels during childhood and adolescence, and these behaviors are known to persist from childhood into adulthood.[31] The association between adverse childhood SES and coronary heart disease may therefore be explained in part by adverse behavioral risk factors that persist from childhood into adulthood among those who are from the poorest backgrounds.

Most investigators agree that the direct effects of poverty and material deprivation at an

individual level (e.g., inability to afford a healthy diet) and at a societal level (e.g., living in an area with little investment in housing, transport infrastructure, and community facilities) are important pathways in the association between SES and disease outcomes.[32,33] Poverty and material disadvantage may act in different ways at different stages of the life course. For example, poor intrauterine nutrition and childhood diet may lead to insulin resistance that persists into adulthood and increases the risk for coronary heart disease, whereas poor diet in adulthood may have an effect, via obesity, on coronary heart disease. The attenuation of the association between childhood SES and adult coronary heart disease after we adjusted for components of insulin resistance syndrome and biomarkers of childhood nutrition supports a role for early-life nutritional programming of insulin resistance as an intermediary in the causal pathway. The association between childhood SES and adult coronary heart disease was independent of birthweight, and these results are consistent with a previous prospective study.[4] Our findings indicate that intrauterine nutritional deprivation may be less important than postnatal nutritional effects in the association between childhood SES and risk for coronary heart disease.

The association between childhood SES and FEV_1 among a subgroup of women who had little or no lifetime exposure to tobacco, and the attenuation of the association between childhood SES and coronary heart disease after we adjusted for FEV_1 among this same subgroup, indicate that adverse childhood SES affects lung growth independently of smoking and that the childhood exposures that affect lung growth have a detrimental effect on risk for coronary heart disease. FEV_1 during adulthood is affected by exposure to maternal smoking in childhood,[23] and exposure to environmental tobacco smoke during childhood may therefore have a long-term effect on adult risk for coronary heart disease that is independent of whether the individual smokes.

In addition to the direct effects of poverty, it has been hypothesized that relative poverty leads to increased risk for coronary heart disease, because the emotional stress of recognizing relatively inferior SES leads to the neuroendocrine responses hypothesized to increase risk for coronary heart disease.[33] The graded association across the whole distribution between job grade and coronary heart disease mortality in the Whitehall study has been used to support the hypothesis that relative poverty in adulthood is important.[34,35] We found that the association between childhood SES and prevalence of adult coronary heart disease was graded across the childhood social classes. However, we found only weak and inconsistent associations between childhood SES and indicators of adult psychological distress, and adjustment for these indicators did not affect the association between childhood SES and coronary heart disease. Therefore, our findings do not support adult psychological distress as an important variable in the association between childhood SES and risk for coronary heart disease.

Study Limitations

Our response rate (60%) was moderate but consistent with other baseline data used in large epidemiological surveys.[37] Compared with those who did not respond, participants tended to be younger and less likely to have diabetes, although prevalence of coronary heart disease was similar among participants and nonresponders.[18] The possibility that our cohort was healthier than the general population of older British women should not have affected our results—it would only do so if the associations we examined were in the opposite direction or were markedly weaker among nonresponders, which is unlikely.

The women in our study who did not have data on childhood and adult SES were more likely to have coronary heart disease and to be smokers, and they had larger waist-hip ratios than those who did have these data. A large percentage of the women who did not have occupational data were likely to be those whose fathers and husbands were long-term unemployed—this would be consistent with the high prevalence of coronary heart disease and smoking among those who did not have these data. Including these women with those who were in manual social classes in our analysis slightly strengthened the association between adverse childhood SES and adult coronary heart disease (data not shown) and did not alter our overall conclusions. We relied upon self-report for occupational data, which may have been less accurate for father's than for husband's occupation, although any misclassification would weaken associations. All occupations were classified in accordance with the Registrar General's Classification of Occupations for 1980, which may have introduced inaccuracies in SES classification for fathers' occupations from the 1930s to 1950s. However, over the last century, very few occupations have substantially changed status, particularly jobs classified as manual or nonmanual, which have not changed between these broad categorizations.[38] Our study is cross-sectional, and although reverse causality as an explanation for the association between childhood SES and adult coronary heart disease is implausible, survivor bias could have been a problem. However, mortality caused by coronary heart disease among women before the age of 70 years (mean age of women in our study) is uncommon; therefore, survivor bias is an unlikely explanation for our results. Our results are consistent with a number of prospective studies.[1]

CONCLUSIONS

The participants in our study were born during a time of economic deprivation in Britain, and it could be argued that contemporary British children are unlikely to be exposed to such adverse circumstances. The association with coronary heart disease was linear across the distribution of childhood social classes, which indicates that increased risk does not only occur with extreme deprivation. Furthermore, among the 1946 British birth cohort—a group born into the post–World War II welfare state and greater prosperity—premature mortality was strongly influenced by adverse childhood SES,[39] and among a cohort aged 26 years from New Zealand, adverse childhood SES was associated with adverse coronary heart disease risk factors.[40] Randomized control trial evidence would provide the strongest evidence of the effectiveness of specific policies aimed at reducing the effects of childhood poverty on risk for coronary heart disease, but the pathways identified in our study show the types of intervention that might be most beneficial. ■

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Contributors

All the authors developed the study's objectives. S. Ebrahim and D.A. Lawlor managed the data collection and storage for the British Women's Heart and Health Study. D.A. Lawlor performed the analysis and coordinated the writing of the article. All the authors contributed to the article.

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Human Participant Protection

We obtained approval from the ethics committees of each of the 23 towns from which the study sample was drawn.

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