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Life-course influences on health in British adults: effects of socio-economic position in childhood and adulthood

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Background Little evidence exists on the role of socio-economic position (SEP) in early life on adult disease other than for cardiovascular mortality; data is often retrospective. We assess whether childhood SEP influences disease risk in mid-life, separately from the effect of adult position, and establish how associations vary across multiple measures of disease risk.

Methods Prospective follow-up to adulthood of all born in England, Scotland and Wales during 1 week in 1958, and with medical data at age 45 years ($n = 9377$). Outcomes include: blood pressure, body mass index (BMI), glycosylated haemoglobin (HbA1c), total and high density lipoprotein (HDL) cholesterol, triglycerides, fibrinogen, total immunoglobulin E (IgE), one-second forced expiratory volume (FEV1), hearing threshold (4 kHz), visual impairment, symptoms of depression and anxiety, chronic widespread pain.

Results Social class in childhood was associated with blood pressure, BMI, HbA1c, HDL cholesterol, triglycerides, fibrinogen, FEV1, hearing threshold, depressive symptoms and chronic widespread pain, with a general trend of deteriorating health from class I to V. Adult social class was also associated with these measures. Mutually adjusted analyses of child and adult social class suggest that both contribute to disease risk in mid-life: in general, associations for childhood

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class were as strong as for adult class. Individuals with a manual class at both time-points tended to have the greatest health deficits in adulthood.

Conclusions Adverse SEP in childhood is associated with a poorer health profile in mid-adulthood, independently of adult social position, and across diverse measures of disease risk and physical and mental functioning.

Keywords Social class, child and adult, cardio-respiratory disease, health inequalities, birth cohort

Introduction

The influence on adult disease and health functioning of factors from earlier life stages is a major research focus. Some evidence has come from studies examining effects of childhood and adult socio-economic position (SEP), because these studies provide clues on the life stages when factors might act to affect later disease risk. The most extensive research on associations with child and adult SEP has been undertaken for mortality, especially from cardiovascular disease.[1–3] Studies of morbidity and of risk factors such as blood pressure, are often in older populations or reliant upon retrospective reports of social position in early life.[3–6] Little evidence is available from prospective studies of childhood social position and disease risk and physical and mental functioning in early to mid-adult life, even though associations with adult social position are well-established for such outcomes.[7,8]

We obtained key biological markers at 45 years for the 1958 British birth cohort in order to investigate when in the life-course adult disease risk and health function is determined. Our specific aim here is to establish whether childhood SEP is associated with disease risk and health function, and whether effects operate primarily through their influence on adult SEP. To investigate whether factors in childhood contribute to the health burden in mid-adulthood, and to related inequalities, we assess associations for childhood SEP using disease risk measures for major chronic disease and for physical and mental functioning. Elsewhere we investigate place of residence at different life stages in the same cohort and using the same measures of disease risk and health function.[9]

Methods

Study sample

Participants were originally enrolled in the Perinatal Mortality Survey (PMS) of all born in England, Scotland and Wales, during 1 week in March 1958[10,11] with follow-up throughout childhood and adulthood, most recently at 44–45 years.[12] 17 415 individuals participated in the PMS from an eligible sample of 17 638. Immigrants with the same birth dates were recruited up to age 16 years ($n = 920$), thus 18 558 individuals in total were eligible study participants. At 44–45 years, from a target sample of 12 069 participants still in contact with the study, and who at 42 years had not required a proxy interview, were invited to a clinical examination undertaken in their home by a trained nurse; 9377 participants were seen from September 2002 to March 2004. In analyses reported here, fewer participants had an unskilled manual class (IV or V) in childhood, compared with the original sample, although the difference was small: 22.3 *vs* 26.9%.

Measures

Blood pressure was measured three times with the participant seated and rested for 5 min, using an Omron 705CP automated sphygmomanometer (Omron, Tokyo, Japan); a large cuff was used when mid-upper arm circumference $\geqslant$32 cm. Mean blood pressure was calculated from measurements considered by the nurse to be reliable. Standing height was measured using a Leicester portable stadiometer; participants were unshod, lightly clothed and stood upright with their head in the Frankfort plane. Weight was measured to the nearest 0.1 kg with shoes removed. Self-reported weight ($n = 100$) or height ($n = 84$) was recorded when accurate measurements or consent were unavailable. Body mass index (BMI, kg/m^2) was calculated. Spirometry was performed whilst standing, without noseclips, using the Vitalograph Micro spirometer. At least three blows were recorded, and up to five were performed if the best-test variation (assessed by the sum of one-second forced expiratory volume (FEV1) and forced vital capacity) was >5%. Readings with a best-test variation >10% or values with standardized residuals $>\pm 3$ SDs were excluded. Non-fasted venous blood samples were obtained. Glycosylated haemoglobin (HbA1c) was assayed by ion exchange high performance liquid chromatography on whole blood.[13] Triglycerides, total and high density lipoprotein (HDL) cholesterol were measured by autoanalyser. Fibrinogen was assayed by the Clauss assay in an MDA-180 automated coagulometer (Biomerieux, Basingstoke). Total immunoglobulin E (IgE) was measured using the HYTEC enzyme immunoassay; values >2000 were recoded to 2000; zeros were recoded to 0.5.

Pure tone audiometry was performed by air conduction in both ears, with test tones at 1 kHz and 4 kHz according to the British Society of Audiology's recommended procedure.[14] MA25 portable audiometers with TDH 49 earphones in audiocups were used, calibrated to British Standard BS EN ISO 389–1 (2000).[15] We used hearing level at 4 kHz in the better ear, if hearing at 1 kHz was <30 dB. Best distance visual acuity in each eye was measured, using 3 m Keeler crowded logarithm of the minimum angle of resolution (log MAR) test, with pinhole and distance correction if prescribed (glasses/contact lenses); impairment was defined as log MAR >0.2 in the better eye. Symptoms of depression and anxiety in the previous week were measured by the revised Clinical Interview Schedule[16] administered by the nurse. Participants reporting $\geqslant$2 items for the depressive symptoms or anxiety modules were identified. Chronic widespread pain was identified from a manikin (shaded by participants who located pain lasting

$\geqslant$24 h during the previous month) and defined as pain $\geqslant$3 months duration, in the axial skeleton and two contralateral body quadrants.[17]

SEP in childhood is based on father's occupation in 1958, (or 1965 if data was unavailable at birth; $n = 422$). Adult position is based on the participant's current or most recent occupation at 42 years (or 33 years if data were unavailable at 42 years; $n = 1142$). Six Registrar General's occupational groups were used: professional (I), managerial/technical (II), other non-manual (IIInm), skilled manual (IIIm), partly skilled (IV) and unskilled manual (V); those with no male head of household in childhood were grouped with class V. Participants lacking information on both child and adult class ($n = 17$) are excluded.

Analysis

Sex-adjusted means or proportions of health outcomes were calculated for each social class in childhood and adulthood. For continuous outcomes we used linear regression [95% confidence intervals (CI)] to examine separately the association with child and adult social class, and then, in mutually adjusted models. Following the same procedure, logistic regression (giving odds ratios, 95% CIs) was used for categorical outcomes. When examined for main effects, social class was treated as an ordinal variable; when examined as a confounding factor it was treated as a nominal categorical variable, with a separate category for missing information in order to minimize attrition in mutually adjusted models.

Associations between social class and health outcome could, potentially, vary by sex. For example, adult social position is more consistently related to obesity in women than men[18]; sex differences have also been reported for socially patterned early life risk factors for adult chronic disease, such as birthweight.[19] Thus, interactions between sex and class were tested and where $P \leqslant 0.05$ results are presented separately for men and women; otherwise results are reported for the combined sample of men and women, adjusted for sex.

HDL cholesterol, triglycerides, fibrinogen, IgE and hearing threshold were handled using a log normal transformation; geometric means are presented and percentage change in outcome calculated.[20] For HbA1c, geometric means are presented but analyses use untransformed data with robust estimators. Throughout FEV1 was adjusted for height. To compare the strength of association for child and adult social class across different outcomes, we repeated analyses for continuous outcomes using SD scores. Where appropriate, analyses were adjusted for factors that might affect the measurement of outcome (treatment for hypertension or diabetes, use of respiratory inhaler, recent food consumption, recent chest infection, flooring, air temperature, background noise, time or month of interview, laboratory batch and delay in receiving blood sample). These analyses and additional analyses of potential nurse effects, modelled as a random effect, showed negligible effects on associations with social class (data not shown).

Finally, we examined combined effects of child and adult class on health outcomes, first using tests for interaction, and then, using a cross-classification. For the latter, child and adult class were dichotomized into non-manual and manual categories and sex-adjusted means or prevalence were calculated. All analyses were conducted in Stata 9.1 (StataCorp, Texas, USA).

Results

Table 1 shows the distribution of the cohort by social class in childhood and in adulthood: mobility between the two life stages was substantial, with over two-thirds (68% of men; 70% of women) having a manual class in childhood, compared with only 42% of men and 27% of women in adult life.

Associations with childhood class were found for systolic and diastolic blood pressure, BMI, HbA1c, HDL cholesterol, triglycerides, fibrinogen, FEV1, hearing threshold at 4 kHz, depressive symptoms and chronic widespread pain, with a general trend of deteriorating disease risk from class I to V (Table 2). No association was found for total cholesterol, IgE, visual impairment or anxiety symptoms. For fibrinogen the association with childhood class was stronger for women than men (interaction $P = 0.0008$); weaker interactions with sex were found for BMI and anxiety ($P = 0.054$ and $P = 0.050$, respectively). Table 2 shows that after adjustment for adult class, most associations for childhood attenuate. SD scores suggest that the strongest associations are for BMI, HDL cholesterol and hearing, and among women, for fibrinogen; using ORs, the strongest associations are for chronic widespread pain and depressive symptoms (Table 2).

Table 3 shows associations between adult class and systolic and diastolic blood pressure, BMI, HbA1c, fibrinogen, FEV1, hearing threshold, visual impairment, depressive symptoms,chronic widespread pain and anxiety symptoms. For women, associations were also observed for HDL cholesterol and triglycerides ($P = 0.0005$ and $P = 0.048$, respectively for interactions between sex and adult class). Adult class was also more strongly associated with BMI among women (interaction $P = 0.002$); for hearing the association was stronger in men (interaction $P = 0.017$). No association was found for total cholesterol or IgE. When adjusting adult class for childhood class, associations weakened slightly. SD scores suggest that the strongest associations with adult class were for hearing and fibrinogen, whilst ORs indicate stronger associations for visual impairment and chronic widespread pain. Inspection of SD scores and ORs (Tables 2 and 3) suggest that, for several outcomes, associations with childhood class were at least as strong as for adult class.

From mutually adjusted models of both child and adult class, some outcomes were associated only with adult class (visual impairment) and some with neither (total IgE). But for most outcomes an association with child and adult class suggests that influences may be acting at both life stages. Cross-classification of non-manual and manual classes in childhood and adulthood illustrates their combined effect: the general trend was for participants with a manual class at both time-points to have the greatest health deficit, for example, their mean sex-adjusted systolic blood pressure was 128.1 mm/Hg; non-manual classes at both times had the most favourable health profile, with for example, an average systolic pressure of 125.1 mm/Hg. Those changing social class between childhood and adulthood were mostly intermediate on each outcome (data not presented).

Table 1 Social class in childhood and in adulthood for 4665 men and 4712 women aged 44–45 years

	Adult social class							
Childhood social class	I (%)	II (%)	III Non-manual (%)	III Manual (%)	IV (%)	V (%)	Unknown (%)	**Total (%)**
Males								
I	50 (20.0)	130 (52.0)	22 (8.8)	26 (10.4)	7 (2.8)	4 (1.6)	11 (4.4)	250 (5.4)
II	78 (12.3)	351 (55.4)	57 (9.0)	100 (15.8)	27 (4.3)	7 (1.1)	14 (2.2)	634 (13.6)
III Non-manual	43 (9.4)	214 (46.5)	53 (11.5)	104 (22.6)	26 (5.7)	8 (1.7)	12 (2.6)	460 (9.9)
III Manual	125 (5.7)	737 (33.5)	210 (9.6)	778 (35.4)	199 (9.1)	64 (2.9)	87 (4.0)	2200 (47.2)
IV	20 (3.7)	161 (30.1)	34 (6.4)	201 (37.6)	72 (13.5)	27 (5.1)	20 (3.7)	535 (11.5)
V	14 (3.2)	121 (27.6)	43 (9.8)	163 (37.2)	49 (11.2)	18 (4.1)	30 (6.9)	438 (9.4)
Unknown	17 (11.5)	54 (36.5)	11 (7.4)	39 (26.4)	13 (8.8)	4 (2.7)	10 (6.8)	148 (3.2)
Total	347 (7.4)	1768 (37.9)	430 (9.2)	1411 (30.3)	393 (8.4)	132 (2.8)	184 (3.9)	4665
Females								
I	21 (9.7)	107 (49.5)	47 (21.8)	10 (4.6)	21 (9.7)	0	10 (4.6)	216 (4.6)
II	38 (6.0)	284 (44.9)	181 (28.6)	32 (5.1)	68 (10.7)	11 (1.7)	19 (3.0)	633 (13.4)
III Non-manual	13 (2.9)	154 (34.3)	152 (33.9)	35 (7.8)	59 (13.1)	8 (1.8)	28 (6.2)	449 (9.5)
III Manual	48 (2.2)	665 (29.8)	744 (33.4)	163 (7.3)	394 (17.7)	107 (4.8)	109 (4.9)	2230 (47.3)
IV	10 (1.8)	128 (23.2)	196 (35.6)	46 (8.4)	110 (20.0)	33 (6.0)	28 (5.1)	551 (11.7)
V	6 (1.2)	133 (26.6)	154 (30.8)	37 (7.4)	95 (19.0)	35 (7.0)	40 (8.0)	500 (10.6)
Unknown	9 (6.8)	45 (33.8)	41 (30.8)	9 (6.8)	20 (15.0)	2 (1.5)	7 (5.3)	133 (2.8)
Total	145 (3.1)	1516 (32.2)	1515 (32.2)	332 (7.1)	767 (16.3)	196 (4.2)	241 (5.1)	4712

Those with no child and no adult social class information were excluded from analyses.

Interactions between child and adult class were not significant for any health measure.

Discussion

Our study demonstrates social inequalities across several measures of disease risk and physical and mental functioning in mid-adult life. This is noteworthy in part because at 45 years clinical disease and preclinical functional decline are uncommon. Importantly, for most health outcomes, associations were found for both child and adult socio-economic position: in other words, exposures in early life appear to be compounded by those occurring later. In several instances, associations with social class in childhood were as strong as for adult class. This is of interest, given that associations with adult class might partly be due to health-related social mobility, whereas those for childhood class cannot. Our findings therefore support the argument that influences on adult disease risk and function are to be found in childhood, as well as in adulthood. In relation to proposed life-course models[21,22] our findings are most supportive of cumulative effects, in that child and adult class both contribute to health outcomes at 45 years, but vulnerability (interactive) effects were not evident. Programming effects of early life exposures might be operating, as part of an accumulation of influences across the life-span. A corollary of the cumulative effects demonstrated here is that social inequalities in adult health are to some extent due to factors in childhood.[9]

Methodological considerations

Social position was ascertained prospectively in a large, nationwide sample followed over 45 years, and most outcomes were objective measures of disease risk and health function. Effects of childhood SEP could be over-estimated in our analyses if women's own adult occupation is a weak measure of her social position. However, when repeating analyses using the occupation of head of household for women living with a partner, or own class for women not with a partner, changes to the results were found to be negligible. A further consideration is sample attrition occurring by age 45 years, however, only small biases by childhood class were observed. Other methodological issues concern the complexity of disentangling early and later life influences on adult health, one potential problem being collinearity of SEP measures across time. Importantly in our study the distribution of social class changed substantially from childhood to adulthood, as reported previously,[23] thereby permitting comparisons at different life-stages. Nonetheless, more detailed analyses that focus on a broader range of potential explanations are needed to understand the associations shown here.

Comparison with other studies

We confirm previous findings showing associations with both child and adult SEP for cardio-respiratory risk in adult life.[5,6,24,25] Whilst many studies focus primarily on blood pressure and FEV1, we include several indicators of cardio-respiratory risk. The plausibility of early life influences on cardiovascular disease is supported by autopsy studies showing asymptomatic atherosclerosis in young people.[26] Some of the associations with childhood class shown in our study are relatively large effects. There is, for example, an estimated BMI increase from class I to class V of about 2 kg/m^2, over the approximate BMI range, 26–28 kg/m^2. Recent estimates of the risk of death for women aged 50 years with a BMI 25–26.4

Table 2 Disease risk factors at 45 years related to childhood social class, values are sex-adjusted means or prevalence (%)

	Childhood social class						Difference (95% CI) per increase in social class grade		SD score
Risk factor	I	II	IIIn	IIIm	IV	V	Adjusted for sex[a]	Adjusted for sex and adult social class[b]	**Adjusted for sex and adult class[b]**
Systolic pressure (mm/Hg)	123.8	125.7	125.8	126.9	127.6	127.5	0.67 (0.42, 0.92)	0.52 (0.27, 0.78)	0.032 (0.016, 0.047)
Diastolic pressure (mm/Hg)	76.9	78.0	78.4	79.1	79.2	79.7	0.49 (0.32, 0.66)	0.43 (0.26, 0.61)	0.040 (0.024, 0.056)
BMI (kg/m^2)	26.1	26.4	26.8	27.7	28.2	27.8	0.45 (0.36, 0.53)	0.41 (0.32, 0.49)	0.080 (0.064, 0.097)
HbA1c (% total)[c]	5.16	5.17	5.18	5.22	5.27	5.28	0.034 (0.022, 0.047)	0.026 (0.013, 0.039)	0.036 (0.018, 0.054)
Total cholesterol (mmol/L)	5.87	5.81	5.85	5.91	5.92	5.88	0.018 (−0.001, 0.037)	0.021 (0.001, 0.041)	0.019 (0.001, 0.038)
HDL cholesterol (mmol/L)[d]	1.60	1.58	1.54	1.51	1.46	1.48	−0.019 (−0.023, −0.015)	−0.016 (−0.021, −0.012)	−0.067 (−0.084, −0.050)
Triglycerides (mmol/L)[d]	1.51	1.54	1.62	1.72	1.80	1.77	0.039 (0.030, 0.049)	0.036 (0.025, 0.046)	0.060 (0.043, 0.077)
Fibrinogen (g/L)[d]									
Males	2.74	2.79	2.79	2.84	2.83	2.90	0.009 (0.043, 0.014)	0.005 (−0.001, 0.010)	0.024 (−0.001, 0.049)
Females	2.77	2.80	2.93	3.00	3.06	3.05	0.023 (0.018, 0.028)	0.020 (0.015, 0.025)	0.098 (0.072, 0.125)
FEV1 (L)[c]	3.32	3.33	3.31	3.29	3.27	3.25	−0.031 (−0.039, −0.022)	−0.022 (−0.030, −0.013)	−0.029 (−0.040, −0.017)
Total IgE (kU/L)[d]	28.4	31.2	30.1	31.0	29.1	32.7	0.01 (−0.02, 0.04)	0.01 (−0.02, 0.03)	0.005 (−0.013, 0.023)
Hearing at 4kHz (dB)[d]	3.57	3.85	4.24	6.26	6.85	6.66	0.030 (0.024, 0.036)	0.023 (0.017, 0.029)	0.064 (0.048, 0.081)
							Odds ratios (95% CI) per increase in social class		
Visual impairment (%)	2.5	1.7	2.0	2.4	2.2	2.4	1.04 (0.93, 1.16)	0.99 (0.88, 1.11)	–
Depressive symptoms (%)	6.0	6.2	7.5	8.6	8.8	10.6	1.14 (1.07, 1.21)	1.09 (1.03, 1.16)	–
Anxiety symptoms (%)	7.3	7.0	6.8	7.0	6.7	6.8	0.99 (0.93, 1.05)	0.97 (0.91, 1.03)	–
Chronic widespread pain (%)	8.1	9.5	9.6	12.5	15.0	16.5	1.19 (1.12, 1.25)	1.13 (1.07, 1.19)	–

[a] Regression coefficients per increase in social class for continuous variables and odds ratios per increase in social class for categorical variables, sex adjusted except for fibrinogen where sex specific results are presented;

[b] Includes missing class at 42 years as an additional category.

[c] Geometric means presented but regression uses untransformed values with robust estimators.

[d] Geometric means presented with log transformed regression coefficients. Multiplied by 100 these represent the % change per increase in social class i.e. for HDL a 1.9% decrease per social class.

[e] Means and regression coefficients adjusted for height at 45 years.

(relative to the reference group of 23.5–24.9 and adjusted for confounding factors) was 1.08 (1.00–1.17) and amongst never smokers the relative risk (RR) was 1.21 (1.05–1.41).[27] In comparison with BMI, associations with childhood class were weaker for other outcomes such as blood pressure. The adjusted effect of childhood class on diastolic blood pressure of 0.43 mmHg for each increase in class from I to V was similar to an estimate of 0.37 mmHg (0.06, 0.67) reported for men in the West of Scotland Collaborative study,[6] although our effects for systolic pressure in the 1958 cohort (0.52 mmHg, 95% CI 0.06, 0.67) appear to be weaker than in the Young Finns Study.[28] The adjusted absolute difference in systolic pressure between classes I and V in childhood was 2.6 mmHg in our study. This difference is in excess of the estimated increase of 0.6 mmHg associated with a 1 kg increase in birth-weight,[29] for which in turn, the RR of death from coronary heart disease is estimated to be 0.77 (0.67,0.90) for men and 0.83 (0.62,1.10) for women.[30]

Few studies report on social position in early life and measures of glucose metabolism, hence our finding on a childhood as well as adult association with HbA1c at 45 years contributes to the literature. The effect of childhood class was small, with a difference between class I and V of 0.13%. From a recent study showing that an increase of 1% in Hba1c is associated with a 28% increase in death from all-causes, independent of age, blood pressure, serum cholesterol, BMI and cigarette smoking,[31] a social class difference of 0.13% of HbA1c is equivalent to an increase in mortality of 3.6%.

Our study also suggests that early life factors may affect physical function in mid-adulthood, notably hearing thresholds, and chronic widespread pain. Evidence regarding the role of early influences for these outcomes is currently less abundant than for cardio-respiratory risk and our study therefore adds important new information. For adult hearing, the major environmental factor in recent decades is noise exposure, mostly related to occupation and this may explain at least partly the association seen here with adult social position and stronger associations for men. Socially patterned early life factors, such as low weight in infancy and poor child health (including ear disease) may explain the association with childhood class and there are already reports suggesting that some of these factors are associated with higher hearing thresholds in later life.[32] The plausibility of effects on adult hearing of early life factors related to social position is supported by reports based on the 1958 cohort of social inequalities in hearing thresholds in adolescence.[33,34] Explanations underlying the associations with child and adult class need to be identified. With regard to chronic widespread pain, adversities in early life and associated emotional status as well as physical development could potentially affect health status generally as well as specifically be linked to the reporting

Table 3 Disease risk factors at 45 years related to adult social class, values are sex-adjusted means or prevalence (%)

	Adult social class						Difference (95% CI) per increase in social class grade		SD score
Risk factor	I	II	IIIn	IIIm	IV	V	Adjusted for sex[a]	Adjusted for sex and childhood class[b]	**Adjusted for sex and childhood class[b]**
Systolic pressure (mm/Hg)	124.9	125.6	126.6	128.2	127.4	127.0	0.68 (0.43, 0.93)	0.55 (0.30, 0.81)	0.034 (0.018, 0.049)
Diastolic pressure (mm/Hg)	78.2	78.3	79.1	79.3	79.5	79.1	0.33 (0.16, 0.50)	0.23 (0.06, 0.40)	0.021 (0.005, 0.037)
BMI (kg/m^2)[c]									
Males	27.1	27.7	27.9	28.1	28.3	27.7	0.19 (0.09, 0.28)	0.08 (−0.02, 0.19)	0.016 (−0.004, 0.037)
Females	25.9	26.8	26.6	28.1	27.4	28.2	0.34 (0.21, 0.47)	0.24 (0.11, 0.37)	0.048 (0.022, 0.074)
HbA1c (% total)[d]	5.15	5.19	5.19	5.26	5.28	5.30	0.035 (0.022, 0.047)	0.031 (0.017, 0.044)	0.053 (0.034, 0.072)
Total cholesterol (mmol/l)	5.88	5.91	5.84	5.86	5.87	5.93	−0.007 (−0.026, 0.012)	−0.013 (−0.033, 0.006)	−0.012 (−0.030, 0.006)
HDL cholesterol (mmol/l)[e]									
Males	1.44	1.40	1.36	1.39	1.40	1.39	−0.005 (−0.010, 0.001)	−0.0005 (−0.006, 0.005)	−0.002 (−0.026, 0.022)
Females	1.73	1.69	1.66	1.62	1.58	1.56	−0.021 (−0.027, −0.015)	−0.017 (−0.023, −0.011)	−0.070 (−0.094, −0.045)
Triglycerides[e]									
Males	2.00	2.10	2.04	2.10	2.06	2.21	0.006 (−0.009, 0.020)	−0.003 (−0.018, 0.012)	−0.005 (−0.030, 0.021)
Females	1.20	1.31	1.34	1.38	1.46	1.51	0.038 (0.024, 0.051)	0.029 (0.016, 0.043)	0.049 (0.026, 0.072)
Fibrinogen (g/l)[e]	2.79	2.84	2.87	2.94	2.97	3.05	0.016 (0.013, 0.020)	0.014 (0.010, 0.017)	0.067 (0.049, 0.085)
FEV1 (L)f	3.36	3.32	3.29	3.26	3.25	3.22	−0.040 (−0.048, −0.031)	−0.036 (−0.044, −0.027)	−0.047 (−0.058, −0.036)
Total IgE (kU/l)[e]	33.5	30.7	28.8	30.7	33.7	33.2	0.01 (−0.01, 0.04)	0.01 (−0.01, 0.04)	0.010 (−0.009, 0.028)
Hearing at 4 kHz (dB)[e]									
Males	5.90	6.76	6.78	10.40	9.43	10.68	0.414 (0.326, 0.502)	0.035 (0.026, 0.044)	0.095 (0.070, 0.121)
Females	0.95	2.89	3.45	4.05	4.19	5.74	0.023 (0.015, 0.031)	0.018 (0.010, 0.026)	0.049 (0.027, 0.071)
							Odds ratios (95% CI) per increase in social class		
Visual impairment (%)	1.9	1.6	1.7	2.0	3.5	5.0	1.28 (1.15, 1.43)	1.29 (1.16, 1.45)	–
Depressive symptoms (%)	7.0	6.2	8.4	8.0	9.8	16.6	1.19 (1.12, 1.26)	1.17 (1.10, 1.24)	–
Anxiety symptoms (%)	7.2	6.0	6.7	6.0	7.2	10.0	1.06 (1.00, 1.13)	1.07 (1.00, 1.14)	–
Chronic widespread pain (%)	7.9	9.0	11.8	15.0	15.2	19.3	1.24 (1.18, 1.30)	1.21 (1.15, 1.28)	–

[a] Regression coefficients per increase in social class for continuous variables and odds ratios per increase in social class for categorical variables, sex adjusted except for [c] below;
[b] Includes missing class at 42 years as an additional category.
[c] Significant interaction between gender and adult social class, associations presented separately for men and women.
[d] Geometric means presented but regression uses untransformed values with robust estimators.
[e] Geometric means presented with log transformed regression coefficients. Multiplied by 100 these represent the % change per increase in social class i.e. for males a 0.5% decrease in HDL per social class.
[f] Means and regression coefficients adjusted for height at 45 years.

of pain through somatization in childhood. In turn, the early experience of pain and responses to it may affect the reporting of pain later in life: indeed the strongest predictor amongst adults of the onset of pain is the prior experience of pain.[35] Socially patterned adulthood factors, including smoking and psychological distress, were found to increase risk of low back pain onset in this cohort,[36] and the latter has been found to be important also in the development of chronic widespread pain.[37]

Not all measures were associated with social class in childhood: visual impairment was associated only with adult class, although we acknowledge the short-comings of our measure, which fails to discriminate across a range of visual function. Also for mental health, there was no association with childhood class for anxiety symptoms and it was borderline for adult class; whereas depressive symptoms were associated with both child and adult class. Other studies of mental health tend to show that depressive symptoms are more strongly associated with adult than child class.[24,38]

Nonetheless, childhood SEP predicted multiple disease risk factors and functional measures at age 45 years. A key question therefore concerns when and how childhood influences affect later outcomes, as the processes involved may differ for specific health outcomes. Potential childhood influences on adult health, such as birth-weight, weight gain, height and exposure to parental smoking, are differentiated by social origins in this cohort.[39] Adult influences on health are also socially patterned, hence a further question concerns how early life influences combine with adult exposures. Our study does not address such issues, but suggests that in seeking to understand the development of disease risk and functional status in adulthood, future studies should consider explanations from early life. This is an important task because our markers of disease risk and functional status are predictive of chronic conditions associated with a substantial disease burden. Whilst identification of specific influences underlying the childhood effect requires further elucidation, our findings suggest that policies to redress socio-economic adversity and inequalities in childhood may have the potential to improve disease risk and function in mid-adult life.

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Author contributions

CP conceived the idea for the current article. KA carried out the statistical analyses. CP, DPS, PS, EF, AD, IG, GM, JR, BR, SS were responsible for the design and conduct of the medical examination of the British 1958 Birth Cohort. CP and KA wrote the article and all authors contributed to revising the manuscript. All authors approved the final version. CP is the guarantor who accepts responsibility for the conduct of the study, had access to the data and controlled the decision to publish.

Ethics

Ethical approval for the medical examination of the 1958 British birth cohort was obtained from South East MREC (ref: 01/1/44).

Conflict of interest: None declared.

KEY MESSAGES

- Several studies of child and adult social position suggest that mortality from cardiovascular disease is influenced by social position in early life in addition to that in adult life. Evidence on the role of child and adult social position is sparse for measures other than cardiovascular disease and associated risk factors.
- Childhood social class was associated with cardio-respiratory risk factors and sensory, physical and mental function in mid-life, before clinical and pre-clinical disease has become common. Individuals with the most disadvantaged backgrounds had a poorer health profile across multiple, although not all, measures of disease risk and health function: associations for childhood tended to be as strong as those for adult social position.

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