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Life Course models of socioeconomic position and cardiovascular risk factors: 1946 Birth Cohort

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Abstract

Purpose—To identify the life course model that best describes the association between life course socioeconomic position (SEP) and cardiovascular (CVD) risk factors [body mass index (BMI), systolic and diastolic blood pressure, total cholesterol, low-density lipoprotein, high-density lipoprotein, triglycerides, glycated haemoglobin] and explore BMI across the life course as mediators of the relationship.

Methods—The Medical Research Council (MRC) National Survey of Health and Development was used to compare partial F-tests of simpler nested life course SEP models corresponding to critical period, accumulation, and social mobility modes with a saturated model. Then, the chosen life course model for each CVD risk factor was adjusted for BMI at age 53 and lifetime BMI (ages 4, 26, 43, and 53 years).

Results—Among women, SEP was generally associated with CVD risk factors in a cumulative manner; while childhood critical period was the prominent model for men. Using the best fitting SEP models, adjustment for BMI at age 53 reduced associations for all outcomes in both genders. Further adjustment for lifetime BMI (4, 26, 43 and 53 years) did not substantially alter most associations (except for triglycerides).

Conclusion—SEP at different points across life influences CVD risk factors differently in men and women.

Keywords

UK; social class; life course; social mobility; accumulation; cardiovascular risk

Introduction

Numerous studies have shown that socioeconomic position (SEP) at various points in the life course are associated with cardiovascular disease (CVD) risk, subclinical disease, and

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CVD risk factors [1-4]. The mechanisms by which adverse social circumstances over the life course increase risk of cardiovascular outcomes in later life, however, remain unclear. The possible pathways may be better understood by examining at which stages during the life course SEP most strongly influences CVD risk.

Various life course models for SEP have been proposed. The critical (sensitive) period life course model posits that adverse exposures in a specific time window, have an adverse effect on CVD risk development, with no (or less) excess disease risk outside this developmental window [5, 6]. Many studies have found associations between childhood SEP and adult CVD risk, which are independent of adult SEP, suggesting that childhood may be a critical period [3, 7-12].

The accumulation model proposes that adverse exposures over the life course cause cumulative damage to biological systems. The accumulation model has been widely supported in the literature with greater time spent in adverse SEP translating to worse CVD outcomes [3]. This has usually been tested by summing or averaging SEP from separate time points to create a measure of life course socioeconomic disadvantage. However, the cumulative score may be significant when SEP at only a single time period is independently associated with the outcome [13].

A few studies have suggested that SEP mobility across the life course has an impact on CVD risk factors [3]. The evidence presented to support this hypothesis is a comparison of stable high, stable low, upward mobility, and downwardly mobile groups showing that the CVD risk of the socially mobile groups are intermediate to the two stable groups. However such a pattern of association could also be the result of an accumulation model.

Hence, support for a particular model has been largely dependent on the a priori hypothesis being tested. Only one study, to our knowledge, has attempted to distinguish between these three SEP life course models in relation to a CVD outcome, incidence of first myocardial infarction [2]. This previous study did not explore potential pathways linking SEP with the outcome.

One potential explanation for how social conditions across the life course translate into differences in CVD health in later life is through a body mass index (BMI) pathway. BMI, as well as being a risk factor for CVD [14, 15], is also associated with other CVD risk factors such as blood pressure (BP) [17] and lipid levels [17] and has been found to be related to both adult and childhood SEP [18, 20]. Evidence suggests that BMI (and other risk factors for CVD) collected at the same time as SEP do not appear to fully explain the relationship between SEP and CVD incidence [21, 22]. BMI, however, changes across the life course [23, 24] so it is possible that more of the association between life course SEP and CVD risk factors could be explained by lifetime BMI, rather than a single adult measure of BMI. Only one study of which we are aware has investigated the role of BMI between SEP and other CVD risk factors [25].

Using data from the Medical Research Council (MRC) National Survey of Health and Development (NSHD), this study uses a structured modelling approach to contrast a series of nested life course SEP models with the saturated model to assess which is most consistent with the data. We apply a method, developed by Mishra et al [13] using BMI in the NSHD as an illustrative example, to now consider other CVD risk factors; blood pressure, lipid levels and glycated haemoglobin. We then investigate whether BMI at age 53 or 'lifetime' BMI is a stronger mediator of the association between SEP and the other CVD risk factors.

Methods

The MRC NSHD is a longitudinal study of health based on a social class stratified sample of 5362 singleton UK births during one week in March 1946. There have been 21 follow-ups of the entire cohort between birth and age 53 years, with the cohort still relatively representative of the British population of the same age [26]. For the most recent completed data collection when the cohort were 53 years old, 3035 respondents (1472 men, 1563 women) were interviewed and examined in their homes by trained research nurses (98%) or completed a postal questionnaire (2%).

Socioeconomic position

SEP at three time points, represented by occupational social class, were chosen in these analyses to represent different stages of the life course: childhood SEP (S1) [father's occupation when cohort member was age four], young adult SEP (S2) [cohort member's own occupation at 26 years], and later adult SEP (S3) [own occupation at 43 years]. If SEP was missing then the SEP at the closest available age was imputed (n=125 at age 4 from age 7, n=239 age 26 from 36 years, and n=241 age 43 years from age 53 years). Results with and without imputation were similar. Those who had never been in paid work were excluded from the analysis (n=208; 126 women and 65 men). Occupation was based on the Registrar General's six group classification [27]. At each time point, binary indicators of SEP were used in analysis: non-manual (I and II, III non-manual) = 0 and manual (III manual, IV and V) = 1. Of the 4631 with information on SEP at age 4 years, 125 were missing data on young adult SEP, 853 on later adult SEP, and 682 at both years, leaving a total sample for inclusion in analyses of 2971 (1526 men, 1445 women). Those with manual childhood SEP were more likely to be lost to follow-up but differences were small (37% manual compared to 34% of non-manual were missing young adult and/or later adult SEP).

Cardiovascular risk factors

During the home visits when participants were 53 years of age, height and weight were measured according to standardised protocols. BMI was defined as weight (kg)/height (m)². Seven additional CVD risk factors were chosen which have been shown to be related to SEP assessed at any period in the life course [3] and BMI [14, 15]: triglycerides (TRIG), glycated haemoglobin (HbA1c), systolic and diastolic blood pressure (SBP/DBP), total cholesterol (CHOL), and low-density and high-density lipoprotein cholesterol (LDL/HDL-C). During the same home visit, blood pressure and non-fasting venous blood samples taken according to standardised protocols, with details of the assessment of CHOL, HDL-C, TRIG, and HbA1c reported elsewhere [28]. LDL was calculated using the Friedewald formula: [CHOL - HDL-C - (0.45*TRIG)]. The second of two blood pressure readings was used in this analysis, taken after 5 minutes of rest with the participant sitting using the validated Omron HEM-705 (Omron Corp., Tokyo, Japan) automated digital oscillometric sphygmomanometer [26].

Covariates

Additionally, BMI recorded at the same ages as the SEP indicators used in analyses (4, 26 and 43 years) were used to represent 'lifetime' BMI. This was calculated from measured height and weight at ages 4 and 43 years and from self-reports at age 26 years. Cohort members also reported whether they were taking medications. This information was coded according to the British National Formulary (BNF) Number 40 (2000). Any cohort member taking any drug from the BNF section 2.12 and 2.25 were considered to be on lipid-lowering (n=75) and anti-hypertensive drugs (n=402), respectively.

Statistical Analysis

This analysis comprises two parts: (1) selection of the most appropriate life course model for each CVD risk factor and (2) investigation of current BMI and 'lifetime' BMI as mediators in the relationship between SEP and the other CVD risk factors.

For section one, a structured modelling approach developed by Mishra et al [13] was used to examine three different hypothesized life course SEP models in relation to each CVD risk factor. The basic premise of the approach is that, given three binary SEP variables, a saturated model would allow all 8 possible SEP trajectories to have a different mean outcome. The saturated model for each outcome was modelled with 3 main effects, all 2 way interactions, and the 3 way interaction, where α is the expected mean for the CVD risk factor outcome for the trajectory where persons were non-manual at all three time points.

It has been demonstrated that by comparing the model fit of a set of nested models – corresponding to the accumulation, critical period, and social mobility models – to the saturated model, the nested model that best describes the data can be selected. Here we consider two examples of an accumulation hypothesis: (1) A strict version where it is assumed that, regardless of the time period, the longer a person spends in a manual SEP, the worse the CVD risk and (2) a relaxed model where all time periods are contributing to risk but not necessarily in an equal manner. The critical period hypothesis assumes that SEP influences CVD risk only in a certain time period, irrespective of any other time periods. For purposes of presentation we refer to this as the 'critical period', even though in some cases a sensitive period model may be more realistic. We also consider two examples of social mobility: intra-generational mobility and a general mobility. The intra-generational mobility model hypothesises that downwards mobility between S2 and S3 would be harmful to health, and upwards mobility would be beneficial, irrespective of S1, such that those who remain in a manual social class at both S2 and S3 have equal expected means to those who remain in a non-manual social class at both time points. The general mobility model is where all downward and all upward changes are hypothesized to be equally harmful or beneficial. Each life course model specification and constraints are detailed in Appendix 1.

For each risk factor, F-tests were conducted comparing each life course model to the saturated model. Higher p-values represent a better model fit since this indicates that the simpler nested model fit the data as well as the saturated model. When more than one life course model had a p-value greater than 0.05, we selected the model with the largest p-value where there was a clear difference in p-values. Where two models had similar p-values, we chose the simpler model. We also compared the saturated model with the null model (i.e. $E(Y) = \alpha$). In situations where the null model, and all SEP models, produced a non-significant p-value when compared with the saturated model, it was concluded that SEP was not associated with that CVD risk factor and we did not proceed with further analysis. In addition, a sensitivity analyses was conducted substituting SEP at age 53 years in lieu of SEP at 43 years in order to assess whether results were dependent on the age at which the midlife measure of SEP was recorded.

Because of potential differences in associations of SEP with the CVD risk factors between men and women, all analyses are stratified by gender. A constant of 10 and 5 mm/Hg to SBP and DBP [29], respectively, and 1.24 mmol per liter (35%) to LDL [30] was added to those taking specified lipid and blood pressure medications. A log transformation was applied to TRIG and HbA1c to achieve normality.

In the second part of the analyses, each life course SEP model selected in the first part (except those for BMI) was first adjusted for BMI at age 53 and then additionally for

lifetime BMI (ages 4, 26 and 43). We then examined whether lifetime BMI explained more of the SEP-risk factor association than BMI at 53 years.

Results

A larger percentage of women than men were in non-manual SEP groups at all three time points [32.6% vs 29.4%], while more men than women experienced a manual SEP at all time points [27.2% vs 13.0%]. Men and women had similar mean values for BMI, HbA1c, CHOL, and LDL; while men had higher TRIG, SBP and DBP and women higher HDL-C. There was heterogeneity of CVD risk factor means by the 8 SEP trajectories with a test for heterogeneity being highly significant for all outcomes except CHOL and LDL [table 1].

For women, the strict accumulation model was the best fitting model for TRIG, SBP, DBP, and HDL-C (p-values: 0.941, 0.658, 0.188, and 0.123). While both the strict and relaxed accumulation models explained the data as well as the saturated model, both p-values were relatively low (strict: 0.063, relaxed: 0.292). For simplicity and consistency with the other outcomes, we have chosen the strict accumulation model. For men, the childhood critical period model was the best fitting for BMI and DBP. The relaxed accumulation model had the highest p-value for TRIG and SBP but the p-value for the simpler childhood critical period model was almost as large. Further investigation of the 2 models indicated that the relaxed accumulation model did not fit the data significantly better than the critical period model, and thus the simpler model was selected. For both genders, the relaxed accumulation model had the highest p-value for HbA1c (women 0.537, men 0.814) with the later adult critical period model fitting the data almost as well. Again the more complex model showed no significant improvement in fit and thus the later adult critical period model was chosen. CHOL and LDL in both men and women and HDL-C in men produced non-significant p-values and non-significant estimates for all models, suggesting that SEP has no effect on these outcomes. Therefore, no further analyses were conducted on these outcomes. Sensitivity analyses substituting SEP at age 53 years in lieu of 43 years altered individual p-values but did not change basic conclusions in relation to model selection [table 2].

In women, accumulation of manual SEP across the life course was significantly associated with increasing BMI, SBP, DBP and TRIG and decreasing HDL-C. For example, 1-unit increase in SEP score (number of time points 0-3 in manual social class) was associated with an increase in BMI at age 53 of 0.91 (95% confidence intervals [CI]: 0.66, 1.61) [Table 3]. In men, manual SEP in childhood was associated with a significant increase in mean BMI, SBP, DBP and TRIG. Mean SBP was 6.02 mm/Hg (95% CI: 3.38, 8.66) higher in those from a manual SEP in childhood compared with those from a non-manual SEP; similarly the difference was 3.31 mm/Hg (1.74, 4.87) for DBP. ²Manual SEP in later life was associated with a 2.95% (1.45, 4.44) and 2.87% (1.44, 4.30) increase in HbA1c, respectively, for men and women.

Adjustment for BMI at age 53 reduced associations for all outcomes in both genders [Table 3], with generally a larger percentage drop in women than men. For example, in women a 1-unit increase in accumulated manual SEP was associated with an increase in SBP of 1.59 mm/Hg (0.31, 2.86) after adjusting for BMI at age 53, which was a reduction of 32% (21 to 70) in comparison to the crude model. In contrast, men only experienced a drop of 22% (16 to 36). In contrast, adjustment for BMI at age 53 years reduced estimates for TRIG more in men than women [% change in estimate from crude model: childhood critical period men: 49% (31 to 139), accumulation 1-unit women: 34% (26 to 53)] but crude estimates were also higher in men. Additional adjustment for lifetime BMI (at ages 4, 26 and 43 years) did not substantially reduce estimates

Discussion

Using a new structured approach to distinguish between theoretical life course models of SEP, we found gender differences in the way life course SEP is associated with cardiovascular risk factors. However, within genders results were generally consistent that SEP effects accumulated across the life course in women but were more influential during childhood for men. And while adjustment for BMI at age 53 years reduced the associations between SEP and blood pressure, lipids and glycated haemoglobin, there was no additional impact of adjustment for 'lifetime' BMI.

Our findings are consistent with previous studies that have shown that childhood and adult SEP in women are associated with later life obesity [13, 28, 32-38]. The literature on whether childhood social conditions influence adult obesity in men is less consistent, with only 3 out of 14 studies other than NSHD [33, 35, 37-38] showing childhood SEP effects in men after adjustment for age and adult SEP. However, the likelihood of finding an association in men increased when prospectively collected SEP was utilized, as in our study, suggesting null childhood SEP findings may be a result of recall bias [38].

Similar to findings for BMI, the current study also showed that SBP, DBP, and triglycerides in women were affected by SEP across the life course while for men SEP in childhood was the most pertinent exposure period. Gender-stratified examinations of childhood and adult SEP effects on blood pressures and triglycerides are rare [39] with inconsistent results of SEP effects in men-only [3, 40, 41], women-only [32, 42], and pooled gender [43-45] studies. Almost all previous studies only assessed a single life course model. Most considered a relaxed accumulation model with one childhood and one adult time period [3, 32, 39, 40, 42, 44-45] while one study tested a strict accumulation model [43]. Our study improves upon these methods by the addition of SEP exposure at an additional period in the life course and the simultaneous assessment of multiple life course models.

For HbA1c, we found that SEP in time closest to measurement (here being later adulthood) might be more important than other time periods. One explanation is that compared to other CVD risk factors, blood glucose levels do not track so well over the long term [46], reflected in the current test for HbA1c only measuring average blood glucose over the previous 3-11 weeks [47]. Lastly, we did not find an association between SEP at any point in the life course and total cholesterol or LDL. This is consistent with findings from the Northern Swedish Cohort [48], but in contrast to other studies using the accumulation [32] and childhood/adulthood [35] life course models. This could be because, unlike the latter two studies mentioned, we adjusted for cholesterol-lowering medications, which if not accounted for could lead to those in a higher SEP appearing to have lower cholesterol levels because they are more likely to take medications. When analyses were re-fitted with those on medication excluded, p-values for F-tests did not explain the data as well (lower values) but conclusions did not change.

Why SEP effects accumulate over the life course in women but only the effect of childhood SEP is important for men remains unclear. One explanation is that women may be more susceptible than men to BMI change due to events over the life course, such as adolescence [49], pregnancy [50], and menopause [51] and these BMI changes may be socially patterned. Men have been found to be more susceptible to BMI increase only in the period after marriage [52]. There are also clear associations in the NSHD cohort between lower SEP in adulthood and lower fruit and vegetable consumption for women but not men, with low consumption of fruit and vegetables being associated to with higher BMI [58]. Hence, greater SEP differences in diet among women than men may lead to adult SEP being more important in women. BMI throughout the life course is positively associated with blood

pressure [37, 53-54], and lipid and glycemic profiles [55-57] supporting the role of BMI as a major link between low SEP and poor cardiovascular health; thus explaining the consistency of life course SEP patterns on the risk factors by gender. There is also greater tracking of SEP over the life course in men than women, which may be why childhood SEP is more important than adult SEP in men.

Adjusting for BMI over multiple periods in the life course did not reduce associations between SEP and most CVD risk factors more than adjustments for BMI at 53 years only. Strong tracking of BMI throughout adulthood in NSHD [17] and the small numbers of persons in the general population once overweight or obese whom drop weight categories [59], corroborate evidence that BMI in later life is a good proxy for lifetime BMI.

The application of this novel structured approach to modelling is a strength of the current study, allowing us to examine heterogeneity of life course models by gender and cardiovascular risk factor. However, situations arose where two or more models explained the data as well as the saturated model. Where the p-values, although both non-significant, were clearly very different in size, we chose the model with the best fit. In cases where two models produced similar p-values we investigated the two models in more detail. Unless, there was clear evidence that the more complex model of the two was a better fit than the more parsimonious one, we selected the simpler model. Small numbers of socially mobile individuals also limited our ability to test mobility models. Despite these issues surrounding model selection, our approach is preferable to simply interpreting the results from a single pre-specified model (such as accumulation) without considering the relative merits of alternative models.

Multiple testing may have increased our chances of rejecting a model which fits the data well, but the clear SEP patterns within genders across CVD risk factors suggests that this is unlikely to be the case. Six categories of occupational social class are available in NSHD, but the models become exponentially complex with each increase in the number of categories used and a lack of observations in some SEP trajectories reduce power substantially. Associations of SEP in six categories with the CVD risk factors did however tend to cluster into the manual and non-manual groupings (data not shown).

Using a novel structured approach to modelling, our study finds that SEP at different points across life influences CVD risk factors differently in men and women. This study supports gender-specific interventions, particularly early in life and potentially through BMI pathways, to improve cardiovascular risk profiles in later life.

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Appendix

Appendix 1 Model specification and constraints for given life course models

	Life-course model specification	Constraints
Saturated model	$\alpha + \beta_1 S_1 + \beta_2 S_2 + \beta_3 S_3 + \theta_{12} S_1 S_2 + \theta_{13} S_1 S_3 + \theta_{23} S_2 S_3 + \theta_{123} S_1 S_2 S_3$	
No effect	α	

	Life-course model specification	Constraints
Accumulation, strict	$\alpha + \beta_1(S_1 + S_2 + S_3)$	
Accumulation, relaxed	$\alpha + \beta_1 S_1 + \beta_2 S_2 + \beta_3 S_3$	
Critical Period		
Childhood	$\alpha + \beta_1 S_1$	
Early adult	$\alpha + \beta_2 S_2$	
Later adult	$\alpha + \beta_3 S_3$	
Adult social mobility	$\alpha + \beta_2 S_2 + \beta_3 S_3 + \theta_{23} S_2 S_3$	$\theta_{23} = -(\beta_2 + \beta_3)$
Any social mobility	$\alpha + \beta_1 S_1 + \beta_2 S_2 + \beta_3 S_3 + \theta_{12} S_1 S_2 + \theta_{23} S_2 S_3$	$\beta_2 = (\beta_{1+} \beta_3)$, & $\theta_{12} = -\beta_2$ $\theta_{23} = -\beta_2$

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Table 1

Cardiovascular Risk Factors by Socioeconomic Position (SEP) Permutation and Gender. Data are Expressed by Means (Standard Errors), National Survey of Health and Development, United Kingdom (1946-1999).

Gender	N (%)	SEP [‡]			Mean BML, kg/m ²	Geometric Mean TRIG	Geometric Mean HbA1c	Mean SBP [†]	Mean DBP [†]	Mean CHOL [†]	Mean LDL [†]	Mean HDL-C
		SEP1	SEP2	SEP3								
Women												
	414 (32.6)	0	0	0	(n=1270)	(n=1085)	(n=1089)	(n=1243)	(n=1243)	(n=1085)	(n=1047)	(n=1050)
					26.0 (4.4)	2.2 (3.2)	51.5 (1.2)	130.8 (19.8)	81.0 (11.7)	6.0 (1.1)	3.4 (1.0)	1.9 (0.5)
	358 (28.2)	1	0	0	27.6 (5.4)	2.5 (3.3)	52.1 (1.2)	133.7 (20.7)	82.6 (11.1)	6.1 (1.1)	3.5 (0.9)	1.8 (0.4)
	47 (3.7)	0	1	0	26.9 (5.1)	2.7 (3.2)	52.4 (1.2)	134.1 (20.5)	80.7 (10.7)	6.2 (1.0)	3.5 (1.0)	2.0 (0.5)
	51 (4.0)	0	0	1	28.3 (5.4)	2.3 (3.5)	55.1 (1.4)	137.8 (18.1)	85.4 (12.7)	6.3 (1.1)	3.7 (1.1)	1.9 (0.5)
	101 (8.0)	1	1	0	28.3 (6.2)	2.8 (2.9)	52.8 (1.2)	135.8 (21.3)	83.6 (12.7)	6.1 (1.1)	3.7 (0.9)	1.6 (0.4)
	104 (8.2)	1	0	1	29.1 (6.2)	3.4 (3.7)	53.9 (1.3)	135.0 (20.8)	81.7 (11.1)	6.1 (1.1)	3.4 (0.9)	1.8 (0.5)
	30 (2.4)	0	1	1	27.2 (4.9)	3.6 (3.2)	54.7 (1.2)	141.3 (23.6)	87.2 (19.2)	6.5 (1.6)	3.8 (1.5)	1.8 (0.4)
	165 (13.0)	1	1	1	28.6 (6.3)	3.7 (3.7)	58.1 (1.4)	137.7 (23.0)	84.3 (12.5)	6.0 (1.2)	3.4 (1.0)	1.7 (0.5)
	1270	TOTAL			27.4 (5.5)	2.6 (3.3)	53.0 (1.3)	133.9 (20.9)	82.5 (12.0)	6.1 (1.1)	3.5 (1.0)	1.8 (0.5)
Men												
					(n=1281)	(n=1122)	(n=1135)	(n=1271)	(n=1271)	(n=1116)	(n=1006)	(n=1013)
	377 (29.4)	0	0	0	26.7 (3.8)	4.2 (3.5)	51.4 (1.3)	137.5 (20.3)	86.2 (12.3)	6.0 (1.2)	3.5 (1.0)	1.5 (0.4)
	228 (17.8)	1	0	0	27.9 (3.9)	5.9 (3.6)	52.6 (1.2)	142.4 (22.1)	88.6 (13.1)	6.0 (1.0)	3.6 (1.0)	1.5 (0.6)
	53 (4.1)	0	1	0	27.4 (3.7)	5.1 (3.3)	53.3 (1.2)	137.2 (19.1)	86.2 (10.0)	6.2 (0.9)	3.7 (0.8)	1.4 (0.3)
	26 (2.0)	0	0	1	27.0 (3.9)	4.1 (2.7)	57.1 (1.4)	140.2 (17.8)	88.8 (12.7)	5.8 (1.1)	3.5 (1.1)	1.4 (0.3)
	143 (11.2)	1	1	0	27.8 (3.8)	5.9 (4.4)	53.2 (1.2)	144.6 (20.3)	89.4 (11.4)	5.9 (1.1)	3.5 (1.0)	1.6 (0.8)
	35 (2.7)	1	0	1	26.3 (3.5)	3.8 (4.5)	54.5 (1.4)	142.1 (22.4)	88.5 (12.7)	5.6 (1.1)	3.3 (1.1)	1.6 (0.5)
	71 (5.5)	0	1	1	27.2 (4.7)	4.6 (4.3)	55.5 (1.3)	141.2 (19.9)	84.9 (10.9)	5.9 (1.3)	3.5 (1.1)	1.4 (0.4)
	348 (27.2)	1	1	1	27.9 (4.2)	5.8 (4.2)	55.6 (1.3)	145.0 (20.1)	89.9 (12.9)	6.0 (1.1)	3.5 (1.0)	1.5 (0.6)
	1281	TOTAL			27.4 (4.0)	5.1 (3.9)	53.5 (1.3)	141.6 (20.7)	88.0 (12.5)	6.0 (1.1)	3.5 (1.0)	1.5 (0.5)

[‡] 1 refers to manual SEP, 0 refers to non-manual SEP: SEP1=father's social class age 4; SEP2=own social class age 26; SEP3=own social class age 43.

[†] Adjusted for medication use: SBP and DBP hypertensive medications; CHOL & LDL lipid-lowering medications.

Abbreviations: BMI, Body mass index kg; TRIG, triglycerides; HBA, glycated haemoglobin; SBP, systolic blood pressure; DBP, diastolic blood pressure; CHOL, total cholesterol; LDL, low-density lipoprotein; HDL-C, high-density lipoprotein.

P-values for F-tests Comparing Given Life-Course SEP models Ability to Explain Observed Data as Well as the Saturated Model for CVD Risk Factors, by Gender, National Survey of Health and Development, United Kingdom (1946-1999).

Table 2

	BMI, kg/m ²	TRIG, mmol/L	HBA, %	SBP, mmHg [†]	DBP, mmHg [†]	CHOL, mmol/L [†]	LDL, mmol/L [†]	HDL-C, mmol/L
Women								
No effect	<0.001	0.001	<0.001	0.016	0.025	0.156	0.034	<0.001
Accumulation, strict	0.063	0.941	0.327	0.658	0.188	0.174	0.048	0.123
Accumulation, relaxed	0.292	0.839	0.537	0.465	0.078	0.062	0.018	0.096
Critical Period								
Childhood (4 yrs)	0.006	0.023	<0.001	0.045	0.035	0.143	0.044	0.016
Early adult (26 yrs)	<0.001	0.038	0.013	0.181	0.083	0.126	0.038	0.002
Later adult (43 years)	<0.001	0.153	0.339	0.245	0.084	0.139	0.021	<0.001
Adult social mobility	<0.001	<0.001	<0.001	0.015	0.008	0.162	0.078	<0.001
Any social mobility	<0.001	<0.001	<0.001	0.018	0.008	0.480	0.084	<0.001
Men								
No effect	0.001	0.030	0.002	0.001	0.004	0.380	0.730	0.570
Accumulation, strict	0.056	0.239	0.703	0.579	0.121	0.279	0.633	0.465
Accumulation, relaxed	0.283	0.670	0.814	0.884	0.438	0.186	0.450	0.395
Critical Period								
Childhood (4 yrs)	0.317	0.606	0.011	0.639	0.673	0.279	0.689	0.496
Early adult (26 yrs)	0.009	0.122	0.151	0.030	0.008	0.290	0.623	0.493
Later adult (43 years)	0.001	0.023	0.770	0.009	0.007	0.311	0.647	0.531
Adult social mobility	0.001	0.030	0.001	<0.001	0.001	0.642	0.684	0.338
Any mobility	0.002	0.051	0.002	<0.001	0.006	0.252	0.491	0.780

[†] Adjusted for medication use: SBP and DBP hypertensive medications; CHOL & LDL lipid-lowering medications.

Bolded: No significant difference of the nested life course model to the saturated model; higher p-value = better model fit.

Abbreviations: BMI, Body mass index kg; TRIG, triglycerides; HBA, glycated haemoglobin; SBP, systolic blood pressure; DBP, diastolic blood pressure; CHOL, total cholesterol; LDL, low-density lipoprotein; HDL-C, high-density lipoprotein.

Table 3

Adjusted Mean differences (95% CI) in Chosen CVD Risk Factors for Strongest Fitting Life Course Model, National Survey of Health and Development, United Kingdom (1946-1999).

	Women					Men						
	Life course model chosen	SEP variable	N	Adjustment	Estimate	% change in estimate compared to crude (95% CI)	Life course model chosen	SEP variable	N	Adjustment	Estimate	% change estimate compared crude (95% CI)
Mean BMI, kg/m ²	Accumulation	S1+S2+S3	979	Crude	0.91 (0.66, 1.16)	-	Childhood	S1	953	Crude	1.46 (1.14, 1.77)	-
				-	-	-	critical	-		-		
				-	-	-	period	-		-		
Mean SBP, mm Hg	accumulation	S1+S2+S3	958	Crude	2.32 (1.04, 3.61)	-	Childhood	S1	945	Crude	6.02 (3.38, 8.66)	-
				BMI 53	1.59 (0.31, 2.86)	32 (21 to 70)	critical	BMI 53		4.71 (2.15, 7.28)	22 (16 to 36)	
				Lifetime BMI [†]	1.63 (0.34, 2.92)	30 (19 to 68)	period	Lifetime BMI [†]		4.60 (2.02, 7.18)	24 (17 to 40)	
Mean DBP, mm Hg	accumulation	S1+S2+S3	958	Crude	0.83 (0.10, 1.55)	-	Childhood	S1	945	Crude	3.31 (1.74, 4.87)	-
				BMI 53	0.36 (-0.36, 1.08)	56 (31 to 460)	critical	BMI 53		2.47 (0.96, 3.97)	25 (18 to 45)	
				Lifetime BMI [†]	0.36 (-0.36, 1.08)	57 (30 to 469)	period	Lifetime BMI [†]		2.60 (1.08, 4.11)	21 (16 to 38)	
Mean HDL-C, mmol/L	accumulation	S1+S2+S3	808	Crude	-0.07 (-0.10, -0.04)	-	none	-	-	-	-	-
				BMI 53	-0.05 (-0.08, -0.02)	31 (23 to 53)	-	-		-	-	
				Lifetime BMI [†]	-0.05 (-0.08, -0.02)	31 (22 to 53)	-	-		-	-	
TRIG, %	accumulation	S1+S2+S3	840	Crude	8.90 (5.35, 12.45)	-	Childhood	S1	835	Crude	11.57 (3.73, 19.40)	-
				BMI 53	5.86 (2.49, 9.23)	34 (26 to 53)	critical	BMI 53		5.93 (-1.46, 13.32)	49 (31 to 139)	
				Lifetime BMI [†]	6.62 (3.21, 10.02)	26 (20 to 40)	period	Lifetime BMI [†]		7.27 (-1.19, 14.65)	37 (24 to 103)	
HBA, %	Later adult critical period	S3	839	Crude	2.95 (1.45, 4.44)	-	Later adult	S3	843	Crude	2.81 (1.40, 4.22)	-
				BMI 53	2.37 (0.90, 3.84)	20 (13 to 38)	critical	BMI 53		2.50 (1.09, 3.91)	5 (4 to 10)	
				Lifetime BMI [†]	2.35 (0.75, 3.73)	24 (16 to 48)	period	Lifetime BMI [†]		2.38 (0.96, 3.80)	15 (10 to 31)	

⁷ Lifetime BMI: ages 4, 26, 43, and 53 years.

Abbreviations: BMI, Body mass index kg; TRIG, triglycerides; HBA, glycated haemoglobin; SBP, systolic blood pressure; DBP, diastolic blood pressure; CHOL, total cholesterol; LDL, low-density lipoprotein; HDL-C, high-density lipoprotein.