Lifelong Socio Economic Position and biomarkers of later life health: A formal comparison of the critical period, accumulation and chains of risk hypotheses.

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Abstract

The relative contribution of early and mid or later life socio-economic position (SEP) to later life health is not fully understood and there are alternative hypotheses about the importance of direct versus indirect pathways. We use data from the English Longitudinal Study of Ageing to address this issue and to investigate alternative hypotheses about life course influences on biomarkers of later life health. We found that the effect of early life SEP reaches until the beginning of late old age, predicting physical health and fibrinogen levels at least 65 years later. However, a more complicated pattern of associations than what was implied by previous findings was observed. Cohort specific effects emerged, with current SEP dominating the effect on later life physical health and fibrinogen levels in participants under 65, while early life SEP had a more prominent role in explaining later life inequalities in physical health for men and women over 75. We extend previous findings on mid adulthood and early old age to old age and the beginnings of late old age. The complexity of our findings highlights the need for further research on the mechanism that underlies the association between SEP and later life health.

Introduction

It is well recognised that differences in health and in mortality by indicators of Socio-Economic Position (SEP) persist at older ages, although relative differentials appear lower than younger age groups [1-5]. The observations that older people account for the majority of those in poor health and that the economic costs of socioeconomic inequalities in health are in the order of €1000 billion, or 9.4% of European GDP [6] suggest that there is great potential for shifting the overall distribution of risk and improving average population health [7] by eliminating or reducing the socioeconomic health gradient [8, 9]. Plenty of evidence suggests that the mechanism through which SEP influences health acts over the life course and several explanations have been offered to account for its effect. These include early life/critical period effects, the chains of risks/pathways, accumulation of risk and social drift hypotheses [10-15]. It has been suggested that the social environment children experience may put them at risk of exposures to known and unknown factors during childhood and adolescence and have causal direct effects on health in adulthood [10, 11]. It has also been shown that those from more advantaged family backgrounds have a much better chance of achieving a high socioeconomic position in adult life and adult SEP may then in turn affect disease risk by determining exposure to causal factors in adult life, whereas poor health in childhood may lead to downward social mobility as well as poor health in adulthood [12-14].

However, the relative contribution of the proposed explanations which is of great importance in the design of successful policy interventions aiming to reduce inequalities has not been thoroughly assessed. The challenge is in assigning appropriate parameters that represent each hypothesis/pathway [16], an endeavour that requires suitable tools, beyond standard analytical methods. With some exceptions [17-19] the vast majority of the evidence concerning life course processes comes from studies that have relied on standard regression models [20, 21]. In such simplified statistical models, only limited conclusions can be drawn with respect to the life course pathways between which we would like to discriminate in order to understand the mechanism that underlies socio economic disparities in later life health. In the present study we attempt to unify the mechanistic view of causality implied by life-course theory with a formal approach for the identification of mediating factors in order to meaningfully compare the relative contribution of four explanations of the effect of SEP over the life course on later life health.

Methods

Sample

We used data from the fourth wave (2009) and the life course interview (2007) of the English Longitudinal Study of Ageing (ELSA), a nationally representative multi-purpose sample of the population aged 50 and over living in England. The ELSA sample was drawn from households that responded to the 1998, 1999 or 2001 rounds of the Health Survey for England (HSE), a stratified random sample of all households in England. Response rates to these HSE rounds were 69%, 70% and 67% respectively [22]. A comparison of the socio-demographic characteristics of this sample with national census data indicated that the ELSA sample remained representative of the non institutionalised population [22]. Our analytic sample included participants with at least one valid observation in the early life SEP indicators measured in the ELSA life-course interview (N = 7758). Considering that unbiased estimates of pathways cannot be obtained without properly addressing the implications of incompleteness we employed the Full Information Maximum Likelihood method which is naturally incorporated into structural equation models. In this full likelihood context model

parameters and standard errors are estimated directly from the available data and the selection mechanism is ignorable under the Missing at Random (MAR) assumption [23, 24].

Measures

Recollection of early life SEP and health – ELSA Life history interview

We combined several indicators of participants' recall of early life SEP (at age 10) and early life health (childhood and early adolescence) in latent summaries that capture the common variance between these indicators. Combining several indicators in latent variables controls for random error which may be present in the recalled indicators of early life SEP and health since participants' childhood was at least four decades in the past. We have also controlled for current health status (presence of chronic illness and depression at ELSA Wave 3) as drivers of early life SEP and early life health recall in a Multiple Indicators Multiple Causes (MIMIC) model in order to capture the influence of systematic recall bias, since it is plausible that current health status may bias the recall of early life circumstances. High scores in the latent summaries represent high SEP and optimal health (see Appendix I for frequency distribution of all early life indicators).

Later life measures – ELSA Wave 4.

We employed a latent summary of later life SEP based on net individual income and total net wealth using the approach described in Ploubidis, DeStavola & Grundy [1], with high scores representing high SEP. As a measure of physical health we used a latent summary of six indicators using the procedure proposed by Ploubidis & Grundy [25] in surveys where both self and observer measured health indicators are available. Three observer measured (grip strength; a measure of respiratory function -Forced Vital Capacity – FVC; and chair rise speed) and three self reported health indicators (self rated health, presence of long standing illness, and the presence of one or more functional limitations) were combined in the latent health dimension, with high scores representing optimal physical health (see Appendix I for frequency distribution of all later life physical health indicators). Our second health outcome was fibrinogen which has been linked to is an approximate doubling in risk of major cardiovascular disease outcomes (such as coronary heart disease and stroke) and of aggregate nonvascular mortality (mainly comprising cancer deaths).

INSERT TABLE 1 ABOUT HERE

Confounders

Age, gender, marital status, retirement status, number of children, cognitive ability and ethnicity were included as they are thought to be important confounders of the association between later life SEP and later life health.

INSERT GRAPH 1 ABOUT HERE

Statistical Modelling

In order to formally compare the relative contribution of the four hypotheses to later life health inequalities over the life course, direct and indirect effects and their standard errors need to be appropriately quantified. In the causal mediation literature several approaches have been proposed for the estimation of direct and indirect effects with an emphasis on different aspects of mediation [26, 27]. These include marginal structural models, structural

nested models, principal stratification and g-computation. What these methods have in common is that under certain assumptions they are concerned with the identification of the direct effect of an exposure to the outcome[28], or can only identify a single indirect effect [29] and therefore are not appropriate when multiple indirect effects are of substantive interest. In the present paper two of the four hypotheses we wish to compare (chains of risk and social drift) involve the estimation of indirect effects. We have therefore opted for the Linear Structural Equation Modelling (LSEM) approach, which is best suited to answer the research question at hand. LSEM estimates have been shown to be valid under the sequential ignorability assumption and linearity of the models for the outcomes and mediators [30]. Preliminary analysis showed weak or no evidence for interactions with respect to both health outcomes. Taking this into account the parameterisation of the four hypotheses is shown in Graph 1. All models were estimated with the robust maximum likelihood (MLR) estimator in Mplus 6.1 [18] and all reported model parameters are standardized so that their relative sizes can be compared.

INSERT TABLE 2 ABOUT HERE

Results

Recollection of early life SEP and health

In Graph 2 we present the standardised factor loadings of all early life SEP indicators, that can be interpreted as correlations between the indicators and the latent variable. All loadings were satisfactory > |0.4| (see Graph 2), but none exceeded |0.662|, indicating the presence of random error in all early life SEP indicators that was excluded from valid early life SEP variance which is represented by the latent variable. With respect to systematic error in the form of recall bias due to current at the time of recall chronic illness and depression, the presence of one or more chronic illnesses was negatively associated with early life SEP, b = -0.076 (-0.107 - -0.044) as was depression b = -0.101 (-0.134 - -0.069). In Graph 3 we present the standardised factor loadings of all early life health indicators. All standardised loadings were satisfactory > |0.74|, with the exception of self rated health during childhood, which was the only early life health item affected by substantive random error. The presence of one or more chronic illnesses was negatively associated with recollections of early life health, b = -0.129 (-0.160 - -0.098), as was depression b = -0.049 (-0.079 - -0.019).

INSERT TABLE 3 ABOUT HERE

Predictive models

Table 1 shows descriptive statistics of the exogenous variable, mediators and outcomes in the model. Women had higher early life SEP, but similar early life health with men. As excepted they also had worse later life physical health and higher fibrinogen levels compared to men. Cohort differences were observed in both genders. Younger cohorts had higher early life SEP and better early life health. Similarly they had higher later life SEP, better physical health and had lower fibrinogen levels. Preliminary analysis showed that all mean differences were significant.

Tables 2 and 3 and Figures 1 & 2 show the standardised parameters and corresponding 95% confidence intervals In men, early life SEP had a strong positive association with later life SEP, with the association becoming increasingly stronger in the older age groups. Early

life health was not associated with later life SEP in any group. We found evidence of a positive association between early life health and later life physical health in all age groups. Similarly, later life SEP was positively associated with later life physical health in all age groups, with the effect being considerably weaker in the 75+ age group. The accumulation hypothesis had the strongest contribution to later life physical health inequalities in all age groups. Later life SEP dominated the effect in participants up to 74 years old, but early life SEP in men over 75. The total effect of early life SEP was significant in all age groups and had the second strongest contribution to later life physical health inequalities. In men under 75, the effect was mostly due to chains of risk, but for men over 75 most of the effect was due to early life SEP since there was a direct significant effect of early life SEP on later life physical health in the 75+ group. We did not observe any contribution of the social drift hypothesis to physical health inequalities over the life course. With respect to fibrinogen, there was no association between early life SEP and later life fibrinogen in any age group, neither was any direct association between early life health and later life fibrinogen in those over 65. We observed a negative association between early life health and fibrinogen levels in the younger cohort (50-64). We also observed a negative association between later life SEP and later life fibrinogen in those under 65 and over 75, but not in the 65-74 age group.. The accumulation hypothesis dominated by later life SEP had the strongest contribution to later life inequalities in fibrinogen levels while none of the other hypotheses had a significant contribution.

INSERT FIGURE 1 ABOUT HERE

In women, early life SEP had a strong positive association with later life SEP, but the association became weaker in the 75+ group. Early life health was not associated with later life SEP in any age group. Similarly with men, we found evidence of a positive association between early life health and later life physical health in all age groups. Later life SEP was positively associated with later life physical health in all age groups, with the effect being weaker in the older cohorts. Early life SEP had a positive association with later life physical health in age groups, with the effect being stronger in the 75+ group. The accumulation hypothesis had the strongest contribution to later life physical health inequalities in all age groups. Later life SEP dominated the effect in participants up to 74 years old, but early life SEP in women over 75. The total effect of early life SEP was significant in all age groups and had the second strongest contribution to later life health inequalities. In women under 75, the effect was mostly due to chains of risk, but for women over 75 most of the effect was due to early life SEP. Similarly with men, we did not observe any contribution of the social drift hypothesis to physical health inequalities over the life course. With respect to fibrinogen, there was no association between early life SEP and later life fibrinogen in any age group. On the contrary we observed a direct association between early life health and later life fibrinogen as well as a negative association between later life SEP and later life fibrinogen in women under 65. The accumulation hypothesis dominated by later life SEP had the strongest contribution to later life inequalities in fibrinogen levels in women under 65 while none of the other hypotheses had a significant contribution.

INSERT FIGURE 2 ABOUT HERE

Discussion

Our study extends previous findings on the well established association between lifelong SEP and health. We showed that later life physical health is more socially patterned than fibrinogen levels and that early life socio economic circumstances have a strong positive

effect on the later life SEP of men and women over 50. Consistent with the early life/critical period hypothesis, we found that the effect of early life SEP reaches directly until the beginning of late old age, predicting physical health and fibrinogen levels 65 and 55 years later respectively, extending previous findings on midlife health [19, 20, 31]. This effect was more prominent in women, suggesting that early life experiences related to socioeconomic circumstances may have a longer lasting effect in women compared to men. We also found evidence for indirect effects via later life SEP (chains of risk hypothesis) reaching until the beginnings of late old age with respect to physical health for both men and women, but only for men with respect to fibrinogen levels. Early life health had a direct effect on physical health that reached until the beginnings of late old age, but was not associated with fibrinogen levels in any age group. The observed association with physical health is in accordance, with previous findings on midlife and early old age [32], but extends these to older age groups of both genders. Despite this, the effect of early life health on later life SEP was negligible in men and women of all age groups, challenging the notion of reverse causality on the association between SEP and health. Ours and previous findings in younger cohorts do not support this explanation and maintain that that health selection explains only a small portion of the observed social gradient in health [33, 34].

With the exception of the negligible contribution of the social drift hypothesis, we confirmed that the chains of risk, critical period/early life and accumulation hypotheses all significantly contribute to later life health inequalities. However, a complex pattern of associations was observed and cohort differences not implied by previous findings emerged making the comparison of the relative importance of each hypothesis tedious, since their effect was also health outcome depended. There was considerable heterogeneity between cohorts with respect to the magnitude of the contribution of each hypothesis. For example, in women under 65, both critical period and chains of risk hypotheses were confirmed and had a similar contribution to later life health inequalities, whereas in men only the chains of risk hypothesis was supported. However in both genders for participants under 65, the accumulation of risk dominated by the effect of later life SEP had the most prominent role in explaining socioeconomic disparities in physical health and fibrinogen levels. On the contrary, in both men and women over 75, accumulation of risk mostly due to the effect of early life SEP was the dominant explanation of later life inequalities in physical health. A different pattern was observed for men with respect to fibrinogen levels, where later life inequalities were solely attributed to later life SEP.

There are several explanations for the observed cohort differences, as for example they could be attributed to the ageing process, with the younger cohorts being expected to exhibit similar patterns of associations as they grow older. Another explanation is that differences in the effects of early life SEP on later life health may be attributed to cohort specific effects due to the observed differences in early life SEP that shaped participants living conditions with the oldest groups being more disadvantaged during childhood. This explanation is supported by the observation that the 75+ cohort were 10 years old between 1918 and 1942, meaning that the majority of the participants spent their childhood during the great depression of the 1930's which had well documented effects in the living conditions of children in lower socio economic groups whose parents were largely unemployed. Thus, the strong effect of early life SEP on physical health might be the manifestation of a latent effect of the great depression of the 1930's, a finding with important implications for today's climate of financial austerity. Another plausible explanation concerns selection effects. Participants over 75 have lower SEP compared to younger groups but they can be thought of as a selected sample, in which selective attrition of lower SEP participants has already occurred. This is supported by the smaller amount of variance of the later life SEP measure and may – at least partly – explain the weaker effect of later life SEP in this age group.

Strengths of the present study include the availability of a population based dataset, the inclusion of biomarkers of later life health and the formal model based approach in the parameterisation of the various hypotheses that have been proposed to explain the effect of life course SEP on later life health. However, there are some limitations that need to be considered while interpreting our results. First, the problem of unmeasured confounding has yet to be resolved in observational settings and it has been argued that come kind of sensitivity analysis should always be presented when observational data is used [35]. According to the sequential ignorability assumption for our estimated parameters to be valid no unmeasured confounders are allowed in any part of Graph 1. We have adjusted for potential confounders of the later life SEP and health association and the assumption of no unmeasured confounders for this part of Graph 1 is – we believe – sufficiently approximated. However, parental characteristics, such as cognitive ability and health status that may have intergenerational effects are not taken into account in our model and may have introduced bias in our estimates. We attempted to capture these effects by simulating a series of Monte Carlo sensitivity analyses where a continuous variable was added in the models in order to represent unmeasured parental characteristics. Our observed results remained valid even in strong confounding scenarios, indicating that unmeasured parental characteristics do not account for our results (results presented on Appendix II). However, despite our efforts, bias due to unknown unmeasured confounders cannot be ruled out.

Another potential source of bias is the retrospective nature of the early life data. We found evidence – and subsequently controlled for - random error and systematic bias in the recall of early life SEP and health, with recollections of the former being more susceptible to random error. Those in good health at the time of recall reported higher SEP compared to participants with one or more chronic illnesses, a finding in agreement with previous studies [10, 11] according to which healthy adults who are also likely to be socioeconomically advantaged tend to suppress memories of childhood hardship. Consistent with the view that depression leads to negative self evaluation and outlook in life [36, 37] we found that it was negatively associated with recollections of early life SEP and early life health. It appears that the healthy participants' suppression of information related to childhood deprivation extends to negative experiences such as ill health in childhood. However, the systematic bias on early life SEP and health accounted for about 3% -7% of the overall valid –excluding random error - variance of both constructs, indicating that responses to questions in the ELSA life course interview are to a large extend driven by correct recall.

Despite these limitations, our results extend previous findings on mid adulthood and early old age to old age and the beginnings of late old age. The complexity of the observed associations that has not been captured by previous research highlights the need for further research on the mechanism that underlies the links between SEP and later life health in order to identify meaningful target areas for health related policy. This should be attempted with appropriate analytic strategies that formally recognise the various pathways that link SEP and health that have been implied by life-course and social causation theories.

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Table 1 Descriptive statistics of exposure, mediators and outcomes in the model

		Men			Women		
	Age Group	**N**	**Mean**	**Std. Deviation**	**N**	**Mean**	**Std. Deviation**
SEP at Age 10	50-64	1738	0.35	0.75	2143	0.43	0.75
	65-74	920	0.11	0.78	1084	0.21	0.85
	75+	702	-0.04	0.78	971	0.02	0.84
	Total	3360	0.20	0.78	4198	0.28	0.82
Early life health	50-64	1738	1.26	0.76	2143	1.33	0.74
	65-74	919	1.21	0.77	1082	1.13	0.79
	75+	702	1.20	0.73	969	1.16	0.78
	Total	3359	1.24	0.76	4194	1.24	0.77
Current SEP (Wave 4)	50-64	1556	0.15	0.79	1911	0.05	0.82
	65-74	823	0.05	0.75	991	-0.06	0.74
	75+	584	-0.05	0.71	814	-0.32	0.66
	Total	2963	0.08	0.77	3716	-0.06	0.78
Physical Health (Wave 4)	50-64	1557	0.28	0.81	1915	0.16	0.79
	65-74	823	0.00	0.79	991	-0.12	0.76
	75+	587	-0.31	0.73	827	-0.58	0.74
	Total	2967	0.08	0.82	3733	-0.08	0.83
Fibrinogen (Wave 4)	50-64	1001	3.25	0.57	1239	3.36	0.53
	65-74	543	3.39	0.55	651	3.44	0.54
	75+	321	3.44	0.60	447	3.55	0.55
	Total	1865	3.32	0.57	2337	3.42	0.54

Table 2. Standardised parameters and 95% confidence intervals – Men

50 - 64	Physical health		Fibrinogen		SEP Wave 4		Early Health	
SEP Wave 4	**0.35**	**(0.30 - 0.40)**	**-0.13**	**(-0.19 - -0.06)**				
SEP Age 10	0.04	(-0.01 - 0.09)	0.02	(-0.05 - 0.08)	**0.33**	**(0.28 - 0.37)**	0.04	(-0.01 - 0.09)
Early Health	**0.19**	**(0.15 - 0.24)**	**-0.06**	**(-0.12 - -0.01)**	0.02	(-0.03 - 0.07)		
Chains of risk	**0.11**	**(0.09 - 0.13)**	0.01	(-0.02 – 0.03)				
Accumulation	**0.38**	**(0.32 - 0.44)**	**-0.11**	**(-0.19 - -0.03)**				
Total SEP Age 10	**0.15**	**(0.10 - 0.20)**	-0.03	(-0.09 - 0.04)				
Social drift	0.01	(-0.01 - 0.02)	-0.01	(-0.01 - 0.01)				
65 - 74	**Physical health**		**Fibrinogen**		**SEP Wave 4**		**Early Health**	
SEP Wave 4	**0.33**	**(0.26 - 0.40)**	-0.03	(-0.12 - 0.07)				
SEP Age 10	0.03	(-0.04 - 0.10)	-0.06	(-0.16 - 0.03)	**0.39**	**(0.33 - 0.45)**	0.03	(-0.03 - 0.10)
Early Health	**0.19**	**(0.13 - 0.25)**	-0.01	(-0.09 - 0.07)	0.02	(-0.04 - 0.09)		
Chains of risk	**0.13**	**(0.09 - 0.16)**	-0.01	(-0.05 - 0.03)				
Accumulation	**0.36**	**(0.28 - 0.44)**	-0.09	(-0.21 - 0.03)				
Total SEP Age 10	**0.16**	**(0.09 - 0.23)**	-0.07	(-0.17 - 0.02)				
Social drift	0.01	(-0.01 - 0.03)	-0.01	(-0.02 - 0.01)				
75+	**Physical health**		**Fibrinogen**		**SEP Wave 4**		**Early Health**	
SEP Wave 4	**0.18**	**(0.10 - 0.27)**	**-0.14**	**(-0.27 - -0.02)**				
SEP Age 10	**0.11**	**(0.03 - 0.18)**	0.03	(-0.09 - 0.16)	**0.40**	**(0.33 - 0.47)**	-0.04	(-0.11 - 0.04)
Early Health	**0.13**	**(0.05 - 0.21)**	0.04	(-0.06 - 0.14)	-0.03	(-0.10 - 0.05)		
Chains of risk	**0.07**	**(0.04 - 0.11)**	**-0.06**	**(-0.11 - -0.01)**				
Accumulation	**0.29**	**(0.19 - 0.39)**	-0.11	(-0.25 - 0.04)				
Total SEP Age 10	**0.18**	**(0.10 - 0.26)**	-0.02	(-0.14- 0.09)				
Social drift	-0.01	(-0.02 - 0.01)	0.01	(-0.02 - 0.02)				

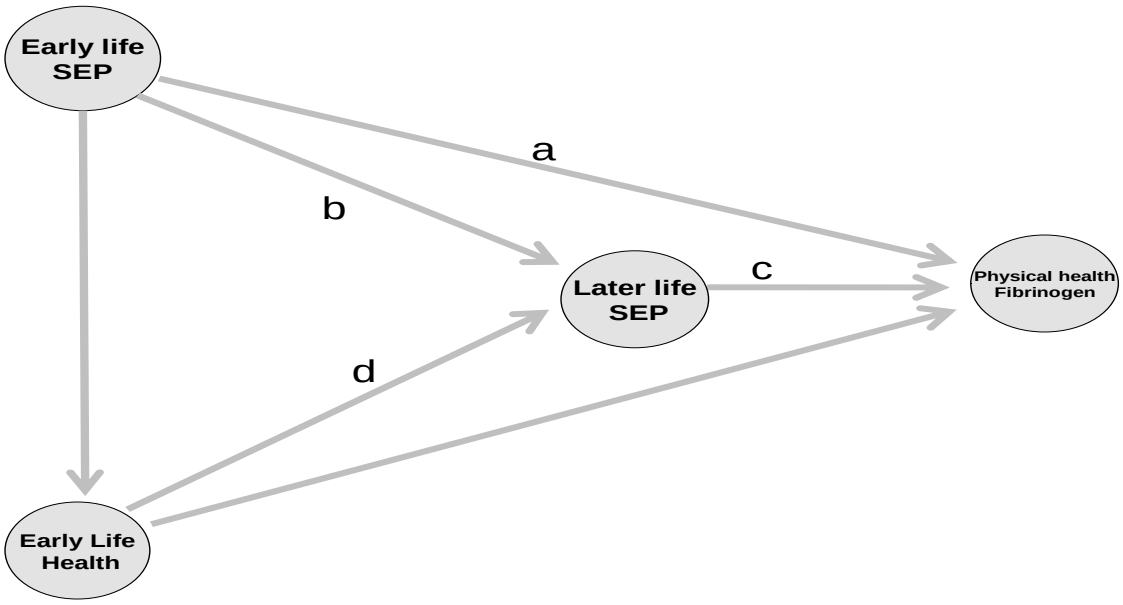
*Model adjusted for age, ethnicity, marital status, number of children and numerical ability (all variables from ELSA Wave 4)

Table 3. Standardised parameters and 95% confidence intervals - Women

50 - 64	Physical health		Fibrinogen		SEP Wave 4		Early Health	
SEP Wave 4	**0.31**	**(0.27 - 0.36)**	**-0.15**	**(-0.21 - -0.09)**				
SEP Age 10	**0.12**	**(0.08 - 0.17)**	-0.03	(-0.09 - 0.03)	**0.34**	**(0.30 - 0.38)**	0.03	(-0.02 - 0.07)
Early Health	**0.20**	**(0.16 - 0.24)**	**-0.06**	**(-0.12 - -0.01)**	**0.06**	**(0.02 - 0.10)**		
Chains of risk	**0.10**	**(0.08 - 0.12)**	-0.01	(-0.03 - 0.01)				
Accumulation	**0.43**	**(0.38 – 0.48)**	**-0.18**	**(-0.26 - -0.11)**				
Total SEP Age 10	**0.22**	**(0.18 - 0.27)**	**-0.08**	**(-0.14 - -0.02)**				
Social drift	**0.02**	**(0.01 - 0.03)**	-0.01	(-0.02 - 0.01)				
65 - 74	**Physical health**		**Fibrinogen**		**SEP Wave 4**		**Early Health**	
SEP Wave 4	**0.24**	**(0.17 -0.30)**	-0.04	(-0.13 - 0.05)				
SEP Age 10	**0.12**	**(0.06 - 0.18)**	0.01	(-0.07 - 0.09)	**0.38**	**(0.32 - 0.43)**	-0.03	(-0.09 - 0.03)
Early Health	**0.22**	**(0.16 - 0.27)**	-0.08	(-0.16 - 0.01)	0.02	(-0.04 - 0.08)		
Chains of risk	**0.09**	**(0.06 - 0.12)**	-0.02	(-0.05 - 0.02)				
Accumulation	**0.35**	**(0.28 - 0.42)**	-0.04	(-0.13 - 0.06)				
Total SEP Age 10	**0.21**	**(0.15 - 0.27)**	-0.01	(-0.09 - 0.07)				
Social drift	0.01	(-0.01 - 0.02)	-0.01	(-0.02 - 0.01)				
75+	**Physical health**		**Fibrinogen**		**SEP Wave 4**		**Early Health**	
SEP Wave 4	**0.13**	**(0.06 - 0.20)**	-0.05	(-0.14 - 0.04)				
SEP Age 10	**0.18**	**(0.10 - 0.25)**	-0.05	(-0.14 - 0.04)	**0.24**	**(0.17 - 0.31)**	0.03	(-0.05 - 0.13)
Early Health	**0.14**	**(0.08 - 0.20)**	-0.07	(-0.16 - 0.02)	-0.02	(-0.09 - 0.05)		
Chains of risk	**0.03**	**(0.01 - 0.05)**	-0.01	(-0.04 - 0.01)				
Accumulation	**0.31**	**(0.22 - 0.40)**	-0.11	(-0.23 - 0.01)				
Total SEP Age 10	**0.21**	**(0.14 - 0.28)**	-0.07	(-0.16 - 0.02)				
Social drift	-0.01	(-0.02 - 0.01)	0.01	(-0.02 - 0.02)				

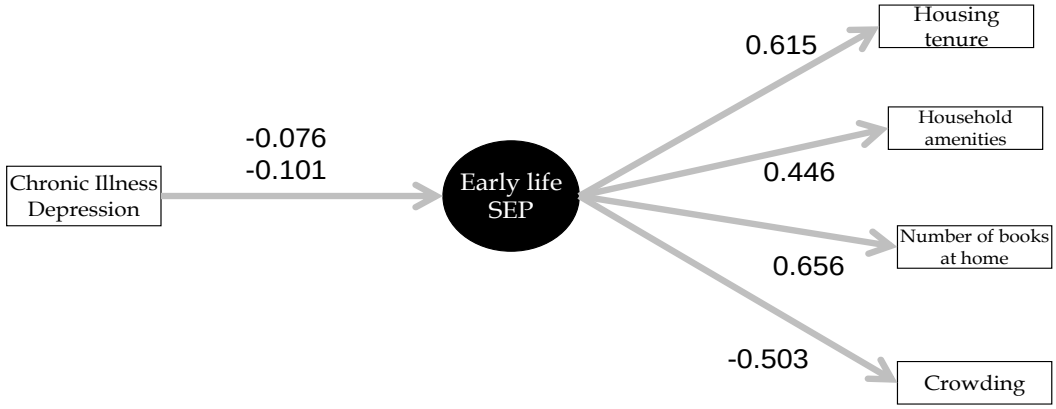
*Model adjusted for age, ethnicity, marital status, number of children and numerical ability (all variables from ELSA Wave 4)

Graph1. Directed Acyclic Graph of the estimated model



Critical period/Early life = a
Chains of risk = b*c
Accumulation = a + c
Social drift = d*c
Total effect of SEP Age 10 = a + (b*c)

Graph 2. Early life SEP (age 10) measurement model



Graph 3. Early life health (childhood and early adolescence) measurement model

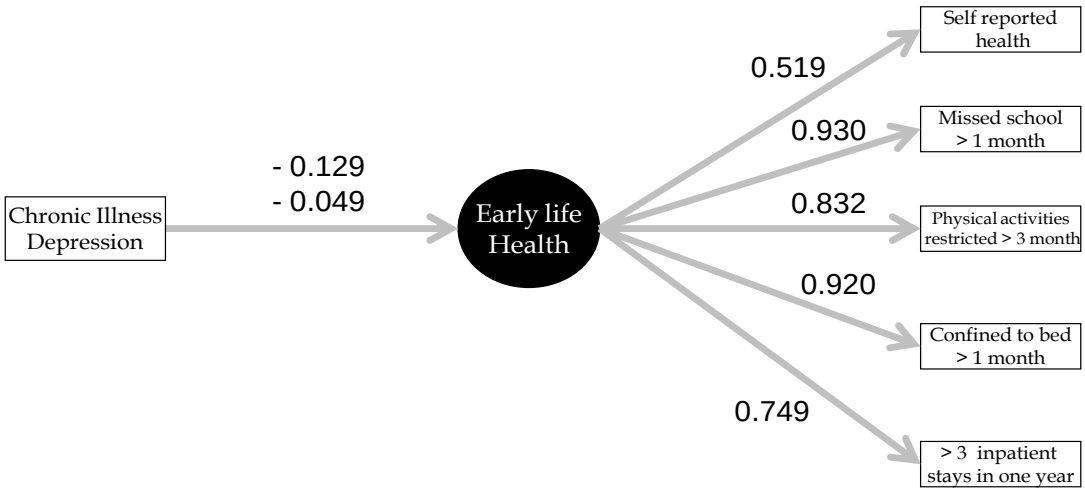


Figure 1. Standardised parameters and 95% confidence intervals – Physical health

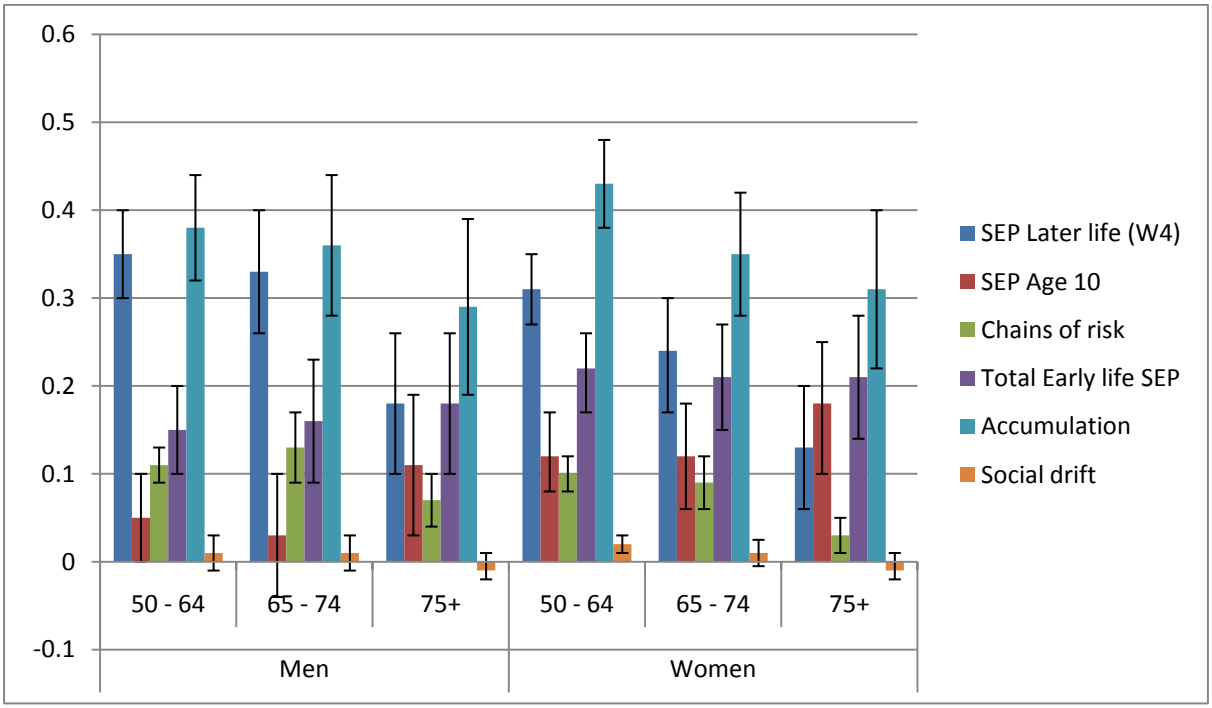
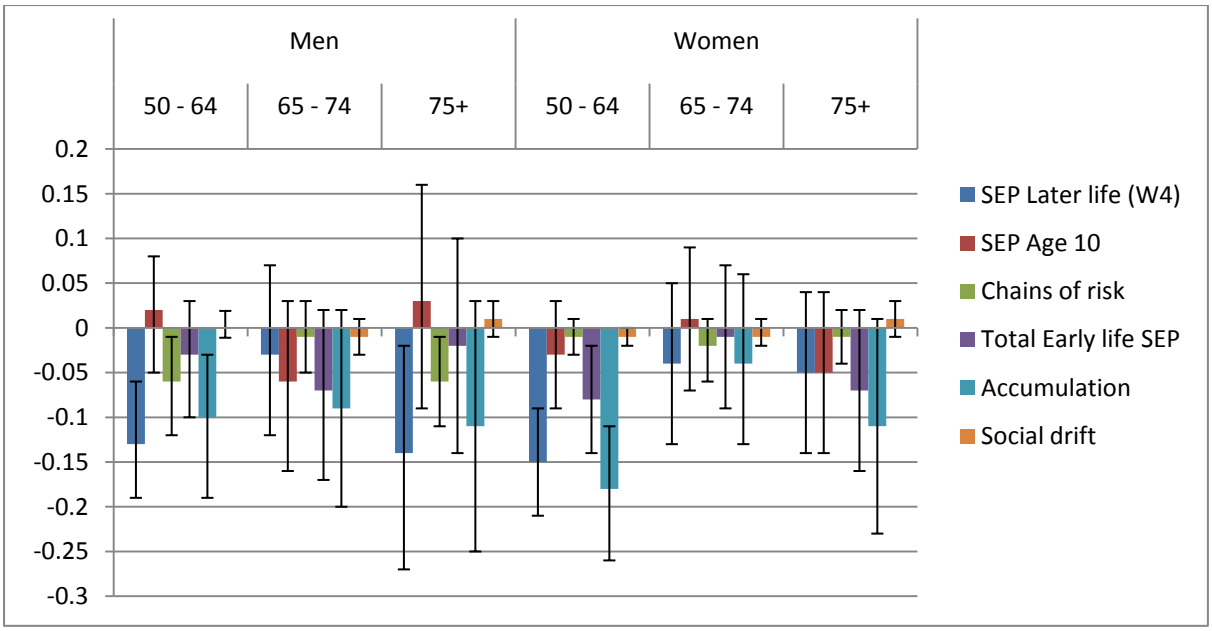


Figure 2. Standardised parameters and 95% confidence intervals – Fibrinogen



Appendix I – Frequency distribution of all indicators of latent variables in the model

Distribution of early life health variables (ELSA lifecourse interview 2007)

Gender	Men								Women								Total	
Age group	50-64		65-74		75+		Total Men		50-64		65-74		75+		Total Women			
	n	%	n	%	n	%	n	%	n	%	n	%	n	%			n	%
n by age and gender	1745		923		702		3370		2149		1089		973		4211		7581	
Self-reported health																		
Excellent	673	38.6	328	35.6	205	29.2	1206	35.8	834	38.8	299	27.4	257	26.4	1390	33.0	2596	34.2
Very good	581	33.3	293	31.7	249	35.5	1123	33.3	666	31.0	371	34.1	370	38.1	1407	33.4	2530	33.4
Good	307	17.6	190	20.6	166	23.7	663	19.7	403	18.8	257	23.6	217	22.3	877	20.8	1540	20.3
Fair	123	7.0	85	9.2	56	8.0	264	7.8	185	8.6	103	9.4	78	8.0	366	8.7	630	8.3
Poor	47	2.7	22	2.4	25	3.5	94	2.8	58	2.7	51	4.7	43	4.4	152	3.6	246	3.3
Health varied a lot	14	0.8	4	0.4	1	0.1	19	0.6	3	0.1	5	0.5	5	0.5	13	0.3	32	0.4
Missing	0	0.0	1	0.1	0	0.0	1	0.0	0	0.0	3	0.3	3	0.3	6	0.2	7	0.1
Missed school for > 1 month																		
Yes	371	21.3	206	22.3	139	19.8	716	21.3	407	19.0	275	25.3	239	24.6	921	21.9	1637	21.6
No	1372	78.6	713	77.3	559	79.6	2644	78.4	1737	80.8	806	74.0	730	75.0	3273	77.7	5917	78.0
Missing	2	0.1	4	0.4	4	0.6	10	0.3	5	0.2	8	0.7	4	0.4	17	0.4	27	0.4
Physical activities restricted > 3 months																		
Yes	229	13.1	115	12.5	75	10.7	419	12.4	204	9.5	141	12.9	105	10.8	450	10.7	869	11.5
No	1515	86.8	805	87.2	627	89.3	2947	87.5	1943	90.4	944	86.7	863	88.7	3750	89.1	6697	88.3
Missing	1	0.1	3	0.3	0	0.0	4	0.1	2	0.1	4	0.4	5	0.5	11	0.2	15	0.2
Confined to bed > 1 month																		
Yes	264	15.1	154	16.7	114	16.2	532	15.8	304	14.1	208	19.1	178	18.3	690	16.4	1222	16.1
No	1478	84.7	766	83.0	582	82.9	2826	83.9	1841	85.7	876	80.4	793	81.5	3510	83.4	6336	83.6
Missing	3	0.2	3	0.3	6	0.9	12	0.3	4	0.2	5	0.5	2	0.2	11	0.2	23	0.3
More than 3 inpatient stays in one year																		
Yes	7	0.4	7	0.8	2	0.3	16	0.5	23	1.1	3	0.3	3	0.3	29	0.7	45	0.6
No	1565	89.7	809	87.6	602	85.8	2976	88.3	1968	91.6	943	86.6	837	86.0	3748	89.0	6724	88.7
Missing	173	9.9	107	11.6	98	13.9	378	11.2	158	7.3	143	13.1	133	13.7	434	10.3	812	10.7

Distribution of early life SEP variables (ELSA lifecourse interview))

Gender	Men								Women								Total	
Age group	50-64		65-74		75+		Total Men		50-64		65-74		75+		Total Women			
	n	%	n	%	n	%	n	%	n	%	n	%	n	%			n	%
n by age and gender	1225		886		646		2757		1482		1048		912		3442		6199	
Housing tenure at age 10																		
Own	441	36.0	276	31.1	171	26.5	888	32.2	524	35.4	340	32.4	242	26.5	1106	32.1	1994	32.2
Rent	736	60.1	583	65.8	438	67.8	1757	63.7	914	61.7	666	63.6	628	68.9	2208	64.2	3965	63.9
Missing	48	3.9	27	3.1	37	5.7	112	4.1	44	2.9	42	4.0	42	4.6	128	3.7	240	3.9
n by age and gender	1745		923		702		3370		2149		1089		973		4211		7581	
Household amenities**																		
0	106	6.1	74	8.0	72	10.3	252	7.5	167	7.8	78	7.2	85	8.7	330	7.8	582	7.7
1	178	10.2	202	21.9	199	28.3	579	17.2	194	9.0	227	20.9	270	27.8	691	16.4	1270	16.8
2	100	5.7	77	8.3	77	11.0	254	7.5	114	5.3	96	8.8	104	10.7	314	7.5	568	7.5
3	155	8.9	126	13.7	95	13.5	376	11.1	172	8.0	132	12.1	134	13.8	438	10.4	814	10.7
4	1055	60.4	404	43.8	209	29.8	1668	49.5	1312	61.1	488	44.8	324	33.3	2124	50.4	3792	50.0
5	89	5.1	12	1.3	5	0.7	106	3.2	119	5.5	23	2.1	8	0.8	150	3.6	256	3.4
Missing	62	3.6	28	3.0	45	6.4	135	4.0	71	3.3	45	4.1	48	4.9	164	3.9	299	3.9
Number of books																		
None or very few (0-10 books)	416	23.8	305	33.0	265	37.8	986	29.3	383	17.8	292	26.8	291	29.9	966	23.0	1952	25.7
1 shelf (11-25 books)	411	23.6	232	25.1	168	23.9	811	24.1	512	23.8	254	23.4	219	22.5	985	23.4	1796	23.7
1 bookcase (26-100 books)	574	32.9	224	24.3	156	22.2	954	28.3	706	32.9	279	25.6	263	27.0	1248	29.6	2202	29.1
2 bookcases (101-200 books)	128	7.3	68	7.4	36	5.1	232	6.9	239	11.1	109	10.0	79	8.1	427	10.1	659	8.7
3+ bookcases (>200 books)	140	8.0	55	6.0	25	3.6	220	6.5	232	10.8	95	8.7	60	6.2	387	9.2	607	8.0
Missing	76	4.4	39	4.2	52	7.4	167	4.9	77	3.6	60	5.5	61	6.3	198	4.7	365	4.8
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
No. of bedrooms (n=7263)	2.96	.93	2.91	.90	2.91	1.00	2.93	.94	3.02	.94	2.89	.89	2.94	.96	2.97	.93	2.95	.94
Household members (n=7287)	4.88	1.70	5.02	1.87	5.10	1.96	4.96	1.81	5.06	1.90	4.91	1.75	5.27	1.99	5.07	1.89	5.02	1.85
Persons per room (n=7258)	1.74	.72	1.83	.79	1.88	.87	1.79	.77	1.78	.76	1.82	.82	1.96	1.05	1.83	.85	1.81	.82

Distribution of confounders in the model (ELSA Wave 4)

	Men								Women								Total	
Age group	50-64		65-74		75+		Total		50-64		65-74		75+		Total			
	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
n	1745		923		702		3370		2149		1089		973		4211		7581	
Marital status																		
Single, separated, widowed	333	19.1	174	18.9	209	29.8	716	21.3	529	24.6	416	38.2	587	60.3	1532	36.4	2248	29.7
Married/in partnership	1224	70.1	649	70.3	378	53.8	2251	66.8	1386	64.5	575	52.8	240	24.7	2201	52.3	4452	58.7
Missing	188	10.8	100	10.8	115	16.4	403	11.9	234	10.9	98	9.0	146	15.0	478	11.3	881	11.6
Ethnicity																		
White	1509	86.5	804	87.1	582	82.9	2895	85.9	1862	86.6	976	89.6	819	84.2	3657	86.8	6552	86.4
Non-white	48	2.7	19	2.1	5	0.7	72	2.1	53	2.5	15	1.4	8	0.8	76	1.8	148	2.0
Missing	188	10.8	100	10.8	115	16.4	403	12.0	234	10.9	98	9.0	146	15.0	478	11.4	881	11.6
Retirement																		
Not retired	1211	69.4	89	9.6	26	3.7	1326	39.4	1338	62.3	175	16.1	108	11.1	1621	38.5	2947	38.9
Retired	345	19.8	733	79.4	560	79.8	1638	48.6	577	26.8	816	74.9	719	73.9	2112	50.2	3750	49.5
Missing	189	10.8	101	11.0	116	16.5	406	12.0	234	10.9	98	9.0	146	15.0	478	11.3	884	11.6
Number of children																		
0	249	14.3	96	10.4	70	10.0	415	12.3	237	11.0	107	9.8	137	14.1	481	11.4	896	11.8
1	290	16.6	113	12.2	108	15.4	511	15.2	342	15.9	149	13.7	172	17.7	663	15.7	1174	15.5
2	644	36.9	362	39.2	253	36.0	1259	37.4	817	38.0	410	37.7	333	34.2	1560	37.1	2819	37.2
3	300	17.2	198	21.5	145	20.7	643	19.1	423	19.7	243	22.3	189	19.4	855	20.3	1498	19.8
4+	262	15.0	154	16.7	126	17.9	542	16.0	330	15.4	180	16.5	142	14.6	652	15.5	1194	15.7
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
n	1534		819		563		2916		1905		981		800		3686		6602	
Numeracy	4.85	1.12	4.42	1.19	3.96	1.29	4.56	1.22	4.18	1.19	3.75	1.21	3.33	1.31	3.88	1.27	4.18	1.29

Distribution of health characteristics used in the derivation of physical health (ELSA Wave 4)

	Men								Women								Total	
Age group	50-64		65-74		75+		Total		50-64		65-74		75+		Total			
	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
n	1745		923		702		3370		2149		1089		973		4211		7581	
Self-rated health																		
Excellent	243	13.9	95	10.3	34	4.8	372	11.0	293	13.6	90	8.3	40	4.1	423	10.0	795	10.5
Very good	499	28.6	227	24.6	144	20.5	870	25.8	616	28.7	280	25.7	169	17.4	1065	25.3	1935	25.5
Good	483	27.7	273	29.6	200	28.5	956	28.4	603	28.1	354	32.5	267	27.4	1224	29.1	2180	28.8
Fair	220	12.6	173	18.7	139	19.8	532	15.8	314	14.6	207	19.0	228	23.4	749	17.8	1281	16.9
Poor	93	5.3	51	5.5	51	7.3	195	5.8	81	3.8	54	5.0	101	10.4	236	5.6	431	5.7
Missing	207	11.9	104	11.3	134	19.1	445	13.2	242	11.2	104	9.5	168	17.3	514	12.2	959	12.6
Difficulties in ADL																		
Yes	257	14.7	221	23.9	272	38.8	750	22.3	368	17.1	298	27.4	482	49.5	1148	27.3	1898	25.1
No	1299	74.4	602	65.2	315	44.9	2216	65.7	1547	72.0	693	63.6	345	35.5	2585	61.4	4801	63.3
Missing	189	10.9	100	10.9	115	16.3	404	12.0	234	10.9	98	9.0	146	15.0	478	11.3	882	11.6
Chronic illness																		
Yes	761	43.6	469	50.8	359	51.1	1589	47.2	926	43.1	557	51.2	550	56.5	2033	48.3	3622	47.8
No	795	45.6	354	38.4	227	32.4	1376	40.8	989	46.0	434	39.8	277	28.5	1700	40.4	3076	40.6
Missing	189	10.8	100	10.8	116	16.5	405	12.0	234	10.9	98	9.0	146	15.0	478	11.3	883	11.6
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
n	1315		716		487		2518		1551		863		648		3062		5580	
Grip strength	40.02	8.68	34.84	7.74	27.41	7.60	36.11	9.52	23.39	5.76	20.05	5.54	15.35	5.20	20.75	6.40	27.68	11.04
n	1194		649		364		2207		1412		745		398		2555		4762	
Chair rise (5 rises)	10.05	3.08	11.75	3.57	13.71	4.56	11.15	3.76	10.47	3.38	12.32	4.14	15.30	5.79	11.76	4.42	11.48	4.14
n	1238		673		434		2345		1486		811		551		2848		5193	
Lung function	4.19	1.00	3.69	0.91	3.07	0.87	3.84	1.04	2.99	0.75	2.55	0.66	2.01	0.64	2.68	0.80	3.20	1.08

Appendix II - Sensitivity analysis – Strong confounding scenario

Graph A shows a strong confounding scenario. U represents unmeasured parental characteristics and their effect is given in standardised parameters (assumed to be similar for all age groups). Figures B and C show the standardised estimated parameters derived from this model.

Graph A

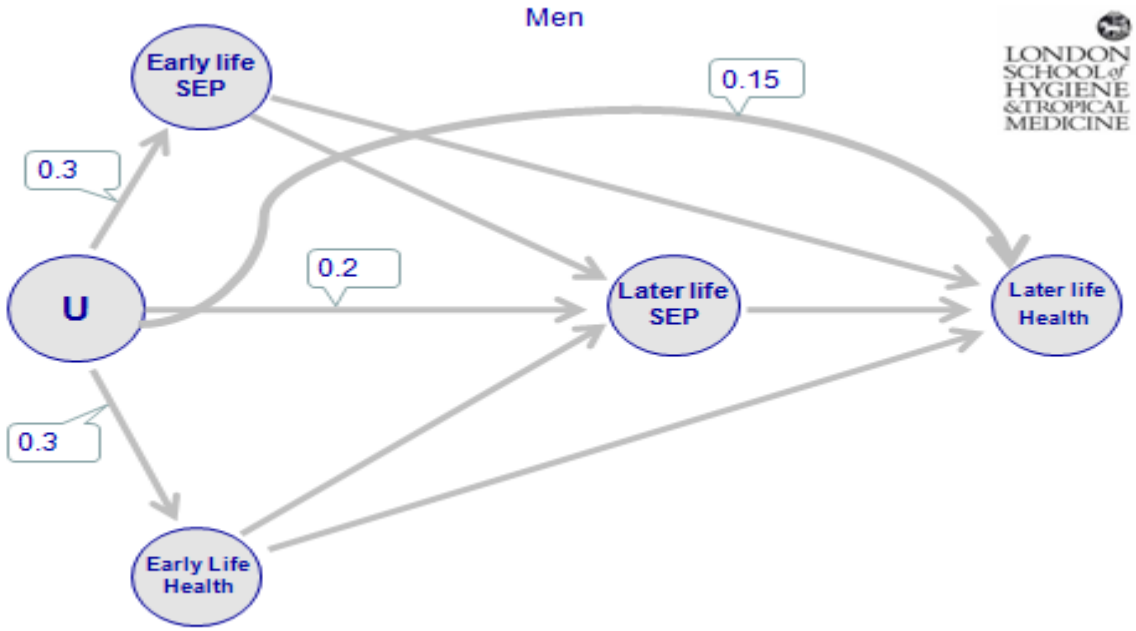


Figure B

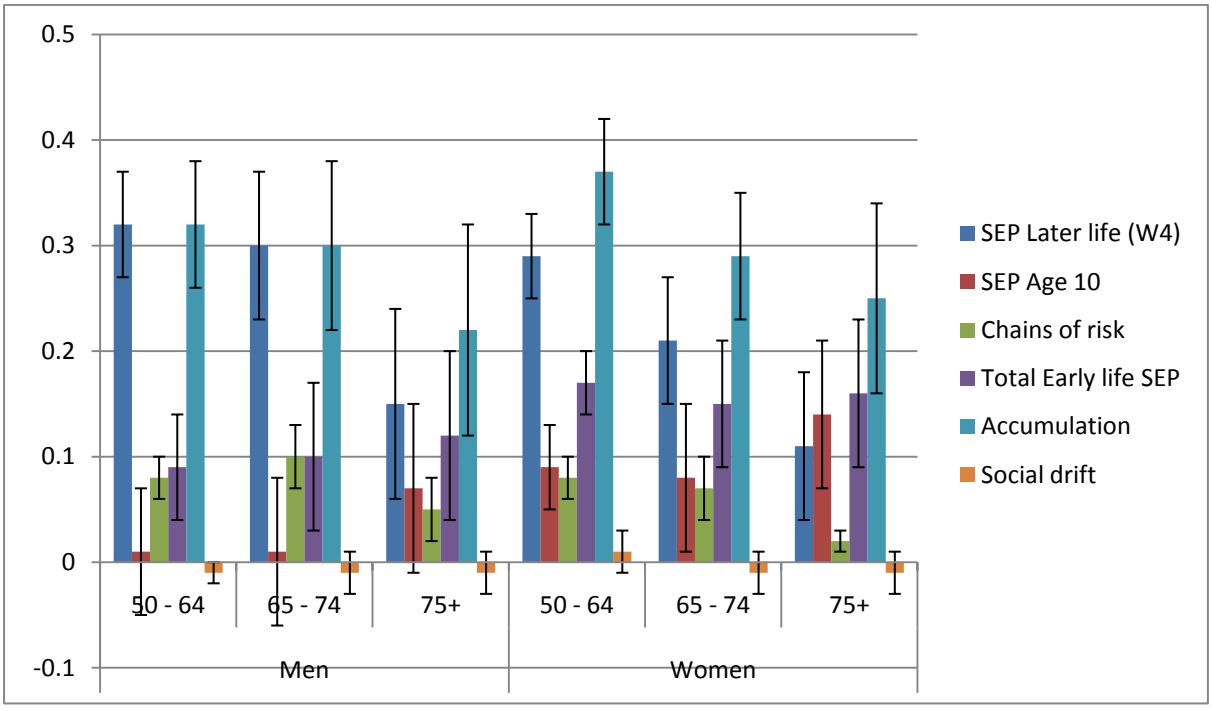
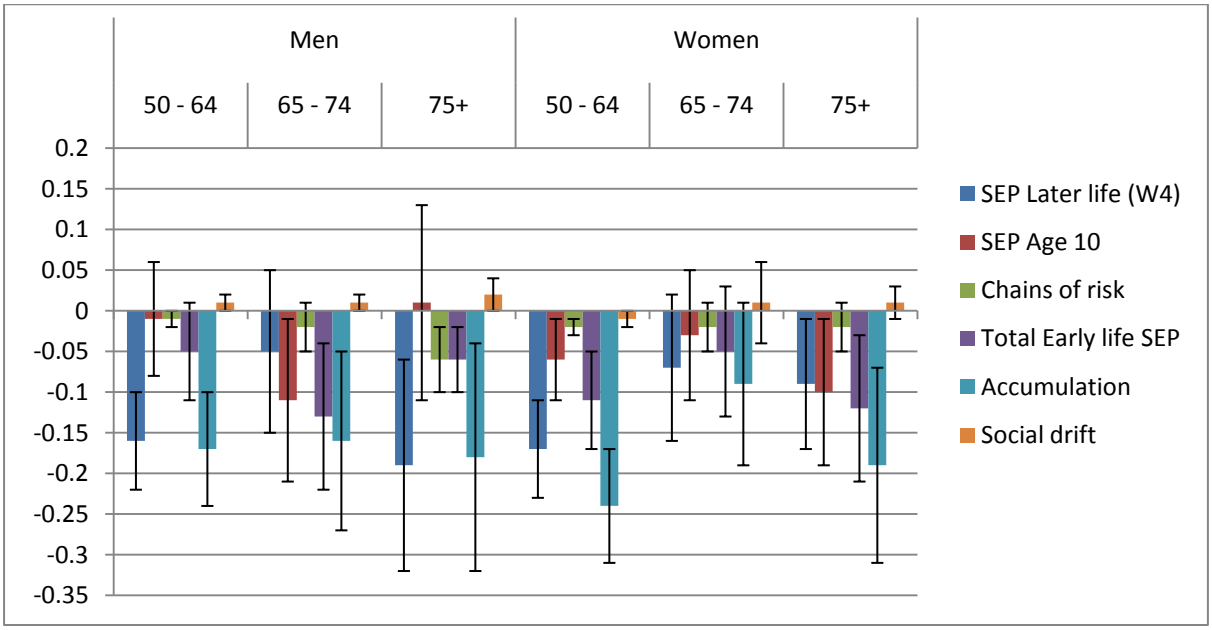


Figure C


Men
Women
50 - 64
65 - 74
75+
50 - 64
65 - 74
75+
0.2
0.15
0.1
0.05
0
-0.05
-0.1
-0.15
-0.2
-0.25
-0.3
-0.35
SEP Later life (W4)
SEP Age 10
Chains of risk
Total Early life SEP
Accumulation
Social drift