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Lifelong Socio Economic Position and biomarkers of later life health: Testing the contribution of competing hypotheses

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ABSTRACT

The relative contribution of early or later life Socio Economic Position (SEP) to later life health is not fully understood and there are alternative hypotheses about the pathways through which they may influence health. We used data from the English Longitudinal Study of Ageing with a formal approach for the identification of mediating factors in order to investigate alternative hypotheses about life course influences on biomarkers of later life health. We found that early life SEP predicts physical health at least 65 years later. However, a more complicated pattern of associations than that implied by previous findings was also observed. Age group 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 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 mechanisms that underlie the association between SEP and later life health.

1. 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 in younger age groups (Demakakos et al., 2008; Gjonca et al., 2009; Grundy and Sloggett, 2003; Huisman et al., 2003; Ploubidis et al., 2011). 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 the European GDP (Mackenbach et al., 2011) suggest that there is great potential for shifting the overall distribution of risk, and therefore improving average population health (Rose, 1985), by eliminating or reducing the socioeconomic health gradient in older age groups (Marmot et al., 1984; Marmot et al., 1991).

However, in order to achieve this, a better understanding of the mechanisms that underlie the association between life-course SEP and later life health is needed. The complexity of these mechanisms calls for an interdisciplinary approach where ideas from life course epidemiology and social science are combined in a unified theoretical framework. The former body of literature has proposed several explanations for the association between SEP and health over the life course (Ben-Shlomo and Kuh, 2002; Hayward and Gorman, 2004; Kuh et al., 2003; Lynch and Smith, 2005; Poulton et al., 2002; Shanahan, 2000). These can be summarised in four hypotheses that are presented in Graph 1. i) Early life/critical period hypothesis: According to this explanation, early life SEP directly influences later life health (path "a"); ii) Chains of risk: Early life SEP is thought to influence later life health indirectly, via later life SEP (paths "b" and "c"); iii) Accumulation of risk: Both early life and later life SEP synergistically influence later life health (paths "a" and "c"); iv) Social drift: According to this explanation, early life health indirectly influences later life health via later life SEP (paths "d" and "c").

Crucial to the interpretation and meaningful comparison of the explanations offered by life-course theory is a better understanding

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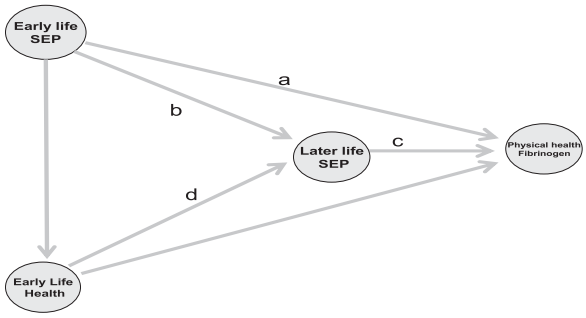


i) Critical period/Early life = a
ii) Chains of risk = b*c
iii) Accumulation = a + c
iv) Social drift = d*c
v) Total effect of SEP Age 10 = a + (b*c)

Graph 1. Directed acyclic graph of the association between life-course SEP and later life health.

of the pathways through which SEP is linked to health. Within the social causation framework theories of explanation of the relation between SEP and health at any stage of the life course essentially focus on three mechanisms (Smith et al., 1994). The first is a neo-materialist one; suggesting that health gradients arise because those with higher incomes are able to purchase better food, better housing, live in safer environments and have better access to health care (Kaplan et al., 1996; Lynch et al., 1997). The second emphasises the social patterning of health related behaviour, such as smoking, physical activity, alcohol consumption and appropriate use of health care (Schrijvers et al., 1999). The third explanation places more emphasis on psychosocial factors such as empowerment and exposure to stressful events (Wilkinson, 1997) and suggests that perceptions of relative SEP produce negative emotions which are translated into poorer health through stress thus giving rise to health inequalities (Pearlin, 1989; Steptoe et al., 2003).

We combined insights from these social theories and the hypotheses offered from life-course epidemiology in the framework adopted here in an attempt to shed further light on the mechanisms underlying associations between life course SEP and later life health. For example, according to the chains of risk hypothesis parental SEP may influence adult SEP through access to social and economic resources and adult SEP may in turn influence health. There is plenty of evidence showing 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 influencing health related behaviour, access to health care and/or causing elevated stress levels (Ben-Shlomo and Kuh, 2002). Similarly, according to the social drift hypothesis, poor health in childhood may lead to downward social mobility as well as poor health in adulthood (Hayward and Gorman, 2004; Poulton et al., 2002; Shanahan, 2000). Downward social mobility will result in relatively lower SEP in later life, which in turn will increase risks of unhealthy behaviour, life events that may elevate stress levels and/or restrict access to appropriate health care. Similarly, early life socioeconomic circumstances may directly influence health related behaviour in later life. We note that a similar mechanism may operate from "critical periods" other than childhood but most evidence to date concerning direct effects from specific periods to adult and later life health has focused on childhood and adolescence. Finally, the accumulation of risk hypothesis posits that SEP and SEP transitions in various stages of the life course synergistically influence later life health (Ben-Shlomo and Kuh, 2002; Kuh et al., 2003). An example of how this mechanism may operate is that SEP and SEP transitions at any stage of the life course may increase or decrease the probability of smoking and it is well known that chronic smoking increases the risk for various diseases (Doll et al., 2004; Pirie et al., 2013).

Despite the extensive research on the association between life-course SEP and health, the relative contribution of the proposed hypotheses, which is of potential importance in the design of successful policy interventions aiming to reduce inequalities, has not been thoroughly assessed. The challenge lies in the appropriate parameterisation of each hypothesis (De Stavola and Daniel, 2012), an endeavour that requires suitable statistical tools, beyond standard analytical methods. With some exceptions (Bartley et al., 2012; Chandola et al., 2006; Nandi et al., 2012), the vast majority of the evidence concerning life course processes comes from studies that have relied on standard regression models (Birnie et al., 2011; De Stavola et al., 2006). Such models allow only limited conclusions to be drawn about the relative importance of the hypothesised life course pathways which lead to socioeconomic disparities in later life health. In the present study we adopt a formal approach to the identification of mediating factors in order to meaningfully compare the relative contribution of the hypothesised mechanisms whereby life course SEP influences later life health.

2. Methods

2.1. Sample

We used data from the fourth wave (2009) and the life course interview (third wave – 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 (Marmot et al., 2002). A comparison of the socio-demographic characteristics of the ELSA with 2001 national census data indicated that the ELSA sample was representative of the non institutionalised population (Marmot et al., 2002). In ELSA Wave 3 a refreshment sample of from the Health Survey for England was recruited to maintain the representation of people aged 50–53 years (Steptoe et al., 2012). Comparisons of the socio-demographic characteristics of participants against results from the 2011 national census indicated that the ELSA sample remains broadly representative of the English older population (Steptoe et al., 2012).

Our analytic sample included participants with at least one valid observation for the early life SEP indicators measured in the ELSA Wave 3 life-course interview ($N = 7578$, out of 7855 eligible participants). From the participants that took part on the ELSA life-course interview at Wave 3, some information was lost due to attrition by Wave 4 (74% of participants in Wave 3 participated in Wave 4). Consequently for the variables measured at Wave 4, valid information was available for 6683 participants. Preliminary results (not shown here) indicate that the distribution of demographic characteristics such as gender and marital status, as well as self rated health status of the participants in our analytic sample was similar with 2011 national census data, indicating that it is broadly representative of the non institutionalised older population of England.

2.2. Measures

2.2.1. Recollection of early life SEP and health – ELSA life history interview (Wave 3 – 2007)

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. Paternal occupational social class, housing tenure, access to household amenities, the number of books in the household and number of persons per room (crowding) were used as indicators of early life SEP. Self reported health at age 10, whether the participants had missed school for more than one month, whether physical activities were restricted for more than 3 months, whether the participants recall being confined to bed for more than one month and an indicator of more than three inpatient stays in a year were used as indicators of early life health status. Combining several indicators in a latent variable that captures the common variance between these controls for random error in the form of unique variance (variance not shared with other indicators) that may be present in each of the recalled indicators of early life SEP and health since participants' childhood was at least four decades in the past. Furthermore, the use of latent variables also accounts for potential unobserved heterogeneity due to unmeasured early life indicators, which in practice means that any excluded indicator of early life SEP or health would have correlated highly with the latent summary and therefore would not alter the latent score (ranking of individuals). It is also possible that current health status may influence recall and reporting of retrospectively collected information on health and SEP in childhood. We therefore also controlled for health status at the time of recall (in Wave 3) using a binary item indicating the presence of any long standing illness and depression at the time of recall in a Multiple Indicators Multiple Causes (MIMIC) model in order to capture the influence of systematic recall bias. The questions on early life circumstances, depression and presence of chronic illness used in this model were asked at Wave 3 (2007), whereas our health outcomes were derived from ELSA wave 4 which took place two years later (2009). High scores on the latent summaries represent high SEP and optimal health (see Electronic Supplementary Material – I, for frequency distributions of all early life indicators).

2.2.2. Later life measures – ELSA Wave 4 (2009)

We employed a latent summary of later life SEP based on net income and total net wealth measured at the benefit unit level (a couple or a single person plus any dependent children they may have) using the approach described in Ploubidis et al. (2011), with high scores representing high SEP. As a measure of physical health (first later life health outcome) we used a latent summary of six indicators using the procedure proposed by Ploubidis and Grundy (2011) for surveys where both self reported 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 good physical health (see Appendix I for frequency distributions of all later life physical health indicators). Our second later life health outcome was fibrinogen, a well known haemostatic marker (Danesh et al., 2005; Moller and Kristensen, 1991).

2.2.3. Confounders

Age, gender, marital status, employment status, number of children and cognitive ability were included in the model as they are thought to be potentially important confounders of the association between the main mediator of interest, later life SEP, and the two later life health outcomes.

2.3. 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 (Snowden et al., 2011; Ten Have and Joffe, 2012; Vanderweele, 2012). We employed a Linear Structural Equation Modelling (LSEM) approach which returns valid parameter estimates under the sequential ignorability assumption (no unmeasured confounding for the exposures–outcomes and mediators–outcomes associations) and linearity of the models for outcomes and mediators (Imai et al., 2010). The application of a valid causal mediation method such as LSEM in these data allows us in addition to the estimation of direct effects – which is common in any regression model – to reliably quantify indirect effects and their standard errors. This is crucial for the present study since two of the four hypotheses we wish to compare (chains of risk and social drift) require the estimation of indirect effects. Preliminary analysis showed weak or no evidence for interactions of the exposure and mediators with respect to both health outcomes. Taking this into account the parameterisation of the four hypotheses is shown in Graph 1. We note that this parameterisation is valid only within a LSEM. If binary or ordinal mediators and/or outcomes were present, a different parameterisation and modelling framework would have had to be used (Daniel et al., 2011; Ten Have and Joffe, 2012).

The latent summaries of early life SEP, early life health, later life SEP and the measure of physical health, were derived with latent variable models appropriate for combinations of binary, ordinal and continuous indicators (Rabe-Hesketh and Skrondal, 2008). The latent variables represent continuous variables that underlie observed "coarsened" responses such as binary or ordinal responses. In the case of binary or ordinal indicators, their associations with the latent variable were modelled with a 2 parameter logit regression. In this instance, factor loadings represent the strength of the association between the indicator and the latent variable, whereas the thresholds represent the level of the latent variable that needs to be reached for a particular response in a binary or ordinal indicator to be endorsed. In the instances where continuous indicators were linked with continuous latent factors a traditional confirmatory factor analytic model with linear regressions between observed and latent variables was estimated. In this instance factor loadings were estimated and as in the binary/ordinal case they capture the association between the observed indicators and the continuous latent variable.

All models were estimated with the robust maximum likelihood (MLR) estimator in Mplus 7.0 (Muthen & Muthen, 1998-2013) and all reported model parameters are standardized so that their relative sizes can be compared. 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 (Little and Rubin, 1989, 2002). In this case MAR implies that the if all the variables that are responsible for the missing data generating mechanism are included in the model, then this can be ignored and parameter estimates can be robustly computed for participants with missing data. Practically, in our analysis MAR translates to the following assumption: all systematic missingness is due to variables included in our models, the exogenous variable (early life SEP), mediators (early life health and later life SEP) and mediator–outcome confounders (later life cognitive ability, age, marital status, number of children, and employment status). Any other missingness that is not accounted by these variables is thought to be random (thus

missing at random, since we assume that all systematic causes of attrition have been included in the model). We believe that this is a reasonable assumption since it has been shown that SEP and age and are the main drivers of attrition in population surveys in the UK (Durrant and Goldstein, 2008; Noah Uhrig, 2008), as well as in the ELSA (Steptoe et al., 2012).

3. Results

3.1. Measurement models for recollection of early life SEP and health

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

3.2. LSEM results

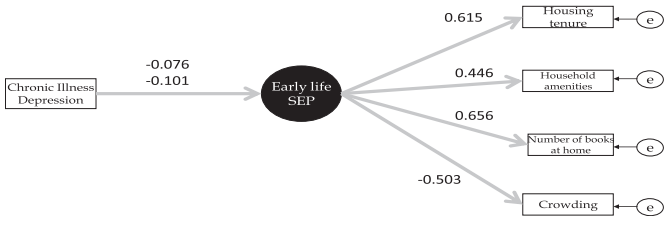
Table 1 shows descriptive statistics (mean and standard deviation) for the exogenous variable (early life SEP), mediators (early life Health and later life SEP) and outcomes (physical health and fibrinogen) in the model. Women had higher mean early life SEP, but similar mean early life health, than men. They also had worse later life physical health and higher mean fibrinogen levels compared to men. Cohort differences were observed in both genders. Younger age groups had higher mean early life SEP and better early life health. Similarly, they had higher mean later life SEP, better physical health and had lower fibrinogen levels. Preliminary analysis showed that all mean differences were significant.

In Tables 2 and 3 and Figs. 1 and 2 we present the standardised parameters and 95% confidence intervals for the six groups (models were estimated separately for each group).

3.3. Men

3.3.1. Early life SEP, chains of risk and total early life SEP effects

Early life SEP had a strong positive association with later life SEP, with the association becoming increasingly stronger in older age groups. The total effect of early life SEP was significant in all age groups and made the second strongest contribution to later life physical health inequalities. In men under 75, the effect was mostly indirect (supporting the chains of risk hypothesis), but for men over 75 most of the effect was direct (supporting the early life/critical period hypothesis). With respect to fibrinogen, there was no direct association between early life SEP and later life fibrinogen in any age group, but for men under 65 and over 75 the chains of risk hypothesis was supported, since early life SEP had an indirect effect (via later life SEP) on fibrinogen in both age groups.



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

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

3.3.2. Early life health and social drift hypothesis

Early life health was not associated with later life SEP in any group. Due to the lack of this association, the indirect effect representing the social drift hypothesis (effect of early life health on later life health via later life SEP) was not significant for either physical health or fibrinogen, indicating no support for the social drift hypothesis as an explanation of health inequalities over the life course. On the contrary, early life health had a positive direct effect on later life physical health in all age groups. We also observed a negative direct association between early life health and fibrinogen levels in the younger age group (50–64).

3.3.3. Later life SEP and accumulation hypothesis

Later life SEP had a positive direct effect on later life physical health in all age groups, with the effect being considerably weaker

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

Age group	Men			Women		
	N	Mean	Std. deviation	*N*	Mean	Std. deviation
Early life SEP (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
Later life 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.

	Physical health	Fibrinogen	Later life SEP (Wave 4)	Early life health
50–64 (*N* = 1738)*				
Later life SEP (Wave 4)	**0.35 (0.30 to 0.40)****	**−0.13 (−0.19 to −0.06)**		
Early life SEP (age 10)	0.04 (−0.01 to 0.09)	0.02 (−0.05 to 0.08)	**0.33 (0.28 to 0.37)**	0.04 (−0.01 to 0.09)
Early life health	**0.19 (0.15 to 0.24)**	**−0.06 (−0.12 to −0.01)**	0.02 (−0.03 to 0.07)	
Chains of risk	**0.11 (0.09 to 0.13)**	0.01 (−0.02 to 0.03)		
Accumulation	**0.38 (0.32 to 0.44)**	**−0.11 (−0.19 to −0.03)**		
Total SEP age 10	**0.15 (0.10 to 0.20)**	−0.03 (−0.09 to 0.04)		
Social drift	0.01 (−0.01 to 0.02)	−0.01 (−0.01 to 0.01)		
65–74 (*N* = 920)				
Later life SEP (Wave 4)	**0.33 (0.26 to 0.40)**	−0.03 (−0.12 to 0.07)		
Early life SEP (age 10)	0.03 (−0.04 to 0.10)	−0.06 (−0.16 to 0.03)	**0.39 (0.33 to 0.45)**	0.03 (−0.03 to 0.10)
Early life health	**0.19 (0.13 to 0.25)**	−0.01 (−0.09 to 0.07)	0.02 (−0.04 to 0.09)	
Chains of risk	**0.13 (0.09 to 0.16)**	−0.01 (−0.05 to 0.03)		
Accumulation	**0.36 (0.28 to 0.44)**	−0.09 (−0.21 to 0.03)		
Total SEP age 10	**0.16 (0.09 to 0.23)**	−0.07 (−0.17 to 0.02)		
Social drift	0.01 (−0.01 to 0.03)	−0.01 (−0.02 to 0.01)		
75+ (*N* = 702)				
Later life SEP (Wave 4)	**0.18 (0.10 to 0.27)**	**−0.14 (−0.27 to −0.02)**		
Early life SEP (age 10)	**0.11 (0.03 to 0.18)**	0.03 (−0.09 to 0.16)	**0.40 (0.33 to 0.47)**	−0.04 (−0.11 to 0.04)
Early life health	**0.13 (0.05 to 0.21)**	0.04 (−0.06 to 0.14)	−0.03 (−0.10 to 0.05)	
Chains of risk	**0.07 (0.04 to 0.11)**	**−0.06 (−0.11 to −0.01)**		
Accumulation	**0.29 (0.19 to 0.39)**	−0.11 (−0.25 to 0.04)		
Total SEP age 10	**0.18 (0.10 to 0.26)**	−0.02 (−0.14 to 0.09)		
Social drift	−0.01 (−0.02 to 0.01)	0.01 (−0.02 to 0.02)		

* All models adjusted for age, marital status, employment status, number of children and cognitive ability (all confounders from ELSA Wave 4).
** Highlighted parameters denote significant results at the 0.05 and 0.001 levels.

in the 75+ age group. The accumulation hypothesis (sum of early life and later life SEP direct effects) had the strongest contribution to later life physical health inequalities in all age groups. Later life SEP dominated the accumulation effect in participants of all age groups. 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. Similarly with physical health, the accumulation hypothesis dominated by later life SEP had the strongest contribution to later life inequalities in fibrinogen levels.

3.4. Women

3.4.1. Early life SEP, chains of risk and total early life SEP effects

In women, early life SEP had a strong positive association with later life SEP, but the association was weaker in the 75+ group. Early life SEP had a positive association with later life physical health in all age groups, with the effect being stronger in the 75+ group. The total (direct and indirect via later life SEP) effect of early life SEP was significant in all age groups and had a stronger effect relative to later life SEP in the 75+ group. In women under 75, the effect was mostly due to chains of risk, ie the path from early life SEP to later life physical health via later life SEP, but for women over 75 most of the total effect of early life SEP was direct (early life/critical period hypothesis). With respect to fibrinogen, the total effect of early life SEP was significant for women in the 50–64 age group.

3.4.2. Early life health and social drift hypothesis

Early life health was not associated with later life SEP in any age group. Due to the lack of this association, the indirect effect representing the social drift hypothesis (effect of early life health on later life health via later life SEP) was not significant for either physical health or fibrinogen, indicating that as in men, there was no support for the social drift explanation for health inequalities. We found evidence of a positive direct association between early life health and later life physical health in all age groups as well as a direct association between early life health and later life fibrinogen in the younger age group (50–64).

3.4.3. Later life SEP and accumulation hypothesis

Later life SEP was positively associated with later life physical health in all age groups with the effect being weaker in the older group. The accumulation hypothesis (early life SEP and later life SEP effects combined) had the strongest contribution to later life physical health inequalities in all age groups, with later life SEP dominating the effect in women up to 74 years old, but early life SEP in women over 75. A negative direct association between later life SEP and later life fibrinogen was observed in women under 65. The accumulation hypothesis dominated by later life SEP made the strongest contribution to later life inequalities in fibrinogen levels in women under 65.

4. Discussion

Our results extend 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 socioeconomic circumstances have a strong effect on the SEP of men and women over 50. Consistent with the early life/critical period hypothesis, we found that the effect of early life SEP extends directly until the beginning of late old age, predicting physical health 65 later, a finding that extends previous findings on midlife health (Birnie, Cooper, et al., 2011; Galobardes et al., 2004; Nandi et al., 2012). 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 both physical health and fibrinogen levels in men. Early life health had a direct effect on physical health that reached until the beginnings of late old age, but was associated with fibrinogen levels only in the younger age group (50–64). The observed association with physical health is in accordance with previous findings on midlife and early old age (Wadsworth and Kuh, 1997), but extends these to older age groups of both genders. 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

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

	Physical health	Fibrinogen	Later life SEP (Wave 4)	Early life health
50–64 (*N* = 2143)*				
Later life SEP (Wave 4)	**0.31 (0.27 to 0.36)**	**−0.15 (−0.21 to −0.09)**		
Early life SEP (age 10)	**0.12 (0.08 to 0.17)**	−0.03 (−0.09 to 0.03)	**0.34 (0.30 to 0.38)**	0.03 (−0.02 to 0.07)
Early life health	**0.20 (0.16 to 0.24)**	**−0.06 (−0.12 to −0.01)**	**0.06 (0.02 to 0.10)**	
Chains of risk	**0.10 (0.08 to 0.12)**	−0.01 (−0.03 to 0.01)		
Accumulation	**0.43 (0.38 to 0.48)**	**−0.18 (−0.26 to −0.11)**		
Total SEP age 10	**0.22 (0.18 to 0.27)**	**−0.08 (−0.14 to −0.02)**		
Social drift	**0.02 (0.01 to 0.03)**	−0.01 (−0.02 to 0.01)		
65–74 (*N* = 1084)				
Later life SEP (Wave 4)	**0.24 (0.17 to 0.30)**	−0.04 (−0.13 to 0.05)		
Early life SEP (age 10)	**0.12 (0.06 to 0.18)**	0.01 (−0.07 to 0.09)	**0.38 (0.32 to 0.43)**	−0.03 (−0.09 to 0.03)
Early life health	**0.22 (0.16 to 0.27)**	−0.08 (−0.16 to 0.01)	0.02 (−0.04 to 0.08)	
Chains of risk	**0.09 (0.06 to 0.12)**	−0.02 (−0.05 to 0.02)		
Accumulation	**0.35 (0.28 to 0.42)**	−0.04 (−0.13 to 0.06)		
Total SEP age 10	**0.21 (0.15 to 0.27)**	−0.01 (−0.09 to 0.07)		
Social drift	0.01 (−0.01 to 0.02)	−0.01 (−0.02 to 0.01)		
75+ (*N* = 971)				
Later life SEP (Wave 4)	**0.13 (0.06 to 0.20)**	−0.05 (−0.14 to 0.04)		
Early life SEP (age 10)	**0.18 (0.10 to 0.25)**	−0.05 (−0.14 to 0.04)	**0.24 (0.17 to 0.31)**	0.03 (−0.05 to 0.13)
Early life health	**0.14 (0.08 to 0.20)**	−0.07 (−0.16 to 0.02)	−0.02 (−0.09 to 0.05)	
Chains of risk	**0.03 (0.01 to 0.05)**	−0.01 (−0.04 to 0.01)		
Accumulation	**0.31 (0.22 to 0.40)**	−0.11 (−0.23 to 0.01)		
Total SEP age 10	**0.21 (0.14 to 0.28)**	−0.07 (−0.16 to 0.02)		
Social drift	−0.01 (−0.02 to 0.01)	0.01 (−0.02 to 0.02)		

* All models adjusted for age, marital status, employment status, number of children and cognitive ability (all confounders from ELSA Wave 4).
** Highlighted parameters denote significant results at the 0.05 and 0.001 levels.

causality in the association between SEP and health, with our results not supporting the social drift hypothesis. This is consistent with results from previous findings for younger age groups that have also concluded that health selection explains only a very small portion of the observed social gradient in health (Chandola et al., 2003; Krieger et al., 1997).

The other three hypothesised explanations for mechanisms underlying later life health inequalities – the chains of risk, critical period/early life and accumulation hypotheses – were supported by our results. However, a complex pattern of associations was observed and age group as well as gender differences not implied by previous findings emerged. This made the evaluation of the relative importance of each hypothesis more challenging than expected, since their effects were also health outcome dependent. First, there was considerable heterogeneity between cohorts with respect to the magnitude of the contribution of each hypothesised mechanism. For example, in women under 65, both the critical period and chains of risk hypotheses were supported and made a similar contribution to later life health inequalities, whereas in men only the chains of risk hypothesis was supported. Furthermore, 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 seemed the dominant explanation of later life inequalities in physical health. A different pattern was observed with respect to later life fibrinogen levels, where inequalities were attributed to later life SEP, with the exception of women aged 50–64 and men of the same age where the total effect of early life SEP for the former and the indirect effect of early life SEP for the latter were significant.

There are several possible explanations for the observed age group differences. 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. Members of the 75+ age group spent at least part of their childhood during the great depression of the 1930s, which had well documented effects in the living conditions of children in lower socioeconomic groups. Thus, the strong effect of early life SEP on physical health might be the manifestation of a lagged effect of the great depression of the 1930s, a finding with important implications in today's economic climate of financial austerity. Another plausible explanation concerns selection effects. Participants over 75 have on average lower SEP compared to younger groups since average living standards improved in the mid 20th century but they can also be thought of as a sample in which more selective attrition of lower SEP participants has already occurred due to the worse mortality regime they experienced. 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 on both health outcomes in this age group.

The pattern of associations between SEP and our outcomes differed considerably between men and women, a finding that has not been indicated in previous research. The most important

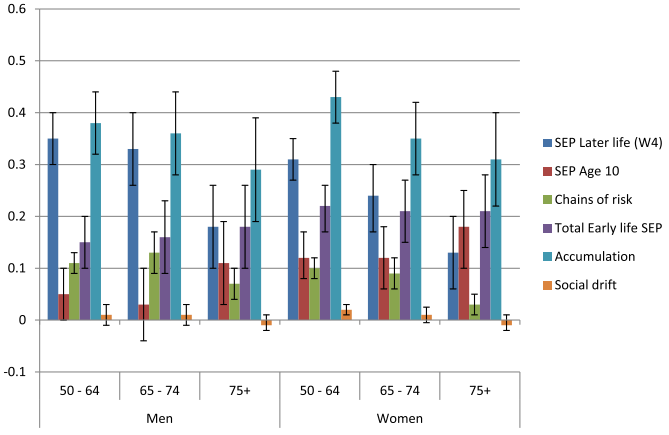


Fig. 1. Standardised parameters and 95% confidence intervals – physical health.

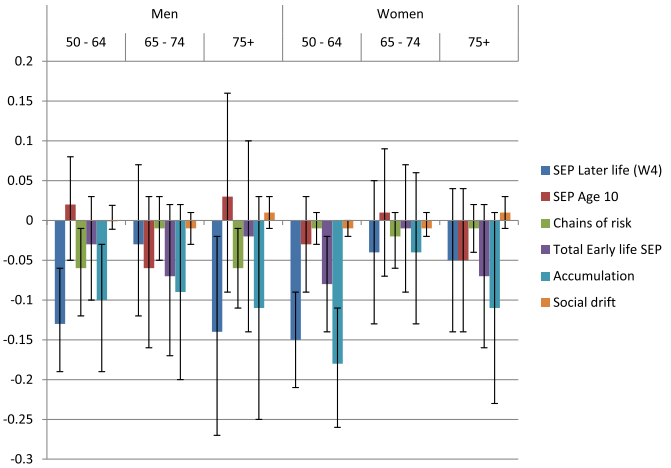


Fig. 2. Standardised parameters and 95% confidence intervals – fibrinogen.

difference concerns the effect of early life SEP, either directly or indirectly via later life SEP, which was stronger in women for both physical health and fibrinogen levels. A potential explanation for this finding is that women were exposed to more extreme circumstances during their childhood. However this explanation is not supported by our data since women had higher early life SEP in all age groups compared to men. Another potential explanation is gender differences in social mobility. However, there is evidence that men and women experienced similar upward social mobility in the UK during the 20th century (Paterson and Iannelli, 2007), making this explanation improbable. We speculate that the more pronounced effect of early life SEP on later life health in women could be related to a gender specific impact of early life SEP on health related behaviour. For this to be true, the direct link between early life socioeconomic circumstances and later life health related behaviour has to be stronger in women. Indeed, in our sample it appears that early life SEP has a stronger association with smoking and physical activity in women (results not presented here, available from corresponding author), but further research is needed to investigate this possible explanation.

Strengths of the present study include the availability of a population based dataset, the inclusion of biomarkers of later life health as outcomes 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 when interpreting our results. First, the problem of unmeasured confounding has yet to be resolved in observational settings and it has been argued that some kind of sensitivity analysis should always be presented when observational data are used (Greenland, 1996). According to the sequential ignorability assumption, for our estimated parameters to be valid no unmeasured confounders are allowed in any part of Graph 1. For example, parental characteristics, such as cognitive ability and health status were not taken into account in our model (information on these variables is not available in the ELSA) and may have introduced bias in our estimates. We attempted to capture these effects by a series of sensitivity analyses where a continuous latent 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 Electronic Supplementary Material II). We have adjusted for potential confounders of the later life SEP and health (mediator–outcome) association and the assumption of no unmeasured confounders for this part of Graph 1 is – we believe – sufficiently approximated. Despite this, we also simulated the effect of potential unmeasured confounders on this association (results presented in Electronic Supplementary III). Our results were supported even under strong mediator–outcome confounding conditions. 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 (ELSA life-course interview at Wave 3 – 2007) reported higher early life SEP compared to participants with one or more chronic illnesses, a finding in agreement with previous studies (Ben-Shlomo and Kuh, 2002; Kuh et al., 2003) 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 (Teasdale, 1983; Wenzlaff et al., 1988) we found that at the time of recall (ELSA Wave 3 life-course interview) being depressed was negatively associated with recollections of early life SEP and early life health. However, the systematic bias on recall of 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 extent 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 which 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. For example the observed cohort and gender differences in the mechanism that links life course SEP and health, as well as the different socioeconomic patterning observed in fibrinogen and physical health warrant further research. This should be attempted with appropriate analytic strategies that formally recognise the various pathways that link SEP and health as they have been suggested by life-course and social causation theories.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.socscimed.2014.02.018.

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