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Life course socioeconomic position and general and oral health in later life: Assessing the role of social causation and health selection pathways

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ABSTRACT

Objective: To examine the pathways between life course socioeconomic position (SEP) and general and oral health, assessing the role of two competing theories, social causation and health selection, on a representative sample of individuals aged 50 years and over in England.
Methods: Secondary analysis from the English Longitudinal Study of Ageing Wave 3 data (n = 8659). Structural equation models estimated the social causation pathways from childhood SEP to adult self-rated general health and total tooth loss, and the health selection pathways from childhood health to adult SEP.
Results: There were direct and indirect (primarily via education, but also adult SEP, and behavior) pathways from childhood SEP to both health outcomes in older adulthood. There was a direct pathway from childhood health to adult SEP, but no indirect pathway via education. The social causation path total effect estimate was three times larger for self-rated general health and four times larger for total tooth loss than the health selection path respective estimates.
Conclusions: The relationship between SEP and health is bidirectional, but with a clearly stronger role for the social causation pathway.

1. Introduction

Health inequalities are well established and characterized by social gradients, with lower socioeconomic position groups having higher risk of disease and lower life expectancy (Wilkinson & Marmot, 2003). The analysis of the relationship between socioeconomic position and health poses a key question -what is the direction of the association? The association between socioeconomic position (SEP) and health may occur in two competing directions: SEP may influence health, known as the social causation theory, and health may influence SEP, known as the health selection theory. The social causation theory states that people from higher SEP groups are exposed to more favorable social determinants of health, leading to better adult health; conversely people from lower SEP groups are exposed to more disadvantaged conditions increasing the disease risk in adulthood. The health selection theory states that health determines whether people move up or down through the socioeconomic hierarchy. The theory purports that healthier individuals move upwards while those less healthy move downwards (Dahl, 1996; Mulatu & Schooler, 2002).

Although over the last 50 years a considerable amount of research has focused on these theories, the direction of the association between SEP and health and the underlying pathways are still not clearly understood. Clarifying the direction of the association has considerable public policy implications. If SEP causes health differences in adulthood, policy makers should advocate policies focused on issues such as equal access to high quality education and equal employment opportunities. However, if differences in health cause changes in SEP, policy actions should be focused on improving health conditions at key life stages (e.g., in childhood), such as improving access to health care, or improving education/employment prospects for those with chronic diseases (Kröger, Pakpahan, & Hoffmann, 2015).

Comprehensively assessing the bidirectional association of SEP and health is complex. Most studies have focused on just one of these theories, either health selection (Blane, Davey Smith, & Bartley, 1993; West, 1991) or social causation (Feinstein, 1993; Williams, 1990), without comparing the size effect of each theory and using conventional statistical methods such as regression models. Although regression is a strong analytical approach to test associations, this approach does not

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enable empirical disentanglement of competing pathways between SEP and health. Alternative analytical approaches, such as structural equation models (SEM), are more appropriate in this context. SEM allows combined modelling of two pathways simultaneously enabling their direct comparison, while considering potential errors of measurements in all observed variables (dependent and independent) (Raykov & Marcoulides, 2006).

Existing evidence comparing the role of the social causation and health selection theories is scarce and has yielded mixed results. A systematic review of the competing theories by Kröger et al. (2015), found six studies that supported both social causation and health selection with no consensus on which theory has the larger effect. Moreover, most relevant research has been conducted on younger populations (aged under 50 years), where the effect of lifetime SEP on adult health might be underestimated as chronic conditions are highly prevalent in older adulthood and have high impact on people's quality of life (ONS, 2018; Petersen & Ogawa, 2005). One of the few studies that has tested both theories simultaneously in an older population using SEM is the study conducted by Warren (2009) who compared the effect of causation and selection on self-reported health, musculoskeletal health, and depression. The findings provided strong support for social causation, but no evidence for the health selection theory. In contrast, Hoffmann, Kröger, and Geyer (2019) reported a significant effect of both social causation and health selection on self-reported health and physical function on a sample of older individuals living in Europe with a stronger effect of social causation irrespective of the SEP indicator used. A Finnish study on adults aged between 17 and 66 years showed social causation playing a slightly larger role than health selection (Aittomäki, Martikainen, Laaksonen, Lahelma, & Rahkonen, 2012). However, the indicator for health was absence from work due to sickness, which cannot be used at childhood and retirement ages. A study of respondents aged 30 to 60 from the British Household Panel Study using fixed effects panel models provided no support for the social causation theory and limited evidence for the health selection theory in men. The authors suggested that indirect causation is the most likely determinant of adult health, itself determined before age 30 (Foverskov & Holm, 2016). The heterogeneity among studies may be explained by cohort differences. The pathways and mediators involved in these relationships may vary by socio-historical contexts. Studies on different populations and cohorts can therefore add valuable information on how life course SEP and health relate. With a worldwide ageing population, it is more relevant than ever to study these mechanisms in older samples.

To understand the mechanisms of the association between SEP and health it is relevant to include the time component. Health and SEP should not be understood as single events but as longitudinal phenomena. A life course perspective allows a more dynamic and nuanced understanding of the interrelationship between SEP and health, by modelling exposure to socioeconomic factors throughout different life stages. Broadly, there are two approaches to study the association between SEP and health over the life course. First, the critical periods approach posits that negative/positive events during key life stages may affect the risk of disease in later life. Second, the accumulative model approach underscores the length of exposure to protective/adverse experiences through the accumulation of relative advantages/disadvantages over specific domains (Ben-Shlomo & Kuh, 2002) The number, duration, and severity of exposures across life, would determine risk of disease.

Fig. 1 provides an illustration of the potential pathways between life course SEP and health. The model combines the insight of the social and selection theories, the life course perspective and the role of social determinants experienced across the life span. The solid arrows depict the pathways linked to social causation. The first pathway (a) it is based on the materialist mechanisms initially identified in the Black report (Black, Morris, Smith, & Towsend, 1982). Those in higher SEP are exposed to safer environments, better access to goods like housing and food, and better access to health care (Kaplan, Pamuk, Lynch, Cohen, &

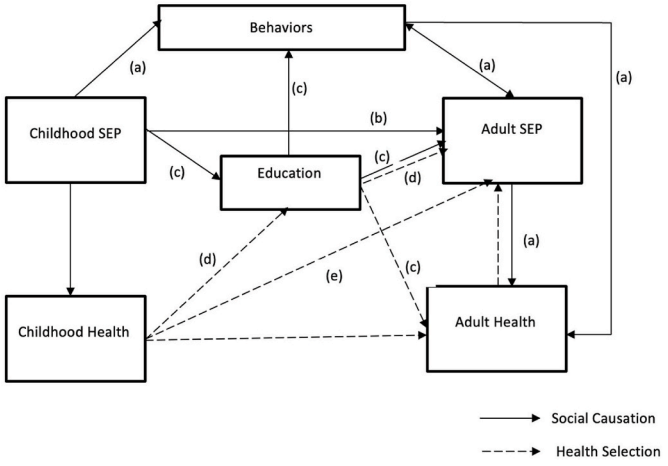

Fig. 1. Social causation and health selection pathways.

Balfour, 1996). Path b is based on the inheritance of the parental background. Born and growing up in a disadvantaged SEP increases the risk of low adult SEP (Breen & Goldthorpe, 2001). A third pathway (c) emphasizes the role of education. Education is linked to employment and income, which in turn is related to more favorable circumstances and healthier lifestyles (Conroy, Sandel, & Zuckerman, 2010). The dashed lines represent the health selection pathways. The first pathway (d) emphasizes the potential role of education; it might be that poor childhood health affects adult SEP through educational attainment. A chain reaction may occur: poor health affects education, affecting employment, income and wealth (Eiser & Vance, 2002). The second pathway (e) suggests that childhood health directly determines adult SEP and subsequently affect adult health (Manor, Matthews, & Power, 2003).

In sum, although over the last 50 years a large number of research has shown a relationship between SEP and health, the direction of the association is still not clearly understood. The scare literature testing simultaneously health selection and social causation theories has mainly focused on young-working populations disregarding how these pathways can affect older populations. Understand the difference in size effect between social causation and health selection is relevant for the design of successful health policies. The current study aims to examine the pathways between life course socioeconomic position and health, assessing two competing theories simultaneously, social causation and health selection, on a representative sample of individuals aged 50 years and over living in England. As the role of these theories may vary across different aspects of health, this study uses two distinct health outcomes: general health and oral health.

1.1. Study setting

Many countries are facing rapidly growing healthcare demands of an ageing population. This puts greater pressure on the public finances, spending on health, social care and pensions. Many contextual and biological factors can affect health in older adulthood (Bernardi, Huinink, & Settersten, 2019), at least some of the negative impacts could be prevented if understood better.

England provides an apt setting to examine the association between lifetime SEP and health among older adults for several reasons. England is going through a fast demographic transition. Projections suggest that by 2050 the proportion of individuals aged 85 and over will double (Age UK, 2019). Longer lives are associated with increased healthcare utilization and costs (de Meijer, Wouterse, Polder, & Koopmanschap, 2013). The prevalence of long-term health conditions increases with age; it is estimated that chronic conditions account for 70% of total health and social care spending in England. The cost of providing health care for a

person aged 85 years and over is three times greater than for a person aged 65 years old (Age UK, 2019). Moreover, the fast aging process occurs under conditions of limited social equality. For example, there is substantial inequality in healthy life expectancy, with differences of 70 in Richmond versus 53 in Towers Hamlets for men and 71 in Wokingham versus 56 in Manchester for women (UK Parliament, 2015). Health inequalities impact on individuals' financial capacity (Health, 2020) leaving many older adults in a situation of serious financial and social vulnerability. Understanding the pathways to healthy aging is more important than ever.

2. Methods

Data came from the English Longitudinal Study of Ageing (ELSA). ELSA is an on-going longitudinal study following the lives of a representative sample of approximately 12,000 individuals living in private households in England aged 50 and over since 2002. Data from Wave 3 (2006/07) were used with a sample of 9771 completed face-to-face interviews. Respondents were invited to participate in a separate life history interview, which retrospectively collected detailed information about their childhood and important life-time events (Marmot & Breeze, 2008).

Two outcomes, assessing different dimensions of health in older adulthood, were used. First, self-rated general health is a holistic measure of health that captures aspects of physical, mental and social well-being (Idler & Benyamini, 1997). It was assessed by the question: "Would you say that your health is … ?" (answers: very good; good; fair; bad; very bad). The sample was dichotomized into those who reported having very good/good against those reporting fair/bad/very bad health. Second, self-reported total tooth loss was assessed by the question: "In relation to oral health, which of the following applies to you"; with four possible answers that were dichotomized into dentate (only natural teeth/both natural teeth and denture) against edentate (no natural teeth and wearing denture/neither natural teeth nor denture) (Tsakos, Demakakos, Breeze, & Watt, 2011). This broad and robust measure of edentulousness reflects the accumulation of oral disease and dental treatment throughout the life course (Gilbert, Chavers, & Shelton, 2002). Childhood health was assessed in the life history interview to test the health selection theory. Participants were asked: "Please rate your health before age 16", with five possible answers that were dichotomized into poor/fair versus excellent/very good/good.

Occupational social class in childhood and adulthood was used to test the social causation theory. Respondents were asked about their childhood SEP through the question: "What was your father's main occupation when you were 14?". Childhood SEP was categorized into three groups: high SEP (managerial, professional, technical, own business), middle SEP (administrative, clerical and secretarial, skilled trade, service sector), and low SEP (manual occupations, casual jobs, unemployed, sick or disabled and lived in children's home). Parental occupation classified as armed forces, retired, refusal, not applicable, unknown, something else or other were treated as missing values (Demakakos, Marmot, & Steptoe, 2012). Occupational social class was used to measure adult SEP. Current or most recent occupation was classified using the three-category version of the UK National Statistics Socioeconomic Classification scheme (NS-SEC). Retired participants were classified according to their last occupation. Responses of "other", "not applicable" or "incomplete" were coded as missing values. Adult SEP was assessed through the highest occupation in the household, mainly because an individual occupation-based SEP approach may not be representative of the household SEP of women (Galobardes, Shaw, Lawlor, Lynch, & Davey Smith, 2006).

Education and health-related behaviors were included as potential mediators. Education has been recognized as a relevant factor that can independently influence SEP (Erikson & Goldthorpe, 2002) and health (Kawachi, Adler, & Dow, 2010; Ross & Wu, 1995). Therefore, childhood conditions may influence adult health and SEP via an indirect pathway through education. Educational attainment was measured according to the highest qualification achieved and categorized into three groups: higher qualification (university degree, other post-secondary qualification and A-level education or equivalent), secondary qualification (certificate of secondary qualification or other), no qualification (Tsakos et al., 2011). Two health-related behaviors were used: smoking and physical activity. Smoking was categorized as: current smoker, ex-smoker and non-smoker. Recreational physical activity was dichotomized into: physically active (taking part in physical activities at least once a week) and physically inactive (taking part in physical activities less often than once a week). Age, gender, employment and marital status were considered as confounders. Age was measured in years, gender was binarized into men and women, employment status was dichotomized into employed versus unemployed (including retired), marital status was dichotomized into married and non-married. The variables were coded so that a higher value was indicative of lower SEP, lower education level, poorer health or health-related damaging behavior.

Among the 9771 individuals, 3540 (36.2%) had missing data on at least one of the variables. To account for missingness, multiple imputation was conducted following the missing at random (MAR) assumption. In large samples this imputation is at least as good as listwise deletion even when the MAR assumption is violated (Graham, 2009). We used the "chained equation" approach. The imputation was informed by all the variables used in the analysis and some auxiliary variables associated with non-responses: ethnicity, limiting long-standing illness, housing tenure, type of household, institutional interview and principal carer at childhood. The number of imputations performed was 60. After imputation, inclusion/exclusion criteria were applied. Only core members aged 50 and over were included in the analysis and institutionalized participants were excluded, resulting in an analytical sample of 8659 individuals.

Cross-lagged panel models were used to assess the contribution of social causation and health selection to the association between SEP and life course health. SEM models were fitted using the software MPlus 7. A separate SEM model was constructed for each outcome, i.e. self-rated general health and total tooth loss. The relative contribution of the health selection and social causation paths was tested by a series of models with the sequential inclusion of mediators (Appendix A). All models were adjusted for age, sex, employment and marital status.

We hypothesized that the influence of childhood SEP on adult health (social causation theory) is both direct and indirect through education and health behaviors. The influence of childhood health on adult SEP (health selection theory) was hypothesized to be direct and indirect via education. Correlations are illustrated with double headed arrows. Causal paths are drawn by single headed arrows. The diagonal arrow from childhood SEP to adult health (x) reflects the direct effect social causation path; and from child health to adult SEP (y) reflects the direct effect health selection path (Appendix A, Figs. 4-6). The weighted least squares with mean and variance bias correction (WLSMV) estimation was used. Additionally, 15,000 iterations were specified to avoid statistical dependence between datasets (Kline, 2011). The root mean square error of approximation (RMSEA) and the comparative fit index (CFI) were used to assess model fit. To facilitate the comparison between both pathways (x and y) and determine which estimate was larger, the standardized regression estimates are reported.

Owing to the wide age range of the sample the models were stratified by age group. One previous study using ELSA (Ploubidis, Benova, Grundy, Laydon, & Destavola, 2014) described cohort differences in childhood and adult circumstances, reporting that younger age groups had higher mean childhood and adult SEP and better childhood and adult health. The individuals were stratified in three age groups: 50 to 64 (probably still employed); 65 to 74 (age close to retirement); 75+ (retired).

Additionally, as mentioned, we used a household SEP approach. To test the robustness of the used approach and compare the results if a

different approach is adopted, a sensitivity analysis was performed using just one person per household (randomly chosen).

3. Results

Table 1 presents the descriptive statistics of the sample. Women were slightly overrepresented (53.2%). Mean age of respondents was about 66 years (65.8, SD 11.6). Most individuals had some educational qualification (no qualifications: 32.9%) were un-employed (retired or economically inactive: 65.0%) and were married (68.5%). Additionally, 33.6% of individuals reported poor self-rated general health, and 17.9% had no natural teeth. During childhood, about 12% of individuals experienced poor general health. In terms of socioeconomic position, at childhood, most individuals were classified within the middle SEP (50.5%). However, the distribution changed at adulthood, the proportion of individuals at high SEP and low SEP increased, resulting in a dramatic decrease in the middle SEP (25.5%).

Table 2 displays the standardized regression estimates of the social causation and health selection direct and indirect pathways via education and behaviors for both outcomes. Figs. 2 and 3 illustrate the final models for self-rated health and total tooth loss respectively. Appendix B contains the Figs. 7-14 illustrating the sequential models showing the standardized estimates at every stage. All SEM models had an excellent fit to the data with RMSEA values below 0.05 and CFI values above 0.95 (Appendix C, Table 3).

Table 1
Socio-demographic characteristics and outcomes distributions of analytic sample (%) n = 8659.

Gender	
Men	46.8
Women	53.2
Age group	
50 to 64	51.9
65 to 74	25.3
74+	22.7
Age continuous: Mean (SD)	65.8 (11.6)
Childhood SEP	
Managerial/Professional (High)	30.5
Intermediate (Middle)	50.5
Routine/Manual (Low)	19.0
Adult SEP	
Managerial/Professional (High)	39.4
Intermediate (Middle)	25.5
Routine/Manual (Low)	35.1
Self-rated Health	
Good health	66.4
Poor health	33.6
Total tooth loss	
Dentate	82.1
Edentate	17.9
Education	
High degree or post-secondary qualification	36.7
Secondary qualification	30.4
No qualification	32.9
Employment status	
Employed	35.0
Unemployed	65.0
Marital Status	
Married	68.5
Non-married	31.5
Childhood self-rated health	
Good health	87.7
Poor health	12.3
Smoking	
Non-smoker	34.9
Ex-smoker	35.8
Current smoker	40.5
Physical activity	
Active	34.4
Non-active	49.7

Weighted percentages of imputed data.

Table 2
SEM social causation and health selection direct and indirect pathways standardized estimates (S.E) for adult poor self-rated general health and total tooth loss. n = 8659.

	Poor self-rated health models	Total tooth loss models
Model 1		
Social causation direct path	.155 (.015)**	.197 (.019)**
Health selection direct path	.045 (.014)*	.045 (.014)*
Model 2		
Social causation direct path	.069 (.016)**	.079 (.020)**
Health selection direct path	.031 (.013)*	.031 (.013)*
SC: Child SEP > education > adult health	.086 (.006)**	.117 (.007)**
HS: Child SRH > education > adult SEP	.013 (.007)	.013 (.007)
Social causation total effect	.155**	.196**
Health selection total effect	.044*	.044*
Model 3.1 including smoking		
Social causation direct path	.063 (.016)**	.072 (.020)**
Health selection direct path	.031 (.015)*	.030 (.015)*
SC: Child SEP > education > adult health	.083 (.006)**	.113 (.007)**
HS: Child SRH > education > adult SEP	.013 (.007)	.013 (.007)
SC: Child SEP > smoking > adult health	.004 (.002)*	.005 (.002)*
SC: Child SEP > adult SEP > smoking > adult health	.002 (<.001)**	.002 (<.001)**
SC: Child SEP > education > adult SEP > smoking > adult health	.002 (<.001)**	.004 (.001)**
Total indirect effect social causation	.091**	.121**
Total indirect effect health selection	.013	.013
Total social causation effect	.154**	.193**
Total health selection effect	044*	.043*
Model 3.2 including physical activity		
Social causation direct path	.072 (.017)**	.081 (.019)**
Health selection direct path	.030 (.013)*	.030 (.013)*
SC: Child SEP > education > adult health	.060 (.006)**	.103 (.007)**
HS: Child SRH > education > adult SEP	.016 (.008)*	.016 (.008)*
SC: Child SEP > education > physical activity > adult health	.022 (.003)**	.012 (.002)**
SC: Child SEP > adult SEP > physical activity > adult health	.003 (.001)*	.002 (.001)*
SC: Child SEP > education > adult SEP > physical activity > adult health	.004 (.002)*	.002 (.001)*
Total indirect effect social causation	.089**	.119**
Total indirect effect health selection	.016*	.016*
Total social causation effect	.161**	.200**
Total health selection effect	.046*	.046*

*p-value <0.05; ** p-value <0.001.
SC: social Causation; HS: Health Selection; SEP: socioeconomic position; SRH: self-rated health.
Social causation direct path: diagonal arrow: child SEP→adult health.
Health selection direct path: diagonal arrow: child health→adult SEP.
Model 1: model including childhood SEP, childhood self-rated health, adult SEP and adult health outcome.
Model 2: Model 1 + education included as mediator.
Model 3.1: Model 2 + smoking status included as mediator.
Model 3.2: Model 2 + physical activity included as mediator.
Model 3.1 and 3.2 illustrated in Figs. 2 and 3.
These paths are illustrated from Figs. 7-14 in Appendix B.

The main objective was to compare the coefficients that represent social causation and health selection. There were significant direct and indirect effects for both pathways. Model 1 in Table 2 shows the direct effects. Lower childhood SEP was associated with poorer self-rated health (regression coefficient: 0.155) and higher total tooth loss (regression coefficient: 0.197) in older adulthood. The health selection direct estimate was much smaller (regression coefficient: 0.045).

The inclusion of education into the models (Table 2, Model 2) considerably attenuated the health selection and social causation direct path estimates for both outcomes. However, even after accounting for education, the magnitude of the social causation estimate was more than twice as strong as the health selection estimate; the direct estimate of childhood SEP on self-rated health was 0.069 and on total tooth loss 0.079; while the direct estimate of childhood health on adult SEP was 0.031. The estimate of childhood SEP on education was 0.299 (Figs. 2

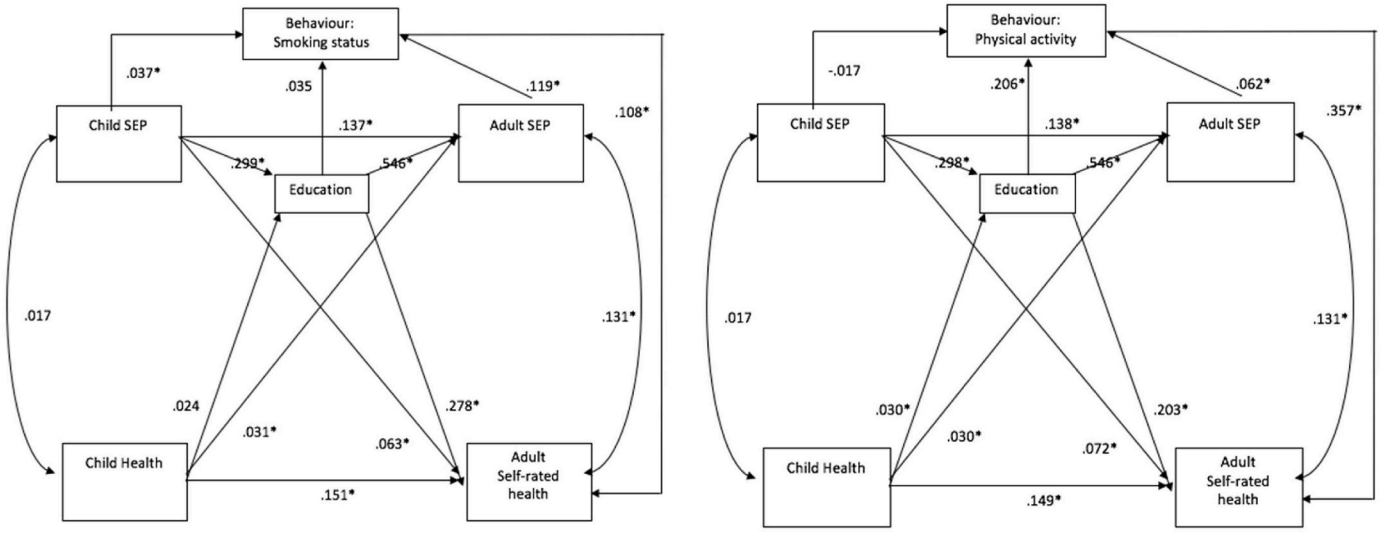

Fig. 2. Poor self-rated health Model 3.1 and Model 3.2: SEM standardized regression estimates of the pathways between childhood SEP and health and adult SEP and self-rated general health including education level and smoking status or physical activity as mediator, adjusted for age, sex, employment and marital status($p^*<0.05$).

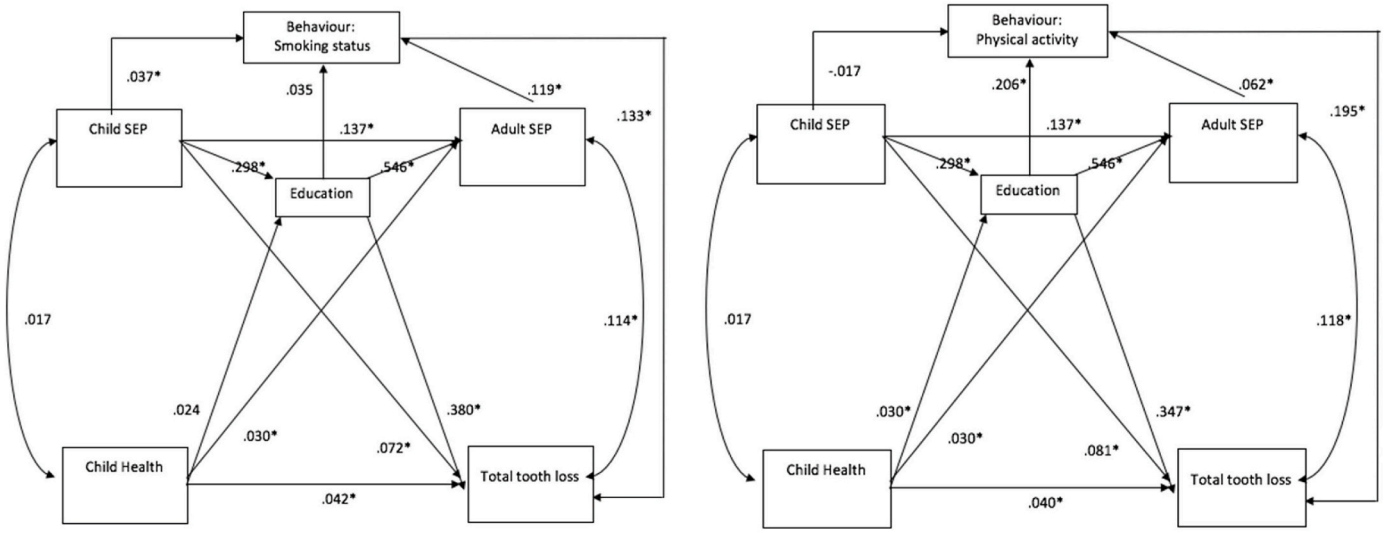

Fig. 3. Total tooth loss Model 3.1 and Model 3.2: SEM standardized regression estimates of the pathways between childhood SEP and health and adult SEP and total tooth loss including education level and smoking status or physical activity as mediator, adjusted for age, sex, employment and marital status ($p^*<0.05$).

and 3). The estimates for the pathways from education to adult health and SEP were significant and larger than the estimates of other paths in the models; overall, the estimate from education to adult SEP was 0.546 (education to self-rated health = 0.278; education tooth loss = 0.380; Figs. 2 and 3). Looking at the social causation indirect pathway via education (child SEP→education→adult health), lower childhood SEP was associated with lower educational level, which in turn was associated with poor self-rated health (regression coefficient: 0.086) and higher likelihood of total tooth loss (regression coefficient: 0.117). However, there was no statistical evidence confirming a childhood health pathway through education.

The inclusion of smoking (Table 2, Model 3.1) and physical activity (Table 2, Model 3.2) generally attenuated the estimates of the social causation direct and indirect paths via education for both self-rated health and total tooth loss. But again, social causation estimates remained larger than the health selection estimates. Several pathways through education and/or behaviors were identified. Generally, lower childhood SEP was related to less healthy behaviors, which in turn were associated with poor self-rated health and total tooth loss (Table 2, Figs. 2 and 3).

In all models, adult SEP and adult health were significantly correlated, implying that lower SEP in adulthood was associated with poor self-rated adult general health and total tooth loss. Also, childhood SEP was associated with adult SEP and child health was associated with adult health.

The stratified analysis showed no substantial differences between age group (Table 4 in Appendix D). Both theories operate in all age groups with a predominance of social causation. The results are consistent across groups, but with a stronger health selection effect in the younger group.

The SEM analysis was repeated using only one individual per household (Appendix E, Table 5). Results showed that considering only one person per household slightly underestimated the magnitude of the social causation pathway compared to the adopted analysis. However, the results showed the same direction than the used approach.

4. Discussion

This analysis simultaneously assessed two competing theories (health selection and social causation) about the association between SEP and two health outcomes, representing general and oral health. This study is one of the few studies testing both competing theories and the first to provide evidence of a social causation direct and indirect effect of lifetime SEP on oral health in older adulthood. Using a large, nationally representative sample of older adults living in households in England, the current study supports the idea that the two theories are not mutually exclusive and can operate simultaneously. On the one hand, health inequalities emerge as a consequence of social inequalities over the life course, but on the other hand, health inequalities also contribute to the formation of socioeconomic hierarchies.

When comparing both theories, the effect of the social causation path (childhood SEP ⇒ older adulthood health) was considerably stronger than the estimate of the health selection path (childhood health ⇒ adult SEP). Looking at the total effect estimates, the social causation estimate was three times larger than the health selection estimate for self-rated general health, and four times larger for total tooth loss.

Regarding the social causation theory, we showed that SEP in childhood impacts on adult self-rated general health and total tooth loss directly and indirectly through education, behavior and adult SEP. The strongest pathway was through education. Health-related behaviors were also identified as mediators, although they had a smaller mediating and direct effect on adult general and oral health than education. Regarding the health selection theory, there was a significant direct estimate of childhood health on adult SEP, even after accounting for education. Poor childhood health was associated with poor adult SEP. There was very weak evidence of an indirect estimate of childhood health on adult SEP via education.

Our findings suggest that health inequalities associated with lifetime SEP persist into older age. Most previous studies examining the relationship between SEP and health through similar analytical models but on younger populations report similar findings, supporting the coexistence of both social mobility theories. However, there is little consensus on which pathway plays the more important role (Huurre, Rahkonen, Komulainen, & Aro, 2005; Kröger et al., 2015; Palloni, Milesi, & Turner, 2009; Warren, 2009). Three previous studies have explored these competing hypotheses using similar age groups and methods. The first study, conducted by Warren (2009), aimed to quantify the effect of SEP on health and vice versa using a population cohort of American individuals aged between 18 and 65 years (Warren, 2009). In line with our findings, it provided strong evidence for a social causation effect of childhood SEP and adult SEP on adult general health; however, it reported no health selection effect while our results showed a selective effect of childhood health on adult SEP. Similarly, Ploubidis et al. (2014) using ELSA data reported a direct and indirect effect via adult SEP of early life circumstances on a latent measure of adult biomarkers, self-reported illness and physical function, but no effect of early life health on later life health via adult SEP (Ploubidis et al., 2014). This divergence may be due to different outcomes and SEP indicators. Ploubidis et al. (2014) used a latent variable including seven measures of health and biomarkers. Different health domains can be more or less related to lifetime experiences. This is in line with the conclusion postulated by Kröger et al. (2015) that the effect of the social mobility theories, social causation and health selection, might differ depending on the studied outcome. Also, Ploubidis et al. (2014) used income and wealth as SEP indicators and did not account for mediator factors such as education. The third study, conducted by Hoffmann, Kröger, and Pakpahan (2018) used retrospective data from European individuals aged 50 years, and like in our study both theories were supported with a larger effect of social causation than health selection (Hoffmann et al., 2018). Exploring these associations in different populations is relevant as the socio-historical context may influence the pathways and mediators.

This study extends previous findings giving a further insight on the direction of the association and pathways in an older population. Interpreting which life course model, whether critical period or accumulation of risk, relates with the observed pathways was beyond the scope of this study. However, this is a relevant discussion and most studies on social causation and health selection have focused their attention on this subject. Consistent with the critical period theory, we found that childhood circumstances had a direct and indirect effect on adult general and oral health suggesting an early-life critical period for the studied outcomes. Nevertheless, the accumulative model approach seems to had the prominent role in explaining socioeconomic disparities in general and oral health. The results showed evidence for direct and indirect effects via education and adult SEP with respect to both outcomes. Observed accumulation effect between lifetime SEP and adult health is in accordance with previous findings on midlife and early old age (Aittomäki et al., 2012). Therefore, it is possible that the constraint of health inequalities is caused by different mechanisms acting at the same time. Nevertheless, although the results suggest the coexistence of both, critical and accumulation model, further research is needed to disentangle the role of each model.

A key strength of this study is the use of ELSA, a large, multidisciplinary dataset including indicators of SEP in childhood and later life for a representative sample of individuals living in England aged 50 years and over. Moreover, this is the first study to analyze the role of social causation and health selection in the same model, using oral health outcomes, improving our understanding of how social determinants of health impact on different aspects of health. Another strength is the inclusion of retired participants allowing to enrich this study field by contributing to the limited literature about the effect of long-term SEP on health, extending the previous findings on younger populations to older adulthood. Lastly, the use of structural equation modelling is a strength by itself. The combination of descriptive analysis and modeling could help reach new milestones in understanding how health inequalities are created and maintained over time.

The analysis is not without limitations. First, childhood measures (SEP and health) were obtained retrospectively by asking people about their circumstances more than 35 years ago. However, studies have documented that individuals are able to appropriately report parental occupation with considerable reliability even in old age (Hout & Hastings, 2016). Indeed, Jivraj et al. (Jivraj, Goodman, Ploubidis, & de Oliveira, 2017) found similar proportions of ELSA sample members' fathers working in manual occupations as data from people roughly the same age from a 1958 British birth cohort study, the National Child Development Study, where data were collected prospectively from respondents' parents. Similarly, older individuals can accurately report their childhood health (Smith, 2009). Second, the use of occupation-based SEP can be a potential limitation for a sample of older adults that includes pensioners, as a considerable proportion of the sample is retired. We used occupation because it was the only SEP indicator available for both childhood and adulthood. Occupational class has been recognized as a reliable indicator of social inequalities in adult health in older samples, being one of the most used SEP indicators in life course research (Krieger, Williams, & Moss, 1997). Moreover, Hoffmann et al. (2019) (Hoffmann et al., 2019) showed that social causation plays a larger role in older adulthood than health selection irrespective of the SEP indicator used. Third, transferability of these findings to later born cohorts is questionable given the very different socio-political context people born since 1960 have experienced. There could well be period and cohort effects that potentially affected the balance of the social causation and health selection effects.

5. CONCLUSION and PUBLIC HEALTH IMPLICATIONS

The results of this study support the co-existence of social causation and health selection effects, but with a clearly stronger role for the former, suggesting that the impact of SEP on health is markedly larger

than the impact of health on SEP. A careful understanding of the associations between lifetime SEP and health is necessary before public health policy makers can design effective policies to reduce health inequalities across the life course. In that context, promoting public health policies targeting the social determinants of health are likely to be effective in reducing health inequalities in older adulthood, especially when applied earlier in the life course. Our results showed that education is a mediating factor in reducing the detrimental influence of the lower SEP in childhood on the health of older adults. Effective policies aimed at weakening the influence of childhood SEP on educational attainment would be desirable in their own right, however as shown in this study, they should also reduce health inequalities in older adulthood. Lastly, the results from this study provide evidence of the existence of common social determinants impacting different health domains (general and oral health).

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Human participant protection statement

This is a secondary analysis of data from ELSA. ELSA obtained ethical approval from the Multi-Centre Research Ethics Committee. NatCen processed the data so is not possible to identify participants.

PS

Concerning the possible reviewers for this manuscript, we have the following suggestions:

CRediT author statement

Alejandra Letelier: Conceptualization, Methodology, Formal analysis, Writing – original draft preparation, Visualization, Project administration. Stephen Jivraj: Methodology, Writing – review & editing, Supervision. Anja Heilmann: Conceptualization, Methodology, Writing – review & editing, Visualization, Supervision. Richard G Watt: Conceptualization, Methodology, Writing – review & editing, Supervision. Georgios Tsakos: Conceptualization, Methodology, Writing – review & editing, Supervision. All authors discussed the results and contributed to the final manuscript.

Declaration of competing interest

None. No competing interests were reported by the authors of this paper.

Appendix A. Sequential hypothesized SEM Models

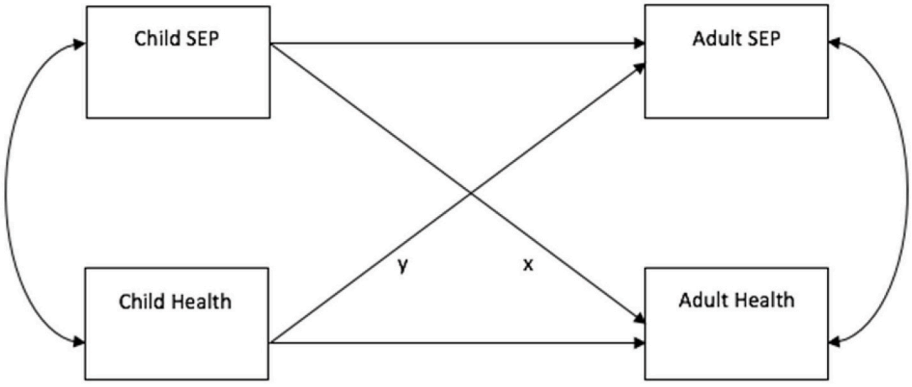

Fig. 4. SEM health selection and social causation theories (Model 1).

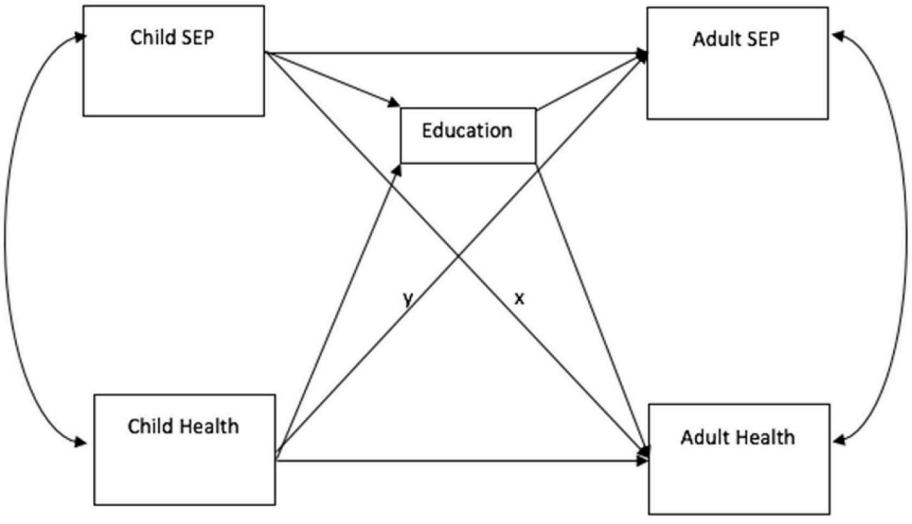

Fig. 5. SEM health selection and social causation theories including education as a mediator (Model 2).

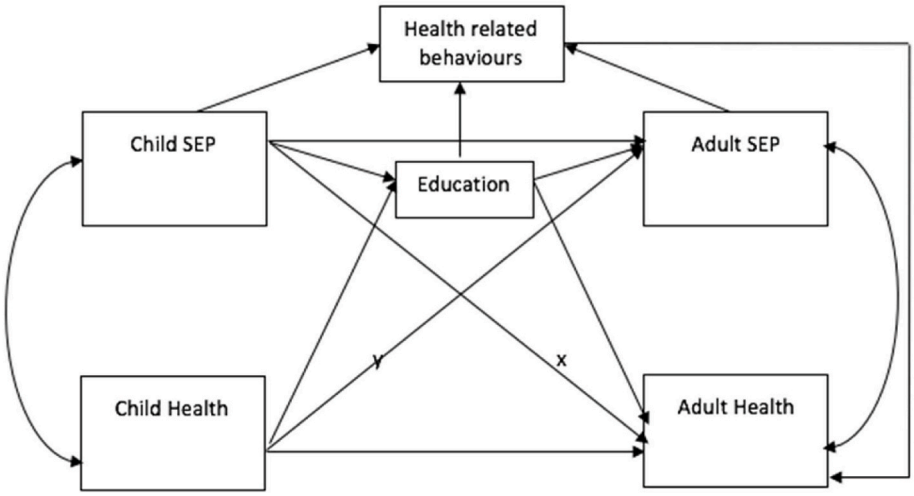

Fig. 6. SEM health selection and social causation theories including education and health-related behaviors as mediators (Model 3).

Appendix B. Sequential SEM models with regression standardized estimatesSelf-rated health

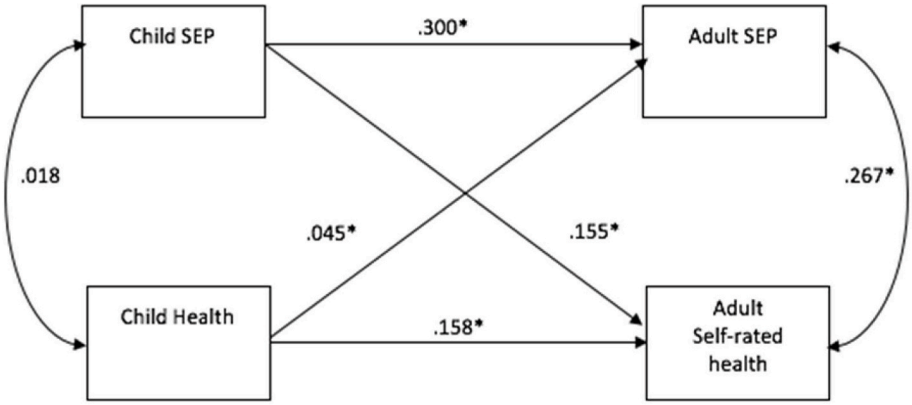

Fig. 7. Poor self-rated health Model 1: SEM standardized regression estimates of the pathways between childhood SEP and health and adult SEP and self-rated general health. Diagonals: social causation direct path (child SEP→adult health) and health selection direct path (child health→adult SEP), adjusted for age, sex, employment and marital status ($p^*<0.05$).

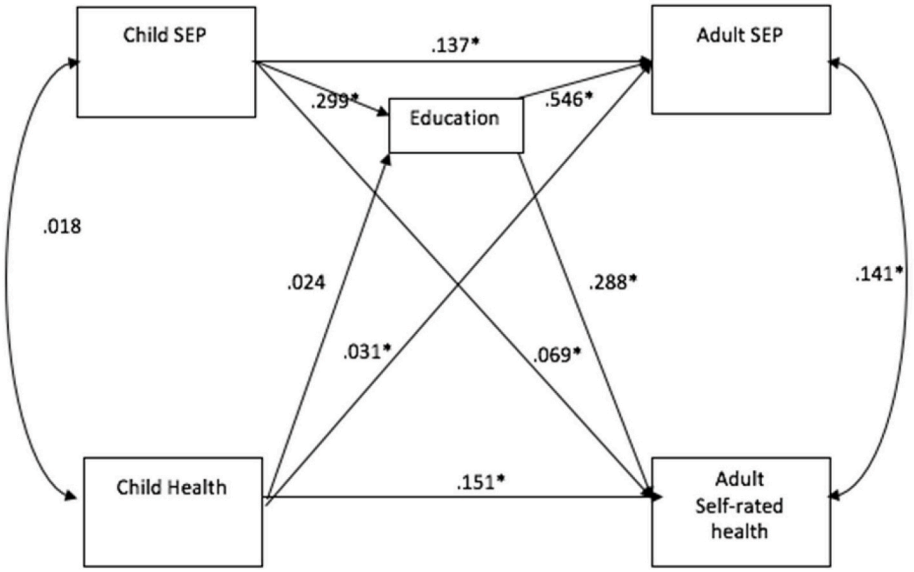

Fig. 8. Poor self-rated health Model 2: SEM standardized regression estimates of the pathways between childhood SEP and health and adult SEP and self-rated general health including education level as mediator, adjusted for age, sex, employment and marital status ($p^*<0.05$).

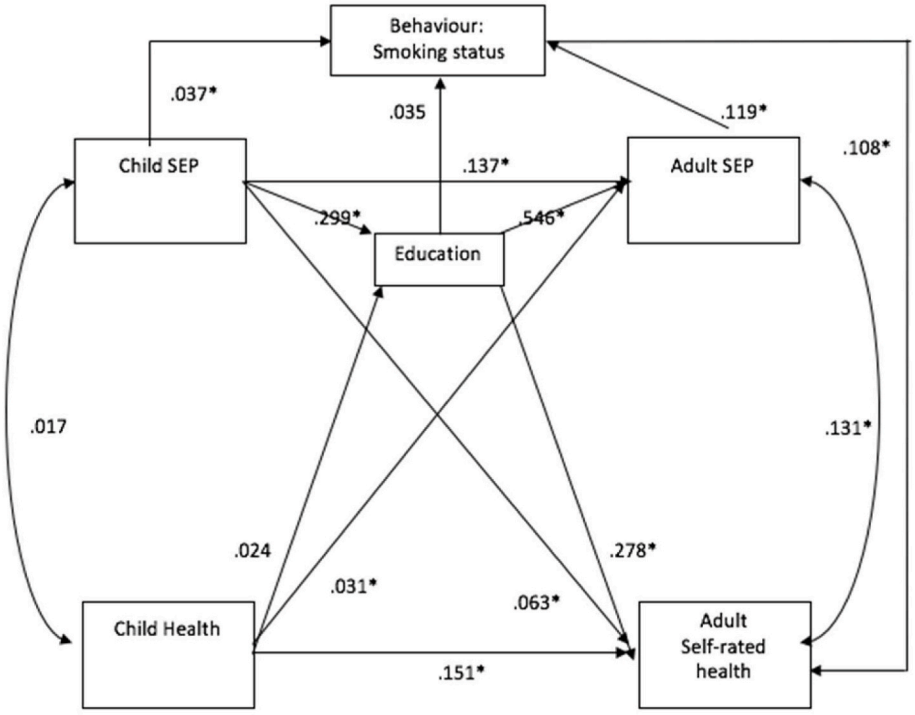

Fig. 9. Poor self-rated health Model 3.1: SEM standardized regression estimates of the pathways between childhood SEP and health and adult SEP and self-rated general health including education level and smoking status as mediator, adjusted for age, sex, employment and marital status (p*<0.05).

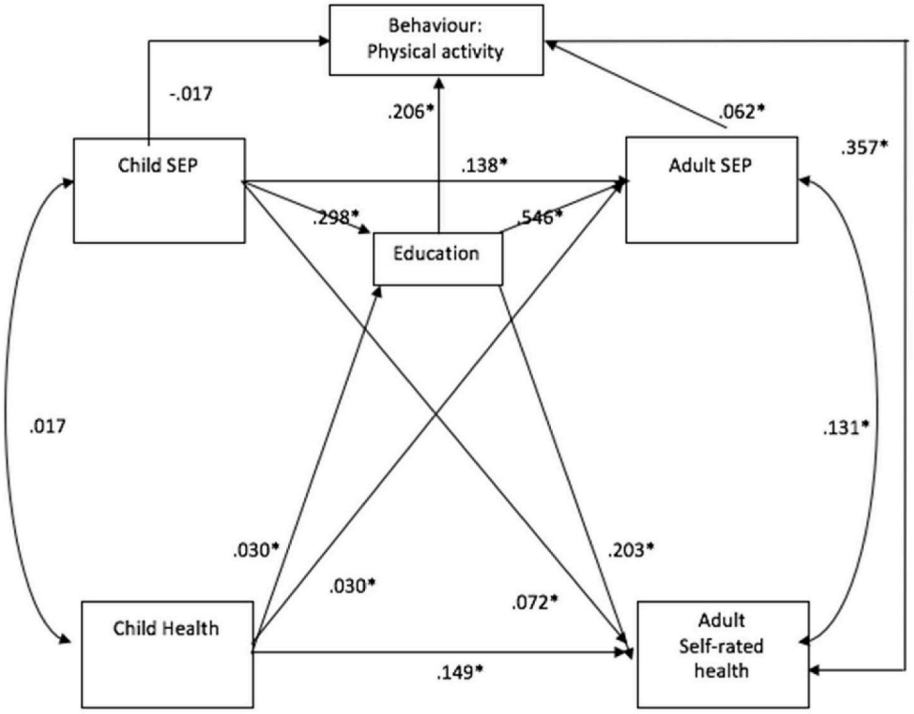

Fig. 10. Poor self-rated health Model 3.2: SEM standardized regression estimates of the pathways between childhood SEP and health and adult SEP and self-rated general health including education level and physical activity as mediator, adjusted for age, sex, employment and marital status (p*<0.05).Total tooth loss.

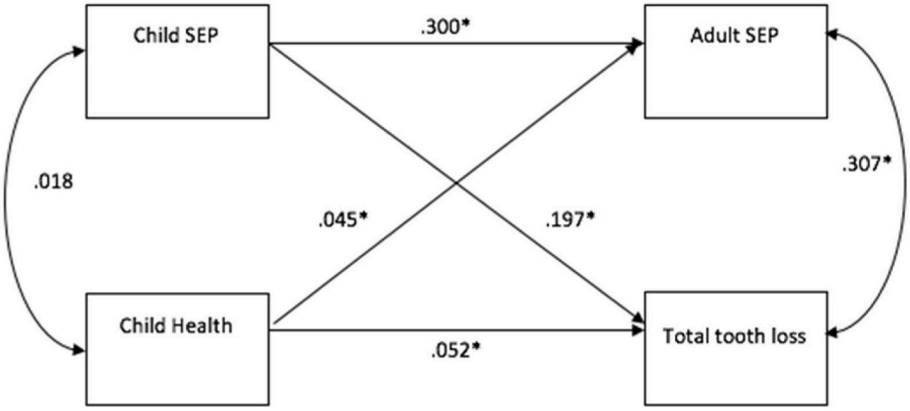

Fig. 11. Total tooth loss Model 1: SEM standardized regression estimates of the pathways between childhood SEP and health and adult SEP and total tooth loss. Diagonals: social causation direct path (child SEP→adult health) and health selection direct path (child health→adult SEP), adjusted for age, sex, employment and marital status (p*<0.05).

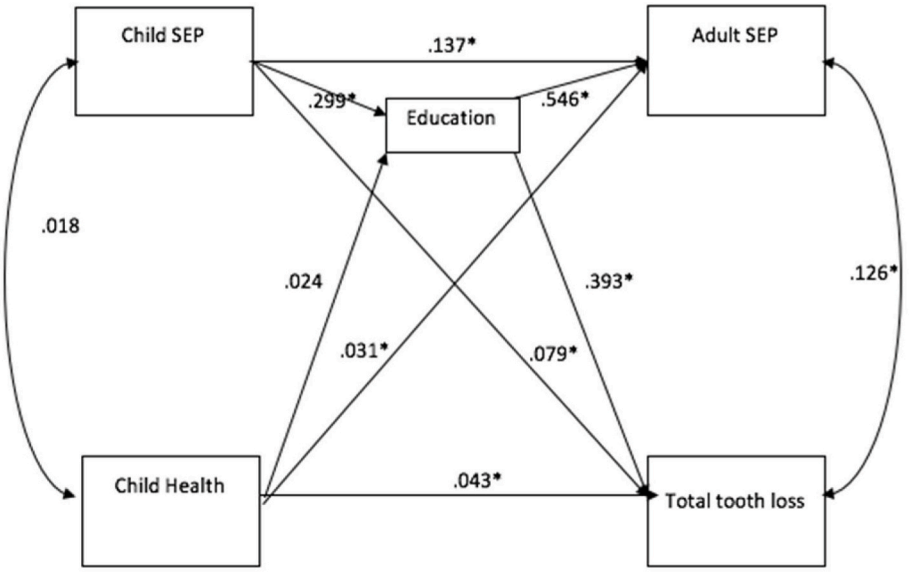

Fig. 12. Total tooth loss Model 2: SEM standardized regression estimates of the pathways between childhood SEP and health and adult SEP and total tooth loss including education level as mediator, adjusted for age, sex, employment and marital status (p*<0.05).

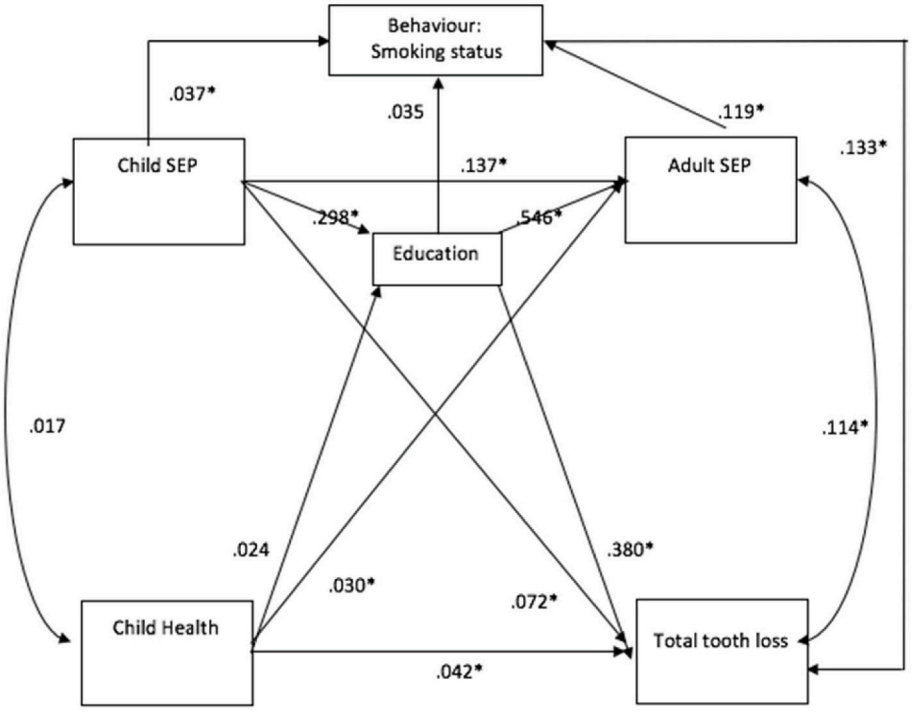

Fig. 13. Total tooth loss Model 3.1: SEM standardized regression estimates of the pathways between childhood SEP and health and adult SEP and total tooth loss including education level and smoking status as mediator, adjusted for age, sex, employment and marital status(p*<0.05).

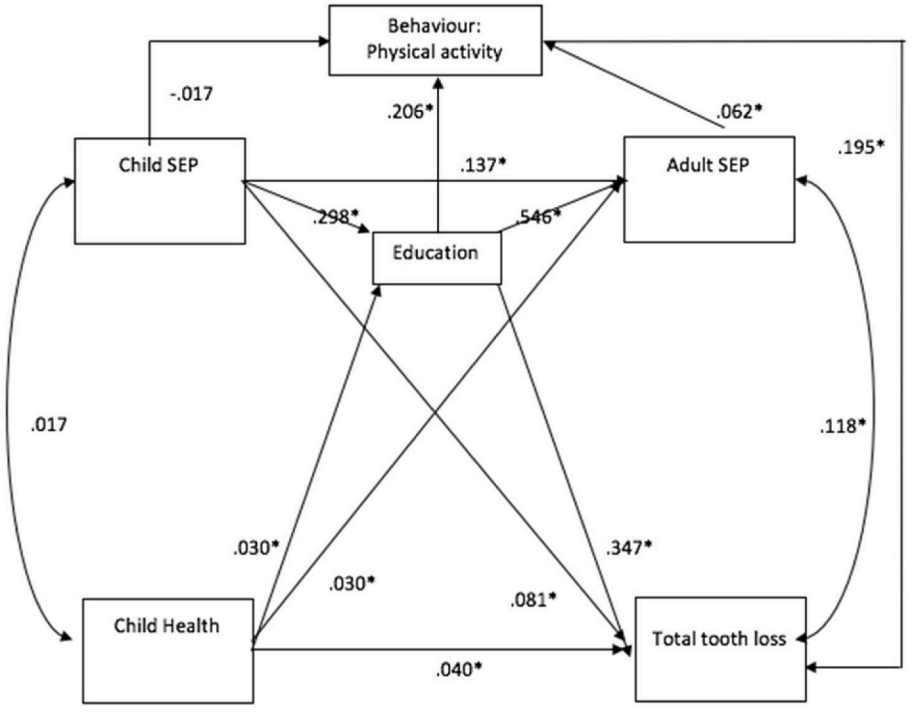

Fig. 14. Total tooth loss Model 3.2: SEM standardized regression estimates of the pathways between childhood SEP and health and adult SEP and total tooth loss including education level and physical activity as mediator, adjusted for age, sex, employment and marital status (p*<0.05).

Appendix C. Fit of the data

Table 3
SEM fit of the models for adult SEP and adult health, oral health and physical function.

		RMSEA	CFI
Self-rated health	Model 1	0.000	1.000
	Model 2: M1+educatio	0.000	1.000

(*continued on next page*)

Table 3 (*continued*)

		RMSEA	CFI
	Model 3.1: M2+smoking	0.003	1.000
	Model 3.2: M2+p.activity	0.035	0.998
Total tooth loss	Model 1	0.000	1.000
	Model 2: M1+education	0.000	1.000
	Model 3.1: M2+smoking	0.003	1.000
	Model 3.2: M2+p.activity	0.035	0.998

Appendix D. Sensitivity analysis: stratification by age group

Table 4
SEM social causation and health selection direct and indirect pathways standardized estimates (S.E) for adult poor self-rated general health and total tooth loss. n = 8659.

	Poor self-rated health models	Total tooth loss models
Age group: 50 to 64 years old		
Model 1		
Social causation direct path	.116 (.014)**	.110 (.019)**
Health selection direct path	.061 (.014)*	.061 (.014)*
Model 2		
Social causation direct path	.069 (.016)**	.072 (.020)**
Health selection direct path	.047 (.013)*	.047 (.013)*
SC: Child SEP > education > adult health	.047 (.007)**	.039 (.007)**
HS: Child SRH > education > adult SEP	.014 (.007)	.014 (.007)**
Age group: 65 to 74 years old		
Model 1		
Social causation direct path	.116 (.015)**	.125 (.019)**
Health selection direct path	.005 (.014)	.005 (.014)
Model 2		
Social causation direct path	.071 (.015)**	.085 (.020)**
Health selection direct path	.003 (.013)*	.003 (.007)*
SC: Child SEP > education > adult health	.045 (.008)*	.040 (.013)**
HS: Child SRH > education > adult SEP	.003 (.007)	.003 (.007)
Age group: 75 and older		
Model 1		
Social causation direct path	.110 (.015)**	.174 (.019)**
Health selection direct path	.034 (.014)**	.034 (.014)*
Model 2		
Social causation direct path	.079 (.015)**	.119 (.015)**
Health selection direct path	.030 (.013)*	.030 (.013)*
SC: Child SEP > education > adult health	.031 (.006)**	.055 (.006)**
HS: Child SRH > education > adult SEP	.005 (.006)	.005 (007)*

*p-value <0.05; ** p-value <0.001
SC: social Causation; HS: Health Selection; SEP: socioeconomic position; SRH: self-rated health.
Model 1: model including childhood SEP, childhood self-rated health, adult SEP and adult health outcome.
Model 2: Model 1 + education included as mediator..
These paths are illustrated in Appendix B.

Appendix E. Sensitivity analysis: Oner person per household approach

Table 5
Sensitivity analysis. SEM social causation and health selection direct and indirect pathways standardized estimates (S.E) for adult poor self-rated general health and total tooth loss. Considering one person per household (n = 5268).

	Poor self-rated health	Total tooth loss
1 person per household		
Model 1		
Social causation direct path	.121 (.009)**	.138 (.007)**
Health selection direct path	.040 (.016)***	.040 (.016)**
Model 2		
Social causation direct path	.068 (.016)**	.075 (.007)**
Health selection direct path	.031 (.013)*	.031 (.013)*
SC: Child SEP > education > adult health	.053 (.009)**	.063 (.010)**
HS: Child SRH > education > adult SEP	.009 (.015)*	.009 (.015)
Social causation total effect	.121 (.009)**	.138 (.009)**
Health selection total effect	.040 (.016)*	.040 (.017)**

*p-value <0.05; ** p-value <0.001.

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