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Mediating pathways between parental socio-economic position and allostatic load in mid-life: Findings from the 1958 British birth cohort

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ABSTRACT

Understanding how human environments affect our health by "getting under the skin" and penetrating the cells, organs and physiological systems of our bodies is a key tenet in public health research. Here, we examine the idea that early life socioeconomic position (SEP) can be biologically embodied, potentially leading to the production of health inequalities across population groups. Allostatic load (AL), a composite measure of overall physiological wear-and-tear, could allow for a better understanding of the potential biological pathways playing a role in the construction of the social gradient in adult health. We investigate the factors mediating the link between two components of parental SEP, maternal education (ME) and parental occupation (PO), and AL at 44 years. Data was used from 7573 members of the 1958 British birth cohort follow-up to age 44. AL was constructed using 14 biomarkers representing four physiological systems. We assessed the contribution of financial/materialist, psychological/psychosocial, educational, and health behaviors/BMI pathways over the life course, in mediating the associations between ME, PO and AL. ME and PO were mediated by three pathways: educational, material/financial, and health behaviors, for both men and women. A better understanding of embodiment processes leading to disease development may contribute to developing adapted public policies aiming to reduce health inequalities.

1. Introduction

Redressing the social stratification of ill health is a major concern in public health research. Classic determinants of non-communicable disease, which are mainly behavioral, are insufficient for explaining the large disparities observed in morbidity and mortality (Gallo et al., 2012). The concept of embodiment (Krieger, 2005) and biological embedding (Hertzman, 2012) are similar, and may be useful tools for formulating hypotheses on how health inequalities are produced over the lifecourse. In both concepts, how human environments affect our health by penetrating the cells, organs and physiological systems of our bodies, is a key tenet. We conceptualize embodiment as a dynamic process that summarizes how we become altered by our past experiences and are responding to the present, from how well we feel, down to the molecular modifications in our bodily structures. The process of embodiment being socially stratified (Hertzman and Boyce, 2010) may contribute to explaining the production of social gradients in health.

Growing evidence supports the idea that exposure to stressful conditions over life contributes to physiological dysregulation, subsequently translated into disease, through prolonged activation of stress response systems (McEwen and Stellar, 1993). Allostasis is the process where our body adapts to environmental challenges or stressful conditions in order to maintain physiological stability. The repeated activation of compensatory physiological mechanisms as a response to chronic stress can lead to a physiological wear-and-tear, known as allostatic load (AL) (Juster et al., 2010; McEwen and Stellar, 1993). AL has been strongly correlated with subclinical conditions, cardiovascular events, physical and functioning decline and mortality (Juster et al., 2010; Karlamangla et al., 2002, 2006). As a composite measure, AL performs as a better predictor of subsequent morbidity and mortality over and above each constituent biomarker when analyzed individually (Karlamangla et al.,

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2002). These findings suggest that AL could represent a global physiological state and perhaps even a proxy for an outcome of the embodiment process.

Growing evidence suggests that early life socioeconomic position (SEP) is a distal determinant of AL (Gruenewald et al., 2012), suggesting that poor socioeconomic circumstances early in life could set different population subgroups on life trajectories that are unfavorable for health, increasing their probability of being exposed to unhealthy environmental stressors and lifestyles. Early SEP is often measured using parental education and occupation at birth or in childhood, both of these measures being generally available in birth cohort studies. Education and occupation may operate through both similar and unique mechanisms to influence offspring health. In the current analysis, measures of parental education were limited to maternal education (ME), and occupation to paternal occupation (PO), due to variables availability and due to the historical context in which measures were assessed. Literature suggests that education and occupation do not impact health through the same pathways and should be analyzed separately (Galobardes et al., 2006, 2007). PO may affect health through: i) material resources (that determine material leaving standards), ii) work privileges (i.e. social security), iii) social standing (determining work control/autonomy and work based stress), and iv) toxic occupational exposures (Bartley et al., 1996; Galobardes et al., 2006). ME could affect health through: i) higher score of knowledge/skills and thus cultural capital, ii) material resources, iii) increasing the odds of acquiring better positions in occupation and higher income (Galobardes et al., 2006).

The lifecourse pathways linking early SEP to AL deserve to be disentangled, in particular the specific pathways involving ME and PO respectively. We aim to address this question by exploring four pathways through which ME and PO may be differentially embodied during childhood, adolescence and early adulthood leading to physiological wear-and-tear, as measured by AL. (a) A material/financial pathway: we hypothesize that living in poor material/financial circumstances could increase the risk of exposure to stressful and harmful situations relate to housing, work conditions, neighborhood, etc. (toxins, allergens, overcrowding) (Gustafsson et al., 2012; Lannero et al., 2002; Robertson et al., 2015). (b) A psychosocial/psychological pathway: parental SEP could influence parenting and the creation of a secure social environment buffering toxic stress responses, as well as the set-up of stress responses systems (Repetti et al., 2002; Shonkoff et al., 2012). (c) An educational pathway: higher parental SEP may influence educational outcomes in childhood, impact cognitive functions (Dubow et al., 2009; Kaplan et al., 2001) and later adult SEP (d) a health behaviors & BMI pathway: the accumulation of social disadvantage over life could contribute to adopt risky behaviors, impacting physiological functioning (Adler and Stewart, 2010; Stringhini et al., 2011).

We selected these four pathways based on epidemiological evidence and on empirical studies suggesting a link between SEP and each path. Lynch et al. (Lynch et al., 2000) suggested that health is the result of an accumulation of experiences and exposures due to the material world. Biological (viruses, bacteria) and chemical hazards, are more likely in more deprived homes and neighborhoods, and in some occupational statuses. An inverse relation between SEP and risky health behaviors has been largely demonstrated in empirical research (Stringhini et al., 2011). The adoption of health behaviors may be explained by complex psychological processes (e.g. self-regulation, self-efficacy, locus of control) (Bandura, 1991). Other explanations relate to social norms, tradition, and customs since lifestyle depends on social characteristics established by community social standards (Bartley, 2003). Concerning the psychosocial path, previous research (Marmot and Wilkinson, 1999) suggests that SEP may relate to health through the perception individuals have of their position in a social hierarchy. These perceptions may produce negative emotions resulting in poorer health, through psycho-neuroendocrine mechanisms (Brunner and Marmot, 1999) linked to stress responses (and/or stress-related behaviors such as smoking). Regarding the educational path, Hackman & Farah, showed in a recent review that SEP is linked with neurocognitive performance, such as language and executive function. Cognitive ability appears to be affected by poverty, especially during childhood. Different mechanisms have been suggested (e.g. cognitive stimulation, nutrition, parenting styles).

2. Materials & methods

2.1. Study population

The National Child Development Study (NCDS) is a birth cohort that includes all children born during one week in 1958 (N = 18,558) in Great Britain. Subsequent data collection was carried out on cohort members between 7y and 50y. At age 44-45y a biomedical survey was conducted including a self-reported questionnaire, physical measurements, blood and saliva samples (Power and Elliott, 2006). The sample used for this study is described in Fig. S1. Details about ethics and data are given in Supplementary Material.

2.2. Allostatic load at 44y

Among available biomarkers, we selected fourteen parameters representing four physiological systems: neuroendocrine (salivary cortisol t1 (nmol/L), salivary cortisol t1-t2 (nmol/L)); immune & inflammatory (insulin-like growth factor-1 (IGF-1 nmol/L), C-reactive protein (CRP mg/L), fibrinogen (g/L), immunoglobulin E (IgE KU/L)); the metabolic system (high density lipoprotein (HDL mmol/L), low density lipoprotein (LDL mmol/L), triglycerides (mmol/L), glycosylated hemoglobin (%)); cardiovascular & respiratory: (systolic blood pressure (SBP mmHg), diastolic blood pressure (DBP mmHg), heart rate/pulse (p/min), peak expiratory flow (L/min)). These biomarkers were chosen based on previous measures of AL (Barboza Solis et al., 2015; Seeman et al., 1997) and according to evidence of their relationship to stressful conditions over life and later morbidity and mortality (Butland et al., 2008; Kumari et al., 2013, 2011, 2008). In accordance with the most classical AL operationalization proposed by Seeman et al., our score result from the sum of fourteen parameters for which the subject was rated in the highest-risk quartile ('1' vs low risk '0') according to gender specific quartiles (Seeman et al., 1997). The high risk quartile was the top quartile (cut-off at the 75th percentile) for most biomarkers, for those where a low level confers a greater risk of poor health outcomes (HDL, salivary cortisol t1-t2, IGF1, peak expiratory flow) the first quartile (cut-off at the 25th percentile) was used. Function for individual biomarkers of AL score and measurement in the NCDS 1958 birth cohort is given in Table S1. Table S2 show the descriptive statistics and high-risk cut-points for men and women. Individuals with missing data were considered as not at risk for the missing biomarker adopting a conservative approach (maximum bias). Exclusion criteria for the analysis is shown in the flow chart (Supplementary Material, Fig. S1).

2.3. Parental SEP

We conceptualized parental SEP using ME and PO collected at birth. PO was constructed from the British Registrar General's social class system (RGSC) using mother's partner's social class (recoded

into four categories: I-professional occupations & II-intermediate occupations/III-skilled occupations (non-manual)/III-partly skilled occupations (manual)/IV-partly skilled occupations & V-unskilled occupations.), and if this was unavailable the mother's father's social class was used. The only ME measure available was self-reported and ask if mother left school after legal minimal age (14y) or if the mother left school before legal minimal age. The theoretical differences between these two measures relate to their potential mechanisms affecting health.

2.4. Early life socioeconomic and biological confounders

We selected from a questionnaire completed at birth by the participant's mother variables known to affect later health and representing socioeconomic markers (Table S1): birthweight (Barker, 2001, 2002; Gavin et al., 2012), maternal smoking during pregnancy (Jaakkola et al., 2001; Moussa et al., 2009; Raisanen et al., 2014), mother's body mass index (BMI) (Supplementary Material) (Cooper et al., 2013; Han et al., 2015; Perng et al., 2014) and mother's age at birth.

2.5. Educational pathway

Data on motor ability at 7y and educational attainment at 23y were collected. Motor ability was derived from the Copy-a-Design test at 7y, a measure of a child's capacity to reproduce geometrical figures and used in some studies as proxy of cognitive ability (Lacey et al., 2011). It ranged from 0 to 12, with higher scores relating to better perceptual motor skills and the child's ability to adapt to the school environment. Educational attainment at 23y was coded into three categories: passed the Advanced Level (A level)//passed the Ordinary Level (O levels)/No qualifications. The O levels represents the minimum leaving school age and corresponds to age 15-16y. The A level represents the final high school degree, and corresponds to age 18 years.

2.6. Psychosocial/psychological pathway

Extracted from the parental and teacher's questionnaires at ages 7, 11, and 16, we selected variables approaching parental involvement at 7y, social adjustment (Bristol Social Adjustment Guide) at 7y and family structure at 7 & 11 and malaise inventory at 23y. Parental involvement was constructed *via* five variables evaluating the time spend with the child (mum outing, dad outing, mum reading, dad reading and paternal role in the management of the child) categorized into three final items: most frequent/occasionally/hardly ever (Supplementary Material). Social adjustment is a teacher-rated measured that aims to characterize the child's behavior in school (Shepherd, 2013). Higher scores potentially indicate psychosocial and behavioral "maladjustment" (Stott, 1963). Family structure informed about parental separation at 7y and presence of a father figure at 11y. Rutter's Malaise Inventory comprise 24 yes/no items on both emotional and somatic symptoms (Rodgers et al., 1999). The individual was considered as having psychological malaise if s/he reported experiencing more than 7 out of 24 symptoms (Rutter et al., 1970).

2.7. Material/financial pathway

We selected variables characterizing material/financial deprivation at ages 7, 11 and 16, and income at 23y. From the parental and teacher's questionnaires childhood material deprivation was derived using a principal components analysis including information about housing deprivation and financial adversity (Supplementary Material). The equivalent net family income, adjusted for family size and composition with weightings from supplementary-benefit (Power et al., 1998), was also added.

2.8. Health behaviors & BMI pathway

From a self-reported questionnaire a variable containing information about physical activity at 23y, alcohol consumption at 23y (House of Commons Science and Technology Committee, 2012), and smoking status at 23y was derived. We created a three-category variable: Least at risk (0–2)/Moderately at risk (3–4)/Most at risk (5–6) (Supplementary Material) considered as a proxy for lifestyle patterns at 23y ranged from 0 to 6. BMI measured at 23y was also added to the health behaviors path to approach nutritional/diet behaviors.

2.9. Statistical analysis

To control for possible bias due to missing data, we imputed data for covariates with missing data using the multiple imputation program ICE in STATA V11 (Supplementary Material) and the analyses were stratified by sex (Supplementary Material). Descriptive statistics (Table 1) were carried out on nonimputed data. We used linear regression path modelling on imputed data to examine the relationship between parental SEP and AL (Barboza Solis et al., 2015; Israels, 1987). Path analysis allows to disentangle indirect mediating pathways. We analyzed the mediation paths between ME and AL and subsequently between PO and AL, in both men and women. Fig. 1 shows the indirect paths chosen a priori in this study. We tested all the mediating pathways, adjusting for ME, PO, confounders and mediators. Bivariate (Table S3) and multivariate linear regression results (Table S4) are shown in Supplementary Material and were carried out on imputed data. Bootstraps on 1000 random samples were ran to calculate the p values for each mediating variable (Tables S5 and S6). We conducted two sensitivity analysis. The first one for studying the stability of the AL score by identifying whether within our score, a parameter was having a stronger association relatively to the others (Supplementary Material, Table S7). The second computed a different AL score calculating a 0–1 risk score within each system, reflecting the proportion of biomarkers within the system for which the participant's values fall into the highest-risk quartile, allowing equal weight for each system. This operationalization; first suggested by (Gruenewald et al., 2012), and later by (Brooks et al., 2014) and (Carroll et al., 2015); did not impact our original results. All analyses were performed using STATA V11 taking a statistical significance level of 0.05.

3. Results

Descriptive statistics of the nonimputed sample are presented in Table 1 for the subsample (n = 3782 for men; n = 3791 for women). Figs. 2 and 3 represent the path analyses results, showing only the mediating variables explaining >5% of the total effect for men and women respectively, first between ME and AL, and second between PO and AL. The sum of all pathways are not equal to 1 due to other small or negative pathways not presented in the figures (Tables S5 and S6).

The path analyses for ME and AL show that for men the link was mainly mediated by the educational pathway [represented by motor ability at 7y (6% of the total indirect effect) and educational attainment at 23y (31% of the total indirect effect)]. The second most important pathway was denoted by a component of the health behavior path represented by BMI at 23y (13%). Finally the third path yield the role of material/financial factors captured by childhood material factors (9%). Overall, 45% of the total effect

Table 1
Descriptive statistics on the subsample for men (n = 3782) and women (n = 3791).

Variable	Sex	
	Men n [%]	Women n [%]
Allostatic load		
0	287 [7.6%]	312 [8.2%]
1	614 [16.2%]	669 [17.6%]
2	789 [20.9%]	727 [19.2%]
3	720 [19.0%]	617 [16.3%]
4	560 [14.8%]	532 [14.0%]
5	359 [9.5%]	380 [10.0%]
6	236 [6.2%]	251 [6.6%]
7	136 [3.6%]	144 [3.8%]
8	61 [1.6%]	99 [2.6%]
9	16 [0.4%]	44 [1.2%]
10	4 [0.1%]	9 [0.2%]
11	0 [0.0%]	5 [0.1%]
12	0 [0.0%]	2 [0.1%]
Maternal education		
Left school at 15 or later	1017 [26.9%]	1017 [26.8%]
Left school before 14	2765 [73.1%]	2774 [73.2%]
Paternal occupation		
I & II (professional/managerial)	739 [19.5%]	706 [18.6%]
IIINM (skilled nonmanual)	378 [10.0%]	388 [10.2%]
IIIM (skilled manual)	1902 [50.3%]	1904 [50.2%]
IV & V (semi-unskilled)	763 [20.2%]	793 [20.9%]
Birthweight		
Q1 – Low weight	792 [20.9%]	849 [22.4%]
Q2	1037 [27.4%]	1002 [26.4%]
Q3	973 [25.7%]	913 [24.1%]
Q4 – High weight	855 [22.6%]	921 [24.3%]
Missing	125 [3.3%]	106 [2.8%]
Mother smoked during pregnancy		
No	2537 [67.1%]	2517 [66.4%]
Sometimes	230 [6.1%]	215 [5.7%]
Moderately	545 [14.4%]	580 [15.3%]
Heavily	428 [11.3%]	438 [11.6%]
Missing	42 [1.1%]	41 [1.1%]
Mother's BMI		
Normal	2618 [69.2%]	2591 [68.3%]
Underweight	138 [3.6%]	180 [4.7%]
Overweight	679 [18.0%]	685 [18.1%]
Obese	148 [3.9%]	141 [3.7%]
Missing	199 [5.3%]	194 [5.1%]
Mother's age at birth		
23 years or less	976 [25.8%]	1023 [27.0%]
24–27 years	1073 [28.4%]	1043 [27.5%]
28–31 years	848 [22.4%]	821 [21.7%]
32 years or more	884 [23.4%]	900 [23.7%]
Missing	1 [0.0%]	4 [0.1%]
Parental involvement at 7		
Most Frequent	1967 [52.0%]	1917 [50.6%]
Occasionally	868 [23.0%]	916 [24.2%]
Hardly ever	548 [14.5%]	584 [15.4%]
Missing	399 [10.6%]	374 [9.9%]
Social adjustment at 7		
Q1 – Least disturbed	956 [25.3%]	1348 [35.6%]
Q2	822 [21.7%]	921 [24.3%]
Q3	829 [21.9%]	664 [17.5%]
Q4 – Most disturbed	826 [21.8%]	540 [14.2%]
Missing	349 [9.2%]	318 [8.4%]
Family structure at 7 & 11		
Presence of father figure/not divorced	3533 [93.4%]	3492 [92.1%]
No father figure or divorced	172 [4.5%]	234 [6.2%]
Missing	77 [2.0%]	65 [1.7%]
Motor ability at 7 (mean)	7.20	7.14
Missing	356 [9.4%]	326 [8.6%]
Childhood material factors at 7–16		
Q1 – Low material deprivation	635 [16.8%]	662 [17.5%]
Q2	612 [16.2%]	615 [16.2%]
Q3	638 [16.9%]	622 [16.4%]
Q4 – High material deprivation	624 [16.5%]	654 [17.3%]
Missing	1273 [33.7%]	1238 [32.7%]
Health behaviours at 23		
Least at risk	1293 [34.2%]	1243 [32.8%]
Moderately at risk	1413 [37.4%]	1550 [40.9%]
Most at risk	534 [14.1%]	556 [14.7%]
Missing	542 [14.3%]	442 [11.7%]
BMI at 23		
Normal	2542 [67.2%]	2677 [70.6%]
Underweight	74 [2.0%]	207 [5.5%]
Overweight	510 [13.5%]	341 [9.0%]
Obese	62 [1.6%]	84 [2.2%]
Missing	594 [15.7%]	482 [12.7%]
Malaise inventory at 23		
No	3123 [82.6%]	3020 [79.7%]
Yes	112 [3.0%]	326 [8.6%]
Missing	547 [14.5%]	445 [11.7%]
Educational attainment at 23		
Passed A levels	789 [20.9%]	762 [20.1%]
Passed O levels	1245 [32.9%]	1511 [39.9%]
No qualifications	1204 [31.8%]	1074 [28.3%]
Missing	544 [14.4%]	444 [11.7%]
Income at 23		
Q1 – Low income (mean = 46.6[a])	531 [14.0%]	843 [22.2%]
Q2 (mean = 93.0)	639 [16.9%]	911 [24.0%]
Q3 (mean = 131.4)	866 [22.9%]	800 [21.1%]
Q4 – High income (mean = 189.0)	1025 [27.1%]	696 [18.4%]
Missing	721 [19.1%]	541 [4.3%]

[a] Pounds per week.

between ME and AL in men remained unexplained by those paths, after adjustment for confounder and mediators. For women, 22% of the total mediated effect between ME and AL was explained by the educational path [represented by educational attainment at 23y (15%)] and by the material/financial path [represented by childhood material deprivation at 7y (7%)]. Overall, 63% of the total effect between ME and AL in women remained unexplained by those paths after adjusting for confounders and mediators.

The relationship between PO and AL in men was mainly mediated by the educational pathway [represented by educational attainment at 23y (IIIM = 17%/IV & V = 17%)]. The second path was denoted by health behaviors [represented by BMI (IIIM = 8%/IV & V = 9%)]. The third path was the material/financial [explained by childhood material deprivation at 7y (IIIM = 6%/IV & V = 9%)]. For the III skilled-manual class 68% of the total effect remained unexplained by the paths, for the IV-V semi/unskilled manual this percentage was 57%. PO for women was mainly mediated by the educational path (IIIM: 12%/IV & V: 14%), and by the health behaviors path [drawn by BMI (IIIM: 12%/IV & V: 14%)]. The third was denoted by the material/financial path [represented by childhood material deprivation (IIIM: 7%/IV & V: 9%)]. For the III skilled-manual class 58% of the total effect remained unexplained by those paths, for the IV-V semi/unskilled manual this percentage was 47%.

Multivariate results are presented in Table S4 for men and women. The full model showed that ME remained statistically linked to later AL only for women (0.39, P = 0.004). PO for both men (IIIM: 0.27; P = 0.004/IV & V: 0.32; P = 0.005) and women (IIIM: 0.29; P = 0.005/IV & V: 0.30; P = 0.016) remained statistically associated with AL. Additionally for men, the full model shows that birthweight and mother's BMI had an important independent association with later AL. Health behaviors, BMI, education level and income at 23y were also found correlated to AL after adjustment for confounder and mediators. For women, variables from early life (birthweight, mother's BMI and mother's age at birth), from childhood/adolescence (social adjustment, family structure and material deprivation) and all variables at 23y (except for income) remained independently associated with later AL.

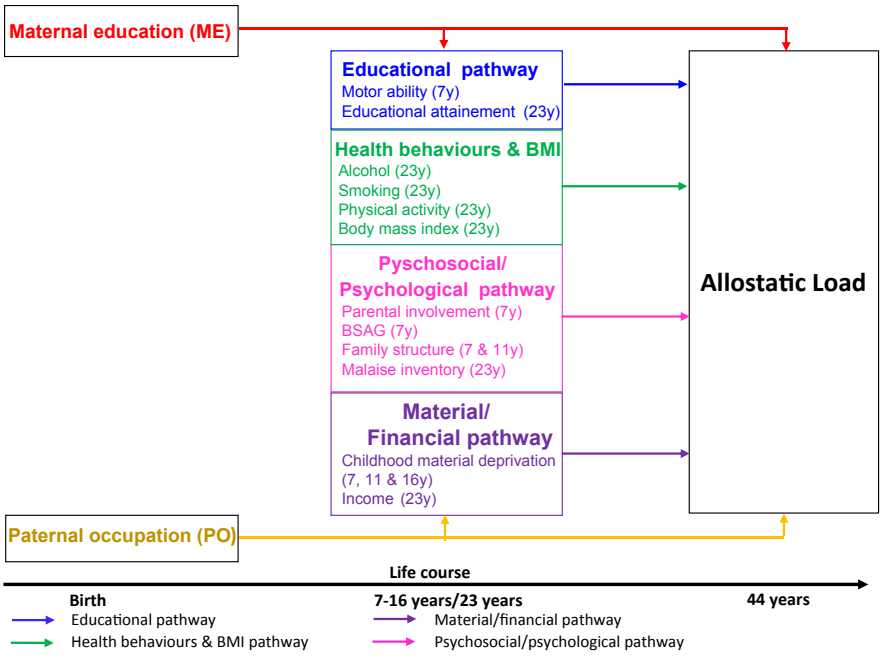
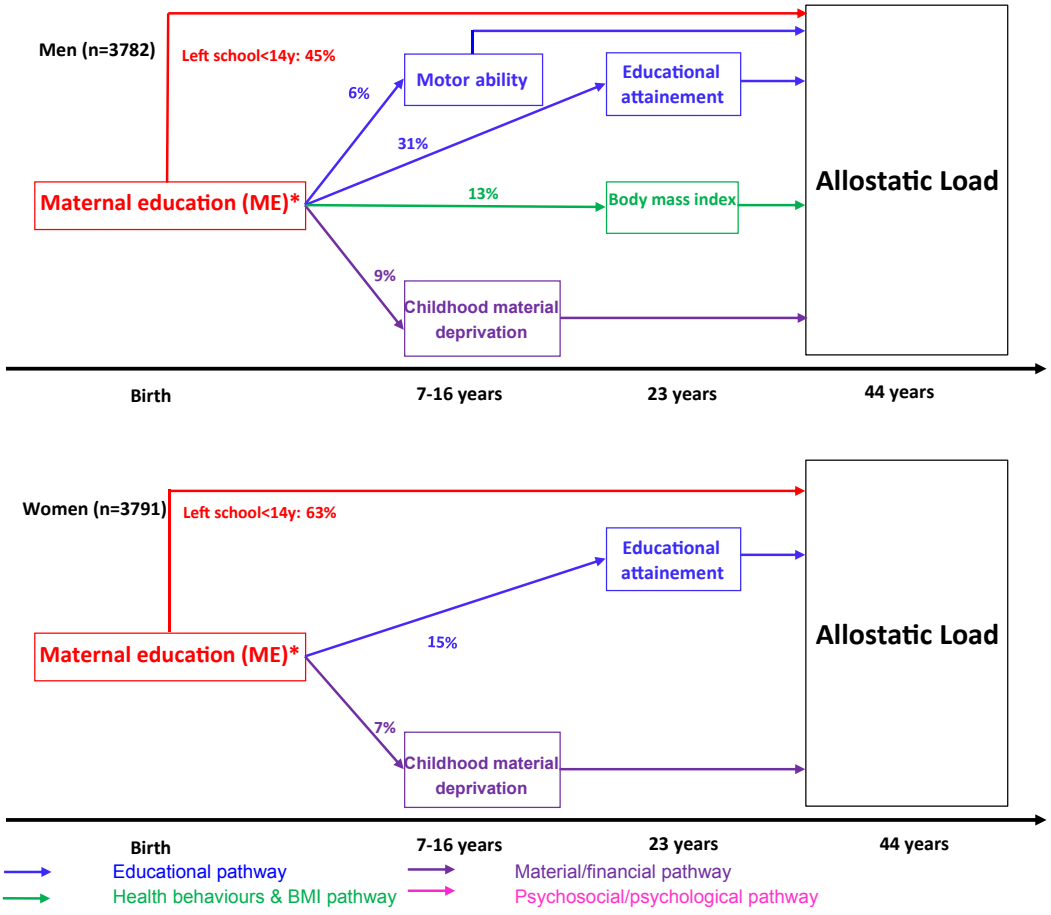


Fig. 1. Path analyses model tested in this work.



Were included in this figure only variables explaining 5% or above of the variability between ME and AL
Model adjusted for paternal occupation, birthweight, mother smoked during pregnancy, mother's BMIn mother's age at birth, parental involvement,
*The sum of all pathways are not equal to 1 due to other small or negative pathways not presented on this figure

Fig. 2. Path analysis results between ME and AL for men (n = 3782) and women (n = 3791).

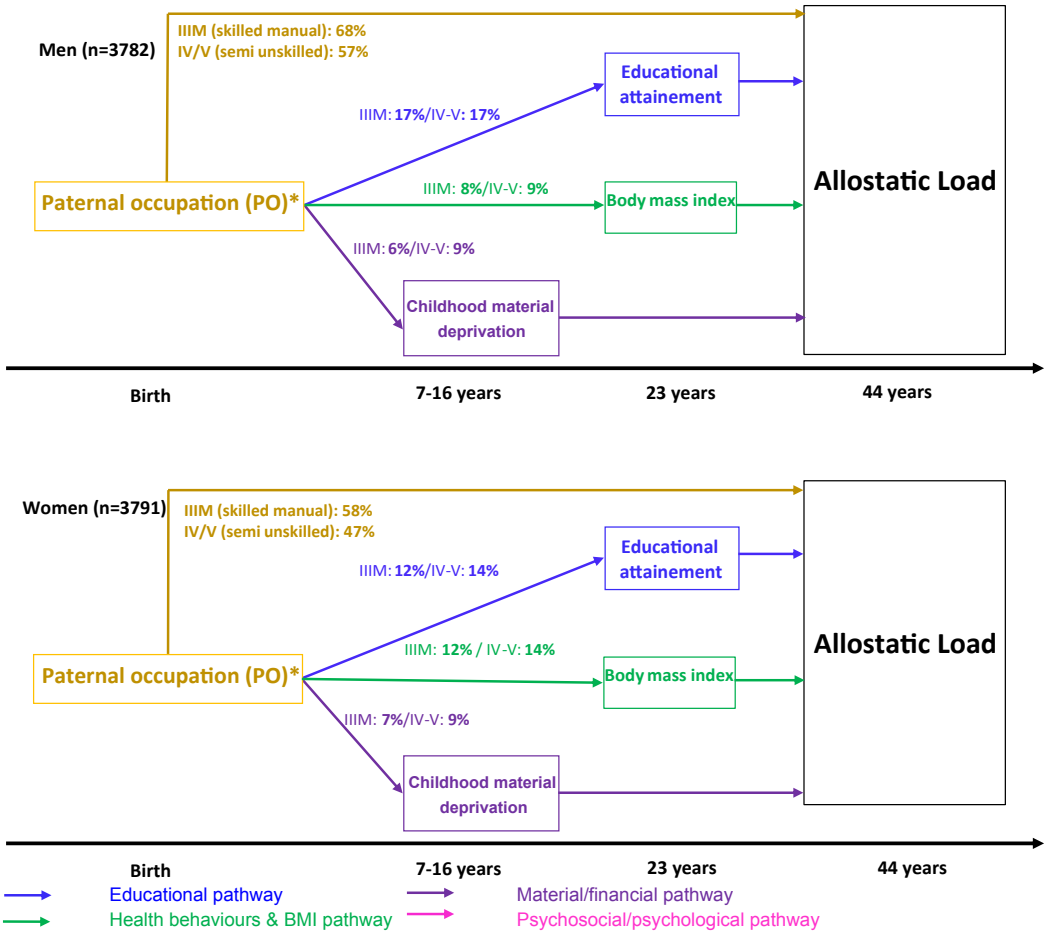


Fig. 3. Path analysis results between PO and AL for men (n = 3782) and women (n = 3791).

4. Discussion

Lower maternal education and manual paternal occupation were associated with a higher allostatic load at 44 years, mainly *via* the educational, material/financial, and health behaviors pathways for both men and women. For the relationship between ME and AL, more than 60% of the link in women and 45% in men, remained unexplained by the mediators. Around 50–60% of the relationship between PO and AL remained unexplained by the mediators in men and in women. In both relationships these pathways may represent differential early life embodiment processes or: i) an underrepresentation, underestimation of the four path we tested due to measurement errors or lack of availability of variables representing better these paths; ii) The lack of identification or omission of other possible paths (e.g. cognitive functioning and social support) (John-Henderson et al., 2015); iii) an early biological impact of parental SEP, having long lasting influence on physiology (Miller et al., 2009; Noble et al., 2015).

Up to 37% of the link between ME and AL observed in men was mediated by the educational path represented by motor ability at 7y (6%) and educational attainment at 23y (31%). The health behaviors path represented 13% of the total effect and the material/financial path 9%. For women, only two paths appeared to be important in the relationship between ME and AL: 15% was explained by educational attainment and 7% by childhood material deprivation. Concerning the link between PO and later AL, men and women showed similar patterns: the main path was educational, followed by the health behaviors and the material/financial pathways.

The most important path revealed in this study for both ME and PO was educational. This suggests a significant protective effect of a favorable health trajectory. Parental education might help and stimulate children, influencing cognitive abilities (Biedinger, 2011), favored a good adaptation to school settings, influence offspring's receptivity to health education messages, and can impact health trough the material circumstances (better jobs, higher household incomes, and better housing) (Dubow et al., 2009; McKenzie et al., 2011). We additionally found a strong correlation between motor ability at 7y and educational attainment at 23y. This provides further evidence suggesting an underlying construct cognitive characteristics, partially influenced by parental SEP early in life and having later consequences on educational attainment (Kaplan et al., 2001).

The second main path was drawn by a particular dimension of adult health behaviors: BMI. The intergenerational influences of parental SEP have been reported to affect offspring's adult obesity (Laaksonen et al., 2004), suggesting that obesity trajectories may be set early in life. Higher SEP could increase awareness concerning health messages, prevention, healthy eating, exercise and weight control efforts (Laaksonen et al., 2004). The third path was the

material/financial pathway. (Gustafsson et al., 2012) suggested that early material circumstances "might be mediated by physiological effects of their emotional and social meaning", it is also conceivable that they could impact AL increasing the probabilities of harmful exposures early in life (e.g. home/neighborhood) (Robertson et al., 2015).

When exploring the link between SEP and AL, (Robertson et al., 2015) showed that behavioral and material factors accounted for much of the association. They also revealed in a previous study that accumulated SEP across the life span was the best fitting life course model (Robertson et al., 2014). (Gruenewald et al., 2012) found that alcohol, tobacco, poor diet and low social support explained an important amount of the link. In this work we contribute to this discussion by testing diverse pathways, taking into account potential sex/gender differences and by testing two dimensions of parental SEP separately.

Other variables characterizing parental SEP were available in the NCDS. We selected ME and PO at birth, because we wanted to include in our analysis the earliest SEP markers available capturing the child social environment where s/he born into. We chose ME since literature suggests that it is a fundamental resource linked to children's health (Currie and Moretti, 2003; Gakidou et al., 2010). ME is strongly associated with improved health outcomes for children and it is inversely correlated with low birth weight and premature birth (Currie and Moretti, 2003). Moreover, at that time women were more likely to have traditional gender roles related to child-rearing in the household. Given the time she was likely to spend on household and childcare activities, we attempted to capture the mother's educational resources that may be transmitted to her children. Concerning occupation, because of gender ascribed social roles, women had less stable occupational trajectories, compared to men. Paternal occupation was the best variable that allowed us to capture the material conditions in the household at birth. In summary, our main goal was to explore the potential pathways through which parental education and occupational status may link to later health. However, due to measurement availability and historical context, the most appropriate measure available for education came from mothers, and likewise, occupation from fathers.

We observed that ME and PO were mediated by the same pathways. However, we expected to see differences concerning the importance of each path impacting AL. For instance, we hypothesized that PO could affect AL through mainly the material path and ME through mainly the educational path. However, our results were similar, suggesting that both may relate in similar ways with AL. Nevertheless, some differences should be mentioned. For ME in men, motor ability had an important role, suggesting perhaps that ME could be positively associated with cognitive ability in offspring (Bartels et al., 2009).

Concerning the path between PO and AL, there was no difference between the III-partly skilled occupations and the IV-V semi/unskilled manual categories. The RGSC is as a general measure of social standing, based on particular skills to perform an occupation. However, research on RGSC have showed a wide range of results sometimes difficult to interpret (Bartley et al., 1996). We speculate that the most important feature when it comes to occupational status and later physiological wear-and-tear is the psychosocial hypothesis of social standing. The psychosocial component may be underestimate using the RSGC, since it may better capture the material advantages bestowed by the world of work. Future research may purposes measures of occupational status based on social standing or social interactions (e.g. Cambridge scale).

The health behaviors path had an important role through BMI (except for ME in women). We hypothesize that the possible impacts of ME and PO may operate through socially stratified parental behaviors and parenting styles, which may in turn impact future life styles in men and in women. For instance, it is known that heritability of BMI is high (Han et al., 2015), and parental socioeconomic factors have been linked with offspring's BMI (Chaparro and Koupil, 2014).

The main weakness of this study is related to attrition, and selection bias, common features related to longitudinal studies. Nevertheless, it has been shown that the NCDS 1958 birth cohort remain broadly representative of the surviving cohort on key childhood and adult characteristics (Atherton et al., 2008). We compared the biomedical survey participants included in our analyses to all of those involved in the cohort at baseline to ascertain differences due to missing data. We observed that the analyzed sample have more educated mothers (27% vs 23%), have fewer fathers in the semi/unskilled category of occupational class (21% vs 24%), are more likely to have a normal BMI at 23y (80% vs 75%), being less in the 'no qualification' at 23y category (34% vs 47%), are more likely to be former smokers (30% vs 25%) or to less smoke heavily (16% vs 20%), compared to their baseline counterparts. In this sense, our results might be rather conservative and they could underestimate the effects of early SEP on AL. Therefore, we imputed the data taking the MAR assumption to preserve important aspects of the distribution, variability, and relationships between variables. The ME variable is unprecise, however it has already been independently related in the NCDS 1958 birth cohort to later AL (Barboza Solis et al., 2015). Concerning the relative minor role of the psychosocial/psychological path, it is possible that we are underestimating its real impact, due to lack of robustness, to variable availability, variable construction, and more importantly, the incapacity to completely disentangle this path from others, especially the material/financial path (Robertson et al., 2015). With family structure, parental involvement, social adjustment and the malaise inventory we tried to capture the nature of the social environment (supportive and secure) that could reverse or buffer potentially damaging stress responses. Indeed, parental warmth, and a supportive social environment, could reduce the extent of physiological wear-and-tear (Carroll et al., 2013; Shonkoff et al., 2012). Additionally, depression and anxiety, approached here with the malaise inventory, has already been found related with AL (McEwen, 2000).

Regarding the composite variable of health behavior, we want to note that the objective was to capture a general profile of lifestyles, hypothesizing that the accumulation of risky behaviors is more likely in socioeconomically deprived population subgroups. Poor population subgroups are more likely to smoke, to have a sedentary life, and to drink more heavily than their well-off counterparts (Jarvis and Wardle, 1999). It is possible that by building this variable we have misclassified individuals, and our statistical power may be decreased, leading to an underrepresentation of the health behaviors path.

We were limited by the availability of variables in our data set. There are some confounders and mediators that may be not taken into account in this analysis, that could better account for other potential mechanisms, like diet, environmental exposures, social support and cognitive skills (Gruenewald et al., 2012). We tried to capture cognitive ability by utilizing the Copy-a-Design test, a concept that aims to "recognize the principle governing different geometric forms and to reproduce them" (Schoon et al., 2002). Higher scores could represent the child's ability to follow instructions, remain concentrated and increasing their probabilities for educational success. Previous studies have used the motor skill test as a cognitive ability proxy (Lacey et al., 2011). A study showed that children whose mothers had no college education showed reduced effects of selective attention on early neural processing of speech and reduced ability to filter irrelevant information (Stevens

et al., 2009). The social, material and behavioral exposures included are self-reported, thus measurement errors are probable. Our AL score remains limited by the pragmatism of variable availability, with a strong focus on the cardiovascular system. Our only available primary mediator biomarker was cortisol, other widely used primary biomarkers are epinephrine and norepinephrine (Juster et al., 2011), however these were not available in the NCDS biomedical survey. With the two cortisol measures (t1 and t1-t2) we tried to characterize the diurnal pattern of the HPA axis. t1 was measure 45 min after wakening and t2, 3 h later (Elliott et al., 2008). A healthy HPA axis is typically characterized by high levels upon waking and a subsequent decline over the day, reaching a low point around midnight (Adam and Kumari, 2009). We tried to capture the cortisol pattern using the t1-t2 measure, however we recognize that the lack of further measures underestimate the impact of the neuroendocrine system inside the AL score.

Additionally, there is currently no consensus regarding the choice of relevant physiological systems, of biomarkers, their interactions (linear relationships), their importance in the chain of physiological stress responses, as well as their measurement, combinations, weighting and the most suitable statistical analysis (Beckie, 2012). Furthermore, as physiological responses to stress may differ according to developmental stage over time, measures of AL may differ in terms of markers and risk thresholds (Barboza Solis et al., 2015). However, the two sensitivity analysis already mentioned showed the stability of the results. Concerning sex stratification, recent population-based studies suggest sex/gender differences in several biomarkers (Goldman et al., 2004; Lakoski et al., 2006), and a study has found sex differences in AL (Yang and Kozloski, 2011). Even if the results do not show large differences by sex, we consider these potential sexual dimorphism should be taking into account in the analyses.

Regarding the significance of each pathway, we used bootstraps on 1000 random samples, to calculate the p values for each mediating variable. However, the significance at 5% was not the main criteria for considering variable as clinically or epidemiologically "relevant" or significant, since we had a large sample and in these cases the statistical significance does not provide information on clinical relevance. It seems to us that in this case we should analyze the change in effect size (Chavalarias et al., 2016). Therefore, we decided to present the percentage for which each variable explained at least 5% of the total variability (Tables S5 and S6).

Despite these limitations, this study has a number of strengths. We used a longitudinal population-based study collecting data prospectively across the life span. This study is further strengthened by analyzing separately two concepts of parental SEP by sex, exploring potential differences in the mechanisms. These findings enlighten us on the important relationship between education and physiological burden. Reducing inequalities in education could contribute to targeting population subgroups, less likely to be educated and at risk of developing physiological wear-and tear. Moreover, identifying sets of biomarkers for capturing embodiment processes early in life may be useful for conceptualizing and assessing early preventive intervention before the initiation of deleterious health trajectories.

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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.2016.07.031.

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