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When in the lifecourse? Socioeconomic position across the lifecourse and biological health score

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ABSTRACT

Purpose: Educational attainment is associated with multiphysiological wear and tear. However, associations with measures of socioeconomic position (SEP) across different life-stages are not established.
Methods: Using regression models and data from 8105 participants from the UK Household Longitudinal Study (*Understanding Society*), we examined associations of lifecourse SEP with an overall biological health score (BHS). BHS is broader than usual measures of biological 'wear and tear' and is based on six physiological subsystems (endocrine, metabolic, cardiovascular, inflammatory/immune, liver, and kidney), with higher scores indicating worse health. Lifecourse SEP was based on respondents' parental, first, and most recent occupations.
Results: Associations with SEP at all life-stages demonstrated higher BHS with increasing disadvantage (e.g. slope index of inequality (SII) (95 % CI) for most recent SEP: 0.04 (0.02, 0.06)). There was little difference in the magnitude of associations for SEP measured at each life-stage. Cumulative disadvantage across the lifecourse showed a stepped association with increasing BHS (SII (95 % CI): 0.05 (0.04, 0.07)). Associations were largely driven by metabolic, cardiovascular, and inflammatory systems.
Conclusion: Our results suggest that disadvantaged SEP across the lifecourse contributes cumulatively to poorer biological health, highlighting that every life-stage should be a target for public health policies and intervention.

What is already known on this topic

- Existing studies have reported that measures of socioeconomic disadvantage at one time point are associated with multi-physiological measures of 'wear and tear' based on biomarkers.

What this study adds

- This research investigates social embodiment theory of social determinants of health by exploring associations of occupational socioeconomic position at three different life-stages with a comprehensive biological health score.
- Results indicate that disadvantage across the life course is associated cumulatively with greater biological ageing and the magnitude of associations at each life-stage is very similar.

How this study might affect research, practice or policy

- Our findings suggest that the social-to-biological processes leading to health inequalities are evident at all points of the lifecourse supporting the theory of social embodiment.
- Interventions to address socioeconomic disadvantage at any life-stage may have the potential to ameliorate accelerated biological ageing to a similar extent.

Introduction

The raised prevalence and incidence of poor health in socioeconomically disadvantaged groups is almost universally observed. For example, a large multi-cohort meta-analysis demonstrated higher mortality in disadvantaged versus more affluent individuals, even after adjusting for 25x25 known risk factors [1]. Evidence also supports socioeconomic inequalities in measures of biological ageing conceptualised as perturbations in

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multidimensional [2] and multiphysiological [3] processes. The best-known multisystem score, allostatic load (AL) [4], captures physiological wear-and-tear mainly related to stress response and has been consistently associated with socioeconomic position (SEP) [4–8] and mortality [9]. However, social-biological associations may be wider ranging than this [10], leading to the theory of the social embodiment of health inequalities [11]. This asserts that people's environments (physical, social, economic) affect all biological systems through dynamic processes across the lifecourse, reflecting interactions between the social environment and biological systems with complex feedback loops, and has led to the development of broader measures of biological health,[12,13] building on AL to include additional key measures of physiological functioning. As well key AL components involving the Hypothalamic-Pituitary-Adrenal (HPA) axis and Sympathetic Nervous System (SNS) [9], the biological health score (BHS) includes liver and kidney function, which are also associated with physiological wear-and-tear but not due to stress response [12]. To our knowledge, BHS has only been examined in two studies, which report associations with educational attainment, particularly for inflammatory and metabolic physiological subcomponents [12], and with all cause and cause-specific mortality and heart disease incidence [13]. .

A number of SEP measures are associated with multiphysiological system scores, including parental [7] and own educational attainment [8], occupational class,[6,14] and income and wealth [5], with education-related differences in BHS apparent from early adulthood [12]. However, while there is evidence for the association of lifecourse SEP and adult AL, [15,16] it is unclear whether similar associations hold for BHS and its sub-components. We examine individual and cumulative associations of SEP at three life-stages based on: (i) early life, (ii) first and (iii) current occupational position with BHS and consider which physiological BHS subcomponents are associated with lifecourse SEP. We hypothesise that SEP at every life-stage is associated individually and cumulatively with the BHS measured in adulthood.

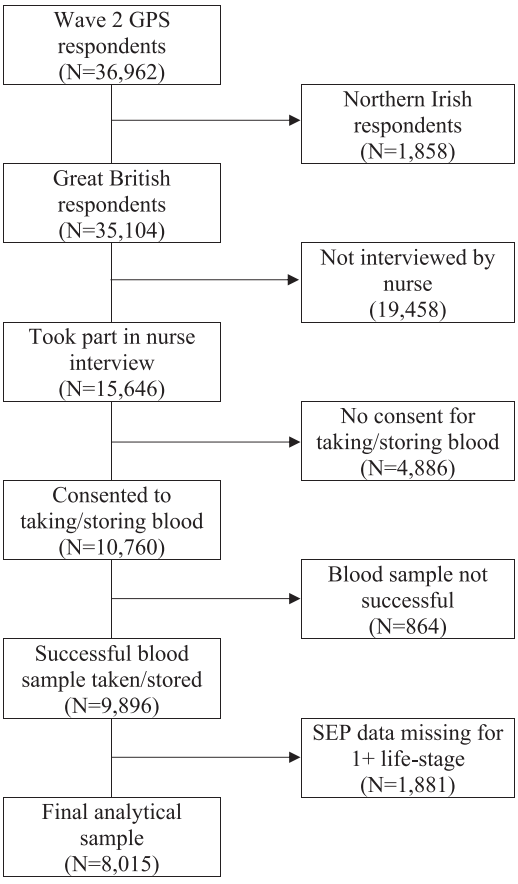

Fig. 1. Derivation of analytical sample.

Methods

Analyses are based on data from *Understanding Society*, the UK Household Longitudinal Study (UKHLS) [17], details of which have been reported previously [18]. The UKHLS is a longitudinal survey of 40,000 households in England, Scotland, Wales and Northern Ireland, with data currently available from thirteen annual collection waves. UKHLS combines four samples [19], the largest of which, the General Population Sample (GPS), is used here. The GPS is a representative probability sample based on households drawn randomly from stratified clustered samples of postcode sectors in England, Wales and Scotland and postcode sectors in Northern Ireland. Wave 2 data collection (2010–11) included a Nurse Health Assessment [20,21] approximately 5 months after the main interview and was restricted to non-pregnant respondents aged 16 + who had completed the main interview in English. The Nurse Health Assessment was restricted to participants from England, Wales, and Scotland. In 2011 81 % of the sample were randomly selected for the interview due to shortages of qualified nurse interviewers. Of those selected for the nurse interview, 58 % took part and 69 % consented to and provided a successful venous blood sample (Fig. 1). Blood samples were posted to a laboratory to be processed, aliquoted and frozen and subsequently 20 biomarkers were produced, according to the Standard Operating Procedures by HCPC Registered Biomedical Scientists with internal and external Quality Controls systems employed. Full details for each biomarker are available in the user guide [22] and are summarised in Appendix Table A1.

Biological health scores

BHS were based on 15 or 16 biomarkers for women and men respectively [12]: DHEA-S, testosterone (men only), glycated haemoglobin, HDL cholesterol, total cholesterol, triglycerides, systolic & diastolic blood pressure (adjusted for medication use), pulse, C-reactive protein, fibrinogen, insulin-like growth factor-1 (IGF-1), alanine transaminase, aspartate transaminase, gamma glutamyltransferase, and creatinine. For each biomarker, individuals were identified as "at-risk" if they were in the highest or lowest quartile for their sex and age group (<40, 40-, 50-, 60-, 70 +) for indicators where a high or low score respectively is associated with poor health (see Appendix Table A2 for details). Individuals' scores were calculated by summing the number of biomarkers in the at-risk quartile and dividing by the total number of biomarkers of interest to obtain a score between 0 and 1. Overall BHS were based on all biomarkers while individual physiological subcomponent health scores were based on specific subsets of biomarkers: endocrine (DHEA-S, testosterone), metabolic (glycated haemoglobin, HDL cholesterol, total cholesterol, triglycerides), cardiovascular (systolic blood pressure, diastolic blood pressure, pulse), inflammatory/immune (C-reactive protein, fibrinogen, IGF-1), liver (aspartate transaminase, gamma glutamyltransferase), and kidney (creatinine). In all cases higher scores indicate poorer outcomes.

Socioeconomic position

SEP at three life-stages was based on own or parental occupation. Parental SEP was derived from father's occupation if available or mother's occupation otherwise. Respondent's first SEP was based on their first occupation after leaving school and current SEP was derived from their occupation recorded at interview or, if retired or currently not working, their last recorded occupation. SEP at all timepoints was coded using five category NS-SEC [23] (management/professional; intermediate; small employers/own account; lower supervisory/technical; semi-routine/routine/never worked). Two commonly used [24–26] cumulative lifecourse SEP measures were also derived from parental, first and current occupations. Cumulative disadvantage was based on

the number of occupations (0, 1, 2, or 3) coded as lower supervisory/technical or semi-routine/routine/never worked and cumulative advantage was based on the number coded as management/professional roles. For brevity we present results for disadvantage across the lifecourse; results for cumulative advantage demonstrated comparable opposite associations.

Statistical analysis

Analyses are based on respondents with complete data on all SEP measures and the outcome of interest. The size of SEP categories at different life-stages varied, making direct comparison of their respective associations with BHS difficult. We therefore derived an Index of Inequality [27] for each measure, putting them all on the same scale and reducing the influence of extremes in the distribution of respondents in each category. The Index of Inequality uses the cumulative proportion ranking of the study population, producing a score between 0 and 1 (the lowest and highest possible respectively) based on the midpoint of the proportion of the population in each category. The Slope Index of Inequality (SII) for each SEP measure was obtained by regressing each BHS on the corresponding Index of Inequality and represents the difference in BHS comparing those with the least versus most favourable SEP. Several BHS models were fitted: (i) unadjusted for each individual life-stage and cumulative SEP measure separately; (ii) for each individual life-stage and cumulative SEP measure separately, adjusted for sex and age group; and (iii) including all three individual life-stage SEP measures simultaneously, adjusted for sex and age group. We adjusted study-provided inverse probability weights for GPS sample inclusion, to create weights for the analytical models employed here. We modelled non-response to wave 1, wave 2, nurse visit participation, and giving a viable blood sample by having a full set of covariates, and combined these probabilities with the provided weights. The models therefore account for unequal selection into the study, differential nonresponse, and covariate data availability, to estimate population level inference. Results are therefore generalisable to the UK population in 2009.

In sensitivity analyses we repeated analyses of physiological subcomponent health scores on the subset of respondents with complete data for all subcomponents. We also repeated analyses restricted to respondents aged < 65, to account for reduced salience of occupation in post-retirement groups and the increasing importance of selective mortality [14]. We also excluded respondents who didn't work, as this group is likely to include those unable to work due to ill health. In addition, analyses of inflammatory health score were repeated (i) excluding C-reactive protein values over 10, as these may represent recent infection rather than chronic processes [28], and (ii) excluding IGF-1 completely, as this biomarker is used to represent a number of physiological processes. Finally analyses of endocrine health were repeated adjusting for the time at which blood was taken to allow for diurnal differences in testosterone levels. Results from all sensitivity analyses were very similar to those presented here (see Appendix for details). Analyses were performed using Stata v17.0 [29]. .

Results

In total 15,646 GPS respondents took part in the Nurse Health Assessment, of whom 9896 (63.2 %) had successful blood samples taken. Characteristics of respondents who had complete data for SEP and at least one biomarker (N = 8015) were very similar to the full sample of Nurse Health Assessment respondents (Table 1). The availability of BHS among respondents with complete SEP varied across the different physiological subcomponents (endocrine: N = 7724 (96 %); metabolic: N = 7242 (90 %); cardiovascular: N = 6658 (83 %); inflammatory/immune: N = 7420 (93 %); liver: N = 7455 (93 %); kidney: N = 7812 (98 %)) and 5579 (70 %) respondents had complete data on overall BHS.

Fig. 2 presents the SEP biographies of respondents across the three life-stages. The figure is composed of horizontal lines, each representing SEP transitions between life-stages for an individual. Changes in colour represent changes in SEP as shown in the legend and demonstrate considerable variation across the lifecourse. Around 20 % of respondents were in the same SEP group across the three life-stages, 20 % moved down, 30 % moved up, and the remainder fluctuated.

Table 1
Characteristics of GPS and Nurse Health Assessment respondents with complete and incomplete SEP data.

	Analytical sample (N = 8015)	Respondents taking part in wave 2 nurse interview (N = 15,646)
% female	50.4	50.4
Mean (SD) age	49.9 (17.5)	48.9 (18.4)
% management/professional parental SEP	27.5	27.6
% management/professional first SEP	17.5	17.0
% management/professional current SEP	35.5	34.0
Mean (SD) overall BHS	0.25 (0.16)	0.25 (0.16)
Mean (SD) endocrine BHS	0.26 (0.38)	0.26 (0.38)
Mean (SD) metabolic BHS	0.25 (0.26)	0.25 (0.26)
Mean (SD) cardiovascular BHS	0.24 (0.31)	0.24 (0.30)
Mean (SD) inflammatory/immune BHS	0.25 (0.29)	0.25 (0.30)
Mean (SD) liver BHS	0.25 (0.33)	0.25 (0.33)
Mean (SD) kidney BHS	0.23 (0.42)	0.23 (0.42)

Fig. 3 shows age- and sex-adjusted means (95 % confidence interval (CI)) for overall BHS according to parental, first, most recent. and cumulative (disadvantaged) SEP. For all individual life-stage SEP measures BHS was greater in respondents with lower SEP. Results for cumulative SEP also showed a clear stepwise increase with higher BHS in individuals with greater disadvantage across the lifecourse. Differences in BHS according to individual life-stage SEP and cumulative SEP are presented in Table 2 along with associated SIIs. Unadjusted SIIs indicate that respondents with the lowest individual life-stage SEP had mean BHS around 0.04/0.05 greater than those with the highest SEP (e.g., SII (95 % CI) for most recent SEP: 0.04 (0.02, 0.06)). Adjustment for age and sex had no impact and associations with all three individual life-stage SEP measures were very similar, albeit slightly attenuated, in reciprocally adjusted models. Notably, associations were very similar for SEP at each life-stage and there was a strong association of increasing BHS with increasing cumulative disadvantage, which remained after adjustment for age and sex (0.05 (0.04, 0.07)).

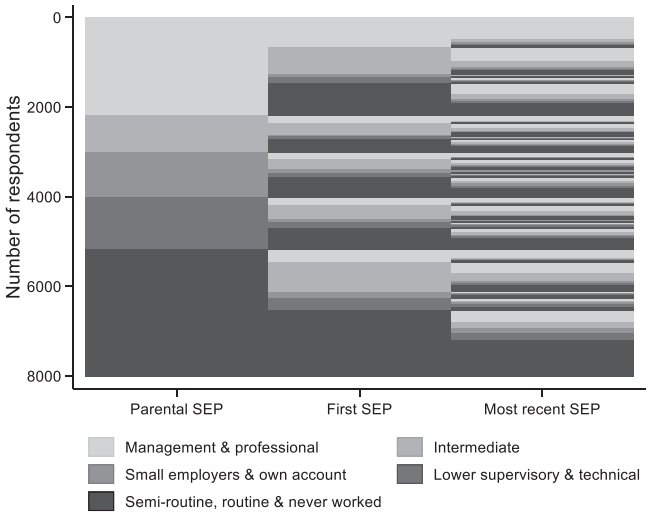

Fig. 2. Respondents' parental, first and most recent SEP.

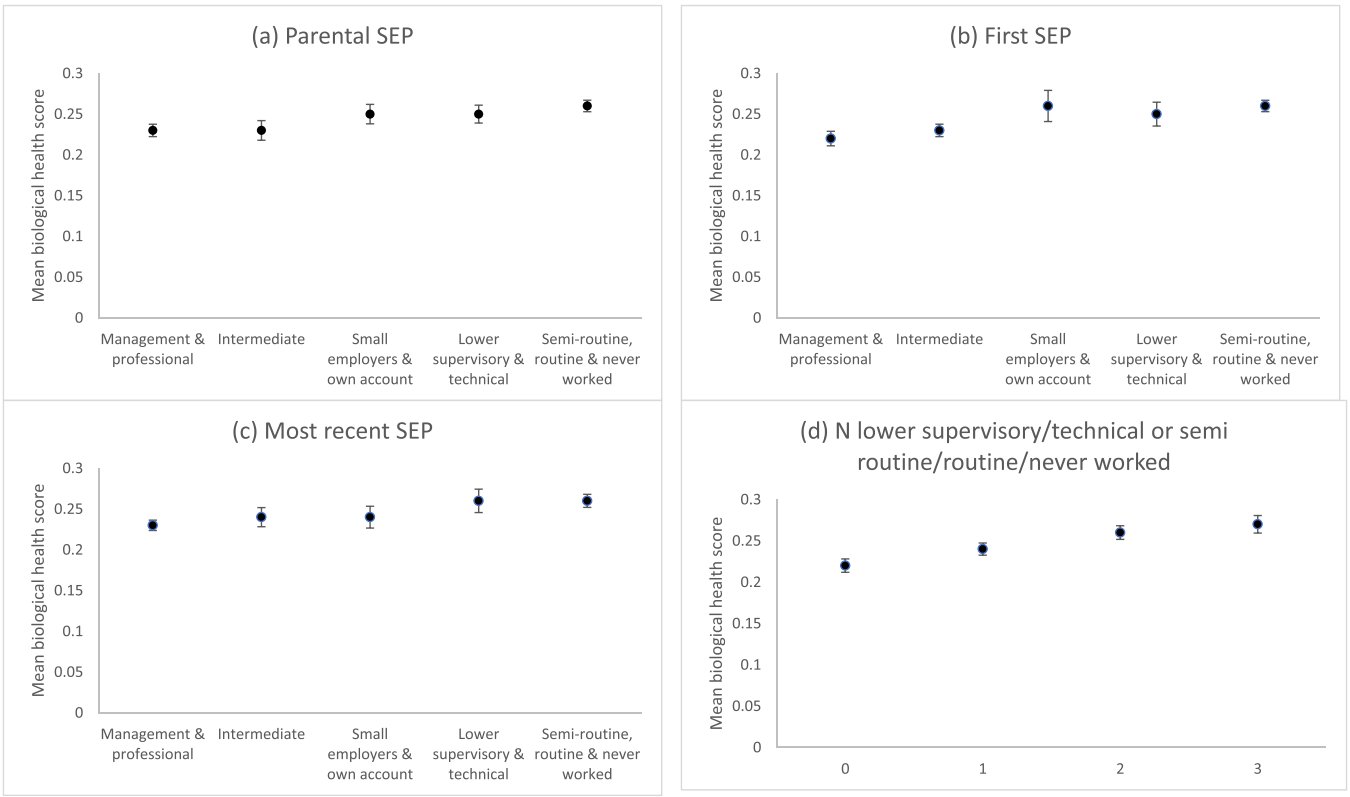

Fig. 3. Age and sex adjusted mean (95 % confidence interval) overall biological health score according to (a) parental, (b) first, (c) most recent and (d) cumulative (disadvantaged) SEP.

Table 2
Overall BHS according to parental, first, most recent and cumulative (disadvantaged) SEP.

SEP	Mean (SD) / Total	Difference (95 % CI) Unadjusted	Difference (95 % CI) Age and sex adjusted	Difference (95 % CI) Reciprocally adjusted
Parental SEP				
Management & professional	0.23 (0.15) / 1495	Reference	Reference	Reference
Intermediate	0.23 (0.15) / 596	0.01 (−0.01, 0.02)	0.01 (−0.01, 0.02)	0.01 (−0.01, 0.02)
Small employers & own account	0.25 (0.16) / 691	0.02 (0.01, 0.04)	0.02 (0.01, 0.04)	0.02 (0.00, 0.03)
Lower supervisory & technical	0.25 (0.16) / 818	0.02 (0.01, 0.04)	0.02 (0.01, 0.04)	0.02 (0.00, 0.03)
Semi-routine, routine & never worked	0.26 (0.16) / 1979	0.03 (0.02, 0.04)	0.03 (0.02, 0.04)	0.02 (0.01, 0.03)
SII (95 % CI)		*0.04 (0.03, 0.06)*	*0.04 (0.04, 0.06)*	*0.03 (0.02, 0.04)*
First SEP				
Management & professional	0.22 (0.15) / 1087	Reference	Reference	Reference
Intermediate	0.23 (0.15) / 1484	0.02 (0.01, 0.03)	0.02 (0.01, 0.03)	0.02 (0.00, 0.03)
Small employers & own account	0.26 (0.16) / 269	0.04 (0.02, 0.06)	0.04 (0.02, 0.07)	0.04 (0.01, 0.06)
Lower supervisory & technical	0.25 (0.16) / 459	0.03 (0.02, 0.05)	0.04 (0.02, 0.05)	0.03 (0.01, 0.05)
Semi-routine, routine & never worked	0.26 (0.17) / 2280	0.04 (0.03, 0.05)	0.04 (0.03, 0.05)	0.03 (0.02, 0.04)
SII (95 %CI)		*0.05 (0.04, 0.07)*	*0.05 (0.04, 0.07)*	*0.04 (0.02, 0.05)*
Most recent SEP				
Management & professional	0.23 (0.15) / 2145	Reference	Reference	Reference
Intermediate	0.24 (0.17) / 806	0.01 (0.00, 0.03)	0.02 (0.00, 0.03)	0.01 (−0.01, 0.02)
Small employers & own account	0.24 (0.15) / 483	0.01 (−0.01, 0.02)	0.00 (−0.01, 0.02)	-0.01 (−0.02, 0.01)
Lower supervisory & technical	0.26 (0.15) / 419	0.02 (0.00, 0.03)	0.02 (−0.00, 0.03)	0.00 (−0.01, 0.02)
Semi-routine, routine & never worked	0.26 (0.17) / 1726	0.03 (0.02, 0.04)	0.03 (0.02, 0.04)	0.01 (0.00, 0.02)
SII (95 % CI)		*0.04 (0.02, 0.06)*	*0.04 (0.03, 0.06)*	*0.02 (0.00, 0.04)*
Cumulative (disadvantaged) SEP (N lower supervisory/technical or semi routine/routine/never worked)				
0	0.22 (0.15) / 1323	Reference	Reference	
1	0.24 (0.16) / 1818	0.02 (0.01, 0.03)	0.02 (0.01, 0.04)	
2	0.26 (0.16) / 1451	0.03 (0.02, 0.04)	0.03 (0.02, 0.04)	
3	0.27 (0.17) / 987	0.05 (0.03, 0.06)	0.05 (0.03, 0.06)	
SII (95% CI)		*0.05 (0.04, 0.07)*	*0.05 (0.04, 0.07)*	

Table 3 presents SIIs for individual physiological subcomponent health scores. There was no strong association between cumulative or individual life-stage SEP and endocrine health scores. In contrast, all individual life-stage and cumulative SEP measures were moderately strongly associated with metabolic, cardiovascular and liver health scores with higher scores in more disadvantaged respondents (e.g. SII (95 % CI) for cumulative SEP: 0.07 (0.05, 0.10), 0.05 (0.02, 0.07), and 0.04 (0.02, 0.07) respectively), although most recent SEP had limited association with liver health scores. Adjustment for age and sex had little impact on these associations; however, reciprocal adjustment of

Table 3
SII (95 % CI) for individual physiological subcomponent health scores according to parental, first, most recent and cumulative (disadvantaged) SEP.

	Difference (95 % CI) Unadjusted	Difference (95 % CI) Age and sex adjusted	Difference (95 % CI) Reciprocally adjusted
Endocrine (N = 7724)			
Parental SEP	0.01 (−0.02, 0.04)	0.01 (−0.02, 0.04)	0.01 (−0.02, 0.04)
First SEP	-0.01 (−0.04, 0.02)	-0.01 (−0.04, 0.02)	-0.02 (−0.06, 0.02)
Most recent SEP	0.01 (−0.02, 0.04)	0.01 (−0.02, 0.04)	0.01 (−0.02, 0.04)
Cumulative SEP	0.00 (−0.03, 0.03)	0.00 (−0.03, 0.03)	
Metabolic (N = 7242)			
Parental SEP	0.07 (0.04, 0.09)	0.07 (0.05, 0.09)	0.06 (0.03, 0.08)
First SEP	0.06 (0.03, 0.08)	0.06 (0.03, 0.08)	0.03 (0.01, 0.06)
Most recent SEP	0.05 (0.03, 0.07)	0.05 (0.03, 0.08)	0.03 (0.00, 0.05)
Cumulative SEP	0.07 (0.05, 0.10)	0.08 (0.05, 0.10)	
Cardiovascular (N = 6658)			
Parental SEP	0.03 (0.00, 0.06)	0.03 (0.00, 0.06)	0.02 (−0.01, 0.05)
First SEP	0.03 (0.00, 0.06)	0.03 (0.00, 0.06)	0.01 (−0.03, 0.04)
Most recent SEP	0.05 (0.03, 0.08)	0.06 (0.03, 0.08)	0.05 (0.02, 0.08)
Cumulative SEP	0.05 (0.02, 0.07)	0.05 (0.02, 0.07)	
Inflammatory/immune (N = 7420)			
Parental SEP	0.09 (0.06, 0.11)	0.09 (0.06, 0.11)	0.06 (0.04, 0.09)
First SEP	0.10 (0.07, 0.12)	0.09 (0.07, 0.12)	0.06 (0.04, 0.09)
Most recent SEP	0.08 (0.06, 0.10)	0.08 (0.05, 0.10)	0.03 (0.01, 0.06)
Cumulative SEP	0.11 (0.09, 0.14)	0.11 (0.09, 0.14)	
Liver (N = 7455)			
Parental SEP	0.05 (0.02, 0.07)	0.05 (0.02, 0.08)	0.04 (0.01, 0.07)
First SEP	0.05 (0.02, 0.08)	0.05 (0.02, 0.08)	0.04 (0.01, 0.08)
Most recent SEP	0.02 (−0.01, 0.05)	0.02 (−0.01, 0.05)	-0.01 (−0.04, 0.02)
Cumulative SEP	0.04 (0.02, 0.07)	0.05 (0.02, 0.07)	
Kidney (N = 7812)			
Parental SEP	0.01 (−0.02, 0.05)	0.01 (−0.03, 0.04)	0.02 (−0.01, 0.06)
First SEP	-0.04 (−0.07, −0.00)	-0.04 (−0.07, −0.00)	-0.02 (−0.06, 0.02)
Most recent SEP	-0.05 (−0.09, −0.02)	-0.05 (−0.09, −0.02)	-0.05 (−0.09, −0.01)
Cumulative SEP	-0.04 (−0.07, −0.00)	-0.04 (−0.07, −0.00)	

individual life-stage SEP attenuated associations of cardiovascular scores with first and parental SEP and of liver scores with most recent SEP. In contrast, there were modest negative associations of first, most recent and cumulative SEP with kidney scores (e.g. unadjusted SII (95 % CI) for cumulative disadvantage: -0.04 (-0.07, -0.00)). The strongest associations with individual and cumulative SEP were those for inflammatory/immune health scores (unadjusted SII (95 % CI) for parental, first, most recent and cumulative SEP: 0.09 (0.06, 0.11), 0.10 (0.07, 0.12), 0.08 (0.06, 0.10), and 0.11 (0.09, 0.14) respectively). Again, these were not impacted by adjustment for age and sex and (attenuated) associations with SEP at all three life-stages remained in reciprocally adjusted models (SII (95 % CI) for most recent, first and parental SEP: 0.06 (0.04, 0.09), 0.06 (0.04, 0.09), and 0.03 (0.01, 0.06) respectively).

Discussion

We show consistent trends of more disadvantaged SEP across all three life-stages having higher overall BHS with the inflammatory/immune system playing a particularly important role. Other literature presents associations of SEP across the lifecourse, captured with parental [7], own educational attainment [8], occupational class [14], and income and wealth, with multiphysiological system scores [5]. Associations of SEP across the lifecourse have been reported for adult AL [15] but it was unclear whether similar associations hold for the recently described BHS [12]. The BHS has been associated with increased mortality independently of measures of education [13], the only measure of SEP to have previously been examined with this score [12]. Our results suggest that additional measures of SEP across the lifecourse are associated with BHS, with differences of around 0.05 units between the most versus least disadvantaged respondents. Chadeau-Hyam et al [13]. estimate that a 0.1 unit increase in BHS is associated with an all-cause mortality hazard ratio of 1.14 and incident cardiovascular disease hazard ratio of 1.15, suggesting that the SEP differences observed here reflect clinically meaningful differences in biological risk.

Our results suggest that SEP associations are stronger with inflammatory markers, which accord with a large literature reporting social patterning of these markers using a variety of SEP measures [6, 30–33]. We included a number of inflammation markers within this subcomponent but biological factors can have different functions. For example, as well as being an inflammatory marker, IGF-1 is an anabolic protein related to insulin and therefore also has metabolic functions, previously demonstrated to be associated with lifecourse SEP [34], although our sensitivity analyses suggest that subcomponent associations are not driven by single variables.

We also observed social patterning of BHS metabolic and cardiovascular components, consistent with previous literature on the social patterning of adiposity [35], blood pressure [36], and other multi-system scores such as AL [14]. The finding of associations of adult SEP but not childhood SEP, in mutually adjusted models, for the cardiovascular subcomponent does not accord with recent literature[37] and may relate to the previous use of educational attainment rather than occupation. We did not find SEP associations with endocrine markers, although previous evidence of social patterning of endocrine markers is equivocal [37–39]. We found an association of worse liver function with increasing disadvantage in childhood and early adulthood that may, partly, reflect people's response to stress, for example heavy alcohol intake, but may also reflect conditions such as fatty liver or infection, which are all socially patterned [40,41]. A separate analysis of these data suggested that educational attainment is associated with liver function measures in young people only [12]. Finally, for kidney function we found either no or a weak association between advantaged SEP and high creatinine and Karimi [12] reports similar results among younger age groups. This may reflect our use of creatinine as a measure of kidney function rather than estimated glomerular filtration rate (eGFR), which is used to detect chronic kidney disease and has been shown to be associated with deprivation [42]. .

Our finding that individual and cumulative lifecourse SEP are associated with BHS suggests that studies of BHS that are limited to adult SEP may not fully account for confounding by early-life SEP and that

considering measures of SEP at one time-point may limit our understanding of associations. Other literature focusing on specific biological systems, e.g. inflammatory,[30,43] cardiovascular [44], and metabolic markers,[45–47] fails to consider the interaction of biological processes meaning that a focus on one system may be impacted by SEP associations in others. The inverse association with creatinine is unexpected, as chronic kidney disease has been reported as being socially patterned [48]. However, much of the USA evidence suggests this is disproportionately associated with ethnicity, and ethnic group was not adequately represented in these data. We examine biomarkers to understand and pinpoint and address pathways of interest. The study suggests that because endocrine markers are not patterned by SEP they may not contribute to the association of SEP with ageing or health.

The study has several advantages. We use a large sample of respondents with extensive interview and biomarker data·We have assessed BHS across the entire adult age-range and have SEP at three life-stages based on repeated longitudinal data, allowing exploration of the individual and cumulative impact·However, there are also a number of limitations to consider·Analyses were restricted to respondents with complete data on SEP, although characteristics of those included were very similar to the full sample·Given UKHLS is a probability-based sample, the use of analytical weights adjusted for being included in the analyses make our results generalisable to the 2009 UK population·However the sample size is not sufficient to explore ethnic group specific associations, which may differ, and groups migrating to the UK since 2009 are not represented·There is considerable evidence of wide health inequalities among different ethnic groups, especially among older populations [49], which this paper cannot investigate·SEP was based on parental and own first and current occupation with potentially greater recall bias in earlier life-stages although the consistency of outcomes might suggest otherwise·Biological data in UKHLS are collected at one wave only and it is not possible to examine change over time, a potential area for future research given the high rate of change in SEP over time·While our measure of BHS covers multiple physiological systems, other broader measures exist, such as the frailty index [50], which, in datasets containing the extensive range of biomarkers and SEP measures required, would be valuable to explore the embodiment of inequalities further·In addition, our analyses may reflect survivor bias in the oldest age groups, and we cannot rule out reverse causality with respondents' higher life-time BHS (reflecting poorer health) potentially impacting their SEP, although, notably, associations with parental SEP, which is least likely to be impacted by respondent ill health, were as strong as or stronger than those with later life SEP·Similarly, it has been argued that the use of occupational class, especially first and last known position, is likely to reduce the possibility of reverse causality as these are less likely to change with ill health than other measures such as income· (1)

While this paper demonstrates strong correlations, and not causality, the evidence of the timing of the SEP measures being before the BHS is suggestive of a causal association. The strength of the association between parental occupation and adult ill health suggests policies that focus on reducing inequalities in child health and education outcomes, and hence creating opportunities for socioeconomic mobility for those who experience disadvantage in childhood, would reduce later life poor health [51].

Conclusion

We have shown that a multidomain biological health score is socially patterned and associated with individual and cumulative social position at each life-stage supporting the concept of social embodiment leading to health inequalities. Associations are particularly apparent for inflammatory and metabolic subcomponents of the BHS.

Research ethics approval

The University of Essex Ethics Committee has approved all data collection on Understanding Society main study. Approval for the collection of biosocial data by trained nurses in Waves 2 and 3 of the main survey was obtained from the National Research Ethics Service (Understanding Society – UK Household Longitudinal Study: A Biosocial Component, Oxfordshire A REC, Reference: 10/H0604/2).

Authorship contribution

All authors conceived the study. EW performed the statistical analyses. All authors contributed to results interpretation. All authors revised the manuscript for important intellectual content and have seen and approved the submission of the manuscript.

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CRediT authorship contribution statement

Elise Whitley: Writing – review & editing, Writing – original draft, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Meena Kumari:** Writing – review & editing, Investigation, Conceptualization. **Michaela Benzeval:** Writing – review & editing, Methodology, Investigation, Funding acquisition, Conceptualization. **Michelle Kelly-Irving:** Writing – review & editing, Investigation, Conceptualization.

Declaration of Competing Interest

The authors declare no conflicts of interest.

Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.annepidem.2024.06.006.

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