RESEARCH REPORT

Socioeconomic status over the life course and allostatic load in adulthood: results from the Northern Swedish Cohort

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ABSTRACT

Background Although several studies have reported rather consistent associations between socioeconomic status (SES) and allostatic load (AL), so far no study has examined the influence of SES over the life course on AL. The aim of the present study was to investigate the association between SES over the life course and AL in mid-adulthood, guided by the conceptual models of cumulative risk, critical period and social chain of risk.

Methods The sample comprises a 27-year prospective cohort (n=1071) from northern Sweden. Participants (n=855, 79.8%) completed questionnaires at the ages of 16, 21, 30 and 43 years. A health examination was performed at age 43 years after an overnight fast, including physical examination and blood sampling, and participants completed 1-day salivary cortisol sampling (four samples). SES was based on parental occupation at age 16 years and participants' own occupation at ages 21, 30 and 43 years. Information on daily smoking, snuff use, high alcohol consumption and physical inactivity was reported by the participants. An AL index was constructed from tertiles of 12 biological parameters.

Results Cumulative socioeconomic disadvantage was related to AL in both women and men. The association was largely explained by health behaviours in men, but was independent of health behaviours in women. In women, an association was observed between AL and SES in adolescence, whereas in men only current SES was related to AL, independently of current health behaviours.

Conclusions SES over the life course influences the level of multi-systemic dysregulation in mid-adulthood, with the strongest support for the cumulative risk model.

A recent review[1] reveals that an inverse relationship between socioeconomic status (SES) and allostatic load (AL) is a fairly consistent finding in the few studies carried out on the subject. However, the review also highlights that all epidemiological studies examining the association have been cross-sectional,[2–6] and that a life-course perspective has been applied only in one small-sized descriptive study[7] or in studies examining only specific aspects of AL.[8 9] The review also concludes that the findings regarding associations between SES and cortisol levels are less consistent. We have recently reported on life-course influences of SES on circadian cortisol levels in adulthood,[10] and in the present report we seek to shed light on the importance of SES over the life course for AL in adulthood.

During the past decade studies on the well-established association between socioeconomic disadvantage and poor health have been extended by the adoption of life-course models.[11 12] Conceptual models of the putative chains of causation have been proposed, including the models of: cumulative risk, focussing on the health effects with respect to the repetitive or chronic nature of exposures; critical or sensitive period, highlighting the importance of exposure at certain developmental stages with latent effects independent of later circumstances; and pathway or social chain of risk, emphasising the continuity of risk trajectories and circumstances over the life course, affecting health over time.[11 13] A variety of empirical support has been presented for these models for cardiovascular health[11 14 15] as well as for other outcomes, such as diabetes,[16] psychosocial functioning,[17] body weight[18] and health behaviour.[19 20]

Whereas life-course models comprise general causal propositions as to how SES may influence health, more specific models of how socioeconomic circumstances influence pathophysiological processes are less well established. The model of AL[21 22] conceptualises the development of chronic disease (eg, cardiovascular disease) within a framework of accumulated physiological 'wear and tear' resulting from the accumulation of stressful exposures. In the short run, such exposures activate physiological stress systems (eg, the hypothalamic–pituitary–adrenal axis), which lead to compensatory changes in secondary physiological systems (eg, metabolic systems).[23] These dynamic responses are necessary for the maintenance of homeostasis, a process called allostasis. However, the model of AL suggests that in the long run the accumulation of adaptations mediated by stress systems develops into AL, a multisystemic pre-disease state represented by subclinical levels of metabolic and cardiovascular parameters. AL has been shown to be a useful concept for predicting future morbidity, mortality and psychosocial functioning.[24–26] The AL model, with its focus on long-term accumulation and gradual development of physiological dysregulations, is conceptually compatible with life-course influences on ill-health, but this issue has so far not been empirically tested.

The present paper aims to examine the life-course influences of SES on AL at age 43 years. More specifically, we aim to examine whether the cumulative risk, critical period and social chain of

risk explain variations in AL. Drawing from our previous findings regarding life-course SES and cortisol levels,[10] our hypothesis is that adolescence would emerge as a critical period for SES exposure, while the other models would be of modest explanatory value.

METHODS

Participants and procedures

The participants were drawn from a prospective cohort study, the Northern Swedish Cohort, approved by the Regional Ethical Review Boards in Uppsala and Umeå. The study has been found to be representative of Sweden in various demographic comparisons.[27] The methods are described in more detail elsewhere.[27 28] Briefly, the cohort comprised all adolescents in the ninth (last) grade of compulsory school, in a middle-sized industrial town in northern Sweden in 1981 (N=1083; 506 girls and 577 boys), when the participants were 16 years of age. The follow-up surveys were conducted in 1983, 1986, 1995 and 2008, when the participants were 18, 21, 30 and 43 years, respectively. At each data collection phase, the participants provided informed consent and completed a questionnaire about demographic and social circumstances, health-related symptoms, labour market history, health behaviour and leisure activities.

A health examination was performed by trained medical personnel in 2008, comprising blood pressure, height, weight and waist circumference measured according to the WHO MONICA manual,[29] and blood samples were drawn after an overnight fast, with subsequent assessment of the following parameters: glucose, total cholesterol, high-density lipoprotein (HDL) cholesterol, low-density lipoprotein (LDL) cholesterol, triglycerides, apolipoprotein A1, apolipoprotein B and C-reactive protein (CRP). The participants also performed saliva sampling, using salivettes, for the assessment of cortisol. Sampling was performed at four time points during one weekday: at awakening, 15 min post-awakening, pre-lunch and at bedtime (for details, see Gustafsson *et al*).[10] The blood and saliva samples were handled, stored and analysed according to the laboratory routines at the Department of Clinical Chemistry, Umeå University Hospital.

The participation rate at the 43 year survey was 94.4% (n=1011) of subjects who were still alive (n=1071). In the present report, data from the 16, 21, 30 and 43 year surveys were included, to represent different periods of adolescence and adulthood. Due to drop-out on one or more key measures (see below), 855 individuals (412 women, 443 men; 79.8%) were included in the analyses.

Measures

Life-course SES

Participants' own occupation at ages 21, 30 and 43 years were coded according to the socioeconomic classification system of Statistics Sweden.[30] The classification is based on the main divisions of manual workers, non-manual employees and self-employed. Manual workers were categorised as low SES (1), while non-manual employees and self-employed were categorised as high SES (0). For participants who were not currently working and for whom information on previous occupation was not available (only at ages 21 and 30 years), for example for participants who were unemployed, studying or doing military service, the highest educational attainment was used as a proxy (n=206 for SES at age 21 years, n=41 for SES at age 30 years): university–preparatory high school or university studies indicated high SES (0), whereas other types of high school education or lower were classified as low SES (1). At 16 years parental occupation was coded into three social groups. Having both parents in social group 3, comparable to manual workers, defined low SES (1), while having at least one parent in social groups 1 or 2 defined high SES (0).

Cumulative socioeconomic disadvantage was operationalised as the number of occasions with low SES (range 0–4).

AL at 43 years of age

The original operationalisation of AL focuses on high-risk parameter levels.[25] However, the AL model conceptualises health and disease as a continuum, and we therefore chose to modify the original operationalisation by including parameter variations across a broader spectrum, thereby combining the general principles of the 'z-score' and 'group' formulations of AL.[22] We regard this as a (patho)physiologically suitable operationalisation, also circumventing the problem of a skewed distribution.

First, biological parameters were selected for inclusion (k=12): systolic blood pressure, diastolic blood pressure (mm Hg), body mass index (BMI, kg/m^2), waist circumference (cm), fasting glucose, total cholesterol, HDL-cholesterol, triglycerides, apolipoprotein A1, apolipoprotein B, CRP (mmol/l), and cortisol (nmol/l). LDL-cholesterol was excluded due to its high correlation with total cholesterol (r=0.93). For cortisol, the diurnal area under the curve (AUC) with respect to ground[31] (log nmol/l×h) was calculated, as an approximation of total cortisol secretion. Each parameter was trichotomised by tertiles, separately for women and men, and coded as 0, 1 or 2 corresponding to low, medium and high contribution to AL. As both hyper- and hypocortisolism can reflect cortisol dysregulation,[32] cortisol AUC was divided into sextiles and coded symmetrically (ie, sextile 1 and 6=2, 2 and 5=1, 3 and 4=0). HDL-cholesterol and apolipoprotein A1 were coded inversely; the highest tertile as 0 and the lowest as 2. Due to CRP levels being truncated at less than 3 mmol/l, there was only a low (0; n=573, 67%) and a high (2; n=282, 33%) category. CRP levels greater than 10 were coded as 0 to reduce influences of ongoing infections.

Second, the parameters were categorised into six physiological systems (see table 1): cardiovascular regulation (k=2), body fat deposition (k=2), lipid metabolism (k=5), glucose metabolism (k=1), inflammation (k=1) and neuroendocrine regulation (k=1).[22] Participants with pharmacological medication were coded as 2 on the following physiological system categories: hypertension medication (n=56, cardiovascular category), dyslipidemia medication (n=25, lipid metabolism) and medication against diabetes (n=10, glucose metabolism). As the main drop-out on biological parameters was due to failure to complete the salivary cortisol sampling (n=130), those without valid cortisol data were assigned the mean value of 1 on the neuroendocrine category.

Third, within each physiological system the codings were weighted with respect to the number of parameters included in the system, by calculating the mean score of parameters, generating a value between 0 and 2 for each physiological system. This was done to reduce the influence of an unbalanced number of parameters measuring similar aspects of physiological dysregulation. Of the means calculated, n=24 had incomplete lipid parameters, and n=27 had either BMI or waist circumference missing. See table 1 for details of the parametrisation.

Finally, the allostatic contributions of the physiological systems (k=6) were summed up into an index (range of 0–12), comprising the final measure of AL.

Potential confounders

Current health behaviour at 43 years (n=967) was identified as potential confounders: daily smoking (no/yes), daily snuff use

Table 1 Operationalisation of individual AL parameters by physiological systems in women and men

	Contribution to AL: women			Contribution to AL: men		
Parameter	**Low (0)**	**Medium (1)**	**High (2)**	**Low (0)**	**Medium (1)**	**High (2)**
Physiological system						
Cardiovascular*						
SBP (mm Hg)	<113	113–124	>125	<120	120–134	>134
DBP (mm Hg)	<71	71–80	>80	<78	78–86	>86
Body fat						
BMI (kg/m^2)	<23.1	23.1–26.8	>26.8	<25.3	25.3–28.7	>28.7
Waist (cm)	<79	79–89	>89	<91	91–101	>101
Lipid metabolism†						
Total cholesterol	<4.7	4.7–5.5	>5.5	<5.0	5.0–5.8	>5.8
HDL-cholesterol	>1.65	1.31–1.65	<1.31	>1.32	1.05–1.32	<1.05
Triglycerides	<0.73	0.73–1.18	>1.18	<1.10	1.10–1.81	>1.81
Apolipoprotein A1	>1596	1380–1596	<1380	>1431	1274–1431	<1274
Apolipoprotein B	<803	803–979	>979	<915	915–1120	>1120
Glucose metabolism‡						
Glucose	<4.7	4.7–5.5	>5.5	<5.0	5.0–5.4	>5.4
Inflammation						
CRP	<3/>10	N/A	3-10	<3/>10	N/A	3–10
Neuroendocrine§						
Cortisol (nmol/l×h)	16–22	15–16/22–26	<15/>26	20-27	17–20/27–29	<17/>29

Unit of measurement is mmol/l unless otherwise noted.
Medication coded as 2:
*Hypertensive medication.
†Dyslipidemia medication.
‡Diabetes medication.
§Missing data coded as 1.
AL, allostatic load; BMI, body mass index; CRP, C-reactive protein; DBP, diastolic blood pressure; HDL, high-density lipoprotein; SBP, systolic blood pressure.

(no/yes), physical inactivity (seldom or never engaging in physical activity during the past 12 months/engaging each month or more often) and high alcohol consumption, measured as annual consumption of pure alcohol derived from questions about typical frequency and quantity of beverage consumption (<80th percentile/>80th percentile). All were coded as low risk 0 and high risk 1.

Data analysis

In total, there were 855 cases comprising our effective sample, see table 2 for descriptive statistics. The N is lowest in analyses simultaneously including all SES, AL and health behaviour measures (n=808, 391 women and 417 men). Those with missing AL did not differ from those with AL regarding cumulative SES or SES at ages 16, 21 or 43 years ($p>0.05$, χ^2 test), but had a higher proportion of low SES at age 30 years (50% vs 42%, n=1030, p=0.043).

In order to specifically test the cumulative risk model, cumulative socioeconomic disadvantage was used as the predictor in a linear regression model, adding health behaviour measures as additional predictors in a separate model. To examine the models of critical periods and social chain of risks, SES at each age was used as the predictor in separate bivariate regression models, with subsequent adjustment for health behaviour in a second model, mutual adjustment for all SES measures in a third model (without health behaviour), and both all SES measures and health behaviour in a fourth model. A significant contribution of early SES in both the first bivariate

Table 2 Descriptive statistics of SES over the life course and health behaviour and AL at age 43 years (N=855)

	Women		Men		Total		
Measure	**n (%)**	**N**	**n (%)**	**N**	**n (%)**	**N**	**p Value***
Low SES, at age, years							
16	154 (38)	407	170 (39)	439	324 (38)	846	0.791
21	229 (57)	404	296 (67)	441	525 (62)	845	0.002
30	155 (38)	403	196 (44)	433	351 (42)	836	0.046
43	125 (30)	412	169 (38)	442	294 (34)	854	0.015
Health behaviour at 43 years							
Smoking	95 (23)	410	74 (17)	439	169 (20)	849	0.021
Snuff	55 (13)	411	131 (30)	434	186 (22)	845	<0.001
Alcohol	45 (11)	411	126 (28)	443	171 (20)	854	<0.001
Inactivity	65 (16)	412	110 (25)	442	175 (20)	855	<0.001
Biological measure at 43 years							
AL, M (s)	5.5 (2.5)	412	5.7 (2.4)	443	5.6 (2.5)	855	0.266

*Difference between women and men; χ^2 test or t test.
Numbers shown are total number of participants (N) and participants with the specified exposure (n (%)) for each exposure, and mean (standard deviation) for allostatic load (AL).
SES, socioeconomic status.

and the third adjusted model is interpreted as indicating a critical period, ie, that the impact of early conditions is independent of later conditions. Conversely, a significant bivariate association between early SES and AL in the first bivariate model, in combination with attenuation of the association and a significant contribution of current SES in the third model, is interpreted as consistent with a social chain of risks, ie, that the impact of early conditions is explained by later conditions.

SES displayed a measure of stability over the life course as indicated by low to moderate correlation between SES measures: 16 years vs 21–43 years (r=0.24–0.25), 21 years versus 30 and 43 years (r=0.37–0.43) and 30 years versus 43 years (r=0.56). Due to multicollinearity (maximum variance inflation factor = 1.67) results are also presented for analyses without SES at age 30 years.

The main analyses were re-run excluding the cases that had been assigned a value for cortisol AUC (n=130), yielding similar results to the analyses on the inclusive sample (data not shown). Therefore, to preserve power and reduce possible selection bias, the more inclusive sample was used in the main analyses. An operationalisation of AL based on parameter dichotomisation at the 75th percentile[25] yielded comparable results (data not shown).

All analyses were performed on the total sample as well as on women and men separately. SPSS version 17.0 was used for all analyses.

RESULTS

Cumulative low SES was related to AL in the total sample (see table 3 and figure 1), an association that was somewhat attenuated but remained significant after the addition of health behaviour measures as covariates. Performing the analyses in women and men separately (table 3), the unadjusted model displayed similar results, but in men the attenuation was particularly strong (mainly explained by physical inactivity) and reduced the contribution of cumulative low SES below significance. In women, health behaviours were not significantly related to AL and the attenuation effect was modest.

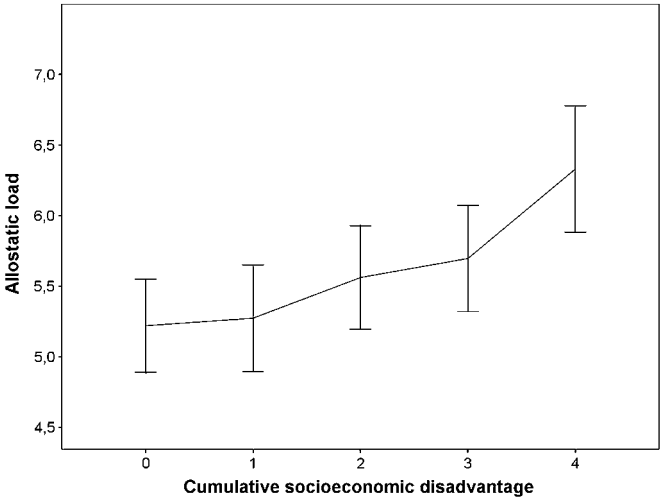

Figure 1 Allostatic load (mean, 95% CI) by cumulative socioeconomic disadvantage, ie, the cumulative number of times with low socioeconomic status at 16, 21, 30 and 43 years.

Cumulative low SES was correlated with the scores of the individual physiological system cardiovascular regulation (r=0.07, p=0.032), body fat deposition (r=0.15, p<0.001), lipid metabolism (r=0.10, p=0.004), glucose metabolism (r=0.09, p=0.010), but not with inflammation (r=0.05, p=0.176) or neuroendocrine regulation (r=0.01, p=0.755). In women, associations were particularly strong for body fat (r=0.20, p<0.0.01) and lipids (r=0.16, p=0.001), whereas in men glucose metabolism (r=0.14, p=0.003) and body fat (r=0.10, p=0.032) were significantly related to cumulative socioeconomic disadvantage.

As an assessment of the critical period and social chain of risk models, the bivariate linear associations between AL and SES measured at ages 16, 21, 30 and 43 years were examined, with adjustment for health behaviour in a second model, for the other SES measures in a third model and both SES measures and

Table 3 Summary of linear regression models with AL on cumulative socioeconomic disadvantage (SES)

	Model 1			Model 2		
Predictor	**b (SE)**	**β**	**p Value**	**b (SE)**	**β**	**p Value**
Total sample						
Cumulative low SES	0.25 (0.06)	0.14	<0.001	0.18 (0.06)	0.10	0.005
Smoking	–	–	–	0.62 (0.22)	0.10	0.004
Snuff	–	–	–	−0.04 (0.21)	−0.01	0.842
Alcohol	–	–	–	0.43 (0.21)	0.07	0.044
Inactivity	–	–	–	0.68 (0.22)	0.11	0.002
Only women						
Cumulative low SES	0.28 (0.09)	0.15	0.003	0.24 (0.10)	0.13	0.015
Smoking	–	–	–	0.44 (0.30)	0.08	0.149
Snuff	–	–	–	−0.45 (0.37)	−0.06	0.219
Alcohol	–	–	–	0.19 (0.40)	0.02	0.638
Inactivity	–	–	–	0.09 (0.34)	0.01	0.799
Only men						
Cumulative low SES	0.21 (0.08)	0.13	0.010	0.12 (0.08)	0.07	0.149
Smoking	–	–	–	0.77 (0.32)	0.12	0.018
Snuff	–	–	–	0.11 (0.26)	0.02	0.662
Alcohol	–	–	–	0.47 (0.26)	0.09	0.068
Inactivity	–	–	–	1.05 (0.28)	0.19	<0.001

Model 1=only socioeconomic status (SES), model 2=SES and smoking, snuff use, alcohol consumption and physical inactivity.
Total sample: model 1: R^2=0.019, p<0.001; model 2: R^2=0.050, p<0.001. Women: model 1: R^2=0.022, p=0.003; model 2: R^2=0.029, p=0.046. Men: model 1: R^2=0.016, p=0.010; model 2: R^2=0.088, p<0.001.
AL, allostatic load; b(SE), unstandardised regression coefficient (standard error); β, standardised regression coefficient.

health behaviour in a fourth model (see table 4). In the total sample, SES at 43 years, and to a lesser degree SES during adolescence and early adulthood, were related to AL. Only SES at 43 years remained influential after mutual adjustment for all SES measures, indicating that early SES did not contribute independently to AL, but through current SES. Analyses performed separately on women and men revealed partly different patterns. In men, only current SES was related to AL throughout the models. Conversely, in women, SES at the ages of both 16 and 43 years were significantly related to AL, with SES at age 30 years displaying a near-significant association, and with SES at age 16 years being the sole significant variable in the fully adjusted fourth model. Due to multicollinearity, the analyses for models 3 and 4 were re-run, dropping SES at age 30 years from the set of predictors. In these complementary analyses (maximum variance inflation factor = 1.2) in women the coefficients were slightly strengthened for SES at age 16 years (model 3: β=0.10, p=0.047; model 4:β=0.12, p=0.025), further indicating SES in adolescence constituting a critical period, and for SES at age 43 years (model 3: β=0.14, p=0.009; model 4: β=0.11, p=0.043). There were no inferential changes in men or in the total sample (data not shown).

DISCUSSION

The main finding of this study was that, contrary to our hypothesis (based on an earlier report on life-course SES and circadian cortisol levels),[10] cumulative socioeconomic disadvantage over the life course was strongly related to AL in adulthood, in both women and men. Adjusting for smoking, snuff use, alcohol and physical inactivity attenuated the effect sizes substantially in men but not in women. SES at all ages contributed to AL in bivariate analyses in the total sample, but after mutual adjustment only current SES remained a significant predictor, consistent with a social chain of risk model. In women only, adolescent SES was independently related to AL, which is consistent with our hypothesis of adolescence as a critical period for socioeconomic influences on later physiological dysregulation.

In the total sample, the support for a social chain of risk suggests that although SES throughout the life course is important, the mechanisms might be largely explained by the tendency for socioeconomic circumstances to be stable over the life course, with current or recent circumstances representing the temporally proximal influence.[13] The importance of current SES can be seen as a corroboration of previous cross-sectional findings of an inverse relationship between SES and AL.[2–6] However, the weak but independent influence of adolescent SES in women is consistent with adolescence or childhood constituting a critical period, with latent effects spanning over decades. This finding is consistent with studies on the diverse adult health effects of socioeconomic disadvantage in childhood.[15] Considering that body fat was more strongly related to life-course SES in women than in men, this might be an indication of obesity driving the associations of early SES and AL in women—the association between childhood SES and adult obesity is generally found to be stronger in women than in men, which might be due to the gendered social pressure against obesity being more marked among women of high SES.[33] This is a possible explanation for our pronounced findings regarding early SES and AL in women only.

The impact of cumulative disadvantage was the most consistent finding of this study, indicating that the accumulation of socioeconomic disadvantage is coupled with physiological dysregulation. AL relates to cardiovascular morbidity[25] and both baseline and increases in AL are prospectively predictive of mortality,[24 26] and our results thus correspond with the wealth of empirical support for the cumulative risk model for manifest, for example cardiovascular, disease.[11] However, it contradicts our own findings of no clear-cut cumulative influences on circadian cortisol levels.[10] Considering the conceptually integral status of these mediators within the AL model, which presupposes neuroendocrine mediation of stressful circumstances, we had expected to find similar life-course influences on AL as on the circadian cortisol rhythm. Even though cumulative effects have been reported by others for life-course SES and cortisol levels,[9] this inconsistency converges with a critique of the empirical status of neuroendocrine mediators within the AL model.[1 34] Possibly, some of the inconsistencies in the literature might stem from the methodological challenges in assessing hypothalamic–pituitary–adrenal axis functioning.

Although a similar association between cumulative SES and AL was observed in both women and men in the bivariate

Table 4 Summary or linear regression models with AL on low SES at ages 16, 21, 30 and 43 years, in the total sample and in women and men separately

Sample SES at age, years	Model 1				Model 2				Model 3				Model 4			
	R^2	b(SE)	β	p Value	R^2	b(SE)	β	p Value	R^2	b(SE)	β	p Value	R^2	b(SE)	β	p Value
Total sample									0.025				0.056			
16	0.005	0.38 (0.18)	0.07	0.031	0.045	0.38 (0.18)	0.08	0.028		0.15 (0.19)	0.03	0.417		0.20 (0.18)	0.04	0.271
21	0.006	0.39 (0.18)	0.08	0.025	0.042	0.25 (0.18)	0.05	0.162		0.09 (0.20)	0.02	0.670		0.05 (0.20)	0.01	0.798
30	0.006	0.43 (0.17)	0.09	0.013	0.040	0.24 (0.18)	0.05	0.172		−0.04 (0.22)	−0.01	0.862		−0.17 (0.22)	−0.03	0.443
43	0.027	0.85 (0.18)	0.16	<0.001	0.052	0.64 (0.18)	0.15	<0.001		0.75 (0.22)	0.15	<0.001		0.66 (0.22)	0.13	0.003
Only women									0.026				0.036			
16	0.012	0.57 (0.26)	0.11	0.025	0.029	0.66 (0.26)	0.13	0.019		0.45 (0.26)	0.09	0.086		0.52 (0.27)	0.10	0.050
21	0.004	0.32 (0.25)	0.06	0.211	0.014	0.21 (0.26)	0.04	0.407		0.03 (0.28)	,01	0.918		0.00 (0.28)	0.00	0.993
30	0.008	0.46 (0.25)	0.09	0.070	0.015	0.34 (0.26)	0.07	0.192		−0.05 (0.33)	−0.01	0.890		0.01 (0.33)	0.00	0.966
43	0.024	0.84 (0.27)	0.15	0.002	0.026	0.71 (0.28)	0.13	0.012		0.68 (0.34)	0.13	0.043		0.53 (0.35)	0.10	0.128
Only men									0.029				0.01			
16	0.001	0.19 (0.24)	0.04	0.422	0.084	0.07 (0.24)	0.02	0.758		−0.16 (0.26)	−0.03	0.535		−0.18 (0.26)	−0.04	0.475
21	0.007	0.43 (0.25)	0.08	0.079	0.084	0.24 (0.24)	0.05	0.327		0.13 (0.29)	0.03	0.661		0.09 (0.28)	0.02	0.762
30	0.006	0.38 (0.23)	0.08	0.108	0.083	0.12 (0.24)	0.02	0.625		−0.03 (0.30)	−0.01	0.925		−0.33 (0.29)	−0.07	0.257
43	0.028	0.84 (0.24)	0.17	<0.001	0.097	0.62 (0.24)	0.12	0.009		0.85 (0.30)	0.17	0.006		0.87 (0.30)	0.17	0.003

Model 1=simple regression, model 2=model 1 + health behaviour (smoking, snuff use, alcohol consumption and physical inactivity; coefficients not shown), model 3 = multiple regression with socioeconomic status (SES) at all ages, model 4=model 3 + health behaviour.
AL, allostatic load; b(SE), unstandardised regression coefficient (standard error); β, standardised regression coefficient.

analyses, health behaviour measures (smoking, snuff use, high alcohol consumption and physical inactivity) largely explained the association in men, while in women the contribution of SES was largely independent of current health behaviour. This suggests that other mechanisms than health behaviours might operate in women. Of particular relevance for the model of AL is that women of low SES are, to a greater degree than men of low SES, exposed to adverse psychosocial circumstances such as low income, unemployment, single parenthood and high depressive symptoms.[35] This potential role of gendered psychosocial exposures over the life course for AL in adulthood is an issue that should be examined in future research.

Methodological considerations

The main methodological strengths of this study include a prospective design spanning over 27 years as well as a high participation rate in a sample representative of Sweden. Analyses indicated that there was no substantial systematic drop-out for those with missing AL data regarding SES, suggesting that selection bias is not an important threat to validity.

Although the use of occupation as a proxy for SES captures only certain aspects of socioeconomic disadvantage,[36] occupation tends to change more during the life course compared with education, making it suitable for studying life-course models. Moreover, there might be a point in using similar operationalisations across the life course, to avoid confounding age with qualities of the measure. Nevertheless, as exemplified by the necessity of using parental occupation at age 16 years and educational attainment for the proportion who were studying or doing military service at age 21 years, the measurement of SES over the life course is problematical and complete correspondence might not be possible.[36] Nevertheless, the rather high correlations between SES at the different time periods indicate that there was a substantial empirical correspondence between the SES measures.

As different life-course models are difficult to disentangle empirically,[37] we examined the compatibility of our data with several different life-course models, as has been recommended by others,[11] while still guided by specific hypotheses. Due to the methodological demands when testing the social mobility model,[11] in combination with the limited sample size, this specific life-course model was not examined. Although the results were independent of some specific health behaviours, other lifestyle or personal characteristics (eg, intelligence) could possibly confound the analyses. Health-rated selection is a potential confounder, and we did not have comprehensive information on physiological dysregulation before the first data collection in adolescence to take this into account. However, our operationalisation of AL, focusing on categorically subclinical levels of biological parameters, makes it unlikely that health-related selection would explain the findings. Our operationalisation of AL differed from the original one[25] by focussing not only on high risk levels but including parameter variations across the normal spectrum. Our operationalisation included the main physiological systems usually included in the concept of AL,[22] which were weighted within each system to reduce the undue influence of different numbers of components measuring related dysregulations. However, there is a considerable heterogeneity in the literature regarding the range of biomarkers included in AL measures and their parametrisation, both in the specific field studying associations between SES and AL,[1] as well as in related fields.[22] This operationalisational heterogeneity hampers direct comparisons between studies because specific biomarkers make different contributions to the overall relationship, and the results thus might differ with the presence/absence of particular biomarkers (such as neuroendocrine markers, which have been criticised for weak empirical evidence).[1] Future research should aim at exploring the influence of different operationalisations and at developing a standardised operationalisation of AL, similar to what has been done for the related clinical concept of the metabolic syndrome.[38]

What is already known on this subject

A few studies have examined the association between cumulative biological dysregulations—AL—and SES. These studies have generally found an inverse relationship between SES and AL. However, although the model of AL presupposes the accumulation of insults over a long time, so far only cross-sectional studies have been conducted on the subject.

What this study adds

This paper extends previous cross-sectional findings by reporting an association between SES over the life course and AL in mid-adulthood, consistent with the life-course model of cumulative risk. These results indicate that socioeconomic circumstances over the life course may be important for the development of AL.

CONCLUSIONS

As the first longitudinal study examining associations between SES and AL, our findings give strong support for the cumulative risk model, moderate support for the social chain of risk model in the gender collapsed analyses only, and moderate support for adolescence as a critical period in women only. These findings indicate that life-course processes might be relevant for socioeconomic influences on AL in adulthood.

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