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History of Socioeconomic Disadvantage and Allostatic Load in Later Life

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Abstract

There is a growing interest in understanding how the experience of socioeconomic status (SES) adversity across the life course may accumulate to negatively affect the functioning of biological regulatory systems important to functioning and health in later adulthood. The goal of the present analyses was to examine whether greater life course SES adversity experience would be associated with higher scores on a multi-system allostatic load (AL) index of physiological function in adulthood. Data for these analyses are from 1,008 participants (92.2% White) from the Biomarker Substudy of the Study of Midlife in the US (MIDUS). Multiple indicators of SES adversity in childhood (parent educational attainment, welfare status, financial situation) and two points in adulthood (educational attainment, household income, difficulty paying bills, availability of money to meet basic needs, current financial situation) were used to construct SES adversity measures for each life course phase. An AL score was constructed using information on 24 biomarkers from 7 different physiological systems (sympathetic and parasympathetic nervous systems, hypothalamic-pituitary-adrenal axis, cardiovascular, lipid metabolism, glucose metabolism, inflammatory immune activity). Analyses indicate higher AL as a function of greater SES adversity at each phase of, and cumulatively across, the life course. Associations were only moderately attenuated when accounting for a wide array of health status, behavioral and psychosocial factors. Findings suggest that SES adversity experience may cumulate across the life course to have a negative impact on multiple biological systems in adulthood. An important aim of future research is the replication of current findings in this predominantly White sample in more ethnically diverse populations.

Keywords

socioeconomic status; SES; allostatic load; biomarkers; health inequalities; life course; USA

A voluminous literature documents an inverse association between socioeconomic status (SES) and health, such that occupation of a lower SES position is associated with greater risk of a wide array of adverse health outcomes (Adler et al., 1994; Kaplan & Keil, 1993). This includes risk of development of infectious illness, as well as chronic health conditions,

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such as heart disease, diabetes, and poor cognitive and physical functioning (see Cohen, 1999; Kaplan & Keil, 1993; Strike & Steptoe, 2004; Tamayo et al., 2010). Those of lower SES are also at greater risk of disease-specific and all-cause mortality (e.g., Lynch et al., 1994; Turrell et al., 2007).

That the potential ill effects of low SES can be observed across such a wide range of conditions suggests common biological mechanisms through which SES adversity is linked to health. A general conceptualization of the mechanisms, offered by many theorists (e.g., Gallo & Matthews, 2003; Seeman & Crimmins, 2001; Williams, 1990), through which SES variations may be linked to variations in biological functioning is provided in Figure 1. SES-patterned environmental exposures, and psychological, social and behavioral processes, are hypothesized to affect the functioning of various biological regulatory systems important to health. These include primary regulatory systems, such as the neuroendocrine and nervous systems which respond to internal and external demands and which, in turn, affect the activity of secondary regulatory systems, such as the immune, cardiovascular and metabolic systems, that carry out biological activities to meet such demands. The underlying hypothesis of this general conceptual model is that those of lower SES are subject to environmental, psychological and behavioral characteristics and experiences that more often put demands on these biological systems, leading to greater system wear and tear over time, and subsequently enhancing risk for poor health and functioning.

Evidence for SES gradients in biomarkers of these potential physiological pathways to disease is accumulating. Lower SES, assessed by a variety of indicators (education, income, occupational status, financial strain), has been linked to more “risky” patterns of biological functioning, including higher levels of hormones hypothesized to be elevated under conditions of stress (e.g., sympathetic nervous system and hypothalamic-pituitary-adrenal hormones, e.g., Cohen et al., 2006; Janicki-Deverts et al., 2007; Rosmond & Bjorntorp, 2000; Steptoe et al., 2003), poorer metabolic profiles (e.g., greater body mass index, higher fasting glucose and insulin and glycosylated hemoglobin, poorer lipid profiles; Danese et al., 2009; Loucks et al., 2007; McLaren, 2007; Senese et al., 2009), and other indicators of cardiovascular disease risk (e.g., high blood pressure, low heart rate variability; Colhoun et al., 1998; Hemingway et al., 2005; Sloan et al., 2005). Circulating levels of C-reactive protein, fibrinogen and other indicators of inflammatory burden, have also been found to be greater in those of lower SES (e.g., Brunner et al., 1996; Gruenewald et al., 2009; Hemingway et al., 2003; Koster et al., 2006).

A number of investigations have also documented SES gradients in multi-system physiological indices, often referred to as measures of allostatic load (AL) (McEwen, 1998; McEwen & Stellar, 1993; Seeman et al., 1997), which assess risk across a wide array of biomarkers or across multiple biological systems. Given that the experiential and behavioral correlates of SES likely affect, and risk for most major morbid conditions is affected by, the functioning of multiple physiological systems, multi-system AL indices may provide a better picture of the physiological toll that SES adversity experience imparts on the body. AL levels, assessed with a variety of scoring methodologies, have been found to be higher in those of lower SES (Crimmins et al., 2009; Geronimus et al., 2006; Kubzansky et al., 1999; Seeman et al., 2004; Singer & Ryff, 1999; Weinstein et al., 2003).

Increasing interest is also being accorded to the time course of such associations, that is, how experience of SES adversity across the life course is linked to biological functioning in adulthood. A number of models have been advanced to explain the potential association between life course SES and health, which may be applicable to the study of physiological pathways to disease, including the *accumulation of risk, status mobility*, and *sensitive* or *critical periods* models (Ben-Shlomo & Kuh, 2002; Pollitt et al., 2005). The accumulation of

risk model posits that greater exposure to SES adversity (e.g., low levels of educational attainment, low occupational status, financial strain) accumulates across the life course to have a more negative impact on physiological functioning and health in later adulthood. This accumulative process is also captured in theories of the *aging* or *weathering* of biological systems under conditions of chronic adversity (e.g., Geronimus et al., 2006). An additive process is also acknowledged in the status mobility framework, in which those who persistently experience a low status position across the life course are expected to fare the worst, while the upwardly mobile are expected to benefit physiologically from status improvements over the life course. Sensitive or critical periods models suggest that SES adversity experience may have a differential effect on physiological functioning depending on the life course phase in which adversity is experienced (e.g., early life SES adversity experience may permanently tune developing biological systems).

Several studies provide support for the accumulation of risk hypothesis in that cumulative measures of SES adversity across childhood and adulthood are stronger predictors of physiological risk, such as high inflammatory burden (e.g., Loucks et al., 2010; Pollitt et al., 2008) and weight gain (e.g., Baltrus et al., 2005; Senese et al., 2009), than measures from single points in the life course. Support has been less consistent for the protective effects of upward mobility (Pollitt et al., 2005), and adult SES measures often have greater explanatory power than childhood measures, although some investigations find significant associations for childhood SES independent of adult SES (e.g., Pollitt et al., 2005, 2007; Tamayo et al.). To date, research on associations between AL indices and life course SES experience is limited, although Singer and Ryff (1999) demonstrated that AL levels were highest in those of low income in adolescence and midlife, lowest in those with persistently high income, and of intermediate levels in the upwardly and downwardly mobile, in a small subsample of participants from the Wisconsin Longitudinal Study.

The goal of the present investigation is to further explore the cumulative risk hypothesis, by examining whether AL levels in adult Americans are greater in those with greater experience of SES adversity across the life course, as measured in childhood and two points in adulthood. The multiple time periods for which SES information is available also allows for explorations of the social mobility and sensitive periods hypotheses, that is, whether specific patterns of SES mobility, or the experience of SES adversity at certain life course phases (e.g., childhood versus adulthood), are differentially correlated with biological functioning in later adulthood.

Methods

Sample

Data come from the Biomarker Substudy of the Study of Midlife in the U.S. (MIDUS), a longitudinal study of psychosocial, behavioral, and sociodemographic correlates of healthy aging. In 1994–1995, a national sample of 3,487 individuals were surveyed via telephone using random digit dialing, with 3,034 of the respondents completing an additional mail survey. Samples of siblings of randomly dialed respondents (n = 950) and twins (n = 1,914) were also included in the baseline cohort. The original cohort was resurveyed via phone (n = 4,474) and mail (n = 3,637) approximately ten years later. The current analyses focus on a subset of individuals (n = 1,054) who participated in the Biomarker Substudy at the second MIDUS wave. Substudy subjects participated in an overnight visit at one of three regional centers (Georgetown, DC; Los Angeles, CA; Madison, Wisconsin), which included a medical exam/history and the collection of a wide array of biomarkers. Substudy participants were comparable to the larger MIDUS cohort on demographic (age, race/ethnicity, marital status, income) and health characteristics (e.g., self-rated health, number of health conditions, impairments in activities of daily living), with the exception of substudy

participants having higher educational attainment (e.g., 42.1% college degree or greater versus 34.5% in the larger sample; see Love et al., 2010, for additional details on sample and substudy protocol). The Biomarker Substudy was approved by the Institutional Review Boards of the University of Wisconsin-Madison, the University of California, Los Angeles, and Georgetown University.

Of the 1,054 individuals from the baseline MIDUS cohort who participated in the Biomarker Substudy, 1,008 had sufficient data to construct SES disadvantage and multi-system physiological risk scores (9 missing sufficient biological data; 37 without complete SES data). Mean or mode substitution was used for the small proportion (.1 – 1.0 %; n = 1 to 10 cases) of missing data on covariates included in multivariate analyses.

Measures

Unless otherwise noted, all measures were collected during the Biomarker Substudy visit.

Physiological biomarkers—A wide range of biomarkers representing different physiological systems were collected during the study visit. Measures of *cardiovascular* functioning included resting systolic and diastolic blood pressure (SBP, DBP) and resting pulse. Indicators of *sympathetic nervous system* (SNS) activity included overnight urinary measures of epinephrine and norepinephrine. Measures of *parasympathetic nervous system* (PNS) activity included the following heart rate variability parameters: low and high frequency spectral power, the standard deviation of R-R (heartbeat to heartbeat) intervals (SDRR), and the root mean square of successive differences (RMSSD). Indicators of *hypothalamic pituitary adrenal* (HPA) *axis activity* included an overnight urinary measure of the hormone cortisol and a serum measure of the hormone dehydroepiandrosterone sulfare (DHEAS). Measures of *inflammation* included plasma C-reactive protein (CRP), fibrinogen, and serum measures of interleukin-6 (IL-6) and the soluble adhesion molecules e-Selectin and intracelleular adhesion molecule-1 (ICAM-1). Indicators of *lipid and general metabolic activity* included high density lipoprotein (HDL) cholesterol, low density lipoprotein (LDL) cholesterol, triglycerides, body mass index (BMI), and waist-hip ratio (WHR). Levels of glycosylated hemoglobin, fasting glucose, and the homeostasis model of insulin resistance (HOMA-IR), served as measures of *glucose metabolism*. Details on the measurement and assay of biomarkers are provided in supplementary data file 1

Allostatic load—A multi-system allostatic load (AL) score was computed as the sum of seven separate physiological system (SNS, PNS, HPA, cardiovascular, glucose metabolism, lipid, and inflammation) risk indices. System risk indices were computed as the proportion of individual biomarker indicators for each system (ranging from 2 to 6 biomarkers) for which participant values fell into high-risk quartile ranges (upper or lower quartile depending on whether high or low values of the biomarker typically confer greater risk for poor health outcomes; see Table 1); scores were only computed for individuals with values on at least half of the system biomarkers. System risk scores could range from 0 to 1 (indicating 0 –100% of system biomarkers in high-risk range for a given participant). As the number of biomarker indicators varied across the seven physiological systems, this average risk scoring method produced a similar scaling of risk scores across the different systems. An AL score was computed as the sum of the seven system scores (possible range: 0 to 7) for participants with information on 6 or 7 of the 7 systems.

SES disadvantage variables—SES disadvantage variables were created for three time periods: childhood, and MIDUS I (MI) and MIDUS II (MII) adult periods. The childhood SES disadvantage score was computed by summing values on 3 indicators: financial level growing up (2 - worse off than others, 1 - about the same as others, 0 - better off than

others), highest level of parental education (2 - less than high school, 1 - high school/GED, 0 - some college or higher), and childhood welfare status (2 - ever on welfare, 0 - never on welfare). Information on childhood SES was collected retrospectively at the MI exam. MI and MII adult SES disadvantage scores were computed by summing values on 5 indicators at each timepoint: education level (2 - high school/GED or less, 1 - some college/associate arts degree, 0 - bachelor's degree or higher), family-size adjusted income to poverty ratio (2 - less than 300%, 1 – 300–599%, 0 – 600% or more), current financial situation (2 - worst possible, 1 - average, 0 - best possible), availability of money to meet basic needs (2 - not enough, 1 - just enough, 0 - more than enough), and difficulty level of paying bills (2 - very or somewhat difficult, 1 - not very difficult, 0 - not at all difficult). A cumulative disadvantage score was created by summing the childhood, MI and MII adult disadvantage scores. Possible score ranges were as follows: 0–6 for childhood SES, 0–10 for MI and MII adult, and 0–26 for the cumulative SES disadvantage score.

Sociodemographic covariates—Age was coded in years. Gender was coded as male or female. Race/ethnicity was coded as White or non-White given the small number of non-White participants (Black/African-American n = 25, multiracial n = 37, other n = 17).

Health conditions—A summary index of 45 major (e.g., cancer, heart disease, stroke, diabetes) and more minor (e.g., migraine headaches) health conditions participants reported ever experiencing was calculated, to capture burden of poor health across a range of conditions.

Health behavior covariates—Alcohol consumption was categorized as: never drink or did not drink in last month, drank less than once a week in last month, or drank once a week or more in last month. Smoking status was coded as non-smoker, ex-smoker, or current smoker. Fast food consumption frequency was rated on a 5-point scale (ranging from 1 - never to 5 – 7 or more times per week). A summary physical activity score was computed as the weighted sum of participants responses to three questions asking about frequency of engagement (6-point scale ranging from 1 - never to 6 - several times a week) in *light* ("that which requires little effort," e.g., light housework, easy walking), *moderate* ("not physically exhausting, but it causes your heart rate to increase slightly and you typically work up a sweat," e.g., light tennis, brisk walking), and *vigorous* ("causes your heart to beat so rapidly that you can feel it in your chest and you perform the activity long enough to work up a good sweat and are breathing heavily," e.g., vigorous swimming, high intensity aerobics) activity as of the MII main study survey (weights of 1, 3, and 5 for light, moderate and vigorous activity, respectively, were used to provide greater weight to relatively more vigorous activity in the summary score; possible score range from 9 to 54).

Distress covariates—Perceived stress was assessed with the Perceived Stress Scale (Cohen et al., 1983), a ten-item scale of perceived frequency (1 - never to 5 - very often in past month) of feelings of stress or strain (possible summary score range of 10 to 50; $\alpha = .86$ for study sample). Depressive symptomatology was measured with the widely used Center for Epidemiologic Studies Depression Scale (Radloff, 1977), which assesses the frequency (0 = rarely or none of the time (less than 1 day) to 3 = most or all of the time (5–7 days)) of twenty depressive symptoms during the past week (possible summary score range of 0 to 60; $\alpha = .89$ for study sample). Anxious symptomatology was measured with the General Distress - Anxious Symptoms subscale of the Mood and Symptom Questionnaire (Clark & Watson, 1991), which assesses the degree of experience (1 - not at all to 5 - extremely) of 11 symptoms of anxiety during the past week (possible summary score range of 11 to 55; $\alpha = .80$ for study sample).

Positive affect and positive life experiences covariates—The Positive Affect subscale of the Mood and Symptom Questionnaire was used to assess the degree of experience (1 - not at all to 5 - extremely) of 14 indicators of positive affect (e.g., cheerful, really happy) during the past week (possible summary score range of 14 to 70; $\alpha = .94$ for study sample). An index of the frequency of positive experiences in the past month was computed as the mean of frequency ratings (1 - never, 2 – 3 to 6 times in last month, 3 – 7 or more times) of 49 positive experiences (e.g., seeing beautiful scenery, taking a relaxing bath, being with happy people) adapted from the Positive Events Schedule (MacPhillamy & Lewinsohn, 1982).

Perceived mastery and constraints covariates—Perceived mastery and constraints were assessed with mean scores for ratings of agreement (1 - strongly disagree to 7 - strongly agree) with four items designed to measure personal mastery (e.g., “I can do just about anything I really set my mind to”) and eight items designed to measure perceptions of constraints in carrying out life activities (e.g., “I have little control over the things that happen to me”; see Lachman & Weaver, 1998) from the main MII study survey (α’s = .74 and .86 for mastery and constraint measures, respectively, in study sample).

Social contact, support and conflict variables—Frequency of contact with family and friends was assessed with an 8-point scale (1 - never/hardly ever to 8 - several times a day). Scores for perceived support and conflict with family and friends were calculated (separately) by taking the mean of four ratings (1 - not at all to 4 - a lot) to assess perceived level of support (e.g., “how much can you rely on family (or friends) for help with a serious problem?”) and four ratings to assess perceived level of conflict/demands (e.g., “how often do family members (or friends) make too many demands on you?”). Social support, conflict, and contact variables were assessed at the MII main study assessment.

Analyses—Generalized estimating equation (GEE) models (specifying an exchangeable correlation matrix), which can account for clustering by family membership (the sample included participants from the sibling/twin subsamples of the main MIDUS Study) and substudy data collection site, were used to assess the association between levels of SES disadvantage and AL. Separate models were run for childhood, MI and MII adult, and cumulative SES disadvantage variables (measures were z-scored to allow comparison of parameter estimates across models). Given the large number of covariates targeted for exploration in analyses, a series of multivariate models was conducted which included different sets of psychosocial, behavioral, and health status covariates in each model. The baseline model (Model 1) for each SES disadvantage predictor included age, gender, and race/ethnicity. Models 2 – 7 added the following covariates to the baseline model: model 2: health conditions; model 3: health behaviors; model 4: distress variables; model 5: frequency of positive experiences and positive affect; model 6: perceived mastery and constraints, and model 7: social contact, support, and conflict. A final multivariate model (model 8) included the baseline model covariates and significant ($p \leq .10$) psychosocial, behavioral and health covariates from models 2 –7. AL levels were graphed according to quintiles of each SES disadvantage variable for descriptive purposes.

Results

Descriptive statistics for individual biomarkers and the multi-system AL score are detailed in Table 1. Sample-derived high-risk quartile cutpoints for the biomarkers were similar to standard clinical risk cutpoints or “borderline” or “moderate” risk clinical cutpoints (see Table 1). Average AL level was rather moderate in the sample (M = 1.72, SD = 1.02; range 0 – 4.8; possible range of 0–7), although there was considerable variability in the range of

scores. Descriptive statistics for demographic, SES disadvantage, psychosocial, behavioral and health status variables are depicted in Table 2. Mean levels of SES disadvantage fell on the lower end of the scale of each measure, however, there was substantial variability in the range of scores (see Table 2).

As detailed in Figure 2, mean AL levels (derived from demographic-adjusted GEE models) were higher in those in higher quintile brackets on each SES disadvantage measure (childhood, MI and MII adult, and cumulative). As detailed in Table 3, greater levels of all four of the SES disadvantage variables (continuous z-scored measures) were significantly associated with greater AL levels in GEE models controlling for demographic variables. Comparison of parameter estimates indicates slightly stronger associations for the cumulative and adult as compared to childhood SES disadvantage measures.

Results for models 2 to 7 indicate minor reductions in the magnitude of associations between SES disadvantage variables and AL levels with the inclusion of different sets of health status, health behavior and psychosocial variables in GEE models. A final multivariate model (Model 8) for each SES disadvantage variable including demographic and significant ($p \leq .10$) covariates from models 2 to 7, led to a moderate reduction in the parameter estimate for each SES disadvantage predictor (reductions of 36%, 40%, 38%, and 35%, for childhood, MI adult, MII adult, and cumulative SES disadvantage variables, respectively), although associations between SES disadvantage variables and AL remained statistically significant in each model. Significant covariate predictors in this final model included age, number of health conditions, current smoker status, anxiety (marginally significant) and frequency of fast food consumption (associated with higher levels of multi-system physiological risk), and light alcohol consumption and frequency of contact with friends (associated with lower AL levels).

Additional demographically-adjusted GEE analyses examined AL levels by four patterns of SES mobility from childhood to MII adulthood: (1) low SES in both childhood and MII adulthood, (2) downwardly mobile (high childhood SES/low MII adult SES), (3) upwardly mobile (low childhood SES/high MII adult SES), and (4) high SES in both childhood and MII adulthood (median splits on SES disadvantage scores for childhood (score splits: 0–1, 2–6) and the adult MII measure (score splits: 0–4, 5–10) were used to construct high and low SES groups). AL levels for each mobility pattern group are depicted in Figure 3. Those with persistent low SES from childhood to MII adulthood had the highest AL levels, followed by the downwardly mobile, then the upwardly mobile, and those with persistently high SES had the lowest AL levels. Pairwise comparisons indicated significantly higher AL levels for those with persistently low SES compared to the the upwardly mobile and those with persistently high SES, and significantly higher AL levels for the downwardly mobile compared to those with persistently high SES (p s < .05).

The question of whether SES adversity experience at certain lifecourse periods is more strongly linked to adult AL levels was examined by including SES disadvantage scores from each lifecourse period (childhood, MI adult, MII adult) simultaneously in a GEE model (including demographic covariates). Each SES disadvantage score was a significant or marginally significant predictor of AL levels. The coefficient for MII adult SES disadvantage was twice the size of that for childhood and MI adulthood (childhood B = .06, p = .04; MI B = .06, p = .10; MII B = .13, p = .001), but specific contrast tests indicated that these differences were not statistically significant (p s > .05).

Supplementary analyses

Although age was included as a covariate in all analyses, it is possible that the association between SES disadvantage history and AL may vary by age. However, age did not significantly interact with SES disadvantage scores to predict AL levels in analyses.

In addition, although our focus in this analysis is to examine associations between SES disadvantage indicators and levels of a multi-system AL index, we acknowledge that associations may vary by the individual physiological systems. Greater cumulative SES adversity was associated with higher scores on each of the physiological subsystem risk scores (p s < .05), with the exception of the SNS and PNS (although trends were evident for these systems; see Figure 1 in supplementary online text). Examination of physiological system scores by patterns of mobility generally indicated higher (significant or marginally significant) system risk scores in the persistently low versus persistently high SES group across childhood and adulthood with the exception of the PNS (see supplemental Figure 2). Analyses did not suggest stronger associations between system risk scores and SES adversity at a specific time period with the exception of inflammation, for which the MII adult SES coefficient was significantly larger in magnitude than the coefficient for childhood SES (see supplemental Table 1)

Additional supplementary analyses (data not shown) indicated that patterns of associations between SES predictors and AL were similar for more traditional AL operationalizations using fewer biomarker indicators and simply counting up the number of biomarker indicators for which participant values fell into high-risk quartiles (original 10-item formulation used in Seeman et al., 1997 and 10-item formulation using biomarkers typically incorporated into AL scores in the National Health and Nutrition Examination Surveys (SBP, DBP, pulse, waist-hip ratio, total cholesterol, HDL cholesterol, triglycerides, body mass index, glycosylated hemoglobin, CRP; e.g., Crimmins et al., 2009; Geronimus et al., 2006; Seeman et al., 2008). These results suggest that observed SES variations in AL levels are fairly robust to different methods of assessing AL, including those which rely on fewer biomarker indicators.

Discussion

Findings indicate higher levels of allostatic load in middle and later adulthood in individuals who have experienced a greater level of SES adversity across the life course from childhood to adulthood. Greater AL in those with greater life course SES adversity was observed whether cumulative SES adversity was assessed as higher scores on a summary measure incorporating SES adversity information from childhood and two points in adulthood, or when assessed as persistent SES adversity in both childhood and adulthood. These findings support the cumulative risk hypothesis, such that greater experience of SES adversity across the life course may cumulate to have a greater negative effect on biological functioning in later adulthood. Higher AL may be one pathway through which greater life course SES adversity leads to greater risk for morbidity and mortality in later adulthood.

When examined simultaneously in analytic models, greater SES adversity at each time period was a significant or marginally significant independent predictor of higher AL. Although the magnitude of association between AL and recent adult SES adversity was twice that of the associations for childhood and earlier adult SES adversity measures, these differences were not statistically significant. Thus, biological functioning in middle and later adulthood may be particularly affected by recent SES adversity experience, but still bear the scars of SES adversity experience earlier in the life course. A number of investigations have found that associations between childhood SES and health indicators in adulthood are attenuated when accounting for adult SES, although, similar to the present findings, some

investigations still find an independent association for childhood SES (e.g., Galobardes et al., 2008; Haas, 2008; Tamayo et al., 2010).

Analysis of AL levels by patterns of SES mobility from childhood to adulthood indicated that downwardly mobile participants had significantly higher AL levels than the persistently advantaged participants. It is not clear whether this indicates a negative influence of recently experienced SES adversity on biological functioning or the negative impact of losing status and resources, or both. The upwardly mobile participants, on the other hand, had only slightly higher AL levels than the persistently advantaged participants, and the difference in scores was not statistically significant, suggesting that those who experienced improvements in SES from childhood to adulthood looked similar biologically to those with persistent high SES.

In general, the inclusion of a wide array of different domains of potential mediators (health status, behavioral, psychosocial) in analytic models resulted in little change in the magnitude of the associations between SES variables and AL. However, in a final multivariate model which adjusted for a number of covariate factors found to be significant predictors in previous models, the reduction in the magnitude of parameter estimates for SES predictors ranged from 35 to 40%. Significant covariate predictors in this final model included age, health condition burden, smoking, frequency of fast food consumption, light alcohol consumption and frequency of contact with friends (both of the latter associated with lower AL). These findings suggest that health behaviors and social contact characteristics may be pathways that explain how those of lower SES have poorer biological functioning, but much of this association remains to be explained. One limitation of these analyses is that measures of some of these covariate factors were only collected as part of the MII Biomarker Substudy. A greater mediating role may have been observed if life course information (i.e., from childhood and the first adult assessment) on all of these characteristics and behaviors had been available.

An additional limitation of the current analyses is that information on biological functioning is not available for childhood and the first adult assessment. Thus, we cannot examine the cross-time and longitudinal patterns of associations that would best inform our understanding of the temporal and directional patterns of associations between SES adversity experience and biological functioning across the life course. The lack of childhood health information also renders it impossible to rule out the possibility that poor physiological/physical health in childhood is the cause, rather than the consequence, of downward SES mobility or persistently low SES.

Another limitation is that the retrospective assessment of childhood SES characteristics raises concerns about the accuracy or reliability of the childhood SES indicators. There has been little empirical evaluation of the accuracy of recall of childhood SES characteristics, although Krieger and colleagues (1998) documented good agreement between adult twins in recall of father's educational attainment (concordance = 91%) and occupational status (concordance = 80%) in childhood. Our own analysis of MIDUS sibling data also indicates moderate to high sibling agreement for the childhood SES indicators used in the present analyses (3-category parental education: intraclass correlation (ICC) = .78, concordance = 78%; childhood family welfare status: ICC = .55, concordance = 95%; 3-category rating of childhood family financial status: ICC = .54, concordance = 60%). In addition, although the multi-indicator childhood SES composite may provide a more comprehensive assessment of SES adversity in childhood, additional investigation of the construct validity of such composite measures is needed. Less than perfect measurement of childhood SES characteristics and measurement error may be potential explanations for the lower

magnitude of association of AL with childhood as compared to adult SES adversity measures.

Although the Biomarker Substudy sample was drawn from the larger MIDUS sample, the MIDUS sample is not nationally representative, with the a lower representation of ethnic minorities and low SES individuals than in the general U.S. population, raising a concern about generalizability of study findings to non-White populations. This concern is underscored by research that documents racial/ethnic variations in the magnitude, direction and/or significance of associations between SES and health indicators, with typically weaker or reverse associations in non-Whites as compared to Whites (see Pearson, 2008, for a review). An important future direction is the replication of current findings in other ethnic/racial populations.

Despite the limitations outlined above, the current analyses provide a number of important contributions to the literature on biological correlates of socioeconomic adversity experience. To our knowledge, this is the first comprehensive examination of variations in allostatic load as a function of socioeconomic adversity at different phases of the life course, cumulatively across the life course, and by patterns of SES mobility from childhood to later adulthood. The AL index used in the current analyses is also the most comprehensive assessment of allostatic load to date, assessing AL as a summary index of average risk across 7 physiological systems, represented by a total of 24 different biomarkers. However, we also find reassuring our supplementary analyses documenting similar patterns of results when using more traditional AL indices, constructed from simple summary counts of a smaller set of biomarker indicators, suggesting that findings are not simply an artifact of the AL measurement used in the this investigation.

An important future direction for research in this area includes concurrent examination of SES adversity experience, psychosocial and behavioral characteristics, and allostatic load, at multiple points in the life course, to better understand how SES adversity experience at different life course phases affects health. Another important future direction is understanding the behavioral and psychosocial trajectories of individuals who experienced upward versus downward SES mobility. As those who moved from low SES in childhood to high SES in adulthood looked similar biologically to those with persistently high SES, a better understanding of the behavioral and psychosocial characteristics of the upwardly mobile may provide information on foci for interventions targeted to improve socioeconomic and biological well-being. The clear finding of greater AL levels in those with persistent experience of SES adversity suggest that such intervention efforts may be quite valuable to individual health and well-being.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Research Highlights

- In a US sample, life course patterns of socioeconomic adversity were examined as predictors of a multi-system allostatic load index.
- Greater cumulative life course socioeconomic adversity is linked to higher allostatic load in later adulthood.
- Findings also suggest a potential protective effect of upward mobility on allostatic load levels in adulthood.

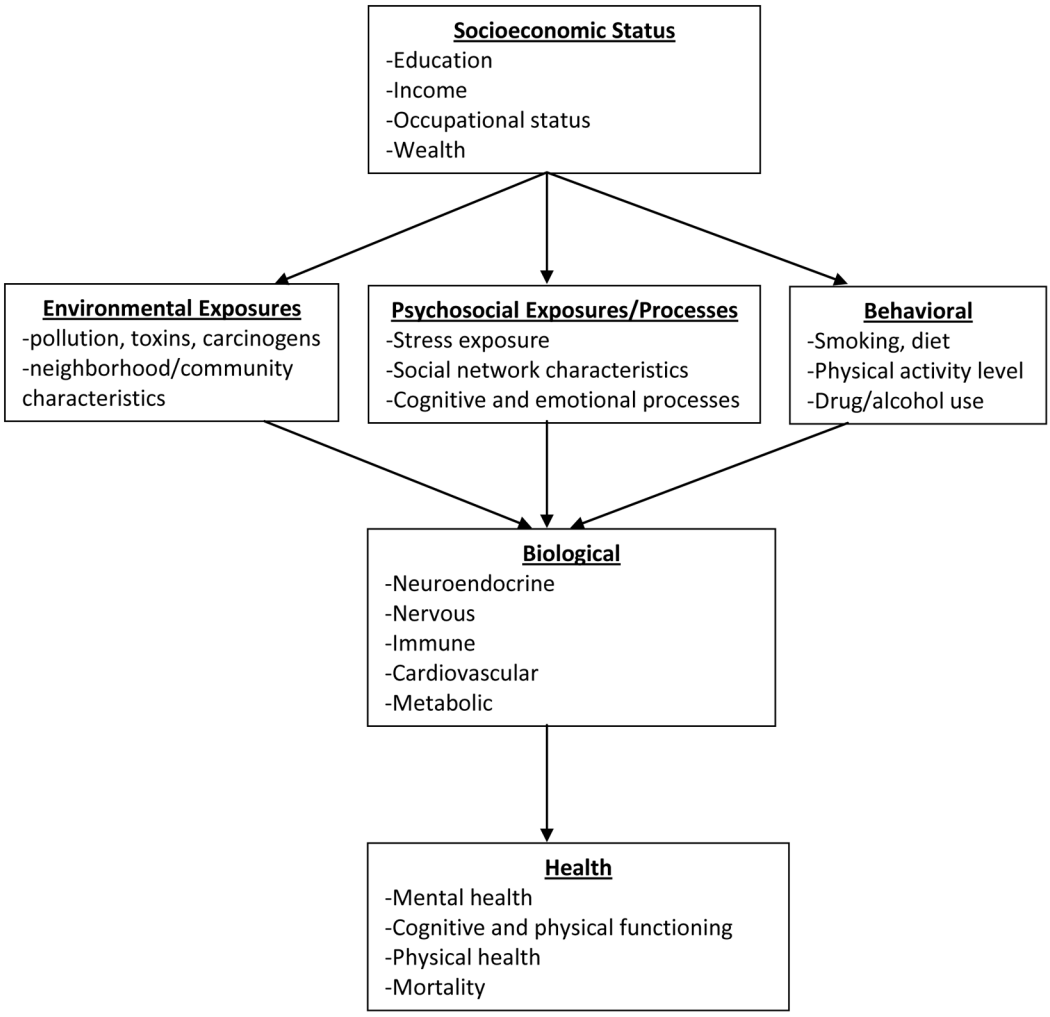

Figure 1.
Conceptual model of potential pathways through which social status is linked to health.

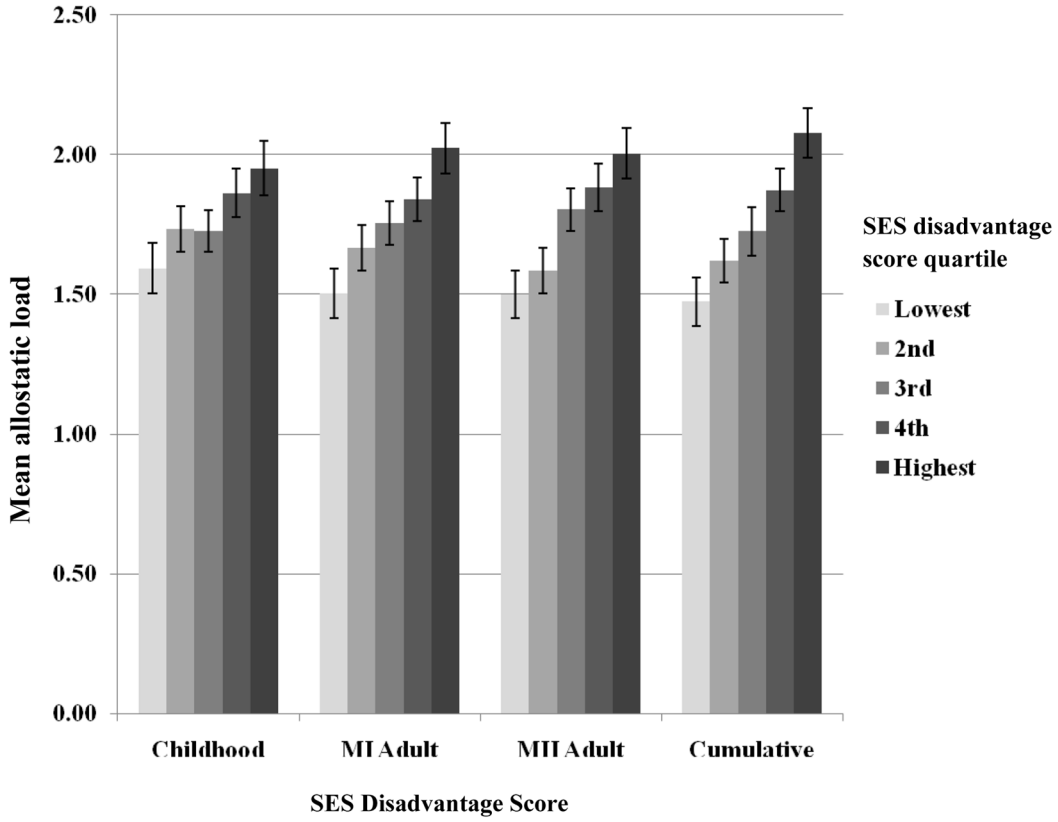

Figure 2.
Mean levels of allostatic load by quintiles of SES adversity measures.

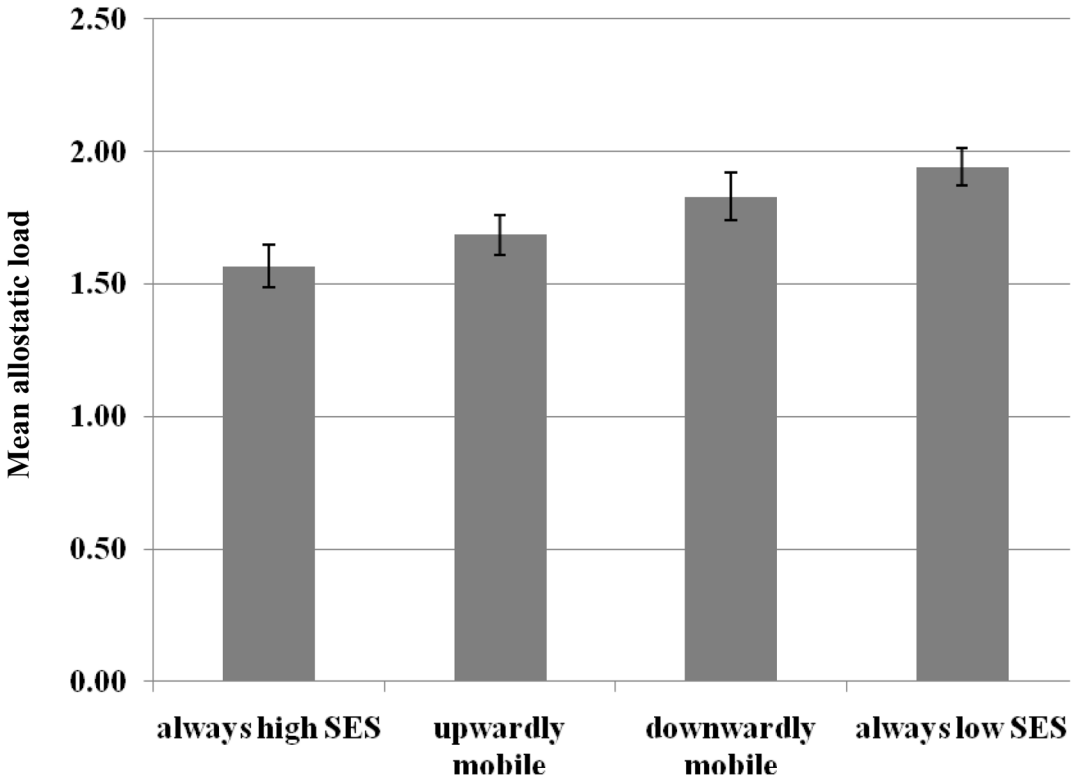

Figure 3.
Mean levels of allostatic load by patterns of SES mobility from childhood to adulthood.

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Table 1

Descriptive statistics and high-risk cutpoint values for individual biomarkers and the multi-system allostatic load index.

System and representative biomarkers	N	M	SD	High-risk cutpoint	Clinical cutpoint
Cardiovascular					
Resting SBP (mmHg)	1008	130.98	17.54	≥ 143.00	≥140 (≥120)
Resting DBP (mmHg)	1008	74.90	10.20	≥ 82.00	≥90 (≥80)
Resting heart rate (bpm)	1007	70.55	11.15	≥ 77.00	> 90 (>80)
Metabolic - lipids					
BMI	1008	29.13	5.95	≥ 32.31	≥25, ≥30
WHR	1007	0.89	0.10	≥ 0.97	>1 (>.9)
Triglycerides (mg/dL)	1006	130.63	80.27	≥ 160.00	≥200 (≥150)
HDL Cholesterol (mg/dL)	1006	54.76	17.64	≤ 41.37	<40
LDL Cholesterol (mg/dL)	1006	106.31	35.00	≥ 128.00	≥160 (≥130)
Metabolic - glucose metabolism					
Glycosylated hemoglobin (HbA1c)	1003	5.98	.89	≥ 6.10	≥7 (> 6.4)
Fasting glucose (mg/dL)	1001	100.23	23.27	≥ 105	≥126 (> 100)
Insulin resistance (HOMA-IR)	1000	3.25	3.11	≥ 4.05	
Inflammation					
CRP (mg/L)	1002	2.66	3.92	≥ 3.18	> 3
IL6 (pg/mL)	1007	2.78	2.78	≥ 3.18	
Fibrinogen (mg/dL)	1003	341.23	84.20	≥ 390.00	
sE-Selectin (ng/Ml)	1007	41.62	20.84	≥ 50.58	
sICAM-1 (ng/Ml)	1007	286.67	100.69	≥ 329.65	
Sympathetic Nervous System					
Urine Epinephrine (ug/g creatine)	991	2.04	1.29	≥ 2.54	
Urine Norepinephrine (ug/g creatine)	997	27.71	13.04	≥ 33.33	
Hypothalamic Pituitary Adrenal Axis					
Urine Cortisol (ug/g creatine)	1006	16.54	16.35	≥ 21.00	
Blood DHEA-S (ug/dL)	1003	105.65	76.43	≤ 51.00	
Parasympathetic Nervous System					
SDRR (msec)	928	35.12	17.02	≤ 23.54	
RMSSD	928	21.40	15.24	≤ 11.83	

System and representative biomarkers	N	M	SD	High-risk cutpoint	Clinical cutpoint
Low frequency spectral power	928	426.65	630.90	≤ 113.96	
High frequency spectral power	928	259.13	446.49	≤ 54.16	
Allostatic load	1008	1.72	1.02		

Clinical cutpoint values in parentheses note cutpoints for borderline or moderate risk for disease outcomes.

Table 2

Descriptive statistics for demographic, SES disadvantage, psychosocial, behavioral and health variables.

	%	M	SD	Range
Demographic covariates				
Age		58.07	11.57	35 – 85
Gender				
Male	45.2			
Female	54.8			
Race/ethnicity				
White	92.2			
Non-white	7.8			
SES disadvantage scores				
Childhood		1.87	1.40	0 –6
MIDUS I Adult		4.58	2.62	0 – 10
MIDUS II Adult		4.30	2.68	0– 10
Cumulative (childhood, MI and MII adult)		10.75	5.30	0 – 24
Health condition index		5.07	3.46	0 – 32
Health behavior covariates				
Physical activity summary score		29.92	10.56	9 – 54
Fast food consumption frequency		2.43	0.91	1 – 5
Alcohol use (last month)				
Non-drinker or rarely drink	32.0			
Light drinker (less than once a week)	28.2			
Moderate+ drinker (once a week or more)	39.8			
Smoking status				
Non-smoker	55.7			
Ex-smoker	32.9			
Current smoker	11.4			
Perceived mastery/control covariates				
Personal mastery		5.79	1.00	1.25 –7.00
Perceived constraints		2.40	1.09	1 – 7.00
Social contact and support covariates				
Frequency of contact with family		5.98	1.44	1 – 8
Frequency of contact with friends		5.71	1.68	1 – 8
Perceived social support - family		3.54	0.59	1 – 4
Perceived social support - friends		3.33	0.64	1 – 4
Distress covariates				
Perceived stress		21.59	6.17	10 – 48
Depressive symptomatology		7.90	7.66	0 – 49
Anxious symptomatology		16.51	4.48	11– 47
Positive experience frequency		2.27	0.28	1.39 – 2.87
Positive affect		44.80	10.18	14 – 70

Table 3

Generalized estimating equation (GEE) model parameter estimates for levels of allostatic load by SES disadvantage variables in multivariate models.

	Childhood disadvantage score (z-scored)	MI Adult disadvantage score (z-scored)	MII Adult disadvantage score (z-scored)	Summary disadvantage score (z-scored)
Model 1: baseline (includes age, gender, race/ethnicity)	.10**	.16***	.18***	.20***
Model 2: add chronic conditions to baseline model	.09**	.14***	.15***	.17***
Model 3: add health behavior covariates to baseline model	.07*	.13***	.15***	.16***
Model 4: add distress covariates to baseline model	.09*	.13***	.15***	.17***
Model 5: add positive life experiences and positive affect covariates to baseline model	.09*	.15***	.17***	.19***
Model 6: add mastery and constraints covariates to baseline model	.10**	.14***	.16***	.18***
Model 7: add social contact, support and conflict covariates to baseline model	.09**	.15***	.17***	.19***
Model 8: final model with significant covariate predictors from models 2–7	.07*	.10**	.11***	.13***

Note 1. Model 8 covariates: chronic conditions, alcohol use, smoking status, frequency of fast food consumption, anxiety, perceived constraints, frequency of positive experiences, frequency of conflict with family, and frequency of contact with friends.

Note 2. Parameter estimates derived from GEE models accounting for clustering by family and data collection site.

* $p \leq .05$,

** $p \leq .01$,

*** $p = \leq .001$