Socioeconomic Status, Positive Experiences, and Allostatic Load

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Objective: Socioeconomic disparities in physiological well-being may be a pathway to the poorer health outcomes observed in those of lower socioeconomic status (SES). The present research examined greater frequency of positive life experiences (POS) as a route through which greater cumulative SES (CSES) may be linked to lower allostatic load (AL), a multisystem index of physiological dysregulation, and assessed whether the association between POS and AL varies along the socioeconomic spectrum. ***Method:*** These associations were examined using data from the Midlife Development in the United States Biomarker Project ($N = 2,096$). Analyses included tests of whether positive experiences mediated the CSES–AL association, whether CSES moderated associations of positive experiences and AL, and whether CSES moderated positive experience mediation of the CSES–AL association (moderated mediation). ***Results:*** The observed association between CSES and AL was weakly mediated by POS. CSES moderated the POS–AL association, such that POS was associated with AL only at lower levels of CSES. The moderated mediation analysis showed that POS mediated the association between CSES and AL only at lower levels of CSES. ***Conclusions:*** The results suggest complexity in associations between cumulative socioeconomic advantage, positive life events, and physiological well-being. Positive life events may play a stronger role in physiological health in those of lower socioeconomic advantage, as one of multiple pathways through which lower SES is linked to poor health. Given the modifiability of access to, and frequency of, positive life events, the potential role of positive experiences in lessening health disparities warrants further study.

Keywords: positive experiences, allostatic load, socioeconomic status, socioeconomic disparities, physiological well-being

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Links between socioeconomic status (SES) and both mental and physical health are well established (Adler, 2013). Lower SES has long been associated with increased mortality (Kitagawa & Hauser, 1973), and recent research shows that socioeconomic disparities in death rates are continuing to increase in the United States, even as they decline in other high-income countries (Bosworth, 2018). There are also socioeconomic disparities in morbidity, long-standing illness (Demakakos et al., 2008; Marmot, 2004), and mental health (Reiss, 2013), particularly depression (Assari, 2017; Everson et al., 2002; Lorant et al., 2003). Socioeconomic disparities are seen in indicators across multiple systems of the body, including heart rate, body fat distribution, cortisol adaptation (Adler et al., 2000), diabetes, and cholesterol (Demakakos et al., 2008). Many of these biomarkers are viewed as preclinical indicators of eventual disease risk, and thus these physiological states may be pathways to the poorer health outcomes observed in those of lower SES.

Given these health disparities, there is an ongoing need to identify the many pathways linking SES to physiological well-being. The current study examines whether frequency of positive life experiences (POS) is a route through which SES is linked to physiological health. We focus on cumulative SES (CSES), as a large body of research indicates that both childhood SES (ChSES) and adult SES (AdSES) are linked to physiological well-being, with the strongest associations often observed for cumulative indices that incorporate SES conditions across these periods of the life course (Luo and Waite, 2005; Pollitt et al., 2005). We also use allostatic load (AL), a multisystem index of physiological dysregulation (Gruenewald et al., 2012; McEwen, 2000; Seeman et al., 1997), because it allows for subclinical observation of biological pathways associated with disparities in physical health (Duru et al., 2012; Gruenewald et al., 2009; Howard & Sparks, 2016; see Beckie 2012). Our analyses

examine multiple ways in which POS and CSES may be linked to levels of AL. We first assess whether POS may be a pathway that links CSES to AL (mediation). We then turn our attention to assessing whether the POS–AL association varies by level of CSES (moderation) as well as whether POS mediation of the CSES–AL association varies along the CSES spectrum (moderated mediation).

Pathways Linking SES to Physiological Well-Being

Many environmental and psychosocial factors likely play mediating roles in the relationship between SES and physiological well-being (Chen & Miller, 2013). For example, poorer people are more likely to live in communities with greater toxic pollution (Bullard & Wright, 1993), leading to health consequences. Those with lower SES experience more stress and possess fewer psychological resources with which to respond to stress (Adler & Snibbe, 2003), which may then play a role in SES disparities in morbidity and mortality (van Oort et al., 2005). Negative emotions such as anger, hostility, hopelessness, depression, and anxiety (Gallo & Matthews, 2003), and health-related behaviors, including smoking, fast food consumption, sleep, and exercise, may also mediate SES-health relationships (Gruenewald et al., 2012; Mulatu & Schooler, 2002).

Positive environmental, social, psychological, and cognitive factors may also play a role, either by mitigating some of the negative effects of stress, distress, and poverty or as pathways through which SES is linked to physiological and physical well-being. For example, socioeconomic disparities in mortality and cardiovascular disease have been found to be weaker for those who live in greener neighborhoods (Mitchell & Popham, 2008), and maternal warmth has been shown to buffer against the impact of poverty on physiological health outcomes in children (Evans et al., 2007) and adults (Miller et al., 2011). Positive psychological states and cognitive strategies have been linked to increased physiological and physical well-being (Billings et al., 2000; Boehm & Kubzansky, 2012; Bussell & Naus, 2010). There is evidence that optimism, sense of control, self-esteem, social support, mastery, and coping style may mediate the relationship between SES and health (Matthews et al., 2010). Positive life experiences, our current focus, may be another pathway linking SES and AL, a multisystem index of physiological dysregulation (Gruenewald et al., 2012), though this mediating pathway has not yet been assessed.

Positive Life Experiences and Health

Positive life experiences are behaviors and events that have the potential for enjoyment and reward, including experiences of relaxation, social interaction, intimacy, entertainment, nature, physical comfort, and achievement. The Pleasant Events Schedule (PES; MacPhillamy & Lewinsohn, 1982) is a commonly used instrument that assesses the frequency and perceived pleasantness of a range of positive life experiences. An obtained pleasure score can be generated for each experience as the product of the event's frequency and perceived pleasantness, allowing for calculation of a total across experiences. Other investigations have used study-specific assessments of engagement in a variety of pleasurable or leisure activities.

Greater frequency of positive experiences has been found to be associated with mental health outcomes, particularly lower levels of depressive symptomatology (Ferreira & Barham, 2018; Rider et

al., 2016). A meta-analysis (Mazzucchelli et al., 2009) indicated a similar efficacy of interventions designed to enhance pleasant experiences and gold-standard depression treatments such as cognitive behavioral therapy. Such associations between positive experiences and depression may affect physical health outcomes over time, particularly in populations experiencing high stress. In a longitudinal study of acute coronary syndrome patients, lower obtained pleasure from positive experiences predicted depression trajectory, which in turn predicted morbidity and mortality (Keegan et al., 2016).

Higher frequency of and obtained pleasure from positive experiences have also been linked to better physiological well-being. Higher obtained pleasure was associated with lower blood pressure in in-home spousal caregivers of Alzheimer patients (Mausbach et al., 2018). An intervention designed to increase the frequency of positive life experiences decreased Interleukin-6 in family caregivers of dementia patients (Moore et al., 2013). Individuals who participate in leisure activities have also been found to have lower blood pressure, cortisol, waist circumference, and body mass index (Pressman et al., 2009).

Socioeconomic Resources, Positive Experiences, and Physiological Health

Although there has been no direct examination to date, there are theoretical and empirical rationales for the hypothesis that positive experiences may play a role in associations of SES and physical and physiological health. Frequency of positive experiences has been found to be lower in those with less education and financial resources (Ferreira & Barham, 2018). Theories that seek to explain the poorer health of those with lower SES have also identified deficits in positive psychosocial resources, including positive experiences, as routes through which SES is linked to mental and physical health (Gallo et al., 2009). A study documenting higher levels of AL in those with greater cumulative (lifetime) levels of socioeconomic disadvantage also observed a slight attenuation of this association when accounting for the frequency of positive experiences (Gruenewald et al., 2012). To date, however, positive experiences have not been directly examined as a pathway through which SES may be linked to health.

The current study aims to clarify associations among CSES, positive experiences, and AL. Building on prior findings that the association between CSES and AL was slightly attenuated with the inclusion of positive experience frequency (Gruenewald et al., 2012) in a cohort of $N = 1,008$ from the Midlife Development in the United States (MIDUS) Study (http://midus.wisc.edu), the present investigation aims to directly examine the frequency of positive experiences as a mediator of the CSES–AL association. Our analysis utilizes a doubly expanded MIDUS sample that includes the prior sample utilized in Gruenewald et al. (2012) and a sample from a new MIDUS Refresher (MR) cohort.

A second aim is to determine whether the hypothesized association between POS and AL is moderated by SES. There is evidence that factors associated with physical health may have a stronger impact at particular levels of socioeconomic advantage, although the literature is mixed with regard to whether associations are stronger at lower or higher ends of the socioeconomic spectrum. The phrase "intervention-generated inequality" (White et al., 2009) describes outcomes of health interventions that wind up having a stronger positive impact on individuals who are already better off

and that therefore serve to increase health disparities. The observed differential impacts may be due to easier access, more frequent adoption, more adherence, or more effectiveness among those of greater advantage (Veinot et al., 2018). Similar moderation effects have been seen among other factors known to increase well-being. For example, social support was found to be associated with both lower blood pressure and lower Epstein–Barr antibody titers only in more-advantaged, not less-advantaged women (Fagundes et al., 2012).

Other studies have found that psychosocial resources were more impactful for those of lower socioeconomic advantage. Vitaliano et al. (2001) found that social relationships that provide emotional and instrumental support were related to a decrease over time in cardiovascular risk and natural killer cell activity only for those with lower incomes. A psychosocial intervention was found to reduce physiological reactivity only in less-advantaged adolescents (Chen, 2007). Factors related to social stress may have a stronger negative impact on those of lower SES as well. In adolescents, the impacts of a chaotic household on proinflammatory immune activity were found to be more pronounced as SES decreased (Schreier et al., 2014). In elders, negative social interaction was found to increase the risk of heart disease only among those of low advantage (Krause, 2005).

In addition to clarifying a potential moderating role of SES in the hypothesized association of positive experiences and AL, we will also examine whether SES might act to moderate the hypothesized mediating role of positive experiences in its own association with AL, which would constitute a type of moderated mediation. In this situation, positive experiences might be a pathway linking SES and AL at only certain points along the SES spectrum, such as only in those with low or high SES. Although most moderated mediation models (Edwards & Lambert, 2007; Preacher et al., 2007) examine a fourth variable as a moderator, the possibility that a causal variable could moderate its own indirect effect on an outcome has been noted by multiple theorists (Hayes, 2022; Judd & Kenny, 1981). Hayes (2022) points out that a number of researchers (Kraemer et al., 2008; Valeri & VanderWeele, 2013) have argued that models such as this should be normative in mediation analysis.

Method

This research uses data from the MIDUS Study (http://midus.wisc.edu). All data, documentation, and data collection instruments are available via the Inter-university Consortium of Political and Social Research (https://www.icpsr.umich.edu/web/pages/NACDA/midus.html) and the MIDUS Portal (https://midus.colectica.org). We report on our data manipulation and exclusion decisions, how we arrived at our sample size, and all measures included in the study. We follow the STROBE guidelines for reporting observational studies. Analysis code is provided at https://osf.io/jbvhm/?view_only=ed32369d72654a6c9f5697f21d23fae7. Data were analyzed using Stata 17 (StataCorp, 2021). The study was not preregistered.

The MIDUS Study was approved by the University of Wisconsin-Madison Institutional Review Board (IRB), as well as the University of California, Los Angeles, and Georgetown University IRBs (Biomarker substudies). The current study was exempt from human subjects IRB review because it relied on publicly available, de-identified data.

Data and Participants

MIDUS was designed to provide data on the behavioral, social, and psychological factors that are associated with health and well-being over the life course. It includes a 3-wave, longitudinal phone and mail survey assessment of a cohort of Americans aged 25–74 ($n = 7,108$) first surveyed in 1995–1997 (M1) and then at subsequent 9- to 10-year intervals (M2 and M3), as well as an MR cohort ($n = 4,085$), added in 2011–2014 to replenish the original cohort. The cohorts consist of national probability subsamples, a twin/sibling subsample, city oversamples, and subsamples of African-American participants from Milwaukee, WI. Figure 1S in the online supplemental materials shows detailed information regarding sample composition and follow-up times for each wave in each cohort.

At the M2 wave of the original cohort and the initial wave of the MR cohort, subsamples were invited to participate in biomarker studies (M2 Biomarker Project, $n = 1,255$; MR Biomarker Project, $n = 863$), which included an overnight visit at one of three clinical research centers in the U.S. The purpose of the Biomarker Project was to collect data that could be used to further examine associations between psychosocial and behavioral factors and physical health, and data collection included comprehensive biological assessments, medical histories, and an additional self-administered questionnaire. SES and demographic and health covariate data were collected during the main MIDUS surveys in the original and Refresher cohorts. Positive experience frequency, AL biomarker, and additional health covariate data were collected during the Biomarker Project visit. Out of the 2,118 participants in the combined Biomarker Project, 22 were dropped from the analyses due to insufficient AL data (see AL score description below and Figure 1S in the online supplemental materials).

Measures

Allostatic Load

AL was computed as a multisystem biological risk score, using 24 biomarkers that represent 7 physiological systems. Measures of the cardiovascular system included resting pulse and systolic and diastolic blood pressure. Measures of lipids and general metabolic activity included body mass index, waist–hip ratio, triglycerides (mg/dl), high-density lipoprotein cholesterol (mg/dl), and low-density lipoprotein cholesterol (mg/dl). Glucose metabolism was measured using glycated hemoglobin, fasting glucose (mg/dl), and the homeostasis model of insulin resistance. Hypothalamic-pituitary-adrenal axis activity was assessed using overnight urinary cortisol adjusted for urinary creatinine (μg/g) and blood level of dehydroepiandrosterone-sulfate (μg/dl). Inflammatory activity was measured using interleukin-6 (pg/ml), fibrinogen (mg/dl), C-reactive protein (μg/ml), e-Selectin (ng/ml), and soluble intercellular adhesion molecule 1 (ng/ml). Sympathetic nervous system measures included overnight urinary norepinephrine (μg/g) and epinephrine (μg/g), both adjusted for urinary creatinine. Parasympathetic nervous system activity was assessed with 4 measures of heart rate variability: low-frequency (0.04–0.15 Hz) and high-frequency (0.15–0.50 Hz) spectral power, the standard deviation of R–R intervals (in milliseconds), and the root mean square of successive differences.

For each of the 24 biological measures, a risk indicator variable was created to identify participants whose scores fell within the

highest-risk quartile (either upper or lower, depending on the measure). Quartile cutoff values were calculated without the M2 and MR Milwaukee subsample data, as participants in these city-specific samples may be less representative of the national population. Some of the assays and labs used to process some of the biomarkers changed between the M2 and MR Biomarker data collection (see online supplemental materials for additional information and location of MIDUS biomarker documentation files). Despite efforts by the MIDUS team to harmonize assay values across the M2 and MR Biomarker samples, our analyses indicated markedly different means and SDs for some biomarkers (e.g., the hormones tested through urine collection) across the samples. The relative age distributions of each biomarker, however, were similar within the two Biomarker samples. Thus, we elected to use subsample-specific quartiles to identify high-risk cut points for each biomarker indicator (see online supplemental materials for additional discussion).

A risk proportion score was calculated for each of the 7 systems as the mean of the risk indicators within that system, for participants who had data on at least half of the system biomarkers. AL was calculated as the mean of the system risk proportion scores multiplied by 7 (range: 0–7) for those with information on 6 or 7 of the system risk scores. This AL scoring method addresses the unequal representation of biomarkers across different physiological systems in MIDUS (see Gruenewald et al., 2012). It has been demonstrated to have similar predictive validity to and capture sociodemographic variations as well as other AL scoring methods in the literature (Kezios et al., 2022; McLoughlin et al., 2020).

Positive Experiences Score (POS)

Frequency of positive experiences was assessed using a set of 49 items, most of which were taken from the PES (MacPhillamy & Lewinsohn, 1982). These items were on a survey given to participants on the first day of the M2 and MR Biomarker lab visits. Participants were asked how often over the past month they had a variety of experiences involving relaxation, recreation, entertainment, green spaces, social engagement, intimacy, achievement, exercise, and physical comfort. The response categories were 0—Never, 1—1 to 6 times, and 2—7 or more times. POS was calculated as the mean of the items (range: 0–2). Scores were calculated for participants who had data on at least half of the items, and only 11 participants with incomplete data had missing data on more than 4 of the 49 items.

Cumulative SES

Cumulative life-course SES was computed for M2 national probability subsample, twin/sibling subsample, and city oversample participants from retrospective childhood items assessed during the main M1 survey study and adult SES items assessed during the main M2 survey study. For M2 Milwaukee and MR survey participants, all items were assessed during the main M2 and MR survey studies. Three measures of childhood SES were summed to form a scale ranging from 0 to 6: parents' highest level of education (0—less than high school, 1—high school/General Educational Development [GED], 2—some college or greater), whether the participant's family ever received governmental welfare (0—yes, 2—no), and a self-assessment of the participant's financial level when growing up (0—worse off than others, 1—same as others, 2—better off than others). A comparison of sibling data in the original MIDUS

sample showed high sibling concordance on the retrospective childhood items (Gruenewald et al., 2012).

Five measures of current adult SES were summed to form a scale ranging from 0 to 10: participant's level of education (0—high school/GED, 1—some college/associate's degree, 2—college degree or greater), a self-assessment of the participant's current financial level (0—worst possible, 1—average, 2—best possible), whether the participant has enough money to meet basic needs (0—not enough, 1—just enough, 2—more than enough), difficulty of paying bills (0—very or somewhat difficult, 1—not very difficult, 2—not difficult at all), and categories of household-adjusted income-to-poverty ratio (IPR; 0, <300%; 1, 300–599%; 2, ≥600%). For the IPR variable, poverty thresholds from the U.S. Census Bureau (https://www.census.gov/topics/income-poverty/poverty/data/tables/cps.html) were assigned to each participant based on year of income assessment and household size and composition. IPR was calculated as total household income divided by poverty threshold.

ChSES scores were calculated for participants who had data for at least 2 of the 3 measures, and AdSES scores were calculated for participants who had data for at least 4 of the 5 measures. Mean imputation was used to fill in the final component when necessary. CSES was computed as the sum of ChSES and AdSES (range: 0–16).

Chronic Conditions Covariate

Indicators for 9 types of major chronic conditions were used to create a sum variable: lung-related conditions, AIDS or HIV, autoimmune disease, high blood pressure or hypertension, diabetes or high blood sugar, neurological disorders, stroke, cancer, and heart trouble. Self-report items from both the M2/MR main survey studies and the M2/MR Biomarker Project (20 items total) were used to create the variable. Participants who reported having a type of condition on any of the multiple measures assessing it were given a 1 on that indicator. The resulting chronic conditions variable was winsorized at 95% (range: 0–4) and added to the analyses to control for the impact of health burden.

Demographic Covariates

Age at time of Biomarker substudy participation, gender (male or female [referent], the only available categories), and race (Black, other, or white [referent]), were included in the analyses.

Analyses

Descriptive univariate statistics and bivariate relationships with AL were examined for each of the model variables. A mediation analysis was conducted to assess whether frequency of POS may mediate the CSES–AL association observed in the prior analysis of the M2 Biomarker cohort (Gruenewald et al., 2012). We used clustered robust standard errors in order to account for correlated errors within families, and we included gender, race, age, and number of chronic health conditions as covariates. Direct and indirect effects were computed for all paths in the mediation model, and a bootstrap estimate was calculated for the indirect effect.

A moderator analysis was conducted in order to assess whether the hypothesized negative association between POS and levels of AL may vary by level of CSES. We used a generalized estimating equation (GEE) model with an exchangeable correlation structure to account for the family level clustering in the data, and the

model included sociodemographic characteristics (gender, race, and age) and number of chronic health conditions as covariates. Simple slopes for POS were examined at one standard deviation below, at, and one standard deviation above the mean of CSES. The Johnson–Neyman (JN) technique was used to further probe the moderating effect of CSES.

We tested moderated mediation by adding an interaction term for CSES × POS to the mediation model. We calculated conditional indirect effects (with bootstrap confidence intervals) at −1 SD, the mean, and +1 SD of CSES. We also carried out supplementary analyses that examined potential differences between the M2 and MR samples, ChSES and AdSES, and the separate subsystems of AL.

All participants with AL scores were included in the analyses, and mean imputation (for numeric variables) or mode imputation (for categorical variables) was used for the small fraction of missing data on the antecedent variables (<1% missing for each variable). Linearity in the CSES–AL, POS–AL, and CSES–POS associations was assessed graphically. POS was mean-centered and CSES was z-scored in all models. The mediation models were run using the -sem- command in Stata 17, and bootstrap estimates were calculated using 5,000 bootstrap samples. The GEE moderation model was run using the -xtgee- command.

Results

Table 1 describes each variable in the model, including demographic characteristics of the sample, and its relationship with AL. Females and males had similar AL, on average, while Black participants had higher average AL than participants from other racial groups. Age and number of chronic conditions were both positively and moderately correlated with AL, while CSES and POS were both negatively and weakly correlated with AL. Table 2 displays the coefficients and 95% confidence intervals (CIs) for each variable in the mediation model, and Figure 2S in the online supplemental materials shows the coefficients for the direct and indirect paths between CSES, POS, and AL, all of which were significant at the .05 level. As depicted in Figure 2S in the online supplemental materials, the

direct effect accounted for much of the total effect of CSES on AL. The bootstrap test of the indirect effect (Indirect effect = −0.01, $p = .049$, 95% CI [−0.02, −0.0001]) indicated that POS played a small mediating role in the relationship between CSES and AL.

Table 2 shows the coefficients and 95% CIs for all variables in the moderation model. As depicted in the table, CSES significantly interacted with POS to predict AL, $b = 0.21$, $p = .014$, 95% CI [0.04, 0.37]. An examination of simple slopes showed that CSES moderated the association between POS and AL as follows: at −1 SD of CSES, POS was a significant predictor of AL ($b = -0.34$, $p = .003$, [−0.55, −0.12]), but POS was not a significant predictor at the mean of CSES ($b = -0.13$, $p = .136$, [−0.30, 0.04]) or +1 SD of CSES ($b = 0.08$, $p = .543$, [−0.17, 0.33]). Figure 1 shows AL as a function of positive experiences for these 3 levels of CSES. A JN analysis showed that the slope for the effect of POS on AL was positive and nonsignificant at values of CSES above $z = 0.621$ and negative at lower z-scored values of CSES. The negative effect reached significance at CSES scores of $z = -0.187$. In other words, greater POS significantly predicted lower AL for participants who were just below the mean of CSES or less socioeconomically advantaged, and this relationship became stronger at lower levels of CSES (Figure 3S in the online supplemental materials shows this significance point on a graph of the conditional effect of POS as a function of CSES).

The moderated mediation model, with path coefficients, is shown in Figure 2. The results showed that CSES significantly interacted with POS ($b = 0.20$, $p = .019$, 95% CI [0.03, 0.37]), meaning that CSES moderated its own indirect effect on AL. The bootstrap test of the indirect effect through POS was significant at 1 SD below the mean of CSES (Indirect effect = −0.02, $p = .004$, [−0.03, −0.01]) but was not significant at the mean of CSES (Indirect effect = −0.01, $p = .106$, [−0.02, 0.002]) or at 1 SD above the mean of CSES (Indirect effect = 0.003, $p = .634$, [−0.01, 0.02]). In other words, POS mediated the association between CSES and AL only at lower levels of CSES.

Table 1

Descriptive Univariate and Bivariate Statistics

Variable	n	Missing[a]	%			AL [M (SD)]
Gender	2,096	0[b]	—			
Male	947	—	45.2%			1.84 (1.08)
Female	1,149	—	54.8%			1.83 (1.11)
Race	2,089	7[b]	—			
White	1,562	—	74.5%			1.80 (1.10)
Black	386	—	18.4%			1.98 (1.08)
Other	148	—	7.1%			1.81 (1.12)

Variable	n	Missing	M (SD)	Min	Max	Correlation with AL
Age at clinic visit	2,096	0[b]	55.44 (12.56)	26	86	0.34
Chronic conditions	2,096	0[b]	1.19 (1.14)	0	4	0.37
Positive experiences	2,088	8[b]	1.24 (0.26)	0.12	1.96	−0.09
CSES	2,081	15[b]	9.23 (3.52)	0	16	−0.18
Allostatic load	2,096	22[c]	1.83 (1.10)	0	5.8	—

Note. AL = allostatic load; CSES = cumulative socioeconomic status. All correlations are significant at the .001 level. There were 2,118 participants in the M2 and MR 1 Biomarker Projects, and 2,096 had sufficient data to compute a biological systems AL score. The remaining 22 were excluded from the analyses.
[a]Frequency missing prior to imputation of the predictor variables.
[b]Frequency missing out of the 2,096 participants included in the analyses.
[c]Frequency missing out of the 2,118 total participants in the M2 and MR 1 Biomarker Projects.

Table 2
Coefficients and 95% Confidence Intervals for the Variables in the Mediation and Moderation Models

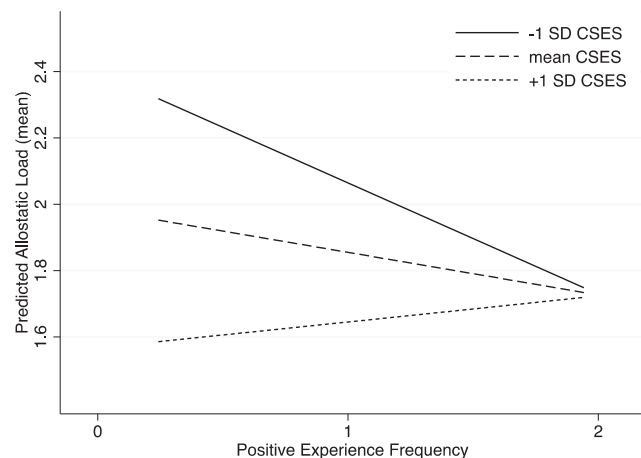|	Mediation model			Moderation model		
		95% CI			95% CI	
Variable	*b*	LL	UL	*b*	LL	UL
Dependent Variable: AL						
Predictors						
POS	−0.170*	−0.338	−0.002	−0.128	−0.297	0.040
CSES	−0.163***	−0.209	−0.116	−0.159***	−0.206	−0.113
CSES × POS				0.207*	0.042	0.372
Covariates						
Age	0.024***	0.021	0.028	0.024***	0.021	0.028
Gender						
Male	0.022	−0.064	0.108	0.029	−0.057	0.114
Race						
Black	0.041	−0.080	0.161	0.044	−0.076	0.164
Other	0.099	−0.065	0.264	0.101	−0.065	0.266
Chronic condition	0.243***	0.202	0.283	0.237***	0.197	0.277
Dependent Variable: POS						
Predictors						
CSES	0.055***	0.043	0.067			
Covariates						
Age	0.000	−0.0004	0.001			
Gender						
Male	−0.043***	−0.065	−0.021			
Race						
Black	−0.024	−0.059	0.010			
Other	−0.064**	−0.110	−0.019			
Chronic condition	−0.010	−0.021	0.001			

Note. CI = confidence interval; POS = positive life experiences; CSES = cumulative socioeconomic status; AL = allostatic load.
*$p < .05$. **$p < .01$. ***$p < .001$.

Supplementary Analyses

The average age was 57 in M2 and 53 in MR. While *t*-tests showed no significant differences in ChSES, AdSES, CSES, or POS between cohorts, the average lag time between the main study survey and biomarker visit was longer by about 5 months in M2 (see

Figure 1
*Predicted Allostatic Load Scores as a Function of Cumulative Socioeconomic Advantage and Positive Experience Frequency*

Note. CSES = cumulative socioeconomic status

Table 1S in the online supplemental materials). When the primary analyses were stratified by cohort, the coefficients followed a similar pattern to those seen in the primary analyses, though not all effects reached significance (see Tables 2S and 3S and Figures 4S and 5S in the online supplemental materials). The results followed the same overall pattern in both subsamples, but the POS–AL association was stronger in MR than in M2.

When entered into GEE models simultaneously, ChSES and AdSES each uniquely predicted variation in both the POS scores and AL scores, though AdSES carried more weight in the models (Table 4S in the online supplemental materials). When the primary analyses were run using ChSES only, the coefficients, simple slopes, and indirect effects followed the pattern seen in the analyses with CSES, though ChSES was not a significant moderator of the POS–AL association (Table 5S and Figure 6S in the online supplemental materials). When the primary analyses were run using adult socioeconomic advantage, the coefficients again followed the pattern seen in the CSES analyses, and there were no differences in significance from the primary analyses (Table 6S and Figure 7S in the online supplemental materials).

When the AL subsystems were examined separately, the moderation was only significant for the cardiovascular subsystem (see Tables 7S and 8S in the online supplemental materials). In the moderated mediation models, examination of conditional indirect effects suggested that the primary results showing a mediation effect for those of lower SES are likely being driven by the cardiovascular, lipid/general metabolic, glucose metabolism, and inflammatory systems (see Table 9S in the online supplemental materials).

Figure 2

Moderated Mediation Model with Unstandardized Coefficients and Conditional Indirect Effects (CIE)
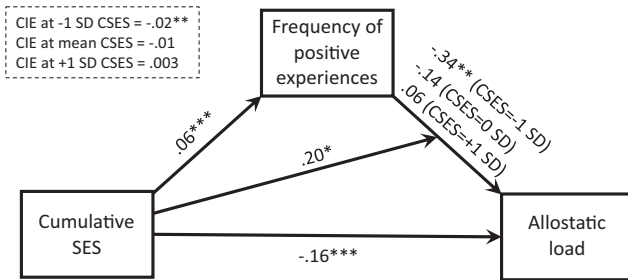
Note. Not shown: This model controlled for gender, race, age, and chronic health conditions. Significance of conditional indirect effects based on 5,000 bootstrap samples. CSES = cumulative socioeconomic status; SES = socioeconomic status.

*p < .05. **p < .01. ***p < .001.

Discussion

Consistent with previous research, our results show a direct association between SES and AL, such that higher CSES over the life course is associated with lower levels of physiological dysregulation. The results of our simple mediation analysis suggest that POS is a weak mediator of this association. This result is in line with prior findings based on a smaller analytic sample (Gruenewald et al., 2012), in which the addition of POS attenuated the SES–AL association. In our moderation analysis, greater POS significantly predicted lower AL only at below-average levels of CSES, and this relationship was predicted to be stronger at even lower levels of CSES. This finding is in line with results from Chen (2007) and Vitaliano et al. (2001), which showed that social and psychosocial factors may be linked to lower physiological stress and dysregulation in those of lower, but not higher, SES. Finally, moderated mediation was present, such that POS significantly mediated the CSES–AL association only at lower levels of CSES. Overall, these results suggest that there is considerable complexity in the associations between SES, positive life events, and physiological well-being.

In line with previous research showing that childhood SES and adult SES work in tandem to impact health (e.g., Gruenewald et al., 2012; Luo and Waite, 2005), ChSES and AdSES each predicted unique variation in both POS and AL in our supplementary analyses. Childhood SES may therefore have a direct impact on both access to positive life experiences and physiological well-being in adulthood that cannot be explained by adult SES. A composite measure of SES across the life course, such as the one used in this study, takes these multiple periods of socioeconomic experience into account.

The effect size for the association between POS and AL in the mediation model was relatively large—a 1 unit increase in POS was associated with a decrease in AL of 0.17, which is similar to the difference predicted for a 7-year age gap. At −1 SD of CSES, a 1 unit increase in POS was associated with a decrease in AL of 0.34, which is similar to the difference predicted for a 14-year age gap. The indirect effect of CSES through POS, by contrast, is a small portion of the CSES–AL association—at −1 SD of CSES, a 1 SD increase in CSES was associated with a conditional indirect effect that is similar to the difference predicted for only a 1-year age gap.

Although this is a small indirect effect, it illuminates one of many pathways through which SES over the life course may impact the level of physiological dysregulation in the body. Many of the other pathways likely have a considerable impact on physiological well-being, such as access to health care and a healthy physical environment, so positive experiences may well play a weaker mediation role. Our finding does represent, however, one mechanism that may lessen disparities instead of increasing them. Positive life experiences may have a stronger positive impact on physiological well-being for those of lower SES, whereas we noted earlier that some health interventions lead to intervention-generated inequalities (White et al., 2009), in which stronger impacts are observed for individuals who are already more advantaged.

Our results show that frequency of positive experiences is associated with better physiological health in a national sample, but this does not mean that top-down, prescriptive positive experience interventions would be associated with the same health outcomes. Positive life experiences are activities in which individuals freely engage, and Hopper and Iwasaki (2017) suggest that an approach that artificially imposes a positive experiences intervention on those at risk (in their context, marginalized youth), may actually be detrimental. Much as Hopper and Iwasaki (2017) discuss the importance of a bottom-up, youth-led approach, the best way to increase the frequency of positive experiences among less-advantaged individuals and communities may be through providing resources, in the form of time, money, and access, so that people have more options for freely choosing to engage in positive life experiences. Individuals who have limited access to certain positive life experiences are often deeply aware of this deficiency. For example, when urban New York City youth were asked in the Intergenerational Change Initiative's 2021 survey what would make things better where they live, the most common responses, right after basic needs (more jobs and affordable housing), were community events, more green spaces, and public art (Zeller-Berkman et al., 2021).

Limitations

In our study, POS was assessed over the previous month and measured within a similar time bracket as the biomarkers in the AL scores. These POS scores likely represent a longer-term characteristic pattern of activity, as the original Pleasant Events Scale showed test–retest reliability of >0.7 within a 2-month period and >0.6 within a 2-to-3-year period (MacPhillamy & Lewinsohn, 1976). Although we included a health condition burden covariate in the models to address the fact that poor health may limit positive activity frequency, we cannot rule out the possibility that individuals with better overall health may simply be able to engage in more activities, including positive life experiences. Although the literature on the relationship between positive experiences and health suggests positive life experiences do impact health and well-being (Keegan et al., 2016), the relationship may be bidirectional.

The MIDUS Study is a mixture of national probability samples and oversamples of both specific cities and Black individuals from Milwaukee, WI. The results may not be generalizable to the population as a whole or to specific racial or ethnic groups in the US. In addition, although there is considerable socioeconomic and health variation in the MIDUS Biomarker data, and though prior research suggests the M2 Biomarker sample had comparable sociodemographic characteristics to the main M2 sample (Love et al., 2010),

there may be other characteristics in those who agree to participate in a two-day protocol that may affect generalizability.

In our study, we pooled together participants from the M2 and MR cohorts. Although the MR study was conducted specifically to replenish the M2 cohort, we found differences between the cohorts. First, the average lag time between the main M2 survey study and M2 Biomarker Project was about 5 months longer than the lag time between the main MR survey study and MR Biomarker Project. This means that, on average, SES was measured earlier before the biomarker readings for M2 participants than for MR participants. In addition, given that the M2 Biomarker Project started 12 years before the MR Biomarker Project ended, assays and labs had changed. As noted in the Methods section, some of the biomarkers had markedly different means in the M2 versus MR cohorts. As a result, we were unable to apply single cut points across the combined sample for determining high-risk quartiles, and our AL scores are based on cut points calculated within the M2 and MR samples.

Our study examined associations between overall frequency of positive life experiences and physiological dysregulation, pooling together disparate experiences such as social gatherings, nature experiences, and spirituality. It did not examine whether different types of positive life experiences may have different impacts on physiological well-being. This is an important objective that is beyond the scope of the current paper. While we did examine the AL subsystems separately in supplementary analyses, these analyses use coarse measures and are very preliminary. Given the limited number of biomarkers in most systems, and given that each biomarker indicator is coded only as 0 or 1, most individual system scores do not have a great amount of variability and are therefore not ideal for regression analyses such as ours where small effects would be expected. This is also a meaningful area for future research.

Similarly, although we elected to use a composite measure of SES that encompasses multiple socioeconomic indicators across different phases of the life course, the use of the composite does obscure whether specific dimensions of socioeconomic experience (e.g., educational attainment, income, and subjective perceptions) may be differentially linked to positive experiences or AL. Finally, while we controlled for demographic characteristics and chronic health burden, it is likely that there are variables that impact the associations between SES, positive life experiences, and AL that remain unaccounted for in our analyses.

Future Research

Given the finding that positive life experiences may provide a larger benefit for members of certain groups, demographic disparities in positive life experiences should be examined. Future research can assess variations in both frequency of positive life events and associated benefits, as well as variations within different types of positive life events. In addition to looking at overall cumulative life advantage, associations between specific aspects of SES (e.g., educational attainment and objective vs. subjective measures of SES) and positive experiences can be examined. Possible mechanisms involving positive life experiences that underlie SES-health associations can be probed further. For example, the role of obtained pleasure from positive experiences can be examined as a potential pathway, and other pathways that vary by type of positive experience and health outcome can be examined as well. Finally, interventions that aim to increase access so that

people can more easily choose to engage in positive life events, or interventions that aim to enhance the pleasure derived from these experiences, can be designed, and associated effects on health and well-being can be assessed.

Conclusion

Our findings showed that the association between SES and allostatic load was weakly mediated by positive life experiences and that greater frequency of positive life experiences predicted lower allostatic load only at lower levels of cumulative socioeconomic advantage. Positive life experiences may therefore play a stronger role in physiological health in those of lower SES. Given that access to positive life experiences may be modifiable, the potential role of positive experiences in lessening health disparities warrants further study.

References

Adler, N. E. (2013). Health disparities: Taking on the challenge. *Perspectives on Psychological Science*, *8*(6), 679–681. https://doi.org/10.1177/1745691613506909

Adler, N. E., Epel, E. S., Castellazzo, G., & Ickovics, J. R. (2000). Relationship of subjective and objective social status with psychological and physiological functioning: Preliminary data in healthy, white women. *Health Psychology*, *19*(6), 586–592. https://doi.org/10.1037/0278-6133.19.6.586

Adler, N. E., & Snibbe, A. C. (2003). The role of psychosocial processes in explaining the gradient between socioeconomic status and health. *Current Directions in Psychological Science*, *12*(4), 119–123. https://doi.org/10.1111/1467-8721.01245

Assari, S. (2017). Social determinants of depression: The intersections of race, gender, and socioeconomic status. *Brain Sciences*, *7*(12), Article 156. https://doi.org/10.3390/brainsci7120156

Beckie, T. M. (2012). A systematic review of allostatic load, health, and health disparities. *Biological Research for Nursing*, *14*(4), 311–346. https://doi.org/10.1177/1099800412455688

Billings, D. W., Folkman, S., Acree, M., & Moskowitz, J. T. (2000). Coping and physical health during caregiving: The roles of positive and negative affect. *Journal of Personality and Social Psychology*, *79*(1), 131–142. https://doi.org/10.1037/0022-3514.79.1.131

Boehm, J. K., & Kubzansky, L. D. (2012). The heart's content: The association between positive psychological well-being and cardiovascular health. *Psychological Bulletin*, *138*(4), 655–691. https://doi.org/10.1037/a0027448

Bosworth, B. (2018). Increasing disparities in mortality by socioeconomic status. *Annual Review of Public Health*, *39*(1), 237–251. https://doi.org/10.1146/annurev-publhealth-040617-014615

Bullard, R. D., & Wright, B. (1993). Environmental justice for all: Current perspectives on health and research needs. *Toxicology and Industrial Health*, *9*(5), 821–841. https://doi.org/10.1177/074823379300900508

Bussell, V. A., & Naus, M. J. (2010). A longitudinal investigation of coping and posttraumatic growth in breast cancer survivors. *Journal of Psychosocial Oncology*, *28*(1), 61–78. https://doi.org/10.1080/07347330903438958

Chen, E. (2007). Impact of socioeconomic status on physiological health in adolescents: An experimental manipulation of psychosocial factors. *Psychosomatic Medicine*, *69*(4), 348–355. https://doi.org/10.1097/PSY.0b013e3180592b20

Chen, E., & Miller, G. E. (2013). Socioeconomic status and health: Mediating and moderating factors. *Annual Review of Clinical Psychology*, *9*(1), 723–749. https://doi.org/10.1146/annurev-clinpsy-050212-185634

Demakakos, P., Nazroo, J., Breeze, E., & Marmot, M. (2008). Socioeconomic status and health: The role of subjective social status.

Social Science and Medicine, 67(2), 330–340. https://doi.org/10.1016/j.socscimed.2008.03.038

Duru, O. K., Harawa, N. T., Kermah, D., & Norris, K. C. (2012). Allostatic load burden and racial disparities in mortality. *Journal of the National Medical Association*, 104(1–2), 89–95. https://doi.org/10.1016/S0027-9684(15)30120-6

Edwards, J. R., & Lambert, L. S. (2007). Methods for integrating moderation and mediation: A general analytical framework using moderated path analysis. *Psychological Methods*, 12(1), 1–22. https://doi.org/10.1037/1082-989X.12.1.1

Evans, G. W., Kim, P., Ting, A. H., Tesher, H. B., & Shannis, D. (2007). Cumulative risk, maternal responsiveness, and allostatic load among young adolescents. *Developmental Psychology*, 43(2), 341–351. https://doi.org/10.1037/0012-1649.43.2.341

Everson, S. A., Maty, S. C., Lynch, J. W., & Kaplan, G. A. (2002). Epidemiologic evidence for the relation between socioeconomic status and depression, obesity, and diabetes. *Journal of Psychosomatic Research*, 53(4), 891–895. https://doi.org/10.1016/S0022-3999(02)00303-3

Fagundes, C. P., Bennett, J. M., Alfano, C. M., Glaser, R., Povoski, S. P., Lipari, A. M., Agnese, D. M., Yee, L. D., Carson, W. E., Farrar, W. B., Malarkey, W. B., Chen, M., & Kiecolt-Glaser, J. K. (2012). Social support and socioeconomic status interact to predict Epstein–Barr virus latency in women awaiting diagnosis or newly diagnosed with breast cancer. *Health Psychology*, 31(1), 11–19. https://doi.org/10.1037/a0025599

Ferreira, H. G., & Barham, E. J. (2018). Relationships between pleasant events, depression, functionality and socio-demographic variables in the elderly. *Paidéia (Ribeirão Preto)*, 28, Article e2815. https://doi.org/10.1590/1982-4327e2815

Gallo, L. C., de los Monteros, K. E., & Shivpuri, S. (2009). Socioeconomic status and health: What is the role of reserve capacity? *Current Directions in Psychological Science*, 18(5), 269–274. https://doi.org/10.1111/j.1467-8721.2009.01650.x

Gallo, L. C., & Matthews, K. A. (2003). Understanding the association between socioeconomic status and physical health: Do negative emotions play a role? *Psychological Bulletin*, 129(1), 10–51. https://doi.org/10.1037/0033-2909.129.1.10

Gruenewald, T. L., Karlamangla, A. S., Hu, P., Stein-Merkin, S., Crandall, C., Koretz, B., & Seeman, T. E. (2012). History of socioeconomic disadvantage and allostatic load in later life. *Social Science & Medicine*, 74(1), 75–83. https://doi.org/10.1016/j.socscimed.2011.09.037

Gruenewald, T. L., Seeman, T. E., Karlamangla, A. S., & Sarkisian, C. A. (2009). Allostatic load and frailty in older adults. *Journal of the American Geriatrics Society*, 57(9), 1525–1531. https://doi.org/10.1111/j.1532-5415.2009.02389.x

Hayes, A. F. (2022). *Introduction to mediation, moderation, and conditional process analysis: A regression-based approach*. Guilford Publications.

Hopper, T. D., & Iwasaki, Y. (2017). Engagement of 'at-risk' youth through meaningful leisure. *Journal of Park and Recreation Administration*, 35(1), 1–33. https://doi.org/10.18666/JPRA-2017-V35-I1-7289

Howard, J. T., & Sparks, P. J. (2016). The effects of allostatic load on racial/ethnic mortality differences in the United States. *Population Research and Policy Review*, 35(4), 421–443. https://doi.org/10.1007/s11113-016-9382-4

Judd, C. M., & Kenny, D. A. (1981). Process analysis: Estimating mediation in treatment evaluations. *Evaluation Review*, 5(5), 602–619. https://doi.org/10.1177/0193841X8100500502

Keegan, C., Conroy, R., & Doyle, F. (2016). Longitudinal modelling of theory-based depressive vulnerabilities, depression trajectories and poor outcomes post-ACS. *Journal of Affective Disorders*, 191, 41–48. https://doi.org/10.1016/j.jad.2015.11.030

Kezios, K. L., Suglia, S. F., Doyle, D. M., Susser, E., Bradwin, G., Cirillo, P., Cohn, B., Link, B., & Factor-Litvak, P. (2022). Comparing different operationalizations of allostatic load measured in mid-life and their patterning by race and cumulative life course socioeconomic status.

Psychoneuroendocrinology, 139, Article 105689. https://doi.org/10.1016/j.psyneuen.2022.105689

Kitagawa, E. M., & Hauser, P. M. (1973). *Differential mortality in the United States: A study in socioeconomic epidemiology*. Harvard University Press.

Kraemer, H. C., Kiernan, M., Essex, M., & Kupfer, D. J. (2008). How and why criteria defining moderators and mediators differ between the Baron & Kenny and MacArthur approaches. *Health Psychology*, 27(Suppl 2), S101–S108. https://doi.org/10.1037/0278-6133.27.2(Suppl.).S101

Krause, N. (2005). Negative interaction and heart disease in late life: Exploring variations by socioeconomic status. *Journal of Aging and Health*, 17(1), 28–55. https://doi.org/10.1177/0898264304272782

Lorant, V., Deliège, D., Eaton, W., Robert, A., Philippot, P., & Ansseau, M. (2003). Socioeconomic inequalities in depression: A meta-analysis. *American Journal of Epidemiology*, 157(2), 98–112. https://doi.org/10.1093/aje/kwf182

Love, G., Seeman, T. E., Weinstein, M., & Ryff, C. D. (2010). Bioindicators in the MIDUS national study: Protocol, measures, sample, and comparative context. *Journal of Aging and Health*, 22(8), 1059–1080. https://doi.org/10.1177/0898264310374355

Luo, Y., & Waite, L. J. (2005). The impact of childhood and adult SES on physical, mental, and cognitive well-being in later life. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, 60(2), S93–S101. https://doi.org/10.1093/geronb/60.2.S93

MacPhillamy, D. J., & Lewinsohn, P. M. (1976). *Manual for the pleasant events schedule*. University of Oregon. https://www.ori.org/files/Static%20Page%20Files/PESManual.pdf

MacPhillamy, D. J., & Lewinsohn, P. M. (1982). The pleasant events schedule: Studies on reliability, validity, and scale intercorrelation. *Journal of Consulting and Clinical Psychology*, 50(3), 363–380. https://doi.org/10.1037/0022-006X.50.3.363

Marmot, M. G. (2004). *The status syndrome: How social standing affects our health and longevity*. Times Books.

Matthews, K. A., Gallo, L. C., & Taylor, S. E. (2010). Are psychosocial factors mediators of socioeconomic status and health connections? A progress report and blueprint for the future. *Annals of the New York Academy of Sciences*, 1186(1), 146–173. https://doi.org/10.1111/j.1749-6632.2009.05332.x

Mausbach, B. T., Romero-Moreno, R., Bos, T., von Kanel, R., Ziegler, M. G., Allison, M. A., Milis, P. J., Dimsdale, J. E., Ancoli-Israel, S., Losada, A., Marguez-Gonzalez, M., Patterson, T. L., & Grant, I. (2018). Engagement in pleasant leisure activities and blood pressure: A 5-year longitudinal study in Alzheimer's caregivers. *Psychosomatic Medicine*, 79(9), 735–741. https://doi.org/10.1097/PSY.0000000000000497

Mazzucchelli, T., Kane, R., & Rees, C. (2009). Behavioral activation treatments for depression in adults: A meta-analysis and review. *Clinical Psychology: Science and Practice*, 16(4), 383–411. https://doi.org/10.1111/j.1468-2850.2009.01178.x

McEwen, B. S. (2000). Allostasis and allostatic load: Implications for neuropsychopharmacology. *Neuropsychopharmacology*, 22(2), 108–124. https://doi.org/10.1016/S0893-133X(99)00129-3

McLoughlin, S., Kenny, R. A., & McCrory, C. (2020). Does the choice of allostatic load scoring algorithm matter for predicting age-related health outcomes? *Psychoneuroendocrinology*, 120, Article 104789104789. https://doi.org/10.1016/j.psyneuen.2020.104789

Miller, G. E., Lachman, M. E., Chen, E., Gruenewald, T. L., Karlamangla, A. S., & Seeman, T. E. (2011). Pathways to resilience: Maternal nurturance as a buffer against the effects of childhood poverty on metabolic syndrome at midlife. *Psychological Science*, 22(12), 1591–1599. https://doi.org/10.1177/0956797611419170

Mitchell, R., & Popham, F. (2008). Effect of exposure to natural environment on health inequalities: An observational population study. *Lancet*, 372(9650), 1655–1660. https://doi.org/10.1016/S0140-6736(08)61689-X

Moore, R. C., Chattillion, E. A., Ceglowski, J., Ho, J., von Känel, R., Mills, P. J., Ziegler, M. G., Patterson, T. L., Grant, I., & Mausbach, B. T. (2013). A

randomized clinical trial of behavioral activation (BA) therapy for improving psychological and physical health in dementia caregivers: Results of the Pleasant Events Program (PEP). *Behaviour Research and Therapy*, *51*(10), 623–632. https://doi.org/10.1016/j.brat.2013.07.005

Mulatu, M. S., & Schooler, C. (2002). Health: Reciprocal effects and mediating mechanisms. *Journal of Health and Social Behavior*, *43*(1), 22–41. https://doi.org/10.2307/3090243

Pollitt, R. A., Rose, K. M., & Kaufman, J. S. (2005). Evaluating the evidence for models of life course socioeconomic factors and cardiovascular outcomes: A systematic review. *BMC Public Health*, *5*(1), 1–13. https://doi.org/10.1186/1471-2458-5-7

Preacher, K. J., Rucker, D. D., & Hayes, A. F. (2007). Addressing moderated mediation hypotheses: Theory, methods, and prescriptions. *Multivariate Behavioral Research*, *42*(1), 185–227. https://doi.org/10.1080/00273170701341316

Pressman, S. D., Matthews, K. A., Cohen, S., Martire, L. M., Scheier, M., Baum, A., & Schulz, R. (2009). Association of enjoyable leisure activities with psychological and physical well-being. *Psychosomatic Medicine*, *71*(7), 725–732. https://doi.org/10.1097/PSY.0b013e3181ad7978

Reiss, F. (2013). Socioeconomic inequalities and mental health problems in children and adolescents: A systematic review. *Social Science & Medicine*, *90*, 24–31. https://doi.org/10.1016/j.socscimed.2013.04.026

Rider, K. L., Thompson, L. W., & Gallagher-Thompson, D. (2016). California Older Persons Pleasant Events Scale: A tool to help older adults increase positive experiences. *Clinical Gerontologist*, *39*(1), 64–83. https://doi.org/10.1080/07317115.2015.1101635

Schreier, H. M., Roy, L. B., Frimer, L. T., & Chen, E. (2014). Family chaos and adolescent inflammatory profiles: The moderating role of socioeconomic status. *Psychosomatic Medicine*, *76*(6), 460–467. https://doi.org/10.1097/PSY.0000000000000078

Seeman, T. E., Singer, B. H., Rowe, J. W., Horwitz, R. I., & McEwen, B. S. (1997). Price of adaptation—allostatic load and its health consequences (MacArthur studies of successful aging). *Archives of Internal Medicine*, *157*(19), 2259–2268. https://doi.org/10.1001/archinte.1997.00440400111013

StataCorp. (2021). *Stata statistical software: Release 17*. StataCorp.

Valeri, L., & VanderWeele, T. J. (2013). Mediation analysis allowing for exposure-mediator interactions and causal interpretation: Theoretical assumptions and implementation with SAS and SPSS macros. *Psychological Methods*, *18*(2), 137–150. https://doi.org/10.1037/a0031034

van Oort, F. V., van Lenthe, F. J., & Mackenbach, J. P. (2005). Material, psychosocial, and behavioural factors in the explanation of educational inequalities in mortality in The Netherlands. *Journal of Epidemiology & Community Health*, *59*(3), 214–220. https://doi.org/10.1136/jech.2003.016493

Veinot, T. C., Mitchell, H., & Ancker, J. S. (2018). Good intentions are not enough: How informatics interventions can worsen inequality. *Journal of the American Medical Informatics Association*, *25*(8), 1080–1088. https://doi.org/10.1093/jamia/ocy052

Vitaliano, P. P., Scanlan, J. M., Zhang, J., Savage, M. V., Brummett, B., Barefoot, J., & Siegler, I. C. (2001). Are the salutogenic effects of social supports modified by income? A test of an "added value hypothesis". *Health Psychology*, *20*(3), 155–165. https://doi.org/10.1037/0278-6133.20.3.155

White, M., Adams, J., & Heywood, P. (2009). How and why do interventions that increase health overall widen inequalities within populations? In S. J. Babones (Ed.). *Health, inequality and society* (pp. 65–82). Policy Press.

Zeller-Berkman, S., Bryer, J., & Elisha, I. (2021). *Youth ask youth census. Amplify data dashboard. Intergenerational change initiative*. https://amplifydata.org/