Annals of Behavioral Medicine, 2023, **57**, 951–964
https://doi.org/10.1093/abm/kaad044
Advance access publication 7 August 2023
Regular Article

Subjective Social Status and Longitudinal Changes in Systemic Inflammation

Emily J. Jones, PhD[1], Anna L. Marsland, PhD, RN[1], Thomas E. Kraynak, PhD[2], Elizabeth Votruba-Drzal, PhD[1], Peter J. Gianaros, PhD[1]

[1]Department of Psychology, University of Pittsburgh, Pittsburgh, PA, USA
[2]Department of Epidemiology, University of Pittsburgh, Pittsburgh, PA, USA

Emily J. Jones
emj63@pitt.edu

Abstract

Background Subjective social status (SSS) refers to a person's perception of their social rank relative to others and is cross-sectionally linked to systemic inflammation independently of objective socioeconomic status.

Purpose We test the extent to which SSS relates to multiyear *changes* in inflammation, or if associations differ by race or sex.

Methods Healthy adults (N = 331; 30–51 years) completed a baseline visit and 278 participants returned for a second visit 2.85 years later. At both visits, participants underwent a fasting blood draw and completed community (SSS_C) and US (SSS_{US}) versions of the MacArthur Scale. Multiple linear regression analyses examined change in interleukin-6 (IL-6) and C-reactive protein (CRP) predicted by each type of SSS, adjusting for time between visits, sex, race, age, body mass index, smoking, baseline inflammation, and objective socioeconomic status. Additional analyses further adjusted for hopelessness and depressive symptoms. Interactions examined moderations by sex and race.

Results Lower SSS_C was longitudinally associated with greater IL-6 independently of all covariates, including education and income ($\beta = -0.06$), hopelessness ($\beta = -0.06$), and depressive symptoms ($\beta = -0.06$). Lower SSS_{US} was longitudinally associated with greater IL-6 independently of demographic covariates including education and income ($\beta = -0.06$), but was slightly attenuated after adjusting for hopelessness ($\beta = -0.06$) and depressive symptoms ($\beta = -0.06$). There were no associations for CRP or moderation by race or sex.

Conclusions Lower SSS may be associated with greater circulating markers of inflammation over time as suggested by increases in IL-6.

Lay summary

Subjective social status (SSS) refers to how people perceive their social rank compared with others and has been linked to meaningful differences in physical health. Increases in inflammation may contribute to associations between lower SSS and poorer physical health. In a sample of healthy adults, we examined whether SSS was associated with prospective, multiyear changes in markers of systemic inflammation and if this differed by sex or race. We found that adults who perceived their social status as lower than peers in their community exhibited an accelerated increase in interleukin-6, a marker of systemic inflammation, over a 3-year period. When participants were asked to compare themselves to people in the broader USA, the pattern was similar but less robust. Results were independent of individual differences in sociodemographic characteristics including family-adjusted income and education. Findings did not differ by sex or race and were not explained by differences in adiposity and symptoms of depression and hopelessness. Effects for C-reactive protein, a second marker of inflammation, were generally nonsignificant. Although preliminary, findings suggest an immune pathway by which perceived social status may relate to chronic diseases of aging.

Keywords Subjective social status · Interleukin-6 · C-reactive protein · Inflammation · Socioeconomic status

Introduction

Cardiovascular disease (CVD) and diabetes are patterned by socioeconomic status (SES) such that these conditions become increasingly prevalent with lower levels of education and income [1–3]. This socioeconomic gradient in cardiometabolic conditions also holds when considering subjective measures of SES, such as subjective social status (SSS) [4–6]. SSS refers to people's perception of where they stand in comparison to others in their local communities or the broader USA [7]. SSS ratings are thought to reflect what has been referred to as a "cognitive averaging" of multiple factors, including objective measures of SES (e.g., educational attainment, income) and related psychosocial constructs, including perceptions of prestige, power, privilege, and respect compared with others [8]. Because of this, SSS has the advantage of capturing aspects of SES that may be missed by traditional, objective measures of SES. Moreover, SSS has been found to uniquely relate to cardiometabolic risk over and above education and income [4].

Age-related increases in systemic inflammation may be one of many pathways connecting SSS to cardiometabolic conditions. Indeed, circulating levels of proinflammatory cytokines, including interleukin-6 (IL-6) and C-reactive protein (CRP), increase with age [9] and are implicated in the pathophysiology of age-related conditions such as CVD [10, 11] and

diabetes [12, 13]. Furthermore, lower SSS is cross-sectionally associated with higher systemic inflammation (e.g., CRP, fibrinogen) among young and midlife adults [12, 14, 15] and risk for metabolic syndrome [16], a cluster of metabolic factors predictive of cardiometabolic conditions that may be preceded by systemic inflammation [17]. Yet, it is not clear whether lower SSS relates to greater increases in circulating inflammatory markers over multiple years and what biopsychosocial mechanisms may underlie these prospective associations. Studies examining associations between SSS and transient changes IL-6 in response to social stress tasks suggests that there may be a link between lower SSS and greater multiyear changes in IL-6. Three different studies found that adults who reported lower SSS had greater IL-6 reactivity (e.g., higher levels of IL-6 2 hr after a social-evaluative laboratory stress task) relative to their peers who reported higher SSS [18–20]. Derry et al. further found that people who rated their SSS lower reported feeling more threatened by the social stress task [19]. This could suggest that adults lower in SSS may experience heightened stress reactivity to everyday social status stressors (such as transient increases in IL-6) that may accumulate over time and lead to physiological wear-and-tear, including greater age-related increases in systemic inflammation [21, 22]. In addition to greater threat and physiological stress reactivity, lower SSS may be associated with greater age-related increases in systemic inflammation through psychological factors and adiposity, as well as changes in these factors over time. For example, lower SSS cross-sectionally associates with greater negative affect and adiposity [14, 23, 24], both of which may fluctuate over the course of a few years and possibly contribute to changes in inflammation [25]. Here, we examine whether SSS relates to *multiyear* changes in circulating levels of IL-6 and CRP among a sample of healthy, midlife adults, and then explore whether depressive symptoms, hopelessness, and adiposity may partially account for these associations.

Importantly, associations between SSS and prospective, multiyear changes in IL-6 and CRP may differ by sociodemographic factors, including race and sex. In the USA, people vary in power and privilege depending on a host of characteristics beyond SES, including whether they identify as White or as a person of color. Although there is considerable heterogeneity within and across different racial groups, people of color share a common experience of facing marginalization in the USA. For example, people from racially minoritized groups may receive societal messages that they do not belong or are less valuable than their White peers as evidenced by racial inequalities in income, unemployment, education, and the distribution of wealth, particularly between Black and White adults [26–29]. They may also face greater exposure to both structural and interpersonal racism [30]. As such, people from racially minoritized groups may rate their SSS as lower than their White peers [23], particularly when asked to compare themselves to other U.S. adults [31].

Nonetheless, even if people from racially minoritized groups report similar SSS ratings as their White peers, the association between lower SSS and prospective changes in inflammation may differ by race as suggested by the proximity to disadvantage and skin-deep resilience theories [32, 33]. More specifically, Black adults and other adults from marginalized racial groups may accrue fewer psychosocial, financial, and physical health benefits of higher SES as compared with their White peers in part because of their proximity to spatial, relational, and intergenerational disadvantages [33]. Moreover, the effort required to be upwardly mobile may come at an unintended physiological cost to young adults from racially minoritized groups [32]. Existing studies on racial differences in associations between SSS and health have generally focused on differences between Black and White adults and findings have been mixed. For example, Adler et al. [34] found that associations between lower U.S. and societal SSS and poorer global health and hypertension were attenuated or nonsignificant among Black but not White adults in England and the USA. Similarly, Allen et al. [35] found that lower community SSS predicted higher Framingham 10-year CVD risk profiles among White, but not Black U.S. midlife adults. Yet, other research suggested the opposite pattern, with lower community SSS relating to greater psychological distress among Black versus White mothers in the USA [36], which could potentially translate to greater systemic inflammation over time [19, 37]. Inconsistent results may reflect differences in the reference group (e.g., comparing to others in the community vs. entire country), outcome measures assessed, and cultural factors [31, 38]. These mixed findings, coupled with the fact that very few studies have examined links between SSS and health among people belonging to other racial minority groups (e.g., Asian Americans [39]), further underscores the need to examine interactions between SSS and race on inflammatory parameters.

In addition to race, associations between SSS and prospective changes in IL-6 and CRP may differ between men and women. Prior research has suggested that men's health may be more susceptible to social status-based stressors whereas interpersonal stressors may matter more for women's health [40]. This could suggest that SSS is more strongly associated with multiyear changes in inflammation among men compared with women. Support for this notion comes from Freeman et al. [41] who found stronger associations between U.S. SSS and concurrent systemic inflammation among young men versus young women. Yet, this contrasts with more recent research on sex differences in SSS and health outcomes more broadly. Lower community and U.S. SSS have been cross-sectionally associated with insulin resistance among Black women but not Black men [42] and lower U.S. SSS has been prospectively associated with higher rates of respiratory infections among female but not male health care professionals [43]. Although different health outcomes, it is possible that sex differences may extend to multiyear changes in IL-6 and CRP given that elevated inflammation may precede insulin resistance [44] and increase the severity of upper respiratory infection symptoms [45]. Similar to mixed findings between race, SSS, and health, inconsistent results between men and women may be due in part to differences in the reference group, racial and ethnic composition of the various samples, and the types of physical health measures assessed. Thus, additional research on sex differences in SSS and health is warranted.

Present Study

To this end, this study examines whether SSS relates to prospective, multiyear changes in circulating levels of IL-6 and CRP independently of income and educational attainment among a sample of healthy midlife adults assessed at two timepoints spaced 2–4 years apart. In line with prior cross-sectional and experimental research, we hypothesize

that lower SSS will be associated with greater prospective multiyear increases in IL-6 and CRP independently of objective measures of SES. We also consider whether these associations differ by race to the extent possible in this sample (White vs. non-White) and sex assigned at birth, however, we do not have clear directional hypotheses given prior mixed findings. We further account for potential psychological factors and emergent health conditions that may contribute to observed associations.

We also conduct two sets of exploratory analyses. First, we examine pathways through which SSS may relate to multiyear changes in circulating levels of IL-6 and CRP. Informed by prior literature [14, 23–25], we focus on baseline psychological factors (i.e., depressive changes and hopelessness) and weight (as indicated by changes in body mass index [BMI]), as well as changes in these factors over time as these factors may plausibly change over the 2–3 years between visits. We hypothesize that lower SSS at baseline will cross-sectionally relate to higher BMI, depressive symptoms, and hopelessness, as well as greater increases in BMI, depressive symptoms, and hopelessness between visits. In turn, baseline differences and changes in these three factors between visits will associate with greater multiyear increases in IL-6 and CRP. It is also possible that SSS and objective SES indicators could increase or decrease between visits and that this change could promote changes in circulating levels of IL-6 and CRP. Thus, a second exploratory analysis will consider whether multiyear changes in SSS and income associate with multiyear changes in circulating levels of IL-6 and CRP. We hypothesize that participants who report a decrease in SSS and income from baseline to follow-up will evidence greater increases in circulating levels of IL-6 and CRP whereas participants who report improvements in SSS and income over time will evidence more modest increases in IL-6 and CRP compared with the rest of the sample. We do not examine changes in educational attainment between visits as we do not expect education to vary substantively given the age of the sample.

Materials and Methods

Participants

Participants were 331 healthy community-dwelling adults between the ages of 30–51 years (50.5% female, 24.2% Black/African American, 69.5% White) who were recruited from Allegheny county, Pennsylvania, to partake in the Pittsburgh Imaging Project (PIP). See Table 1 for additional details on sample characteristics stratified by race and sex. Participation consisted of a series of baseline visits followed by a second visit approximately three years later (median = 2.85 years, interquartile range = 3.47 years). Exclusion criteria at baseline included having a diagnosis of CVD, diabetes, cancer, pulmonary, or respiratory disease, or regularly taking any lipid-lowering, insulin, cardiovascular, hypoglycemic, glucocorticoid, or weight loss medication. These conditions and medications to treat these conditions could impact inflammation. Adults were also ineligible if they were pregnant, had a substance or mood disorder, or were taking psychotropic medications at baseline. Additional exclusion criteria for the larger study that were not specific to the analyses presented here included colorblindness, claustrophobia, or having a neurological condition, cerebrovascular trauma history, or ferromagnetic implants of any kind. Please see Gianaros et al. for more information on study design and recruitment [46, 47]. Participants provided informed consent and the University of Pittsburgh's Institutional Review Board approved the study.

Procedure

At the baseline and follow-up visits, participants provided written consent, underwent a blood draw, had their anthropometric measurements taken, and completed questionnaires and a semistructured interview. They also completed psychophysiological assessments and an fMRI scanning session, but these measures were not part of the present secondary analyses. Baseline and follow-up data were collected between 2008 and 2017.

Measures

Subjective social status

Participants completed the MacArthur Scale of Subjective Social Status [7], which has been shown to have adequate test–retest reliability and predictive utility for a range of health-relevant outcomes among racially diverse samples (e.g., [48]). Participants were presented with two 10-rung ladders. For the first ladder, participants were told that the ladder represented where people stood in the USA. The top of the ladder (rung 9) was said to represent people who were the best off, including those who had the most education, highest income, and best jobs. The bottom of the ladder (rung 0) was said to represent those in the USA who were the worst off, including those who had the least money and education and the worst (or no) employment. Participants were then asked to place an "X" on the rung where they felt they stood in comparison to people in the US (SSS_{US}). Next, participants were shown a second ladder and given similar instructions, except that the ladder now represented people in their community (SSS_{C}). Participants were encouraged to self-define "community" however they wished. SSS_{US} and SSS_{C} were correlated with one another at baseline (r = .51) and follow-up (r = .56) and covered the entire range of possible scores (0–9).

Objective SES

Education

At baseline, participants indicated which of the following categories best matched their highest level of completed education: less than high school (HS), HS diploma or equivalent (general educational development), technical or vocational training, some college, Associate's degree, Bachelor's degree, Master's degree, or Doctoral degree. Based on the sample distribution, educational attainment was collapsed into four categories: HS degree, equivalent or less (n = 27), some postsecondary education (n = 104), Bachelor's degree (n = 99), and graduate degree (n = 101). Dichotomous variables were created with Bachelor's degree as the reference group.

Family-adjusted income

At both visits, participants reported on their family income by selecting which of the following categories best matched their family's yearly income before taxes: <$10,000, $10,000–$14,999, $15,000–$24,999, $25,000–$34,999, $35,000–$49,999, $50,000–$64,999, $65,000–$79,999, $80,000–$94,999, $95,000–$109,999, $110,000–$124,999, $125,000–$139,999, $140,000–$154,999, $155,000–

Table 1 Sample Descriptives

	Entire sample (n_b = 331; n_f = 278)	Racial minority (n_b = 101; n_f = 82)	White (n_b = 230; n_f = 196)	Women (n_b = 167; n_f = 143)	Men (n_b = 164; n_f = 135)
	n (%) or *M* (*SD*)	*n* (%) or *M* (*SD*)	*n* (%) or *M* (*SD*)	*n* (%) or *M* (*SD*)	*n* (%) or *M* (*SD*)
Race					
Asian	15 (4.5)			5 (3.0)	10 (6.1)
Black or African American	80 (24.2)			46 (27.5)	34 (20.7)
Indigenous or Native American	0 (0)			0 (0)	0 (0)
White	230 (69.5)			113 (67.7)	117 (71.3)
Bi- or Multi-racial	3 (0.9)			1 (0.6)	2 (1.2)
Other race	3 (0.9)			2 (1.2)	1 (0.6)
Female sex assigned at birth	167 (50.5)	54 (53.5)	113 (49.1)		
Age (baseline)	40.24 (6.24)	40.45 (6.08)	40.14 (6.31)	40.81 (6.17)	39.65 (6.27)
BMI (kg/m^2; baseline)	26.88 (5.05)	28.12 (5.78)	26.33 (4.60)	27.09 (5.85)	26.65 (4.08)
BMI (kg/m^2; follow-up)	26.60 (5.23)	28.88 (6.18)	26.94 (4.69)	27.89 (5.83)	27.12 (4.51)
Change in BMI	0.42 (1.92)	0.39 (2.14)	0.43 (1.83)	0.65 (2.11)	0.45 (1.71)
Smoker (current or prior)	124 (37.5)	51 (50.5)	73 (31.7)	72 (43.1)	52 (31.7)
Time between visits (in years)	2.86 (0.59)	2.82 (0.53)	2.88 (0.61)	2.84 (0.45)	2.88 (0.70)
SES and SSS (baseline)					
Educational attainment					
Less than high school	2 (0.6)	2 (2.0)	0 (0)	0 (0)	2 (1.2)
High school degree or equivalent	25 (7.6)	13 (12.8)	12 (5.2)	14 (8.4)	11 (6.7)
Technical or vocational training	9 (2.7)	3 (3.0)	6 (2.6)	6 (3.6)	3 (1.8)
Some college (no degree)	51 (15.4)	22 (21.8)	29 (12.6)	22 (13.2)	29 (17.7)
Associate's degree	44 (13.3)	18 (17.8)	26 (11.3)	28 (16.8)	16 (9.8)
Bachelor's degree	99 (29.9)	17 (16.8)	82 (35.7)	47 (28.1)	52 (31.7)
Master's degree	68 (20.5)	17 (16.8)	51 (22.2)	39 (23.4)	29 (17.7)
Doctoral degree	33 (10)	9 (8.9)	24 (10.4)	11 (6.6)	22 (13.4)
Family-adjusted income	$45,994 ($30,342)	$35,044 ($30,158)	$50,715 ($29,244)	$42,252 ($27,883)	$49,830 ($32,311)
SSS_{US}	4.41 (1.64)	3.81 (1.73)	4.67 (1.53)	4.32 (1.51)	4.49 (1.76)
SSS_C	5.07 (1.65)	5.01 (1.69)	5.10 (1.63)	5.09 (1.61)	5.05 (1.68)
SES and SSS (follow-up)					
Family-adjusted income	$53,712 ($33,090)	$36,380 ($28,816)	$60,970 ($32,114)	$48,622 (29,415)	$59,071 (35,901)
SSS_{US}	4.64 (1.66)	4.05 (1.81)	4.89 (1.53)	4.47 (1.54)	4.82 (1.76)
SSS_C	5.09 (1.65)	4.84 (1.75)	5.20 (1.60)	5.03 (1.64)	5.16 (1.66)
Change in family-adjusted income	$6,319 ($20,481)	$950 ($21,798)	$8,559 ($19,533)	$5,049 ($19,813)	$7,657 ($21,157)
Change in SSS_{US}	0.15 (1.34)	0.10 (1.37)	0.17 (1.34)	0.09 (1.29)	0.21 (1.40)
Change in SSS_C	−0.01 (1.60)	−0.22 (1.73)	0.07 (1.54)	−0.13 (1.51)	0.10 (1.68)
Depressive symptoms (baseline)	3.35 (3.38)	3.59 (3.32)	3.24 (3.40)	3.30 (2.92)	3.40 (3.79)
Depressive symptoms (follow-up)	4.47 (4.84)	5.12 (5.94)	4.19 (4.29)	4.91 (4.94)	4.00 (4.70)
Change in depressive symptoms	1.12 (4.14)	1.14 (5.04)	0.99 (3.71)	1.50 (4.34)	0.72 (3.89)
Hopelessness (baseline)	1.38 (1.75)	1.59 (1.75)	1.28 (1.64)	1.29 (1.69)	1.47 (1.81)
Hopelessness (follow-up)	1.32 (1.74)	1.48 (1.97)	1.26 (1.63)	1.17 (1.57)	1.48 (1.89)
Change in hopelessness	−0.04 (1.86)	−0.15 (2.49)	0.01 (1.53)	−0.15 (1.77)	0.09 (1.95)
IL-6 (baseline, pg/mL, raw)	1.50 (1.22)	1.70 (1.52)	1.40 (1.06)	1.53 (1.33)	1.45 (1.12)
IL-6 (follow-up, pg/mL, raw)	1.88 (1.24)	2.44 (1.51)	1.65 (1.02)	1.89 (1.13)	1.87 (1.34)
Change in IL-6 (log)	0.00 (0.58)	0.02 (0.66)	−0.01 (0.55)	−0.01 (0.56)	0.01 (0.66)
CRP (baseline, mg/L, raw)	0.24 (0.38)	0.32 (0.55)	0.21 (0.28)	0.28 (0.38)	0.21 (0.38)
CRP (follow-up, mg/L, raw)	0.29 (0.46)	0.41 (0.60)	0.24 (0.38)	0.35 (0.47)	0.23 (0.44)
Change in CRP (log)	0.11 (0.91)	0.19 (1.00)	0.07 (0.87)	0.07 (0.90)	0.15 (0.92)

b baseline; *BMI* body mass index; *CRP* C-reactive protein; *f* follow-up; *IL-6* interleukin-6; *M* mean; *SD* standard deviation; *SES* socioeconomic status; SSS_C subjective social status relative to others in the participant's community; SSS_{US} subjective social status relative to others in the USA. Family-adjusted income was calculated as the midpoint of annual family income divided by the cubed root of the number of people living in the household. Prior to fitting multiple regression models, IL-6 at both visits was log-transformed and a change score was calculated by subtracting log-transformed IL-6 at baseline from log-transformed IL-6 at follow-up.

$169,999, $170,000–$185,000, >$185,000. The midpoint of these ranges was calculated to estimate each participant's average family income (e.g., $12,500 as midpoint for $10,000–$14,999). To account for family size, family-adjusted income was calculated by dividing the midpoint value for family income by the cubed root of the number of people in the household.

Demographic moderators

At baseline, participants self-identified their race (Asian/Asian American, Black/African American, Indigenous or Native American, White, Bi- or multi-racial, or another race) and sex assigned at birth (female or male). Race is a social construct that was coded as White (0) and non-White (1) to account for the potential role of racial inequality in access to resources and experiences of discrimination among people of color on associations between SSS and changes in IL-6 [49, 50]. Although not ideal, we chose this approach because we were not powered to look at racial minority groups separately given that only 23 participants identified with a racial minority group other than Black/African American. To maintain power, we decided to code race as White or non-White rather than exclude these 23 participants. Then, in post hoc sensitivity analyses, we examined whether associations held when limiting the sample to Black and White adults only (please see below).

Psychological factors

Depressive symptoms and hopelessness, two related but distinct constructs, could relate to associations between SSS and prospective changes in IL-6. As such, participants completed the 21-item Beck Depression Inventory (BDI) [51] and 2-item Hopelessness Scale [52]. These scales had acceptable internal consistency (BDI: $\alpha = 0.84$; Hopelessness Scale: $r = .47$) and were correlated with one another within the sample ($r = .50$).

IL-6 and CRP

Participants underwent a fasting blood draw between 7:00 and 11:00 AM as part of each visit. Participants were advised to refrain from eating, drinking (except water), physical activity, and using tobacco products for 8 hr before the blood draw and were rescheduled if they were currently sick, taking antibiotics or antivirals, or had received a vaccine or tattoo in the two weeks before their appointment. Blood was drawn by antecubital venipuncture into sodium citrate and serum separator tubes that were then centrifuged within 1 hr of collection. Plasma samples were aliquoted and frozen at −80°C before being batch processed by the University of Pittsburgh's Behavioral Immunology Laboratory to quantify levels of IL-6 using a high sensitivity enzyme-linked immunoassay kit (Human IL-6 Quantikine High Sensitivity ELISA, R&D Systems) with a detection range 0.2–10 pg/mL and intra-assay coefficient of variation of 4.24%. Serum samples were sent to the University of Pittsburgh's Clinical Services Laboratory where levels of CRP were measured using a high sensitivity CRP assay with a SYNCHRON LX system (Beckman Coulter) and a CRPH reagent (intra-assay coefficient of variation = 5.0%). All samples were run in duplicate for both IL-6 and CRP, respectively.

When examining IL-6 values, one baseline value (15.22 pg/mL) was removed for being more than 9 standard deviations (*SD*) above the sample mean. Given the participant's BMI (26.4%), this value was unlikely to be biologically possible in the absence of infection. IL-6 and CRP values at both baseline and follow-up were log-transformed to normalize their distributions. Change in circulating levels of IL-6 was then calculated by subtracting log-transformed IL-6 levels at baseline from log-transformed IL-6 levels at follow-up. This same process was done to calculate change in circulating levels of CRP.

Covariates

Participants reported their age and tobacco use and had their height and weight taken to calculate their BMI (kg/m^2). To account for between-person differences in the length of time between baseline and follow-up visits, we calculated the years between the baseline and follow-up assessments.

Statistical Analyses

In transparency, the preregistered analyses presented here deviate from those initially proposed by (i) using multiple regression instead of a series of hierarchical stepwise regression models that would have added SSS, income, and education one at a time and (ii) limiting our scope to psychological factors (i.e., hopelessness and depressive symptoms) versus psychological factors and health behaviors, and (iii) exploring whether baseline psychological factors and BMI, as well as changes in these factors from baseline to follow-up, could be potential pathways through which SSS may relate to multiyear changes in IL-6 and CRP, respectively. The first two steps were taken before analyzing the data in an effort to decrease the risk of Type I error and the third step was taken in response to reviewer interest in exploring mechanisms that could inform future investigations. In the preregistration, we also discussed general associations between SSS and changes in inflammation and did not specify the type of ladder. Here, we examine separate associations between SSS_{US} and SSS_{C} in relation to changes in inflammation given that links between SSS and health-related outcomes may meaningfully differ depending on whether people are asked to compare themselves to others in their direct communities or in the broader USA [31, 38].

Prior to fitting regression models, we examined variable distributions and correlations between study variables. For primary analyses, multiple regressions were used to model independent associations between SSS and change in IL-6 levels from baseline to follow-up, adjusting for baseline IL-6, educational attainment, family-adjusted income, age, race, sex, smoking status, BMI, and time between visits. Separate regression models were fit for SSS_{US} and SSS_{C} given their strong intercorrelation. Next, moderation effects of race and sex were assessed using Hayes' PROCESS macro with SSS as the predictor and either race (1 = non-White; 0 = White) or sex (1 = female, 0 = male) as the moderator. For significant main and moderation effects only, we then added depressive symptoms and hopelessness one at a time to the models to see whether the results were robust to differences in these psychological factors. The above models were then refit with change in CRP as the dependent variable, adjusting for baseline CRP and demographic and psychological covariates.

For our two sets of exploratory analyses, we first calculated change scores for depressive symptoms, hopelessness, BMI, SSS_{US}, SSS_{C}, and family-adjusted income by subtracting participant's baseline values for each of these measures from their values at follow-up. For the first set of exploratory analyses, we used PROCESS (Model 4) to examine whether there

was an indirect effect of SSS on changes in IL-6 and CRP through baseline levels of depressive symptoms, hopelessness, and BMI, as well as changes in these three factors between the baseline and follow-up visit. Models were fit separately for SSS_{US} and SSS_{C} and we estimated standardized indirect effects for each pathway using 5,000 bootstrapped samples and a seed of 5,129. The indirect effect pathway (*ab*) was considered significant if the bootstrapped 95% confidence intervals (boot 95% CIs) did not contain zero. Indirect effect models adjusted for the same covariates included in the primary analyses. To ground the change scores, we further adjusted for baseline depressive symptoms, hopelessness, and BMI.

For the second set of exploratory analyses, we tested whether changes in SSS and family-adjusted income from baseline to follow-up were associated with multiyear changes in IL-6 and CRP. We did this by fitting multiple regression models predicting changes in IL-6 and CRP from changes in SSS and family-adjusted income. Similarly, we adjusted for the same covariates included in the primary analyses and grounded the change scores by additionally adjusting for baseline SSS_{C}, SSS_{US}, and family-adjusted income.

Finally, we ran three post hoc sensitivity analyses for the statistically significant models. First, we used the natural log of family-adjusted income in place of raw family-adjusted income. Second, we limited the sample to Black and White adults to determine whether significant effects held when excluding 23 participants who identified as a racial minority group other than Black or African American. Third, we excluded participants who developed or started taking medications for new chronic health conditions during the time between their baseline and follow-up visits. This included participants who reported a cardiac event (myocardial infarction; $n = 1$), traumatic ischemic attack ($n = 1$), or cancer (adenocarcinoma; $n = 1$) or were told by a doctor on two separate occasions that they had high blood pressure ($n = 5$, including the participant who had a myocardial infarction) or chronic obstructive pulmonary disease ($n = 1$) that required medication management. At follow-up, one participant indicated that they were taking antihypertensive medication for the past decade. Had this participant self-reported this medication use at baseline, they would have been ineligible for the study. Consequently, post hoc sensitivity analyses excluded this participant and the eight other participants diagnosed with and currently taking medications for the abovementioned health conditions. Results remained substantively unchanged with two exceptions noted below. Thus, the results presented here include data on all participants. All statistical analyses were performed in R (R Core Team).

Results

See Table 1 for sample descriptives. Participants were 30–51 years of age (mean = 40.24 years) and the majority identified as White ($n = 230$, 69.5%) and female ($n = 167$, 50.5%). Of the 331 participants who completed the baseline visit, 278 participants were retained for the follow-up visit. There were no sociodemographic differences between those who were retained and those lost to follow-up except that retained participants were slightly older than those who completed the baseline visit only (40.58 vs. 38.37 years; $t[329] = 2.372$, $p = .018$). Change in log IL-6 from baseline to follow-up was minimal but varied across participants (mean = 0.00 pg/mL, *SD* = 0.58 pg/mL), with some participants demonstrating an increase in IL-6 between visits whereas others showed no change or even a decrease in log IL-6 levels over time (*range* = −1.94 to− 3.15 pg/mL). In contrast, there was a slight increase in log CRP, on average, between baseline and follow-up (mean = 0.11 mg/L, *SD* = 0.91 mg/L); however, the extent of this change varied across participants (*range* = −2.59 to 2.944).

Both SSS ladders were correlated with one another and with family-adjusted income, but the strength of these correlations differed. Specifically, correlations between SSS_{US} and income (rs = .48–.53) were stronger than correlations between SSS_{C} and income (rs = .21–.27; ps < .05). See Table 2 for a correlation matrix of all continuous study variables and Supplementary Tables S1 and S2 for correlation tables separated by race and sex, respectively.

Primary Analyses

SSS_{C} was longitudinally associated with a greater multiyear change in IL-6 independently of age, sex, race, years between visit, BMI, smoking status, education, and income (β = −0.06, $p = .021$). This association held when further adjusting for hopelessness (β = −0.06, $p = .021$) and depressive symptoms (β = −0.06, $p = .022$; Table 3) and was substantively unchanged in all post hoc sensitivity analyses.

SSS_{US} was longitudinally associated with a greater multiyear change in IL-6 independently of age, sex, race, years between visit, BMI, smoking status, education, and income (β = −0.06, $p = .047$; Table 3). However, this association was slightly attenuated after accounting for symptoms of hopelessness (β = −0.06, $p = .055$) and depression (β = −0.06, $p = .057$; Table 4). Additionally, this effect was attenuated in post hoc sensitivity analyses using the natural log of family-adjusted income ($p = .070$), when limiting the sample to Black and White adults only ($p = .060$), and when excluding participants who developed chronic health conditions in between the baseline and follow-up visit ($p = .053$).

Neither race nor sex moderated associations between either SSS_{US} or SSS_{C} and prospective multiyear changes in IL-6 (all ps > .10; Supplementary Table S3). There were also no associations between SSS_{C} or SSS_{US} and multiyear change in CRP (ps > .30; Table 5) nor evidence of moderation by race or sex (ps > .30; Supplementary Table S4).

Exploratory Analyses: Pathways

There were no indirect effects of SSS on multiyear changes in IL-6 or CRP through *baseline* levels of depressive symptoms, hopelessness, and BMI (all boot 95% CIs contained zero).

On average, depressive symptoms (mean = 1.12, *SD* = 4.14) and BMI (mean = 0.42, *SD* = 1.92) slightly increased from baseline to follow-up while mean hopelessness decreased (mean = −0.04, *SD* = 1.36). With one exception, there were no indirect effects of SSS on multiyear changes in inflammation through *changes* in depressive symptoms, hopelessness, or BMI from baseline to follow-up. Specifically, there was a significant indirect effect of SSS_{C} on multiyear change in CRP through change in BMI (*ab* = −0.002, Boot SE = 0.001, Boot 95% CI = [−0.004, −0.001]; Fig. 1); however, this was in the absence of a total effect of SSS_{C} on change in CRP (β = −0.072, $p = .317$). Lower SSS_{C} was associated with an increase in BMI from baseline to follow-up ($a = -0.186$, $p = .007$) and an increase in BMI was in turn associated with an increase in CRP from baseline to follow-up ($b = 0.339$, $p < .001$). This indirect effect held in all three post hoc sensitivity

Table 2 Correlation Matrix of Continuous Study Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. SSS_{US} (baseline)	1														
2. SSS_{C} (baseline)	.51**	1													
3. SSS_{US} (follow-up)	.66**	.40**	1												
4. SSS_{C} (follow-up)	.48**	.53**	.56**	1											
5. IL-6 (log; baseline)	−.17**	−.13*	−.17**	−.06	1										
6. IL-6 (log; follow−up)	-.27**	-.21**	-.24**	-.17**	.33**	1									
7. CRP (log; baseline)	−.10	−.03	−.16	−.06	.37**	.33**	1								
8. CRP (log; follow-up)	−.14	−.04	−.18	−.01	.36**	.51**	.68	1							
9. Age	.04	.08	.01	.04	.13*	.10	−.01	.03	1						
10. Body mass index	−.15**	−.10	−.25**	−.05	.34**	.33**	.41**	.47**	.12*	1					
11. Income (baseline)	.49**	.23**	.50**	.27**	−.20**	−.16**	−.11	−.09	.13*	−.06	1				
12. Income (follow-up)	.48**	.21**	.53**	.27**	−.26**	−.23**	−.12	−.18**	.05	−.14*	.80**	1			
13. Years between visits	−.03	−.11	−.08	−.03	.09	−.07	−.03	−.03	−.10	−.01	.00	−.11	1		
14. Depressive symptoms	−.20**	−.18**	−.27**	−.20**	.10	.11	.05	.14*	.02	.03	−.12*	−.21**	.02	1	
15. Hopelessness	−.18**	−.20**	−.23**	−.21**	.16**	.12	.02	.03	.11	.04	−.11	−.20**	.04	.50**	1

CRP C-reactive protein; *IL-6* interleukin-6; SSS_C subjective social status relative to others in the participant's community; SSS_{US} subjective social status relative to others in the USA. Family-adjusted income was calculated as the midpoint of annual family income divided by the cubed root of the number of people living in the household.
*Correlation significant at $p < .05$.
**Correlation significant at $p < .01$.

Table 3 Results of Multiple Linear Regression Analyses Examining Independent Associations Between Subjective Social Status and Multiyear Changes in Interleukin-6 Using the Community Ladder

	Effects of SSS_C independent of objective measures of SES				Independent effects of SSS_C + hopelessness				Independent effects of SSS_C + depressive symptoms			
	B	*SE*	β	*p*	*B*	*SE*	β	*p*	*B*	*SE*	β	*p*
Change in IL-6 from baseline to follow-up												
Intercept	0.231	0.156		.141	0.228	0.157		.149	0.226	0.157		.153
IL-6 (log; baseline)	−0.910	0.025	−0.957	<.001	−0.911	0.025	−0.957	<.001	−0.911	0.025	−0.957	<.001
Years between visits	−0.070	0.027	−0.063	.010	−0.069	0.027	−0.063	.011	−0.069	0.027	−0.063	.011
Female sex	−0.001	0.028	−0.001	.979	0.001	0.029	0.001	.971	0.000	0.029	0.000	.990
Racial minority	0.095	0.033	0.074	.004	0.097	0.033	0.075	.004	0.097	0.033	0.075	.004
Age	0.001	0.002	0.011	.651	0.001	0.002	0.011	.654	0.001	0.002	0.011	.651
Body mass index	0.007	0.003	0.057	.029	0.007	0.003	0.057	.029	0.007	0.003	0.057	.029
Tobacco smoker	−0.004	0.032	−0.004	.890	−0.006	0.032	−0.005	.855	−0.007	0.032	−0.005	.836
SSS_C	−0.021	0.009	−0.059	.021*	−0.021	0.009	−0.059	.021*	−0.021	0.009	−0.059	.022*
HS degree, equivalent or less	0.110	0.062	0.048	.078	0.109	0.063	0.048	.086	0.109	0.062	0.048	.080
Some college or Associate's degree	0.037	0.039	0.029	.344	0.035	0.040	0.027	.378	0.035	0.040	0.027	.375
Bachelor's degree (referent)												
Master's degree	−0.042	0.036	−0.033	.243	−0.042	0.036	−0.034	.241	−0.042	0.036	−0.034	.241
Family-adjusted income	0.000	0.000	0.000	.989	0.000	0.000	0.001	.982	0.000	0.000	0.001	.975
Hopelessness					0.001	0.008	0.002	.936				
Depressive symptoms									0.001	0.003	0.005	.851
Model fit	R^2 = 0.867, adjusted R^2 = 0.860				R^2 = 0.867, adjusted R^2 = 0.860				R^2 = 0.866, adjusted R^2 = 0.860			

B unstandardized beta coefficient; *IL-6* interleukin-6; *SE* standard error; *SES* socioeconomic status; SSS_C subjective social status relative to others in the participant's community; *β* standardized beta coefficient. Family-adjusted income was calculated as the midpoint of annual family income divided by the cubed root of the number of people living in the household.
*$p < .05$ for association between SSS_C and change in IL-6.

Table 4 Results of Multiple Linear Regression Analyses Examining Independent Associations Between Subjective Social Status and Multiyear Changes in Interleukin-6 Using the U.S. Ladder

	Effects of SSS_{US} independent of objective measures of SES				Independent effects of SSS_{US} + hopelessness				Independent effects of SSS_{US} + depressive symptoms			
	B	*SE*	β	*p*	*B*	*SE*	β	*p*	*B*	*SE*	β	*p*
Change in IL-6 from baseline to follow-up												
Intercept	0.232	0.159		.146	0.227	0.160		.157	0.225	0.161		.163
IL-6 (log; baseline)	−0.907	0.025	−0.953	<.001	−0.908	0.025	−0.954	<.001	−0.907	0.025	−0.953	<.001
Years between visits	−0.070	0.027	−0.063	.010	−0.070	0.027	−0.063	.010	−0.070	0.027	−0.063	.010
Female sex	−0.004	0.028	−0.004	.878	−0.003	0.029	−0.002	.924	−0.004	0.029	−0.004	.882
Racial minority	0.083	0.033	0.064	.012	0.084	0.033	0.065	.012	0.084	0.033	0.065	.012
Age	0.001	0.002	0.006	.818	0.000	0.002	0.005	.841	0.001	0.002	0.005	.826
Body mass index	0.007	0.003	0.058	.027	0.007	0.003	0.059	.027	0.007	0.003	0.058	.026
Tobacco smoker	−0.008	0.032	−0.006	.806	−0.009	0.032	−0.007	.788	−0.010	0.032	−0.008	.758
SSS_{US}	−0.021	0.011	−0.057	.047*	−0.021	0.011	−0.056	.055	−0.021	0.011	−0.055	.057
HS degree, equivalent or less	0.116	0.062	0.051	.062	0.114	0.063	0.050	.073	0.116	0.062	0.051	.063
Some college or Associate's degree	0.033	0.040	0.026	.406	0.031	0.040	0.024	.437	0.032	0.040	0.025	.426
Bachelor's degree (referent)												
Master's degree	−0.043	0.036	−0.034	.236	−0.043	0.036	−0.034	.231	−0.043	0.036	−0.034	.233
Family-adjusted income	<0.001	0.000	0.013	.659	0.000	0.000	0.013	.653	0.000	0.000	0.013	.649
Hopelessness					0.002	0.008	0.006	.797				
Depressive symptoms									0.001	0.003	0.008	.744
Model fit	R^2 = 0.866, adjusted R^2 = 0.860				R^2 = 0.866, adjusted R^2 = 0.860				R^2 = 0.866, adjusted R^2 = 0.860			

B unstandardized beta coefficient; *IL-6* interleukin-6; *SE* standard error; *SES* socioeconomic status; SSS_{US} subjective social status relative to others in the USA; *β* standardized beta coefficient. Family-adjusted income was calculated as the midpoint of annual family income divided by the cubed root of the number of people living in the household.
*$p < .05$ for association between SSS_{US} and change in IL-6.

Table 5 Results of Nonsignificant Multiple Linear Regression Analyses Examining Independent Associations Between Subjective Social Status and Multiyear Changes in C-Reactive Protein

	Independent effects of SSS_{US}				Independent effects of SSS_C			
	B	*SE*	β	*p*	*B*	*SE*	β	*p*
Change in CRP from baseline to follow-up								
Intercept	−1.538	0.630		.015	−1.641	0.637		.011
CRP (log; baseline)	−0.416	0.057	−0.511	.000	−0.417	0.057	−0.511	.000
Years between visits	−0.030	0.090	−0.021	.739	−0.029	0.090	−0.020	.751
Female gender	0.051	0.114	0.028	.653	0.054	0.114	0.029	.640
Racial minority	0.180	0.132	0.090	.175	0.198	0.133	0.099	.137
Age	−0.003	0.009	−0.018	.772	−0.003	0.009	−0.018	.768
Body mass index	0.037	0.012	0.212	.003	0.037	0.012	0.212	.003
Tobacco smoker	0.016	0.128	0.008	.903	0.026	0.129	0.014	.843
SSS	−0.040	0.040	−0.072	.317	−0.012	0.036	−0.022	.734
HS degree, equivalent or less	−0.091	0.235	−0.027	.699	−0.095	0.237	−0.028	.690
Some college or Associate's degree	0.069	0.155	0.035	.655	0.088	0.154	0.044	.571
Bachelor's degree (referent)								
Master's degree	0.050	0.143	0.026	.726	0.046	0.145	0.023	.752
Family-adjusted income	0.000	0.000	0.025	.733	0.000	0.000	0.003	.969
Model fit	R^2 = 0.211, adjusted R^2 = 0.168				R^2 = 0.208, adjusted R^2 = 0.165			

B unstandardized beta coefficient; *CRP* C-reactive protein; *SE* standard error; *SES* socioeconomic status; SSS_C subjective social status relative to others in the participant's community; *β* standardized beta coefficient. Family-adjusted income was calculated as the midpoint of annual family income divided by the cubed root of the number of people living in the household.

analyses. All other indirect effect models were nonsignificant as indicated by bootstrapped 95% CIs containing zero.

Exploratory Analyses: Changes in SSS and Family-Adjusted Income

Changes in SSS_{US} (mean = 0.15, *SD* = 1.34), SSS_C (mean = −0.01, *SD* = 1.60), and family-adjusted income (mean = $6,319, *SD* = $20,480.95) were modest. Neither change in SSS_{US}, SSS_C, or family-adjusted income was associated with a prospective change in circulating levels of IL-6 or CRP (all *p*s > .30).

Discussion

This study examined the extent to which SSS was longitudinally associated with multiyear changes in circulating levels of IL-6 and CRP independently of objective measures of SES. We found that individual differences in changes in IL-6 were correlated with SSS. More specifically, complimenting experimental research demonstrating links between lower SSS and transient increases in IL-6 in response to a laboratory stressor [19, 20], we found that lower SSS was associated with greater multiyear increases in circulating levels of IL-6 independently of education and family-adjusted income. Moreover, the association between SSS and changes in IL-6 was not accounted for by psychological factors nor explained by baseline levels of depressive symptoms, hopelessness, BMI, or changes in these three factors between baseline and follow-up visits.

Notably, independent associations between SSS and multiyear increases in IL-6 were more consistent when participants were asked to report on their SSS in relation to others in their direct community versus the broader USA. Specifically, the association between SSS_C and change in IL-6 was robust to the inclusion of psychological factors and held in all post hoc sensitivity analyses, whereas the effect of SSS_{US} on change in IL-6 became statistically nonsignificant when adjusting for hopelessness and depressive symptoms and post hoc sensitivity analyses. Nonetheless, given that the standardized beta coefficients were comparable for SSS_{US} and SSS_C, we suspect that the association between SSS_{US} and multiyear changes in IL-6 would have been statistically significant in a larger sample. In addition, although hopelessness and depressive symptoms explained some of the variance in changes in IL-6 attributed to SSS_{US}, neither psychological factor was an independent driver of changes in IL-6 within this sample. This seems to suggest that the association between SSS_{US} and changes in IL-6 is not attributed to differences in psychological factors, at least in this sample.

It is not clear why there were more consistent associations between SSS_C and changes in IL-6. When looking at prior studies of SSS and inflammation, one focused on SSS_{US} [19], one focused on SSS_C [15], and two used a more general society ladder that did not include a specific referent group [14, 18]. However, a meta-analysis by Zell et al. [6] found comparable small, positive associations of SSS_{US} and SSS_C on health parameters (e.g., health behaviors, self-rated health, biomarkers) even when including both as predictors in the same model. In light of our findings and that the two ladders were strongly (but not perfectly) correlated in our sample, this collectively suggests that future work should consider both ladders and how the type of ladder may modify the relation between SSS and inflammation by collecting qualitative data to elucidate factors that may underlie differences in participants' ratings for each of these ladders [31].

Although purely speculative, one reason for more robust effects of SSS_C versus SSS_{US} on multiyear changes in IL-6 could be reflective of differences in the salience of the self-relevant comparison groups referenced in the instructions of the two ladders scales and to whom participants are comparing

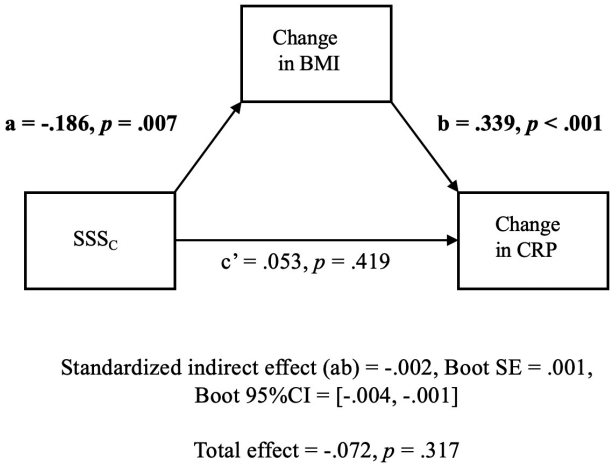

Fig. 1. There was an indirect effect of community subjective social status (SSS_C) on multiyear change in circulating levels of C-reactive protein (CRP) through change in adiposity as measured by body mass index (BMI). Specifically, adults who reported lower SSS_C at baseline had an increase in their BMI between the baseline and follow-up visit. A greater increase in BMI was in turn associated with a multiyear increase in CRP levels. Nonetheless, the total effect of SSS_C on multiyear change in CRP was not significant ($p > .30$).

themselves. To elaborate, people may be reminded of where they stand in their communities on a regular basis through daily interactions with other community members. This may manifest as receiving verbal or financial recognition at work to being acknowledged with a wave or greeting while walking in their neighborhood, all of which serve as reminders of their worth and value. In contrast, SSS in relation to other U.S. adults may be more abstract and may not be as salient if most of people's daily interactions are with others in their direct communities. Thus, it may be that when making U.S. comparisons, people tend to abstractly think about how they compare to other Americans on more objective SES indicators, such as education or income, rather than more subtle aspects of SES such as respect where they may have concrete experiences or referents in their social networks. Indeed, SSS_{US} was more strongly correlated with family-adjusted income compared with SSS_C in the sample. Coupled with the fact that the salience of SSS may change depending on the self-relevant context (e.g., at work or in one's neighborhood), future research could consider whether SSS measured in daily life may relate to changes in inflammation over time and whether these associations differ depending upon to whom people are comparing themselves.

Nevertheless, neither SSS_{US} nor SSS_C was directly associated with changes in CRP even though there was an average increase in CRP levels from baseline to follow-up within the sample. Rather, in exploratory analyses we found that there was an indirect effect of SSS_{US} on changes in circulating levels of CRP through changes in BMI. Specifically, lower SSS_{US} at baseline was associated with an increase in BMI between visits and this increase in BMI was in turn associated with an increase in circulating levels of CRP. This could suggest that SSS may be directly tied to multiyear changes in IL-6 but indirectly to CRP through other means, such as increases in adiposity. Then again, we may have simply missed other pathways, such as changes in sleep and sedentary behavior, that may be more relevant to SSS and multiyear changes in IL-6. Nonetheless, these results should be interpreted with caution and replicated in larger samples given that they were exploratory and that the total effect and all other indirect pathways were nonsignificant. Thus, longitudinal research with multiple waves of data collected over a longer period of time is needed to discern (i) to what extent the multiyear increases in circulating levels of IL-6 that we observed here are reflective of a trajectory of increasing inflammation that ultimately leads to a clinical diagnosis of CVD or diabetes, (ii) whether this pattern of effects eventually emerges for CRP, and (iii) what biopsychosocial mechanisms may account for these observed associations.

In addition, neither race nor sex moderated associations between SSS and prospective changes in IL-6 and CRP. We were likely underpowered to examine these conditional effects, especially for racial differences as only 82 of the 278 participants who completed the follow-up visit identified with a racial minority group. There may have also been meaningful differences that we missed based on how race and sex were operationalized. Unfortunately, given the sample size and distribution, we were not powered to examine associations separately for people from different racial backgrounds nor could we examine the role of ethnic identity as only 5 participants identified as Hispanic or Latine. Similarly, we were not powered to conduct moderated mediation analyses to consider whether indirect effects of SSS on changes in IL-6 and CRP differed by race and sex, which is a needed area for future investigation. We also did not consider nativity status, which has been found to moderate associations between SSS and health among certain racial minority groups [53, 54]. Finally, participants reported on their sex assigned at birth, which may not align with their gender identity. Thus, future research is needed to disentangle the effects of sex and gender identity on associations between SSS and prospective change in IL-6 and CRP.

The conceptualization of SSS may also meaningfully differ by sex and race, and in turn, have different implications for health, even if we did not observe that here. For example, research has found that SSS is not as consistently correlated with education and income among Black Americans relative to their White peers [4, 31, 34, 55] and that associations between SSS and objective SES are modest among Asian Americans [39]. Unfortunately, we cannot adequately address this latter question in our cohort as we only had 15 participants who identified as Asian American. Nonetheless, within our sample, correlations between both SSS ladders and between SSS_C and objective SES were relatively weaker among participants of color compared with their White peers. In addition, the racial difference in mean SSS_{US} was greater than that of SSS_C, perhaps suggesting that comparing oneself to others in the broader USA brings to light more racial inequalities. Alternatively, it may be that when thinking of community, people are comparing themselves to others who share similar identities (e.g., race, sex) whereas they are comparing themselves to people more generally when thinking about the broader USA. There is also some evidence that SSS may capture different aspects of social status for women compared with men (e.g., [55]). Among our participants, SSS_{US} and family-adjusted income were more strongly correlated among men compared with women while correlations between SSS_C and family-adjusted income were relatively similar. Moreover, there were stark differences in objective SES in our sample, with people from racial minorities reporting family-adjusted incomes an average of $15,000 less than their White peers

and women reporting family-adjusted incomes an average of $7,000 less than men at baseline. Because SSS ratings may reflect power, privilege, and respect based on an array of sociodemographic and historical factors that overlap with objective SES, future research should view SSS and health from an intersectional lens. Indeed, gaining a clearer sense of what SSS means to people with different lived experiences and sociodemographic identities is needed to inform our understanding of how SSS may relate to long-term health and how SSS ratings can be leveraged to address socioeconomic inequalities in health.

We also did not find support for our second set of exploratory analyses examining multiyear changes in family-adjusted income and SSS relating to concurrent changes in IL-6. This is likely due to limited variability within the sample. It is possible that SSS and family-adjusted income may become relatively stable with age, especially as adults become more established in their careers. As such, it may be more meaningful to examine changes in SSS and income in relation to health during life stages where greater fluctuations in income and SSS are expected, such as emerging and young adulthood when people are becoming financially independent from their parents. For example, Goodman et al. [56] found that decreasing SSS from adolescence to young adulthood was associated with increased risk of obesity and depressive symptoms. In addition, the time between baseline and follow-up visits may not have been sufficient to capture changes in SSS and income within our sample. Weiss and Kunzmann [57] examined changes in SSS over a decade and found that midlife adults with decreasing SSS had concurrent increases in negative affect and decreases in positive affect. Thus, it may be more valuable to consider how changes in family-adjusted income and SSS relate to prospective changes in IL-6 and CRP over decades rather than years.

Strengths and Limitations

This study has several strengths. It leveraged a prospective design to examine changes in circulating levels of IL-6 across multiple years. We also included assessments of both SSS_{US} and SSS_{C}, and examined whether associations between SSS and multiyear changes in IL-6 and CRP were independent of objective measures of SES including educational attainment and family-adjusted income. Beyond objective SES, we also considered alternative explanations that may have contributed to findings by adjusting for psychological factors and conducting sensitivity analyses excluding participants who developed chronic health conditions in between visits. We also explored potential pathways that may have partly accounted for associations between SSS and prospective changes in CRP and IL-6, which may could serve as potential areas for future investigation. Even though strict inclusion criteria may limit generalizability, we nonetheless found significant associations between SSS and multiyear increases in IL-6 and indirect effects of SSS on changes in CRP through changes in BMI within a relatively healthy adult sample.

Nonetheless, this study is not without its limitations. These include the abovementioned limitations about how race and sex were operationalized as well as the undetermined clinical significance of findings. Namely, it is not clear to what extent the multiyear increases in IL-6 observed here lead to clinically meaningful differences in the prevalence of chronic health conditions of aging. Other limitations include possible batch effects for IL-6 and CRP, and our choice of measures to capture objective SES. Although we considered family-adjusted income and educational attainment, additional measures of wealth (e.g., stocks), assets (e.g., homeownership), and types of debt (e.g., college loans, credit card debt) would strengthen future studies that aim to examine the unique effects of SSS on health that are independent of objective SES. Research is also needed to elucidate additional shared and unique pathways through which SSS may come to be associated with multiyear increases in IL-6 and CRP, respectively. Along with alterations in physiological stress reactivity, psychological factors, and adiposity, recent work has suggested that eating behaviors and physical activity may be other pathways through which SSS relates to health [58, 59]. It would also be important to consider how experiences of discrimination, financial strain, and other psychosocial factors that unfold across time may mediate the SSS to inflammation association. Finally, this was a socioeconomically advantaged sample as evidenced by over 60% of participants having a Bachelor's degree or higher. This restriction in range may help explain why some of the associations were nonsignificant. With relatively few participants of low SES, the range of values may have been restricted and downwardly biased estimates of associations between SSS and IL-6 and CRP. Therefore, replication studies using nationally representative samples with a broader range of SSS, education, and income levels are needed to substantiate findings.

Conclusion

Among a sample of midlife adults, we found that lower SSS was prospectively associated with multiyear increases in IL-6 independently of income and education. Exploratory analyses also revealed a possible indirect effect of SSS on multiyear increases in CRP through increases in adiposity as measured by BMI, although this needs to be replicated. These findings, albeit preliminary, could suggest that lower SSS may increase risk for chronic health conditions of aging (e.g., CVD, diabetes) by exacerbating age-related increases in low-grade inflammation. Once replicated in larger, more racially, ethnically, and gender diverse samples, a next step will be to understand how to intervene on SSS at the individual- and policy level to offset the link between lower SSS and increases in inflammation.

Acknowledgments

We want to thank the participants who volunteered to partake in the Pittsburgh Imaging Project as well as the research assistants and project coordinators who made this study possible. Research reported in this manuscript was supported by grants from the National Heart, Lung, and Blood Institute of the National Institutes of Health under award numbers R01-HL089850 and P01-HL040962 (PI: Peter Gianaros) and 5T32HL007560-37 (PIs: Rebecca Thurston & Peter Gianaros; postdoctoral trainee: Emily Jones). The content of the manuscript is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

Compliance with Ethical Standards

Authors' Statement of Conflict of Interest and Adherence to Ethical Standards The authors report no competing interests and the Institutional Review Board of the University of Pittsburgh approved the study.

Authors' Contributions Emily Jones (Conceptualization [equal], Formal analysis [lead], Writing – original draft [lead]), Anna L. Marsland (Conceptualization [equal], Supervision [equal], Writing – review & editing [equal]), Thomas E. Kraynak (Conceptualization [equal], Writing – review & editing [equal]), Elizabeth Votruba-Drzal (Conceptualization [equal], Writing – review & editing [equal]), and Peter J. Gianaros (Conceptualization [equal], Funding acquisition [lead], Supervision [equal], Writing – review & editing [equal])

Transparency Statement The study and analytic plan were preregistered prior to beginning data analysis at OSF. Deidentified data and the analytic code used to conduct the analyses presented in this study are available at OSF (https://osf.io/5gj4n). Materials used to conduct the study are not publicly available.

Supplementary Material

Supplementary material is available at *Annals of Behavioral Medicine* online.

References

1. Adler NE, Boyce T, Chesney MA, et al. Socioeconomic status and health: the challenge of the gradient. *Am Psychol*. 1994;49(1):15–24.
2. Benjamin EJ, Muntner P, Alonso A, et al.; American Heart Association Council on Epidemiology and Prevention Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics—2019 update: a report from the American Heart Association. *Circulation*. 2019;139:e56–e528.
3. Hill-Briggs F, Adler NE, Berkowitz SA, et al. Social determinants of health and diabetes: a scientific review. *Diabetes Care*. 2020;44:258–279.
4. Cundiff JM, Matthews KA, Matthews KA. Is subjective social status a unique correlate of physical health? A meta-analysis. *Health Psychol*. 2017;36:1109–1125.
5. Tang KL, Rashid R, Godley J, Ghali WA. Association between subjective social status and cardiovascular disease and cardiovascular risk factors: a systematic review and meta-analysis. *BMJ Open*. 2016;6:e010137.
6. Zell E, Strickhouser JE, Krizan Z. Subjective social status and health: a meta-analysis of community and society ladders. *Health Psychol*. 2018;37:979–987.
7. Adler N, Stewart J. *The MacArthur Scale of Subjective Social Status*. San Francisco: MacArthur Research Network on SES & Health; 2007.
8. Kraus MW, Tan JJX, Tannenbaum MB. The social ladder: a rank-based perspective on social class. *Psychol Inquiry*. 2013;24:81–96.
9. Franceschi C, Campisi J. Chronic inflammation (inflammaging) and its potential contribution to age-associated diseases. *J Gerontol: Series A*. 2014;69:S4–S9.
10. Danesh J, Kaptoge S, Mann AG, et al. Long-term interleukin-6 levels and subsequent risk of coronary heart disease: two new prospective studies and a systematic review. *PLoS Med*. 2008;5:e78.
11. Luc G, Bard J-M, Juhan-Vague I, et al.; PRIME Study Group. C-reactive protein, interleukin-6, and fibrinogen as predictors of coronary heart disease: the PRIME Study. *Arterioscler Thromb Vasc Biol*. 2003;23:1255–1261.
12. Freeman DJ, Norrie J, Caslake MJ, et al.; West of Scotland Coronary Prevention Study. C-reactive protein is an independent predictor of risk for the development of diabetes in the West of Scotland Coronary Prevention Study. *Diabetes*. 2002;51:1596–1600.
13. Pradhan AD, Manson JE, Rifai N, Buring JE, Ridker PM. C-reactive protein, interleukin 6, and risk of developing type 2 diabetes mellitus. *JAMA*. 2001;286:327–334.
14. Demakakos P, Nazroo J, Breeze E, Marmot M. Socioeconomic status and health: the role of subjective social status. *Soc Sci Med*. 2008;67:330–340.
15. Yong JC, Hartanto A, Tan JJX. Subjective social status and inflammation: the role of culture and anger control. *Health Psychol*. 2020;40:62–70.
16. Manuck SB, Phillips J, Gianaros PJ, Flory JD, Muldoon MF. Subjective socioeconomic status and presence of the metabolic syndrome in midlife community volunteers. *Psychosom Med*. 2010;72:35–45.
17. Marsland AL, McCaffery JM, Muldoon MF, Manuck SB. Systemic inflammation and the metabolic syndrome among middle-aged community volunteers. *Metabolism*. 2010;59:1801–1808.
18. Brydon L, Edwards S, Mohamed-Ali V, Steptoe A. Socioeconomic status and stress-induced increases in interleukin-6. *Brain Behav Immun*. 2004;18:281–290.
19. Derry HM, Fagundes CP, Andridge R, Glaser R, Malarkey WB, Kiecolt-Glaser JK. Lower subjective social status exaggerates interleukin-6 responses to a laboratory stressor. *Psychoneuroendocrinology*. 2013;38:2676–2685.
20. Muscatell KA, Dedovic K, Slavich GM, et al. Neural mechanisms linking social status and inflammatory responses to social stress. *Soc Cogn Affect Neurosci*. 2016;11:915–922.
21. McEwen BS, Gianaros PJ, Gianaros PJ. Central role of the brain in stress and adaptation: links to socioeconomic status, health, and disease. *Ann N Y Acad Sci*. 2010;1186:190–222.
22. Wirtz PH, von Känel R. Psychological stress, inflammation, and coronary heart disease. *Curr Cardiol Rep*. 2017;19:111.
23. Goodman E, Adler NE, Daniels SR, Morrison JA, Slap GB, Dolan LM. Impact of objective and subjective social status on obesity in a biracial cohort of adolescents. *Obes Res*. 2003;11:1018–1026.
24. Adler NE, Epel ES, Castellazzo G, Ickovics JR. Relationship of subjective and objective social status with psychological and physiological functioning: preliminary data in healthy, White women. *Health Psychol*. 2000;19:586–592.
25. Stewart JC, Rand KL, Muldoon MF, Kamarck TW. A prospective evaluation of the directionality of the depression–inflammation relationship. *Brain Behav Immun*. 2009;23:936–944.
26. Bhutta N, Chang AC, Dettling LJ, Hsu JW. *Disparities in Wealth by Race and Ethnicity in the 2019 Survey of Consumer Finances*. FEDS Notes. Washington: Board of Governors of the Federal Reserve System. 2020. doi:10.17016/2380-7172.2797.
27. Chetty R, Hendren N, Jones MR, Porter SR. Race and economic opportunity in the United States: an intergenerational perspective. *Q J Econ*. 2020;135:711–783.
28. Derenoncourt E, Kim CH, Kuhn M, Schularick M. *Wealth of Two Nations: The US Racial Wealth Gap, 1860–2020*. Cambridge, MA: Nat Bur Econ Res, 2022.
29. McDaniel A, DiPrete TA, Buchmann C, Shwed U. The black gender gap in educational attainment: historical trends and racial comparisons. *Demography*. 2011;48:889–914.
30. Bailey ZD, Krieger N, Agénor M, Graves J, Linos N, Bassett MT. Structural racism and health inequities in the USA: evidence and interventions. *Lancet*. 2017;389:1453–1463.
31. Wolff LS, Acevedo-Garcia D, Subramanian SV, Weber D, Kawachi I. Subjective social status, a new measure in health disparities research: do race/ethnicity and choice of referent group matter? *J Health Psychol*. 2010;15:560–574.
32. Chen E, Brody GH, Miller GE. What are the health consequences of upward mobility? *Ann Rev Psychol*. 2022;73:599–628.
33. Henry DA, Votruba-Drzal E, Miller P. Chapter One—Child development at the intersection of race and SES: an overview. *Advances in Child Development and Behavior*. 2019;57:1–25.
34. Adler N, Singh-Manoux A, Schwartz J, Stewart J, Matthews K, Marmot MG. Social status and health: a comparison of British civil servants in Whitehall-II with European- and African-Americans in CARDIA. *Soc Sci Med*. 2008;66:1034–1045.
35. Allen AJ, McNeely JM, Waldstein SR, Evans MK, Zonderman AB. Subjective socioeconomic status predicts Framingham cardiovascular disease risk for whites, not blacks. *Ethn Dis*. 2014;24:150–154.
36. Michelson N, Riis JL, Johnson SB. Subjective social status and psychological distress in mothers of young children. *Mater Child Health J*. 2016;20:2019–2029.

37. Rohleder N. Stimulation of systemic low-grade inflammation by psychosocial stress. *Psychosom Med*. 2014;76:181–189.
38. Geiger AM, Kirschbaum C, Wolf JM. Comparison group matters for chronic stress effects of subjective social status. *J Health Psychol*. 2019;24:1923–1928.
39. de Castro AB, Gee GC, Takeuchi DT. Examining alternative measures of social disadvantage among Asian Americans: the relevance of economic opportunity, subjective social status, and financial strain for health. *J Immigr Minor Health*. 2010;12:659–671.
40. Helgeson V. Gender, stress, and coping. In: Folkman S, ed. *The Oxford Handbook of Stress, Health, and Coping*. New York, NY: Oxford University Press; 2011.
41. Freeman JA, Bauldry S, Volpe VV, Shanahan MJ, Shanahan L. Sex differences in associations between subjective social status and C-reactive protein in young adults. *Psychosom Med*. 2016;78:542–551.
42. Subramanyam MA, Diez-Roux AV, Hickson DA, et al. Subjective social status and psychosocial and metabolic risk factors for cardiovascular disease among African Americans in the Jackson Heart Study. *Soc Sci Med*. 2012;74:1146–1154.
43. Thompson M, Naleway A, Ball S, et al. Subjective social status predicts wintertime febrile acute respiratory illness among women healthcare personnel. *Health Psychol*. 2013;33.
44. Dandona P, Aljada A, Bandyopadhyay A. Inflammation: the link between insulin resistance, obesity and diabetes. *Trends Immun*. 2004;25:4–7.
45. Wiseman DJ, Thwaites RS, Drysdale SB, et al.; RESCEU Investigators. Immunological and inflammatory biomarkers of susceptibility and severity in adult respiratory syncytial virus infections. *J Infect Dis*. 2020;222:S584–S591.
46. Gianaros PJ, Rasero J, DuPont CM, et al. Multivariate brain activity while viewing and reappraising affective scenes does not predict the multiyear progression of preclinical atherosclerosis in otherwise healthy midlife adults. *Affect Sci*. 2022;3:406–424.
47. Gianaros PJ, Sheu LK, Uyar F, et al. A brain phenotype for stressor-evoked blood pressure reactivity. *J Am Heart Assoc*. 2017;6:e006053.
48. Operario D, Adler NE, Williams DR. Subjective social status: reliability and predictive utility for global health. *Psychol Health*. 2004;19:237–246.
49. Krieger N. Discrimination and health inequities. *Intern J Health Serv*. 2014;44:643–710.
50. Volpe VV, Dawson DN, Rahal D, Wiley KC, Vesslee S. Bringing psychological science to bear on racial health disparities: the promise of centering Black health through a critical race framework. *Trans Issues Psychol Sci*. 2019;5:302–314.
51. Beck A, Steer R, Brown G. *Beck Depression Inventory—Second Edition*. Manual San Antonio, TX: The Psychological Corporation; 1996.
52. Everson SA, Goldberg DE, Kaplan GA, et al. Hopelessness and risk of mortality and incidence of myocardial infarction and cancer. *Psychosom Med*. 1996;58:113–121.
53. Piedra LM, Andrade FCD, Hernandez R, et al. Association of subjective social status with life's simple 7s cardiovascular health index among Hispanic/Latino people: results from the HCHS/SOL. *J Am Heart Assoc*. 2021;10:e012704.
54. Garza JR, Glenn BA, Mistry RS, Ponce NA, Zimmerman FJ. Subjective social status and self-reported health among US-born and immigrant Latinos. *J Immigr Minor Health*. 2017;19:108–119.
55. Shaked D, Williams M, Evans MK, Zonderman AB. Indicators of subjective social status: differential associations across race and sex. *SSM Pop Health*. 2016;2:700–707.
56. Goodman E, Maxwell S, Malspeis S, Adler N. Developmental trajectories of subjective social status. *Pediatrics*. 2015;136:e633–e640.
57. Weiss D, Kunzmann U. Longitudinal changes in subjective social status are linked to changes in positive and negative affect in midlife, but not in later adulthood. *Psychol Aging*. 2020;35:937–947.
58. Rahal D, Chiang JJ, Fales M, et al. Early life stress, subjective social status, and health during late adolescence. *Psychol Health*. 2020;35:1531–1549.
59. Reitzel LR, Nguyen N, Strong LL, Wetter DW, McNeill LH. Subjective social status and health behaviors among African Americans. *Am J Health Behav*. 2013;37:104–111.