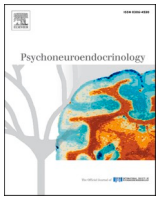
Psychoneuroendocrinology 141 (2022) 105748

Contents lists available at ScienceDirect

Psychoneuroendocrinology

journal homepage: www.elsevier.com/locate/psyneuen



Association of subjective social status with epigenetic aging among Black and White women

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ARTICLE INFO

Keywords:
Socioeconomic status
Subjective social status
Epigenetic clock
Epigenetic age acceleration
Race

ABSTRACT

Objective: Subjective social status (SSS), an individual's assessment of their own social status in relation to others, is associated with health and mortality independently of objective SES; however, no studies have tested whether SSS influences epigenetic aging. The current study examines if SSS is associated with epigenetic age acceleration in both Black and White women, independently of objective SES measured during both childhood and adulthood.
Method: For 9- and 10-year-old Black and White girls, parental education and annual household income was obtained. At ages 39–42, 361 participants (175 Black, 186 White) reported their current education, household income, and SSS, and provided saliva to assess age acceleration using the GrimAge epigenetic clock. Linear regression estimated the association of SSS with epigenetic age acceleration, controlling for objective SES (current education, current income, parents' education, income during childhood), smoking, and counts of cell types.
Results: When all objective SES variables were included in the model, SSS remained significantly associated with epigenetic age acceleration, $b = -0.31$, $p = .003$, $ß = -0.15$. Black women had significantly greater age acceleration than White women, ($t(359) = 5.20$, $p > .001$, $d = 0.55$) but race did not moderate the association between SSS and epigenetic age acceleration.
Conclusions: Women who rated themselves lower in SSS had greater epigenetic age acceleration, regardless of income and education. There was no difference by race for this association.

1. Introduction

Health disparities associated with socioeconomic status (SES) are well established. Household income and parental educational background are not only predictive of childhood health status but continue to play a significant role in morbidity and mortality risks throughout the lifespan (Adler and Rehkopf, 2008). SES is an "overarching determinant of health" that influences a range of health outcomes from cardiovascular disease (CVD), cancer, and diabetes to depression and disability (e.g., Adler and Rehkopf, 2008; Chen et al., 2002, Goodman et al., 2005). The effect size of SES on early mortality is comparable to the effect sizes for smoking, obesity, and overconsumption of alcohol (Fiorito et al., 2017). There is a clear need to reduce health disparities based on SES and understanding the pathways by which SES affects health informs the development of effective interventions to ameliorate and diminish health disparities.

Most research linking SES to health has focused on objective SES indicators (e.g., income, education); however, SES by nature involves judgements of hierarchical comparisons of status to others and cannot be considered a purely objective construct. Subjective social status (SSS), one's assessment of their own social status in relation to others (Diemer et al., 2013), is a subjective measurement of SES that can be obtained by asking individuals to rank themselves relative to others within a reference group (e.g., their country). The most common assessment of SSS asks the individual to place themselves on a ten-rung ladder, where the top rung represents the highest socioeconomic status (e.g., the most

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https://doi.org/10.1016/j.psyneuen.2022.105748
Received 20 November 2021; Received in revised form 21 March 2022; Accepted 22 March 2022
Available online 25 March 2022

income, education, and prestigious occupations) and the bottom rung the lowest status (e.g., the least income, education and least desirable occupations) (Adler et al., 2000). Individuals make this determination based on the factors they deem most important for status, which allows for a more nuanced determination of social status in that respondents may factor in debt or wealth as well as income, or quality of their education as well as years of schooling (Schrock et al., 2018). Meta-analyses support that SSS is associated with physical health, even after accounting for objective SES (Cundiff and Matthews, 2017; Zell et al., 2018). As with objective SES, SSS is a robust predictor of mortality (Demakakos et al., 2018). SSS may be an important aspect of socioeconomic disparities in health; however, the pathways between SSS and health are inadequately understood.

1.1. Social status and cellular aging

In line with the biological embedding model, which suggests that adversity becomes embedded in immune cells (Ehrlich et al., 2016; Miller et al., 2011), SES may recalibrate the immune system, by way of epigenetic changes and other biological processes, to increase vulnerability to later disease and premature mortality. Epigenetic clocks estimate cellular aging using the methylation patterns of specific sites in the genome, CpGs (5′—C—phosphate—G—3′) to robustly predict disease and aging outcomes (Horvath and Raj, 2018). Children whose parents had professional or managerial employment were epigenetically younger as adults than those whose parents had semi-skilled/unskilled employment or were not working (Hughes et al., 2018). Postmenopausal women with less education were found to be older epigenetically than peers of the same age who were more educated; epigenetic age partially mediated the relationship between education and mortality (Liu et al., 2019).

Limited work has linked SSS to aging-related biomarkers. SSS has been associated with inflammation in young adults (Freeman et al., 2016) and the subjective assessment of finances was associated with leukocyte telomere length (Schrock et al., 2018). To our knowledge, the current study is the first to examine whether SSS is associated with epigenetic aging, in women in early middle age. Examining epigenetic aging in midlife allows us to evaluate the influence of social status on health decades before the onset of major disease or disability. Further, few studies have examined both SSS and objective SES in relation to biomarkers, and it is not known if associations with cellular aging will remain significant when both SSS and SES are considered. When both objective SES and SSS were included, SSS (and not SES) remained associated with self-rated health (Singh-Manoux et al., 2005) and this pattern of associations may replicate with epigenetic aging. In the current study, we examined the associations between SSS and epigenetic age acceleration (i.e., epigenetic age relative to chronological age), and included SES measured during both childhood and adulthood.

1.2. Joint consideration of SES and race on cellular aging

Black Americans have worse health compared to White Americans, including higher rates of the leading causes of death in the US, as well as the earlier onset of disease and disability (Adler and Rehkopf, 2008; Jolly et al., 2010; Williams, 2012). Due to a long history of discrimination and racism, on average, Blacks receive lower income, less education, and higher unemployment than Whites. Black Americans show greater epigenetic aging than White Americans and this helps to account for a shorter life expectancy (Liu et al., 2019). In the United States, race and SES are confounded and their unique influences on health can be difficult to disentangle.

SSS is less reliably associated with health for Blacks compared to Whites (Adler et al., 2008; Cundiff and Matthews, 2017). After accounting for objective SES, SSS was associated with CVD risk in middle-aged Whites, but not Blacks (Allen et al., 2014). Similarly, in a sample of older adults with hypertension, the associations between SSS and health were stronger for Whites than for Blacks (Cené et al., 2016). No differences by race have been found in the association of SSS and elevated inflammation; however, this was a sample of young adults and race differences may emerge later in life (Freeman et al., 2016). The current study was the first to examine the relations of both SSS and objective SES with epigenetic age acceleration and is powered to examine race differences.

1.3. The current study

To examine associations between SSS, SES, and epigenetic aging, we used data from a well-characterized longitudinal cohort study, the National Heart, Lung, and Blood Institute Growth and Health Study (The National Heart Lung and Blood Institute Growth and Health Study Research Group, 1992), which assessed Black and White girls annually for ten years and re-recruited them as adults in early middle age. Girls were initially recruited at age 9 or 10 at the first assessment (1987–1988) and age 39–42 at follow-up. The aims of the current study were to establish 1) if SSS influences epigenetic age acceleration independently of childhood and adulthood objective SES, and 2) if there are differences in the association between SSS and epigenetic age acceleration for Black and White women. Based on the limited amount of research looking at SSS and cellular aging, we had no specific a priori hypotheses about the association of SSS with epigenetic age acceleration or the existence of race differences.

2. Method

2.1. Participants and procedure

To examine associations between SSS, SES, and epigenetic aging, we used data from a well-characterized longitudinal cohort study, the National Heart, Lung, and Blood Institute Growth and Health Study (The National Heart Lung and Blood Institute Growth and Health Study Research Group, 1992), which assessed Black and White girls annually for ten years and re-recruited them as adults in early middle age. Girls were initially recruited at age 9 or 10 at the first assessment (1987–1988) and age 39–42 at follow-up.

The initial aims of NGHS were to track cardiovascular risk factors and other health-related variables annually from childhood through young adulthood in 1209 Black and 1166 White girls from Richmond (CA, USA), Cincinnati (OH, USA), and Washington (D.C, USA). In 1987–1988, the NGHS Contra Costa County cohort (887 girls) was recruited at ages 9 and 10 from public and parochial schools in the Richmond Unified School District area. The original investigators chose Richmond, CA based on census data that showed approximately equal percentages of Black and White children with the smallest income and occupational disparity between races. More details about the initial study sample recruitment are available (The National Heart Lung and Blood Institute Growth and Health Study Research Group, 1992). Retention across the 10-year study period was 89% (The National Heart Lung and Blood Institute Growth and Health Study Research Group, 1992).

In 2016, we began a follow-up of the NGHS Contra Costa County cohort to assess health and well-being in midlife (ages 39–42). Eligibility criteria for the follow-up study included: (1) being an original NGHS participant; (2) not pregnant at the time of recruitment, and had not experienced a pregnancy, miscarriage, or abortion within the last three months; and (3) not living abroad, nor incarcerated or otherwise institutionalized. Multiple recruitment strategies were used to re-recruit original NGHS participants from the Richmond site, including mailing and telephone follow-up, social media and electronic outreach, and door-to-door outreach. Eligible participants provided written informed consent and participated in a three-part protocol: (1) baseline survey; (2) home/clinic visit, including saliva collection for TL assessment; and (3) other biospecimen collection (not relevant for the present study).

Table 1
Descriptives of study variables.

		Total	Black	White	
Current education % (n)	High school degree or less[a]	19.7 (71)	24.6 (43)	15.1 (28)	χ^2 = 33.95 ***
	Some college[a]	39.1 (141)	49.7 (87)	29.0 (54)	
	College degree or more[a]	41.3 (149)	25.7 (45)	55.9 (104)	
Current annual income %(n)	< $40,000[a]	27.1 (98)	37.1 (65)	17.7 (33)	χ^2 = 50.09 ***
	$40,000–69,999[a]	28.0 (101)	36.6 (64)	19.9 (37)	
	$70,000–129,999[a]	22.4 (81)	16.0 (28)	28.5 (53)	
	$130,000 +[a]	22.4 (81)	10.3 (18)	33.9 (63)	
Parents' maximum education %	High school degree or less	21.1 (76)	22.3 (39)	19.9 (37)	χ^2 = 22.83 ***
	Some college[a]	48.5 (175)	58.9 (103)	38.7 (72)	
	College degree or more[a]	30.5 (110)	18.9 (33)	41.4 (77)	
Household income in childhood%	< $10,000[a]	19.1 (69)	32.6 (57)	6.5 (12)	χ^2 = 63.66 ***
	$10,000–19,999[a]	18.3 (66)	24.0 (42)	12.9 (24)	
	$2000–39,999	27.4 (99)	24.0 (42)	30.6 (57)	
	$40,000 +[a]	35.2 (127)	19.4 (34)	50.0 (93)	
US SSS*M*(*SD*)	range: 1–10	6.04 (1.98)	6.09 (2.07)	6.01 (1.90)	t = 0.39 d = 0.04
GrimAge acceleration*M* (*SD*)	min = − 11.22	-0.11 (4.25)	0.99 (4.13)	-1.26 (4.07)	t = 5.20 ***
	max = 13.12				d = 0.55
Current/past smoker(% yes)		42.1	37.7	46.2	χ^2 = 2.69

Note. US SSS = subjective social status at the national level. * $p < .05$; ** $p < .01$. *** $p < .001$.
[a] Denotes column proportions differ significantly by race.

This study was approved by the local Institutional Review Board. As a result of extensive recruitment efforts, over 73% of eligible women (307 Black and 317 White) were enrolled in the follow-up study.

The sample for the current study consisted of 361 women (175 Black and 186 White, $M_{age} = 39.3$, $SD_{age} = 1.2$). Overall, the sample was free of major medical conditions and disability. Twenty-two women (6.1%) endorsed having diabetes, 23 (6.4%) endorsed previously having gestational diabetes, and 26 (7.3%) endorsed being prediabetic. Twenty women (6.3%) reported that they took medication for diabetes. Sixty-five women (18.3%) endorsed hypertension, 26 (7.3%) endorsed hypertension only while pregnant, 14 (3.9%) endorsed being pre-hypertensive, and 34 (10.6%) reported taking medication for high blood pressure. Forty-eight women (13.4%) reported that they had high cholesterol and three women (0.3%) reported having heart disease.

There was no significant difference between the baseline sample and the final sample in parents' education, $\chi2(2) = 1.90$, p = .39, childhood income, $\chi2(3) = 6.68$, p = .08, or race, $\chi2(1) = 1.85$, p = .17.

2.2. Measures

2.2.1. Objective socioeconomic status

2.2.1.1. Childhood. In 1987–1988, the highest level of education for each parent (both mother and father, if known) and annual household income were reported by participants' mothers at baseline, when participant was aged 9–10. Parental maximum education level was categorized as a high school degree or less, some college, or at least a college degree. Annual household income was categorized as less than $10,000, between $10,000 and $19,999, between $20,000 and $39,999, and $40,000 or more.

2.2.1.2. Adulthood. Current level of education and annual household income were self-reported by participants at follow-up. Education level was categorized as a high school degree or less, some college, or at least a college degree. Annual household income was categorized as less than $40,000, between $40,000 and $69,999, between $70,000 and $129,999, and $130,000 or more.

2.2.2. Subjective Social Status

The most common assessment of SSS is the MacArthur Scale of Subjective Social Status (Adler et al., 2000). In pictorial format, participants were asked to place an X to demonstrate where they believe they stand on a ten-rung "social" ladder compared to others in the US. On this ladder, the top rung represents the highest status (e.g., the most income, education, and prestigious occupations) and the bottom rung represents the lowest status (e.g., the least income, education and least desirable occupations). The MacArthur Scale of Subjective Social Status has demonstrated good validity and adequate test-retest reliability (Giatti et al., 2012; Operario et al., 2004; Singh-Manoux et al., 2003).

2.2.3. Epigenetic age acceleration

DNA methylation analyses with whole blood samples were performed at the Semel Institute UCLA Neurosciences Genomics Core (UNGC) using the Illumina Infinium HumanMethylation450 BeadChip (Illumina, Inc.). Genomic DNA was isolated using temperature denaturation and subjected to bisulfite conversion, PCR amplification, and DNA sequencing (EZ DNA Methylation-Gold Kit, Zymo Research). To obtain outcome measures, methylation profiles were input to Horvath's online calculator https://dnamage.genetics.ucla.edu/, which automatically imputes any missing CpGs. After selection of the advanced analysis option and normalization based on the BMIQ method (Teschendorff et al., 2013), the output files contain the estimated epigenetic (DNAm) age of each participant and measures of predictive accuracy and data quality (e.g., for identifying array outliers, "corSampleVSgold-standard"). Before data analysis began, participants (n = 26) were excluded due to quality control issues.

DNAm GrimAge is based on 1030 unique CpGs that predict time- to-death (Lu et al., 2019), which are DNAm surrogates of seven plasma proteins and smoking pack years (i.e., the number of packs of cigarettes smoked per day multiplied by the number of years an individual smoked). "AgeAccelerationResidual", the residual resulting from a linear model where DNAm age is regressed on chronological age, was the outcome variable. One participant was excluded as an outlier (AgeAccelerationResidual > 3 SDs above mean). Positive residual values reflect an individual being older biologically than chronological age and negative residual values reflect the reverse. To account for confounding due to blood cell composition, we included estimated cell counts of naïve CD8+ T, exhausted cytotoxic CD8+ T (CD8+ CD28– CD45–), granulocytes, natural killer (NK), and CD4+T as covariates in all analyzes (Horvath and Levine, 2015).

The current utilizes DNAm GrimAge as a longitudinal study of aging outcomes comparing different clocks found that GrimAge evinced the strongest associations with outcomes (Li et al., 2020). The authors concluded that this might be because GrimAge uses the largest number of CpG sites and allows a single CpG site to contribute to estimation via multiple intermediate biomarkers. Another study of comparing clocks also found DNAm GrimAge "stood apart" in having the strongest associations with age-related performance (Maddock et al., 2020). A third study concluded: "Among all of the clocks we examined, GrimAge stands out as being influenced by all lifestyle risk factors in the expected

direction. The above evidence, along with the fact that GrimAge performs better at predicting age-related disease and mortality, suggests that it may be a promising DNAm clock that best mirrors the relationship between environmental risk factors and healthy aging" (Zhao et al., 2019, p. 13–14)".

2.2.4. Data analysis

To determine if there was a significant association between SSS and epigenetic age acceleration, we conducted linear regressions. Smoking status (past/current smoker or non-smoker), cell type counts, race (Black = 1, White = 0), objective SES indicators, and SSS were simultaneously entered into the model. Objective SES in adulthood (current education and income) and objective SES in childhood (parents' education and childhood income) were entered separately in preliminary models. All four objective SES variables were entered simultaneously in the final model. To examine moderation by race, an interaction term representing SSS moderated by race was entered into the model in the next block, after the entry of all other variables.

Meta-analysis has determined that the overall effect of SSS on health is small (Zell et al., 2018). With the model and variables as described, to detect a small effect size of .15 with .80 power and an alpha level of .05, power analysis (G*POWER 3.1; Faul et al., 2009) suggests a minimum sample size of 43.

3. Results

Descriptive statistics are reported in Table 1. As expected, Black women had significantly higher epigenetic age acceleration (epigenetic age relative to chronological age) than White women, $t(359) = 5.20$, $p > .001$, $d = 0.55$. Black women were approximately one year (0.99) older than their chronological age and White women were approximately one year and a quarter (− 1.26) younger than their chronological age.

SSS was normally distributed with a mean of 6.04 and standard deviation of 1.98 (range 1–10). There was no significant difference between Black and White women in SSS at the national level (6.09 vs. 6.01, out of 10).

There were significant differences in current education and household income between Black and White women. White women were more likely than Black women to have a college degree (55.9% vs. 25.7%). Black women were more likely than White women to have some college education (49.7% vs. 29.0%) or a high school degree or less (24.6% vs. 15.1%). White women were more likely than Black women to have annual incomes between $70,000 and $129,999 (28.5% vs. 16.0%) or $130,000 or higher (33.9% vs. 10.3%). Black women were more likely than White women to have annual incomes between $40,000 and $7000 (36.6% vs. 19.9%) or under $40,000 (37.1% vs. 17.7%).

Compared to Black women, White women were significantly more likely to have a parent with a college degree (41.4% vs. 18.9%). Black women were more likely have parents with some college education (58.9% vs. 38.7%). There was no significant difference in the proportions of Black and White women who had parents with a high school degree or less (22.3% vs. 19.9%). There were significant differences in household income in childhood (1988–89) between Black and White women. Compared to Black women, White women were significantly more likely to have had an annual income in childhood of at least $40,000 (50.0% vs. 19.4%). Black women were more likely to have childhood incomes between $10,000 and $20,000 (24.0% vs. 12.9%) or under $10,000 (32.6% vs. 6.5%). There was no significant difference in the proportions of the Black and White women with childhood incomes between $20,000 and $40,000 (24.0% vs. 30.6%).

3.1. Effects of subjective social status on epigenetic age acceleration

When accounting for current income and education (Table 2), SSS at the national level was associated with epigenetic age acceleration, $b = -0.31$, $p = .003$, $\beta = -0.15$, in that higher SSS was associated with lower age acceleration. In addition, having a college degree was significantly associated with lower age acceleration, $b = -1.96$, $p < .001$, $\beta = -0.23$, compared to those with a high school degree or less. There was no relationship between current income and epigenetic age acceleration.

Similarly, the relationship between SSS at the national level and epigenetic age acceleration was significant, $b = -0.39$, $p < .001$, $\beta = -0.18$, when accounting for parents' education and household income in childhood (Table 2). Having parents with some college, $b = -1.24$, $p = .01$, ß = − 0.15, or a college degree, $b = -1.52$, $p = .01$, ß = − 0.17, was associated with lower epigenetic age acceleration compared to having parents with a high school degree or less. Again, there was no relationship between childhood income and epigenetic age acceleration.

Table 2
Subjective social status and objective SES on epigenetic age acceleration (separate models).

	b	*SE*	ß	*t*	*p*
Subjective Social Status & Objective SES (adult)					
Smoker (past/current)	**2.56**	**0.40**	**.30**	**6.40**	**< 0.001**
CD8+ T (cell counts)	-0.01	0.01	-0.09	-1.75	.08
CD8+ CD28– CD45– (cell counts)	0.12	0.10	.06	1.30	.19
NK (cell counts)	-13.51	40.85	-0.02	-0.33	.74
CD4T (cell counts)	**45.44**	**16.08**	**.67**	**2.83**	**.01**
Gran (cell counts)	12.05	8.03	.36	1.50	.13
Race (Black = 1, White = 0)	**2.45**	**0.42**	**.29**	**5.86**	**< 0.001**
US SSS	**-0.31**	**0.10**	**-0.15**	**-3.04**	**.003**
Ed: High school or less[a]					
Ed: Some college	-0.63	0.50	-0.07	-1.26	.21
Ed: College or more	**-1.96**	**0.58**	**-0.23**	**-3.37**	**< 0.001**
Income: < $40,000[a]					
Income: $40,000–69,999	-0.83	0.49	-0.09	-1.69	.09
Income: $70,000–129,999	-0.27	0.54	-0.03	-0.49	.62
Income: $130,000 +.	-0.37	0.64	-0.04	-0.58	.56
Subjective Social Status & Objective SES (child)					
Smoker (past/current)	**2.90**	**0.38**	**0.34**	**7.58**	**< 0.001**
CD8+ T (cell counts)	**-0.02**	**0.01**	**-0.12**	**-2.23**	**0.03**
CD8+ CD28– CD45– (cell counts)	0.15	0.10	0.07	1.55	0.12
NK (cell counts)	-6.00	41.17	-0.01	-0.15	0.88
CD4T (cell counts)	**49.49**	**16.40**	**0.73**	**3.02**	**0.00**
Gran (cell counts)	15.22	8.14	0.46	1.87	0.06
Race (Black = 1, White = 0)	**2.75**	**0.42**	**0.32**	**6.60**	**< 0.001**
US SSS	**-0.39**	**0.10**	**-0.18**	**-4.05**	**< 0.001**
Parent: High school or less[a]					
Parent: Some college	**-1.24**	**0.49**	**-0.15**	**-2.55**	**0.01**
Parent: College or more	**-1.52**	**0.56**	**-0.17**	**-2.70**	**0.01**
Income as child: < $10,000[a]					
Income as child: $10,000–19,999	0.28	0.60	0.03	0.47	0.64
Income as child: $2000–39,999	-0.46	0.57	-0.05	-0.80	0.42
Income as child: $40,000 +.	-0.18	0.59	-0.02	-0.30	0.77

Note: US SSS = subjective social status at the national level; Significant predictors are in bold.
[a] Reference category.

3.1.1. Final model

As seen in Table 3, when all four SES variables (current education, current income, parents' education, childhood income) were included in the model, the association between SSS at the national level and epigenetic age acceleration remained significant, $b = -0.31$, $p = .003$, ß = − 0.15. Education was significantly associated with epigenetic age acceleration; participants with at least a college degree showed less epigenetic age acceleration than those who had a high school degree or less, $b = -1.78$, $p = .004$, ß = − 0.21. In addition, participants whose parents had some college, $b = -1.29$, $p = .008$, ß = − 0.15, or at least a college degree, $b = -1.26$, $p = .03$, ß = − 0.14, showed less epigenetic age acceleration than those whose parents had a high school degree or less. The associations between household income, in either childhood or

Table 3
Subjective social status and objective SES on epigenetic age acceleration (final model).

Subjective social status & objective SES (all)	*b*	*SE*	ß	*t*	*p*
Smoker (past/ current)	2.52	0.40	.29	6.31	< 0.001
CD8+ T (cell counts)	-0.01	0.01	-0.11	-2.02	.04
CD8+ CD28– CD45– (cell counts)	0.13	0.10	.07	1.41	.16
NK (cell counts)	-23.01	41.04	-0.03	-0.56	.58
CD4T (cell counts)	51.02	16.29	.75	3.13	.002
Gran (cell counts)	14.55	8.08	.44	1.80	.07
Race (Black = 1, White = 0)	2.54	0.44	.30	5.76	< 0.001
US SSS	**-0.31**	**0.10**	**-0.15**	**-3.02**	**.003**
Ed: High school or less[a]					
Ed: Some college	-0.59	0.51	-0.07	-1.16	.25
Ed: College or more	**-1.78**	**0.61**	**-0.21**	**-2.89**	**.004**
Income: < $40,000[a]					
Income: $40,000–69,999	-0.94	0.49	-0.10	-1.95	.05
Income: $70,000–129,999	-0.31	0.54	-0.03	-0.57	.57
Income: $130,000 +.	-0.40	0.63	-0.04	-0.62	.53
Parent: High school or less[a]					
Parent: Some college	**-1.29**	**0.49**	**-0.15**	**-2.67**	**.008**
Parent: College or more	**-1.26**	**0.57**	**-0.14**	**-2.22**	**.03**
Income as child: < $10,000[a]					
Income as child: $10,000–19,999	0.50	0.60	.05	0.83	.41
Income as child: $2000–39,999	-0.11	0.58	-0.01	-0.18	.85
Income as child: $40,000 +.	0.31	0.60	.04	0.52	.61

Note: US SSS = subjective social status at the national level; Significant predictors are in bold.
[a] Reference category.

adulthood, and epigenetic age acceleration were not significant.[1]

3.1.2. Moderation by race

The interaction of race and SSS was not significantly associated with epigenetic age acceleration, $b = -0.06$, $p = .77$, ß = − 0.05.

4. Discussion

The current study was the first to find subjective social status (SSS) at the national level (i.e., comparison of self to others in the United States) to be a robust predictor of epigenetic age acceleration, independently of objective SES, for both Black and White women. These findings are in line with evidence (Schrock et al., 2018) that suggests SSS may impact cellular aging independently of SES, and that when both objective SES and SSS have been considered, SSS remains significantly associated with indices of health (Singh-Manoux et al., 2005).

Study findings also suggest that race and SSS are associated with health outcomes in an additive rather than in an overlapping manner. The current study did not find that race moderated the relation between SSS and health. Race remained robustly associated with epigenetic age acceleration when all SES and SSS indicators were included, which supports that SSS does not serve as a proxy of race but has further explanatory value. Race is a social construct associated with both SES (e.g., income disparities) between Blacks and Whites as well as other predictors of poor health (e.g., systemic racism in the criminal justice system), and racism is a fundamental cause of racial inequities in health (Phelan and Link, 2015; Williams et al., 2019). Consequently, Blacks demonstrate worse health than Whites of similar SES and often do not show improved health outcomes after increased SES mobility or higher education as do Whites ("diminishing returns", Farmer and Ferraro, 2005; Surachman et al., 2020).

While a body of evidence has found associations between SSS and health, the exact mechanisms by which SSS exerts its effects on health are not known. Study findings suggest that one route by which SSS impacts health is through epigenetic age acceleration, which leads to earlier onset of disease and dysfunction. SSS may be an important predictor of health and aging as it relates to a relative (rather an absolute) position in the social hierarchy. Social rank has direct effects on physiological processes that influence biological vulnerability to disease (Wilkinson, 1992). low social status may give rise to physiological processes thought to accelerate biological aging, such as oxidative stress (Finkel and Holbrook, 2000). Over time, chronic stress takes its toll on the body in the form of greater allostatic load and cellular aging (Evans, 2003; Seeman et al., 2001). This pathway may be similar in process to "weathering" in which being assigned subordinate societal status due to race involves daily experiences of slights, barriers, and exclusion that take a physiological toll on the body and lead to premature aging (Geronimus, 1992; Geronimus et al., 2006; Simons et al., 2021).

In the current study, objective SES indicators of education, but not those of income, were significantly associated with epigenetic age acceleration. Having completed a college degree or having a parent that had at least some college education was associated with lower epigenetic age acceleration and so appeared to have a protective effect. Previous work has found both income and education to be associated with epigenetic aging with similarly sized effect (Schmitz et al., 2021). Prior research has presented the intriguing suggestion that education is more predictive of the onset of health issues and income more predictive of the progression of health issues (Herd et al., 2007; Zimmer and House, 2003). As with Schmitz et al. (2021), the current study found both participant education level as well as the education level of their parents was associated with epigenetic aging when both were included in the same model, supporting that childhood SES does not act merely as a proxy for current SES.

4.1. Strengths and limitations

The current study has notable strengths including the sufficiently large sample of both Black and White women to examine race as a moderator of the associations between SSS and epigenetic aging. Race and social status are often confounded, and their unique influences can be difficult to separate. Secondly, we had multiple measures of objective SES including current education and income as well as parents' education and household income in childhood. Research has found that historical objective SES variables, those from childhood and young adulthood, demonstrate a stronger association with health outcomes than do current SES variables (Austin et al., 2018). Using childhood income and parents' education in the current study provides a rigorous test of whether SSS has explanatory power above and beyond SES. Finally, examining epigenetic aging as an outcome in midlife allows us to evaluate the influence of SSS on health decades before the onset of major disease or disability. To do so, the current study used GrimAge, a newer epigenetic estimator that outperforms other clocks when predicting healthspan (Ecker and Beck, 2019).

The study also has several limitations. First, the current study was able to include SES in both childhood and adulthood but did not have assessments of SSS or epigenetic age acceleration during childhood. SSS appears to be relatively stable from ages 12 to 28, but evidence suggests it may decline during and soon after high school (Goodman et al., 2015; Rahal et al., 2020). Future research should examine the influence of childhood SSS, relative to SSS in adulthood, on epigenetic aging. Secondly, the current study evaluated the associations between SES, SSS, and epigenetic aging in Black and White women and did not include men. Future studies should examine the associations between SSS and epigenetic age acceleration in men and women of other racial and ethnic backgrounds. Thirdly, it's possible that SSS is a sensitive predictor of epigenetic age acceleration in early middle age and does not predict accelerated aging in older adulthood. Studies should examine the

[1] In response to reviewer concerns, two sensitivity analyses were conducted. The first (Table S1) examined home and car ownership as predictors of epigenetic age acceleration (neither were significant predictors). The second (Table S2) covaried BMI, diabetes, and hypertension in the final model. Results did not change substantively.

associations between SES, SSS, and epigenetic aging in older adults. Lastly, the study was adequately powered to examine the interaction of race and social status on epigenetic aging; however, future research should utilize larger samples to replicate results and explore the use of additional dimensions of SES (e.g., occupation, wealth and assets).

5. Conclusion

Subjective social status is an established predictor of poor health throughout the lifespan, and effects may be partly mediated through epigenetic age acceleration, which predicts earlier onset of disease. When both objective SES and SSS at the national level were considered, SSS remained significantly associated with epigenetic age acceleration, and there was no moderation by race. There was a main effect of race on epigenetic aging, in that Black women had accelerated epigenetic age when compared to White women; however, race did not modify the association between SSS and epigenetic aging. Race and socioeconomic status may be considered "codeterminants" of health disparities (Kawachi et al., 2005) and attention to both simultaneously is necessary to achieve health equity.

Significance statement

Subjective social status is an established predictor of poor health throughout the lifespan, and effects may be partly mediated through epigenetic aging, a form of premature aging on the cellular level. Examining cellular aging in midlife allows us to evaluate the influence of social status on health decades before the onset of major disease or disability. Women who rated themselves as lower in social status had greater epigenetic age acceleration (epigenetic age compared to chronological ages), regardless of income and education. Understanding the pathways by which social status affects health may lead to strategies to reduce health disparities.

Funding

This research was supported by National Institute of Mental Health Grant 5T32MH019391, National Institute on Aging Grant 1R01AG059677 to E.S.E, and National Institute of Child Health and Human Development Grant 1R01HD073568 to B.L.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.psyneuen.2022.105748.

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