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Race/ethnicity, neighborhood socioeconomic status and cardio-metabolic risk

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ABSTRACT

Objective: To determine the association between neighborhood socioeconomic status (NSES) and cardio-metabolic risk and whether this relationship differs by race/ethnicity.
Methods: Participants in the Multi-Ethnic Study of Atherosclerosis (n = 5750), ages 45–84 years, from 6 US counties, including 5 examinations from 2000 to 2012. We calculated a modified allostatic load (AL) index, indicating cardio-metabolic risk. NSES score included census-derived measures at census tract of residence. Mixed effects growth curve models were used to assess linear and non-linear associations between NSES and AL at baseline and over time.
Results: Higher NSES was associated with lower AL across race/ethnic groups; considering NSES quintiles, significant associations were found only for the highest NSES quintiles (difference of -0.86 and -1.15 for white and Hispanic participants) vs. the lowest. We found no significant association between NSES and change in AL over time.
Discussion: Our findings suggest that the relationship between NSES and AL reflects the health benefits of living in the most advantaged neighborhoods.
Public health implications: Understanding the impact of higher NSES on health effects may help identify interventions to effectively target high risk neighborhoods.

A large and growing body of evidence documents the negative effects of lower neighborhood socioeconomic status (NSES) on major cardiovascular risk factors such as diabetes, smoking, high BMI, high blood pressure (Cohen et al., 2011; Cubbin et al., 2001; Diez Roux et al., 2002, 2003), CVD outcomes (Davey-Smith et al., 1997; Kaplan & Keil, 1993; Lynch et al., 1996) and mortality (Gaskin et al., 2019). Multiple pathways have been suggested for neighborhood influences on negative health outcomes. These include fewer physical resources, such as recreational facilities (Gordon-Larsen et al., 2006; Powell et al., 2006) and limited access to healthy and affordable food (Horowitz et al., 2004; Moore & Diez Roux, 2006; Powell et al., 2007). In addition, individuals living in low SES areas are less likely to obtain adequate and preventive health care (Pappas et al., 1997). Moreover, exposure to violence and stressful life events are greater in more disadvantaged neighborhoods (Attar et al., 1994). These factors, individually and synergistically, operate through multiple biological pathways to negatively influence health in disadvantaged neighborhoods.

To assess the full scope of neighborhood influences on health risks, it is thus likely important to move beyond examination of neighborhood associations with individual biomarkers, as health risks accrue from changes in multiple biological systems (e.g., cardiovascular risk accrues from changes in blood pressure, blood glucose, lipids, body fat, and chronic inflammation (McEwen & Stellar, 1993)). A cumulative, multi-system index, known as allostatic load (AL), reflecting multiple biological factors that contribute to health risks, may better capture the global biological impact of living in disadvantaged neighborhoods. Indeed, it has been shown that AL has a stronger association with SES than each of its individual components (Seeman et al., 2004).

One of the first studies to examine the association between living in disadvantaged neighborhoods and increased AL found that while the 3

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major race/ethnic groups examined (white, African American and Mexican American) showed signs of higher AL with lower NSES, the strongest and most significant associations were found among the African American population (Merkin et al., 2009). That study also confirmed the highly confounded relationship between neighborhoods and racial/ethnic groups in the United States, with minority populations generally living in more severely disadvantaged neighborhoods (Merkin et al., 2009). Aside from the differences in levels of socioeconomic disadvantage, factors related to minority race/ethnic status may exacerbate the negative effects of living in disadvantaged neighborhoods. Researchers have described the triple threat of being African American, experiencing structural racism, segregation and living in an area of concentrated poverty as a state of "triple jeopardy." (Do et al., 2019) All these factors may lead to differences in NSES-health effects by race/-ethnicity and emphasize the importance of considering possible race/-ethnic variation in neighborhood effects (LaVeist, 2005; Merkin et al., 2009). While several other studies have examined the association between low NSES and AL, these were generally limited to cross sectional studies, and few examined this association by race/ethnicity (Carbone, 2020; Ribeiro et al., 2018, 2019).

The current study aims to address the limitations of previous analyses and to further test the fundamental hypothesis relating NSES to cumulative biological risk – i.e. that exposure to lower SES neighborhood characteristics is associated with greater/faster accumulation of risk indicators. Building on that earlier work (Merkin et al., 2009), this study examines the association between NSES and a similar index of AL based on a score of cardio-metabolic risk (henceforth described as AL), including investigation of possible associations with actual change in AL over time (the prior analyses examined only cross-sectional data). Data from the Multi-Ethnic Study of Atherosclerosis (MESA), utilized in this study, also include a diverse race/ethnic distribution, including white, African American, Chinese American (missing from most studies on NSES-AL) and Hispanic participants.

The study objectives are to examine the association between living in a socioeconomically disadvantaged neighborhood and a measure of cardio-metabolic risk, as well trajectories of change in cardio-metabolic risk over a ten year period in 4 different race/ethnic groups. We hypothesize that living in a disadvantaged socioeconomic neighborhood is associated with higher cardio-metabolic risk and greater accumulation of risk over time; these associations are hypothesized to be more pronounced among race/ethnic minority populations.

Methods

Study sample

MESA is a prospective cohort study of the determinants of subclinical cardiovascular disease with a multi-ethnic, population-based sample of 6814 men and women. Participants were recruited in 2000 at ages 45-84 years from a range of socioeconomically diverse neighborhoods in six communities in the U.S., including Baltimore, MD; Chicago, IL; Forsyth County, NC; Los Angeles County, CA; New York, NY; and St. Paul, MN. Represented race/ethnic groups include white, African American, Chinese and Hispanic participants. The baseline examination took place between July 2000 and August 2002, with 4 follow-up exams through September 2011. Among those screened and eligible for the baseline examination (including no history of cardiovascular disease), the participation rate was 59.8%, and retention rates were 91%, 87%, 84% and 80% of the original cohort through exams 2–5, respectively. Details of the study design and recruitment for MESA have been published (Bild et al., 2002). MESA was approved by the IRBs at participating institutions, and all participants provided written informed consent.

Our study sample included MESA participants who attended the baseline exam and at least one of the other follow-up exams, had available data for measuring a cardio-metabolic index, geocoded data, and information about socioeconomic factors and nativity status. Of the initial MESA cohort (n = 6814), 623 participants were excluded for missing geocoded neighborhood data, 23 were missing components of the cardio-metabolic index, and 418 were missing nativity status, parental nativity status, parental education, education, income and wealth. The total analytic sample included n = 5750 participants, 84% of the original cohort. Those remaining in the analytic sample included a significantly higher proportion of males, white and Chinese participants, lower percentage of African Americans, and had higher SES and lower AL compared to the original MESA cohort (data not shown).

Outcome

We used a cardio-metabolic index of disease risk to measure AL, at each of the 5 examinations (baseline and 4 follow-up) by incorporating available data on metabolic and cardiovascular measures; similar indices have been used in other studies (Merkin et al., 2014, 2015). Metabolic indicators included waist–hip ratio (WHR), triglycerides, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, and glucose. Triglycerides, LDL cholesterol, and glucose values were considered only for those who fasted for at least 10 h. Cardiovascular measures included systolic blood pressure, resting heart rate, and pulse pressure. For each indicator, we calculated standardized scores to indicate where the individual's value placed them (in standard deviation units) relative to accepted clinical cutpoints for higher risk (with 0 indicating the clinical cutpoint for high risk). These included 0.90 WHR for men and 0.85 for women (Alberti & Zimmet, 1998), 200 mg/dL for triglycerides (National Cholesterol Education Program (NCEP) Expert Panel, 2001), 160 mg/dL for LDL cholesterol (National Cholesterol Education Program (NCEP) Expert Panel, 2001), 40 mg/dL HDL cholesterol (National Cholesterol Education Program (NCEP) Expert Panel, 2001), 4.84 log of glucose (corresponding to 126 mg/dL) (Alberti & Zimmet, 1998), 140 mm of mercury for systolic blood pressure (Chobanian et al., 2003), 60 mm of mercury for pulse pressure (Haider et al., 2003) and 90 beats per minute for heart rate (Seccareccia et al., 2001). Higher standardized scores correspond to higher risk (HDL values were multiplied by -1 since lower values reflect higher biological risk). The total AL score was calculated by summing the standardized scores for the individual parameters. We set the AL score to missing if values were missing for more than half ($\geq$5) of the 8 components in the score (0 missing at baseline, 0.09% at exam 2, 0.02% at exam 3, 0.13% at exam 4, 0.07% at exam 5). For those with 1–4 missing components (8% of the sample), we imputed values at a given examination using the mean of a given component across all available visits. Imputed values were used for n = 1083 participants missing heart rate data at exam 2 due to a delay in heart rate data collection; n = 750 other values were imputed across all 5 exams. We chose to use calculate AL score based on continuous values of biology (Seeman et al., 2004) rather than counting indicators of high risk (Merkin et al., 2014, 2015), to ensure that the score is sensitive to changes in biology that do no cross clinical or high risk thresholds.

Exposure

A NSES index was developed from U.S. Census and American Community Survey (ACS) data at the census tract level, based on data from the U.S. Census 2000 (US Census Bureau. Census, 2000). Using principal factor analysis with varimax rotation, 16 tract-level measures of educational attainment, occupation, income, wealth, poverty, employment status, and housing characteristics were considered; 7 SES variables that loaded together were used to calculate a NSES score by multiplying the standardized variables by the factor weights. These measures include: median home value, percent with $\geq$ high school education, percent with $\geq$ Bachelor's degree, percent with management/professional occupation, median household income, percent with household income>$50,000, percent households with interest/dividend/rental income. This score has been used in other MESA

analyses (Moore et al., 2013); a higher index score indicates greater neighborhood advantage.

Covariates

Additional covariates were based on self-report and mostly obtained at baseline, including age, gender, race/ethnicity, MESA study site, nativity status, parental nativity, socioeconomic factors; some wealth measures were only available at the second visit. Age was centered at the mean (61.8), and nativity status was defined as foreign-born vs US born. Race/ethnic groups represented in MESA include white, Chinese, African American, and Hispanic participants. Parental education was categorized into approximate tertiles: less than high school, complete high school and greater than high school for parental education (highest attained education by either parent). Participant education was collapsed into approximate tertiles: less than or complete high school, some college and complete college education. Wealth measures (yes/no) included information on home ownership at baseline, land or property ownership at visit 2, car ownership at visit 2, and possession of investments, stocks or bonds at visit 2. Medication use was determined at each MESA exam and included self-reported information about hypertensive, statin or insulin medication.

Analysis

In initial descriptive analyses, we examined the distributions of AL, NSES and all covariates by race/ethnicity. We then ran mixed effects models on 29,940 observations from 2 to 5 study visits per each of 5750 participants, with AL as linear function of time elapsed since the baseline examination. The mixed models included random effects for the intercept (AL level) and slope (rate of change in AL over time), at the individual-level and neighborhood-level, thereby accounting for both potential within-person clustering in repeated measures and between-person clustering by neighborhood. The models included fixed effects for the NSES predictor and each covariate on both intercept and slope. In light of prior work suggesting the possibility of a non-linear association (Merkin et al., 2009; Slopen et al., 2014), we examined the functional form of the NSES-AL relationship using restricted cubic splines (adjusting for all covariates) stratified by race/ethnicity. Based on plots of predicted AL obtained from these models, the association between higher NSES and lower AL appeared to be linear at the higher end of NSES (approximately NSES>1.0) but less consistently linear at the lower end of the scale. Based on these results, we examined NSES in two separate models, first as a continuous score and then by quintiles, since the top quintile of NSES in the study sample is > 1.32 and close to the inflection point in the spline plots. All models were stratified by race/-ethnicity per the main objective of this study. Considering previous findings of differential associations between individual-level SES and AL by baseline levels of AL (Merkin et al., 2014), we included adjustment for the effects of baseline AL on changes in AL over time.

Additional sensitivity analyses examined the NSES-AL associations among Hispanic participants stratified by nativity status, informed by studies that have shown health advantages in foreign-born Hispanic populations compared to US-born counterparts (Karlamangla et al., 2010) that may be related to neighborhood-level social support even in impoverished neighborhoods (Eschbach et al., 2004).

Results

As shown in Fig. 1, NSES distributions differed substantially by race/ethnicity. White participants resided in neighborhoods with the highest socioeconomic levels, followed by Chinese, African American and Hispanic participants. A similar pattern was apparent for individual measures of SES, with white participants reporting the highest levels and Hispanic participants the lowest. Whites had the lowest levels of AL (i.e., most negative) at baseline and Hispanics the highest, with similar levels

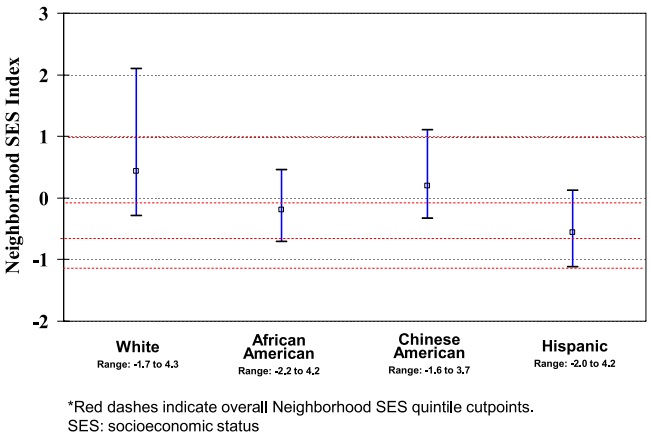

Fig. 1. Interquartile Ranges of Neighborhood SES Index by Race/Ethnic Group: MESA, n = 5750 (analytic sample)*.

for African American and Chinese participants (see Table 1 for all distributions).

Mixed effects models with NSES as a continuous score indicated statistically significant associations between higher NSES score and lower baseline AL for all race/ethnic groups (mean differences ranging from -0.30 to -0.25 per unit increase NSES). There were no statistically significant differences in AL change over time by NSES score (Table 2; Supplementary Table 1 for full set of adjusted covariates).

Examination of NSES by quintiles revealed statistically (or marginally) significant associations between living in the highest NSES quintile (quintile 5 vs. 1) and lower AL in 3 of the 4 groups (the exception being Chinese; highest quintile vs. lowest: 0.86, $p = 0.002$ for white; -0.60, $p = 0.3$ for Chinese; -0.71, $p = 0.07$ for African American; -1.15, $p = 0.002$ for Hispanic participants; see Table 3). Although remaining quintiles mostly indicated lower AL compared to those living in the lowest NSES quintile (i.e. quintiles 2–4 vs 1), these differences were not statistically significant and the magnitude of the differences increased markedly from quintile 4 to 5, indicating stronger health benefits associated with living in the highest NSES quintile compared to the others. To further test whether the major difference was between the highest quintile and the remaining quintiles or whether there were also differences between those remaining quintiles, models were re-run excluding the highest quintile and considering the remaining quintiles as an ordinal variable to assess an overall test of trend; we found no significant trend for quintiles 1–4 ($p > 0.1$ for each race/ethnic group). Interaction terms for race/ethnicity and highest NSES quintile did not show statistically significant differences in these association by race/ethnicity ($p > 0.1$). In addition, interaction terms for race/ethnicity and continuous NSES, as well as a single ordinal measure of NSES quintiles were all not statistically significant ($p \geq 0.1$). There were only two (of 16) statistically significant differences in AL change over time by NSES quintiles (Table 3; Supplementary Table 2 for full set of adjusted covariates).

A final set of models examined the question of possible differences among Hispanic participants by nativity. In models stratified by Hispanic nativity status, we found similar trends for both groups, though associations were stronger among the larger group of foreign-born Hispanic participants (-1.33, $p = 0.006$ and -1.00, $p = 0.2$ for lowest quintile vs highest for n = 828 foreign-born and n = 420 US-born, respectively).

Discussion

Our findings confirm the inverse association between NSES and AL and that this trend is similar across race/ethnic groups. The results suggest that this association is more pronounced among Hispanic populations, with an increase in effect size compared to the other groups and

Table 1
Distributions of selected characteristics by race/ethnic group in MESA, n = 5750[a].

Characteristics	White n = 2305	Chinese n = 706	African American n = 1471	Hispanic n = 1268
	Percentage or mean, median, SD			
Age [range: 44–84]	62.15, 62.0, 10.12	61.75, 62.0, 10.19	61.21, 61.0, 9.79	61.28, 61.0, 10.31
Male	48.63	49.86	45.21	48.19
MESA Site				
Forsyth, NC	21.91	0	23.11	0.24
New York, NY	8.76	0.28	22.16	34.62
Baltimore, MD	19.09	0	28.35	0
St. Paul, MN	23.25	0	0	29.89
Chicago, IL	21.82	39.09	17.54	0
Los Angeles, CA	5.16	60.62	8.84	35.25
Born outside the US	6.68	95.89	9.52	66.88
Parent/s born outside the US	22.73	99.72	11.62	88.49
Income				
≤$24,999	14.88	46.74	28.01	48.26
$25–49,999	26.59	22.80	32.56	33.75
≥$50,000	58.52	30.45	39.43	17.98
Participant's Education				
<=High school	20.43	37.96	27.80	62.78
Some college	27.42	20.54	36.30	26.34
>=College	52.15	41.50	35.89	10.88
Parents' Education				
<High school	27.77	45.33	43.51	72.71
Complete High School	30.72	19.41	30.25	18.45
>High School	41.52	35.27	26.24	8.83
Wealth (yes vs. no)				
Home ownership	85.29	63.17	64.85	45.03
Land/property ownership	35.49	25.21	36.37	18.14
Car ownership	90.07	81.44	82.73	70.19
Investments/stocks/bonds	87.46	45.61	62.34	31.39
Medication use				
Hypertensive medication (exam 1- baseline)	32.41	27.76	47.79	32.97
Hypertensive medication (exam 5)	50.16	47.05	66.45	55.18
Statins medication (exam 1- baseline)	16.68	13.03	14.35	12.80
Statins medication (exam 5)	40.88	30.29	34.37	36.89
Insulin medication (exam 1- baseline)	0.83	0.28	2.26	2.61
Insulin medication (exam 5)	1.65	1.52	4.86	4.86
Neighborhood Socioeconomic Status				
Neighborhood SES score	0.86, 0.43, 1.47	0.39, 0.19, 1.07	-0.01, -0.20, 1.0	-0.37, -0.56, 1.06
Quintiles of NSES [score range; mean]				
Q1 [-2.22 to-0.84; mean -1.13]	12.15	11.33	19.10	41.56
Q2 [-0.84 to -0.29; mean -0.57]	8.63	14.87	26.44	19.09
Q3 [-0.29 to 0.26; mean -0.08]	23.60	26.49	21.14	18.85
Q4 [0.27 to 1.32; mean 0.70]	20.91	26.06	24.47	13.33
Q5 [1.32 to 4.25; mean 2.49]	34.71	21.25	8.84	7.18
Allostatic Load				
Baseline	-8.41, -8.36, 3.83	-7.80, -7.64, 3.61	-7.85, -7.97, 3.82	-6.48, -6.56, 3.87
Exam 2	-8.17, -8.22, 3.70	-7.85, -7.96, 3.35	-7.66, -7.89, 3.68	-6.36, -6.53, 3.67
Exam 3	-8.45, -8.53, 3.67	-7.87, -8.03, 3.50	-7.84, -8.12, 3.66	-6.53, -6.65, 3.58
Exam 4	-8.32, -8.35, 3.57	-7.84, -7.94, 3.32	-7.81, -7.87, 3.50	-6.59, -6.59, 3.55
Exam 5	-8.72, -8.83, 3.38	-8.10, -8.20, 3.34	-8.09, -8.29, 3.5	-7.13, -7.44, 3.52

[a] Based on MESA sample not missing NSES score, baseline AL, nativity information, parental nativity, education, parental education, income, wealth, medication for at least one visit for each type. Characteristics were assessed at baseline unless otherwise noted.

Table 2
Adjusted associations of NSES with allostatic load at baseline and annualized rate of change (slope) in allostatic load, stratified by race/ethnicity.[a].

	Race/ethnicity Adjusted mean AL at baseline (adjusted slope)			
	White -7.51 (0.01)	Chinese -7.28 (-0.14)	African American -7.70 (0.03)	Hispanic -6.71 (-0.03)
Mean difference in baseline AL by NSES Advantage Score	-0.30 (-0.46, -0.13)***	-0.29 (-0.57, -0.001)*	-0.25 (-0.44, -0.06)*	-0.26 (-0.44, -0.09)**
Mean difference in AL slope by NSES Advantage Score	0.003 (-0.01, 0.02)	-0.01 (-0.04, 0.01)	-0.001 (-0.02, 0.02)	0.01 (-0.01, 0.02)

~0.1<p < 0.05 *0.01<p < 0.05 **0.001<p < 0.01 ***p < 0.001.
AL: allostatic load, NSES: Neighborhood Socioeconomic Status.
[a] Race-specific means are adjusted for age, age squared, gender, site, nativity status and parental nativity status, parental and adult education, wealth, income, medication use, baseline allostatic load interacted with time.

that this association is weaker for the African American population and not statistically significant for the Chinese population. However, considering the wide confidence intervals and that the race/ethnic differences were not statistically significant, these results do not provide conclusive evidence supporting race/ethnic disparities in the association between NSES and AL. Moreover, we were unable to replicate previous findings by other studies using NHANES data that found stronger NSES-AL associations among African Americans compared to white and Hispanic groups (Merkin et al., 2009), although NSES distributions across race/ethnic groups differ significantly between the datasets and may partially explain the different results. In light of the consistent finding that race/ethnic minority groups are more likely to live in the most disadvantaged neighborhoods (Fig. 1) that appear to be associated with higher AL, further research is clearly needed to assess whether other neighborhood factors that are not included in our NSES index may be contributing to health disparities across these groups.

We found inconsistent trends in AL change over time, with white and Hispanic participants showing modest change in AL over time (not statistically significant), the African American group experiencing increase in AL over time, and the Chinese group showing a statistically significant decrease in AL over time; we also found almost no indication of significant associations between NSES levels and change in AL over time. Improved or minimal change in AL may be the result of increased medical interventions and medication use of this cohort due to shifting eligibility status for Medicare during this study period (median baseline

Table 3
Adjusted associations of NSES quintiles with allostatic load at baseline and annualized rate of change (slope) in allostatic load, stratified by race/ethnicity.[a].

	Race/ethnicity Adjusted mean AL at baseline (adjusted slope) for the reference quintile			
	White -7.24 (-0.004)	Chinese -7.12 (-0.13)	African American -7.54 (0.06)	Hispanic -6.33 (-0.03)
Mean difference in baseline AL by NSES Quintiles				
Q1 (lowest)	Reference	Reference	Reference	Reference
Q2	-0.23 (-0.82, 0.37)	0.22 (-0.70, 1.14)	0.12 (-0.45, 0.68)	-0.24 (-0.72, 0.24)
Q3	-0.18 (-0.57, 0.21)	-0.18 (-1.08, 0.72)	-0.01 (-0.55, 0.53)	-0.19 (-0.70, 0.31)
Q4	-0.32 (-0.77, 0.13)	-0.30 (-1.20, 0.61)	-0.22 (-0.81, 0.37)	-0.27 (-0.83, 0.28)
Q5 (highest)	-0.86 (-1.41, -0.31)**	-0.60 (-1.66, 0.45)	-0.71 (-1.48, 0.06)~	-1.15 (-1.87, -0.44)**
Mean difference in AL slope by NSES Quintiles				
Q1 (lowest)	Reference	Reference	Reference	Reference
Q2	0.02 (-0.04, 0.09)	0.04 (-0.04, 0.13)	-0.05 (-0.11, -0.003)*	-0.05 (-0.09, -0.002)*
Q3	0.01 (-0.03, 0.06)	-0.04 (-0.12, 0.03)	-0.01 (-0.06, 0.04)	-0.003 (-0.05, 0.05)
Q4	0.02 (-0.03, 0.07)	-0.03 (-0.11, 0.05)	-0.05 (-0.10, 0.002)~	0.02 (-0.04, 0.07)
Q5 (highest)	0.03 (-0.03, 0.08)	-0.004 (-0.09, 0.09)	-0.01 (-0.08, 0.06)	-0.01 (-0.09, 0.07)

~0.1<p < 0.05 *0.01<p < 0.05 **0.001<p < 0.01 ***p < 0.001.
AL: allostatic load, NSES: Neighborhood Socioeconomic Status.
[a] Race-specific means are adjusted for age, age squared, gender, site, nativity status and parental nativity status, parental and adult education, wealth, income, medication use, baseline allostatic load interacted with time.

age of 62) or increased medical intervention associated with participation in a cohort study.

Results presented here provide some suggestive evidence that the relationship between NSES and AL may not be strictly linear, with those living in the highest NSES areas (the top 20th percentile in this case) exhibiting significantly lower cardio-metabolic risk compared to all lower NSES areas. Assessment of model fit based on Akaike information criterion (AIC) (Burnham & Anderson, 2004) however, did not indicate significant improvement over the models utilizing a continuous measure of NSES. Two other studies also considered ordinal categories of NSES, thereby testing non-linear associations with AL (Merkin et al., 2009; Slopen et al., 2014), one considered quintiles (as we do in this current analysis) (Merkin et al., 2009) and the other, tertiles of NSES (Slopen et al., 2014), with the highest NSES level in each study indicating the deflection point of lower biological risk compared to the lower NSES levels. While the current findings, as well as the other studies just mentioned, do not definitively point to a threshold effect, they do suggest that the association between NSES and AL may be driven by the benefits of living in the highest SES neighborhood compared to the others. These health differences likely reflect the large socioeconomic gaps between those in the top NSES neighborhoods and all the others, as evidenced by the relatively large NSES mean score increase at the highest quintile (see Table 1).

Interestingly, in popular culture there has been talk of the top "1%", and more recently, about the affluence of the top 9.9% vs. the socioeconomic stagnation of the rest of the US population (Stewart, 2018). Perhaps with regard to health effects, as indicated in the current analyses, living in the "top" tier of neighborhoods may provide protection or mitigate accumulated biological risk compared to living in lower socioeconomic neighborhoods. Future research should address the underlying factors leading to these patterns-are those at the "top" utilizing available resources to maintain good health that are otherwise not available, or is the relative condition of living in the "best" neighborhood conferring its own impact on positive health outcomes? Ultimately, identifying thresholds of impact may most efficiently inform policy to prevent and/or mitigate the negative impact of low NSES.

The strengths of this study include the large and diverse population-based cohort, as well as multiple waves of AL data. In a recent comprehensive review of studies examining the association between NSES and AL, Ribeiro et al. identified 14 relevant studies published by March 2018 (Ribeiro et al., 2018). That review highlights the contribution of the current study in the following ways. First, our results replicate the most common finding (12 of 14 studies) that NSES is indeed inversely associated with AL. Second, while the review notes 4 "longitudinal" studies of NSES and AL, three of those refer to longitudinal measures of NSES and only one considered longitudinal measures of AL (2 time points). Moreover, the one study that did examine change in AL (Jiménez et al., 2015) simply adjusted for baseline AL, a method that can lead to spurious results when change is the outcome (Glymour et al., 2005). Our study uniquely provides 5 waves of AL data, and adjusts for the effect of baseline AL levels on change in AL over time. Considering that such an adjustment can bias the estimate of NSES effect on AL slope, we ran models with and without adjusting for the effect of baseline AL on change in AL; the true NSES association with AL slope lies somewhere between results with and without this adjustment (Glymour et al., 2005). We did not find statistically significant associations between NSES and change in AL over time with and without adjustment for baseline AL. The general decrease in AL over time and the lack of significant association with NSES levels may reflect the medical intervention and ongoing monitoring that are provided to participants in a longitudinal cohort study. That is, providing equitable care to study participants may have not only resulted in improved biological risk over the study period (i.e. lower AL scores over time), but also mitigated health care disparities related to living in disadvantaged neighborhoods.

Other limitations related to our analyses include a measure of AL limited to cardio-metabolic measures that were available at multiple time points in the MESA dataset. Moreover, medication use was not incorporated into the score since it was calculated using standardized biomarker values. As in other analyses using this score (Merkin et al., 2014, 2015), we adjusted for medication use in the models. An additional limitation includes a single time assessment of neighborhood SES at the MESA baseline. Over the course of the follow-up period, about 32% of the cohort moved, however, those who moved were not significantly different than those who did not with regard to baseline AL and baseline NSES (data not shown). In addition, the lack of data on neighborhood history, as well as the possibility of residual confounding due to improperly measured variables or missing confounders, limits the ability to draw formal causal inferences from these findings.

Conclusions

Our findings confirm a consistent relationship between living in disadvantaged neighborhoods and increased cardio-metabolic risk, as measured by AL, across race/ethnic groups. These results suggest that the association may reflect the benefits of living in neighborhoods at the high end of the NSES distribution. Identifying neighborhood factors that impact health in this way may be crucial to effectively target and improve health outcomes for those living in high risk neighborhoods.

Author contributions

Dr. Merkin designed the study and directed its implementation, data analysis and interpretation, and drafted the manuscript. Dr. Karlamangla contributed to the conception and design, data interpretation, reviewed and revised the manuscript. Dr. Diez Roux contributed to the conception and design, data interpretation, reviewed and revised the manuscript. Ms. Shrager and Dr. Watson contributed to data interpretation, reviewed and revised the manuscript. Dr. Seeman contributed to the conception and design, data interpretation, reviewed and revised the manuscript.

Ethics approval

The parent study (MESA) has obtained IRB approval for collecting all data analyzed in this study and consent for all participants (https://www.mesa-nhlbi.org/MesaInternal/IRBApproval.aspx). In addition, we obtained IRB approval from the UCLA Office of the Human Research Protection Program (IRB#12-001371-CR-00007) for the specific set of analyses in the current manuscript.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ssmph.2020.100634.

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