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Social Stratification and Allostatic Load: Shapes of Health Differences in the MIDUS study in the United States

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Abstract

Social stratification is one important mechanism of human organization that helps to explain health differences between demographic groups commonly associated with socioeconomic gradients in the United States. Individuals, or group of individuals, with similar health profiles may have had different stratification experiences. This is particularly true as social stratification is a significant non-measurable source of systematic unobservable differences in both SES indicators and health statuses of disadvantage. Accordingly, the goal of the present analyses was to expand the bulk of research that has traditionally treated socioeconomic and demographic characteristics as independent, additive influences on health. It is hypothesized that variation in an index of multi-system physiological dysregulation—Allostatic Load—is associated with social differentiation factors, sorting individuals with similar demographic and socioeconomic characteristics into mutually exclusive econo-demographic classes. The data are from the Longitudinal and Biomarker samples of the Study of Midlife in the US (MIDUS). Latent class analyses and regression analyses reveal that physiological dysregulation linked to socioeconomic variation among black people, females, and older adults are associated with forces of stratification that confound socioeconomic and demographic indicators. In the United States, racial stratification of health is intrinsically related to the degree to which black people in general, and black females in particular—as a group—share an isolated status in society. Findings present evidence that disparities in health emerge from group-differentiation processes to the degree that individuals are distinctly exposed to the ecological, political, social, economic and historical contexts in which social stratification is ingrained. Given that health policies and programs emanate from said legal and political environments, interventions should target the structural conditions that expose different subgroups to different stress risks in the first place.

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Conflicts of interest

The authors have no conflicts of interest to declare.

Ethical approval

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

Introduction

Studies of socioeconomic status (SES) and race/ethnicity disparities in health in the United States have traditionally treated SES and demographic characteristics as independent, additive influences on health. However, the effects of SES and of demographic characteristics like race/ethnicity on health are not easily disentangled. The persistence of socioeconomic disadvantages among, for example, African Americans, women, and older adults suggests the possibility that the health characteristics they frequently share may be related to the effect of stratification forces that confound socioeconomic and demographic indicators, and not only from additive effects that arise from socioeconomic and demographic indicators independently.

Vast evidence points to the existence of an SES-health gradient in the United States and abroad. Individuals located in the lower tail of education and income distributions show elevated levels of morbidity and mortality (Adler *et al.*, 1994; Pearlin *et al.*, 2005; Clougherty, Souza and Cullen, 2010; Bound *et al.*, 2014; Bound *et al.*, 2015). Research also indicates that low SES is more heavily concentrated in racial/ethnic minority communities, that there is similar confounding between older age and low SES, and likewise between being a female and low SES (Geronimus *et al.*, 2010; Williams *et al.*, 2010). Additionally, the influence of SES on health risks such as cardiovascular disease, for example, appear to vary significantly by race, age, and gender (Karlamangla *et al.*, 2010; Kwarteng *et al.*, 2016). Much research has found morbidity differences between females and males, mortality and health disparities between blacks and non-blacks, and higher comorbidity among older adults (Black, 1982; Verbrugge, 1989; Lipowicz, Szklarska and Malina, 2013).

Although many analyses find that health outcomes coincide with socioeconomic and demographic subgroups, less attention has been devoted to analyzing how socioeconomic and demographic indicators may segregate into selected typologies with respect to their impact on health outcomes. Establishing econo-demographic typologies associated with the distribution of health could refine and push forward current research on SES health gradients and suggest new ways to address health disparities.

Considerable research has looked at the ways race and SES combine to produce health outcomes in the U.S., especially among African Americans and whites. The unique social, political, and economic histories of African Americans continue to shape individual and structural discrimination effects on their health. Their “migration” experience was involuntary and their participation in the most decisive restructuring episodes of American history (i.e., the writing of the Declaration of Independence and the Constitution, and the Civil War) happened under the jurisdiction of the slavery system. Today’s African Americans are the descendants of those who lived under slavery or who were socialized under the Jim Crow system (Sears, Citrin and Van Laar, 1995; Sears and Savalei, 2006). And in spite of advancements prompted by the Civil Rights Movement, blacks continue to experience socioeconomic, legal, and political disadvantages that manifest in detrimental biological expressions (Collins and Williams, 1999).

The intersection between gender and SES has also been the focus of a large body of research. The role of gender, as a powerful principle for social organization, is well documented in social, economic, and political histories (Crenshaw, 1991; Josephson and Tolleson-Rinehart, 2000; McCall, 2005; Hancock, 2007). From the distribution of rights to access to public resources, and from reproductive roles to unequal labor market opportunities, the life experiences of females differ from those of males (Sapiro, 2003). Not surprisingly, given these differences, research has also established gender-based inequalities in health, with women reporting higher levels of distress and depression and a higher burden of chronic illnesses than males, independent of any genetic factors (Marcus and Seeman, 1981; Denton, Prus and Walters, 2004; Annandale, 2009). Females in general, but especially racial/ethnic minority females, tend to locate at the lower tail of the SES distribution and to experience associated accumulations of negative health outcomes (Geronimus, 1992; Geronimus et al., 2010).

Although reaching older age is selective on health (Markides and Machalek, 1984), there is a strong relationship between aging and functional limitation and disability, especially among older adults. In the United States, for example, more than 60% of individuals over the age of 65 have multiple chronic conditions (Vogeli *et al.*, 2007). As retirees age, they also become more vulnerable to the negative health effects of a lower SES due to higher risk of comorbidity. Apart from functional, cognitive, and productivity declines related to aging, pension allowances that often do not correspond to pre-retirement income levels push new retirees toward the left tail of the SES spectrum. In fact, psychosomatic manifestations, frailty, and multi-system physiological dysregulation are the rule more than the exception in older adults (Fried *et al.*, 2001).

A key argument of this study is that the same social forces that non-randomly sort demographic groups into socioeconomic statuses affect their health in profound ways. The health status of blacks, older adults, and females often concur with the multi-system dysregulation found in lower socioeconomic statuses. Multi-system physiological indicators have evinced poorer metabolic performance, higher inflammatory burden, higher concentration of stress-related hormones, and higher risk of cardiovascular disease among these groups (Geronimus *et al.*, 2006). The purpose of this paper is, therefore, to investigate how allostatic load—an index constructed from biomarkers that assess the orchestration of the nervous, metabolic, immune, cardiovascular, and endocrine systems—may be linked to social stratification as evinced in econo-demographic within-class homogeneity and between-class dissimilarity.

Methods

This study uses data from the National Survey of Midlife Development in the United States (MIDUS), a longitudinal study first fielded in 1995/1996 with a national baseline sample including more than 7,000 participants. The baseline cohort also included a subsample of siblings (n = 950) and a subsample of twins (n = 1,914). Data on psychosocial, physiological, demographic, and behavioral indicators important for the understanding of the long-term pathways that lead to health and illness were collected via random-digit dialing telephone surveys and self-administered questionnaires. A longitudinal follow-up

assessment in 2004/2006 (MIDUS II) included the measures used in the baseline MIDUS plus new biological and neurological measures. The final analytic sample implemented in the present analyses use data from the MIDUS II Biomarker substudy (n = 1,255), which includes a subsample of Milwaukee African Americans (n = 201), participants from the baseline sample (n = 666), and a subsample of twins (n = 388). The Biomarker Project data was collected during a 2-day protocol at Georgetown University, UCLA, and University of Wisconsin. Visit protocols were standardized across the three research centers (Love et al., 2010). The analyzed sample includes 1,244 individuals (of which 199 are from the Milwaukee subsample) who had sufficient data to construct the allostatic load scale.

The analyses implement socioeconomic and demographic indicators, and physiological biomarkers—all of which are standard in the literature. The SES indicators are divided in two objective measures (education and household adjusted poverty-to-income ratio) and three subjective measures (perception of current financial situation, enough money to meet basic needs, and having enough money to pay bills). The demographic indicators are race/ethnicity, gender, and age (Table 1). And finally, this study uses twenty-three physiological biomarkers that represent seven biological systems of the human body (Table 2).

Socioeconomic indicators.

Education was coded from 1 to 5: 1 = less than high school (HS), 2 = HS/GED, 3 = some college/associate's degree, 4 = bachelor's degree, and 5 = graduate degree or higher. The construction of household adjusted poverty-to-income ratio (HHPIR) required three main computations: (1) Household size: Total number of children (individuals 17 years and younger) and adults (individuals 18 years and older) living in the household; (2) Household income adjusted to inflation: The sum of the inflation-adjusted respondent's, spouse's, and other household members' personal earnings, pension income, social security income, and government assistance income; (3) Poverty threshold: Obtained from the U.S. Census Bureau and assigned to participants according to household size and the year their data were collected. The HHPIR was coded from 1 to 5: 1 = less than 250%, 2 = 250–499%, 3 = 500–749%, 4 = 750–999%, and 5 = 1000% or more. Perception of current financial situation was coded from 1 to 3: 1 = worst possible, 2 = average, and 3 = best possible. Having money necessary to meet basic needs was coded from 1 to 3: 1 = not enough, 2 = just enough, and 3 = more than enough. And level of difficulty paying bills was coded from 1 to 3: 1 = very or somewhat difficult, 2 = not very difficult, and 3 = not at all difficult.

Demographic indicators.

Race/ethnicity was coded dichotomously: 2 = black/African American and 1 = non-black (n = 1,025; white (n = 960), multiracial (n = 44), other (n = 21)). Gender was coded dichotomously: 2 = female and 1 = male. Age was coded by decade from 1 to 5: 1 = 34–44 years, 2 = 45–54 years, 3 = 55–64 years, 4 = 65–74 years, and 5 = 75–84 years.

Physiological biomarkers.

The 23 physiological biomarkers represent 7 biological systems: the sympathetic (SNS), parasympathetic (PNS), hypothalamic pituitary adrenal axis (HPA), inflammation, cardiovascular, and metabolic (glucose and lipids) systems. The biomarkers were measured

as follows for the 7 biological systems. (1) *SNS*: Overnight urinary measures of epinephrine and norepinephrine. (2) *PNS*: Included four heart rate variability parameters: (a) low and high frequency spectral power, (b) the standard deviation of heartbeat-to-heartbeat intervals (SDRR), and (c) the root mean square of successive differences (RMSSD). (3) *HPA*: An overnight urinary measure of cortisol and a serum measure of dehydroepiandrosterone sulfare (DHEAS). (4) *Inflammation*: Included plasma C-reactive protein (CRP), fibrinogen, and serum measures of interleukin-6 (IL-6) and the soluble adhesion molecules e-Selectin and the intracellular adhesion molecule-1 (ICAM-1). (5) *Cardiovascular*: Three measures of cardiovascular performance were used: (a) resting pulse, (b) resting systolic blood pressure (SBP), and (c) diastolic blood pressure (DBP). (6) *Glucose metabolism*: Levels of glycosylated hemoglobin, fasting glucose, and the homeostasis model of insulin resistance (HOMA-IR). (7) *Lipids metabolism*: High density lipoprotein (HDL) cholesterol, low density lipoprotein (LDL) cholesterol, triglycerides, body mass index (BMI), and waist-to-hip ratio (WHR).

Allostatic Load (AL).

From these biomarkers, 7 indices of physiological risk were developed and then used to construct the allostatic load (AL) scale. This AL scale was computed as the sum of the seven separate physiological risk indices and a score was assigned to each participant, as it is standard in the literature (Gruenewald *et al.*, 2012; Brooks *et al.*, 2014; Karlamangla *et al.*, 2014; Mori *et al.*, 2014; Seeman *et al.*, 2014; Friedman *et al.*, 2015; Wiley *et al.*, 2016). Each of these 7 indices was calculated as the proportion of the total number of biomarkers for each system (ranging from 2 to 6) that reached high-risk quartile values (upper or lower quartile, depending on the biomarker). Indices thus range from 0 to 1, where 0 = none of the system biomarkers reached high-risk quartile levels, and 1 = all of the system biomarkers reached high-risk quartile levels. Indices were assigned to participants with values on at least half of the respective system biomarkers. The sum of these 7 individual physiological risk indices produced an AL scale with a possible range from 0 to 7, where 0 = none of the biomarkers across the 7 indices reached high-risk quartile levels, and 7 = all of the biomarkers across the 7 indices reached high-risk quartile levels. This AL score was assigned to participants with data on at least 6 of the 7 physiological risk indices. Missing AL data was very low, permitting to assign an AL score to over 99% of the sample participants.

The main objective of this study is to investigate whether variations in AL scores are associated with social differentiation forces that sort individuals with similar socioeconomic and demographic characteristics into mutually-exclusive, latent econo-demographic classes —that is, whether the distribution of manifest characteristics in the sample generates socioeconomic typologies found in demographic subgroups that in turn are associated with variations of multi-system physiological risk. Accordingly, a two-step procedure is applied: first a latent class analysis, and then a series of linear regressions.

The analyses begin by using a Polytomous Variable Latent Class Analysis (hereafter LCA) (Stouffer *et al.*, 1950; Lazarsfeld, 1955; Marcoulides and Moustaki, 2012) to detect patterns of association between manifest SES and demographic indicators. For this analysis, the

statistical package poLCA was ran in R (Linzer and Lewis, 2007; Linzer and Lewis, 2011). This method provides a semiparametric alternative to traditional additive modeling of SES and demographic indicators allowing to unearth latent heterogeneity in respondents' characteristics and to thereby cluster respondents with shared demographic profiles and similar socioeconomic response values into mutually-exclusive, latent econo-demographic classes. These latent classes result from important yet commonly overlooked non-measurable sources of systematic unobservable variation in both SES indicators and demographic subgroups. LCA, therefore, offers an alternative perspective to traditional additive modeling of SES-gradients and demographic differences in health Instead of assuming monotonic associations in the data, LCA permits to sort out the similarities and differences of the patterns that live in the cross-categorizations of the socioeconomic and demographic indicators. The underlying assumption is therefore a different one: That the presumable unobserved sources of heterogeneity that generate the latent econo-demographic classes are related to an important degree to processes of social stratification. Results from this modeling strategy encapsulate a rich interpretation of social stratification and its relationship to health.

LCA models were fitted for 2 to 7 latent classes using Maximum Likelihood estimation. Since the variables are categorical, this was accomplished without making assumptions (other than local independence) about the distribution of the indicators that define the classes (Hagenaars and McCutcheon, 2002; Lanza *et al.*, 2007). To avoid local maxima and increase the probability of a global maximum solution of the log-likelihood function, each model was fitted 100 times (Linzer and Lewis, 2011). Complete data cases were used with no imputation for missing data. Differences between missing and non-missing data cases were trivial.

There are differences in the qualitative nature of the variation that SES and demographic indicators contain, respectively. SES indicators mostly contain a social-type of variation while demographic indicators may contain both social and other (e.g., biological) types of variation. Because fitted classes mostly emerge from correlations among the cross-categorizations of the socioeconomic and demographic indicators, the LCA algorithm would exhaust most of the social-type variance shared by the SES and demographic indicators, increasing the confidence that LCA-fitted classes are representative, at least to an important degree, of *social* processes of differentiation. To account for the non-social variation in demographic indicators, these covariates were also included in the regression models.

Each participant is assigned a posterior probability (PP) of class membership, one for each of the fitted classes. The PP is a measure of similarity between the participant and the prototypical person in the class, and it is estimated via the LCA algorithm. PPs sum to 1 for each individual, and differ from one LCA-fitted class to another precisely because they capture unobservable heterogeneity. In essence, a PP represents the probability that a participant with specific characteristics would belong to a given class.

It is hypothesized that AL scores vary across the LCA-identified classes, and vary among individuals within a class, according to how much class members resemble the prototypical person in the class. To test this hypothesis, OLS estimation was used to run a series of linear

regressions of AL scores on the PPs. All linear regressions report robust standard errors that account for non-normality and that are corrected for possible confounding due to same-family membership in the sample. The Predicted Class Membership (PC) was also estimated, which assigns each participant to exactly one class.

Because our final analytic sample comes from the Biomarker Project (complete financial data n = 1,190)—which is a subsample of the MIDUS II longitudinal study (complete financial data n = 4,332)—the latent classes were fitted using the longitudinal study sample. This step allows us to improve generalizability—i.e., the certainty that our fitted classes are more representative of classes detectable in the national population. A final model with 5 latent classes was selected, and it fits the data very well; models with 2 to 4 latent classes did not fit the data as well as the 5-class model. Models with 6 and 7 classes had a similar fit quality as the 5-class model—that is, they did not add new meaning to the classification—but they generated sub-classes (as oppose to primary divisions in the data) that compromised parsimony. In essence, the 5-class model portrayed the most information with the least number of fitted classes.

To ensure that the 5 latent classes generated using the longitudinal study sample were representative of classes extant in our final analytic sample extracted from the Biomarker Project, a random sample from the longitudinal study was generated as well, with the same size as our final biomarker analytic sample (n = 1,190), and assessed the level of similarity between the PPs of the 5-class model and the PPs estimated using the biomarker sample and the random sample, respectively. No significant differences were detected, indicating that the biomarker sample participants were assigned PPs representative of classes that exist in the national population rather than classes affected by sample variability.

Results

Table 1 contains descriptive statistics for SES and demographic indicators in the Biomarker Project sample. As show, the sample is predominantly non-black (83.4%), with an average age of 54 years, and with a slightly greater proportion of females (56.5%). Levels of education are rather high, with 42.5% of the sample having a Bachelor's degree or higher. Despite educational attainment, HHPIRs are somewhat high, with 29.5% of the sample living around or below the poverty level (HHPIR ≤ 2.5). The three subjective financial indicators show somewhat uniform distributions, indicating considerable variation in their score ranges.

Descriptive statistics for individual biomarkers, high-risk and clinical cutpoints, and AL scores are reported in Table 2. High-risk quartile values in the sample for CRP, Resting SBP, BMI, WHR, and HDL cholesterol were comparable to customary clinical risk cutpoints. High-risk quartile values for glycosylated hemoglobin, fasting glucose, triglycerides, and LDL were moderate in size compared to typical clinical risk cutpoints. The mean AL score was 1.76 (SD = 1.03) from a potential range of 0 to 7, and an actual range of 0 to 5.03. Although the mean AL score ($\overline{AL}$=1.74) indicates a rather healthy sample, the score distribution shows significant variation (SD=1.04).

Table 3 provides the characterization of the 5 fitted latent classes using the longitudinal sample. For example, the subscale Race, which has two possible values (Non-Black and Black) shows that individuals in Class 2 have a .914 probability of being non-black (Pr(1) = .914), meaning that Class 2 highly discriminates between the racial categories. The same cannot be said, however, using the response values of the subscale; for instance, individuals in Classes 3 through 5 have a .865, .895, and .962 probability, respectively, of being non-black—in other words, being non-black is not exclusively descriptive of Class 2.

The top panel of Table 4 depicts specific combinations of traits that approximate the prototypical individual in each class in our analytic sample (n=1,190), and the bottom panel reports the predicted probability of class membership for the respective combinations. As shown, Class 1 individuals are likely to be black, relatively young females with exceptionally low SES. Class 2 individuals are likely to be non-black, relatively young males and females with low SES individuals (relatively high education, but low financial indicators). Class 3 individuals are likely to be older, non-black females of low-to-medium SES (low objective SES, yet slightly better than average subjective SES). Class 4 individuals are likely to be non-black, relatively young males with medium-level level SES (somewhat high HHPIR, yet average subjective SES). Class 5 individuals are likely to be older, non-black males with high SES. Put more succinctly, Class 1 represents disadvantaged blacks, especially females; Class 2 represents young, low-SES non-blacks; Class 3 represents older adults (retirees); Class 4 represents the middle class; and Class 5 represents the privileged class.

These findings suggest that social forces sort individuals into mutually exclusive classes structured in complex interactions between socioeconomic and demographic indicators. These latent classes, not revealed in traditional analyses of the additive, independent associations of SES and demographic indicators, are useful illustrations of the *social* combinatory bounds that exist between these indicators.

Table 4 also illustrates the heterogeneity of income and financial-situation returns to education. For instance, while the prototypical individual in Class 1 has both low education and a poor economic/financial situation, the prototypical individual in Class 2 has at least some college but also low SES. Notably, there is no overlap of race categorizations across the classes. Being black and poor is not the same as being non-black and poor.

SES differences between prototypical individuals in classes 2 and 4 exemplify heterogeneity in economic/financial standing on the basis of gender. Probabilities of class membership increase for young non-blacks in both classes, but while the probability of membership increases about equally by gender in class 2, it increases only for males in class 4. Significantly, the prototypical individual in class 4 is likely to have a higher economic and financial standing despite having a slightly lower level of education than the prototypical individual in class 2.

These two examples corroborate that economic and financial-situation returns to education vary along racial and gender lines, and that these circumstances of disadvantage follow mutually exclusive processes of social differentiation. Privileged class 5 reinforces this

notion: the prototypical individual also tends to be a non-black male, albeit older than the class 4 prototypical non-black male. Females are exposed to greater stratification constraints than males. Being a female increases the probability of belonging to classes 1 through 3, which manifest lower levels of SES, while being a non-black male increases the probability of belonging to higher-SES classes 4 and 5. This pattern coincides with the traditional view of additive effects of race and gender on SES. However, probing these relationships reveal other interactions at work. Females' social class stratification appears to be conditional on their race in class 1, on a lack of positive economic and financial-situation returns to education in class 2, and on longevity in class 3.

Table 5 shows the results of the regression models. Model 1 illustrates the direct relationship between demographic indicators and allostatic load. As expected, there are substantial and statistically significant differences in AL between races and genders and among age groups. Model 2 shows the results of testing for relationships between PPs and AL as demographic indicators were progressively added to Models 3 through 5, and as they were added in combinations to Models 6 through 9.

The results of Model 9 show statistically significant differences in AL associated with the probability of belonging to classes 1 through 4 in comparison to privileged class 5. Predicted average AL scores from Model 9 are 2.08, 1.82, 1.84, 1.72, and 1.44 for classes 1 through 5, respectively. These results illustrate that blacks, and especially black females (class 1), constitute a distinctively separate class in society with the worst socioeconomic standing and the worst health.

Since LCA clusters individuals for the most part using socially relevant variance, further controls for age, gender, and race help to remove bias from estimated PP coefficients due to within-class variance unrelated to social stratification. Accordingly, Model 9 shows that the difference in AL between classes 5 and 1 is substantial considering that it equals half of the difference in AL scores between age groups 75 to 84 and 34 to 44 years ($\beta = 1.30$) that is attributable to aging-variance unrelated to social stratification. In other words, their difference in AL corresponds to about 20 years of non-stratification aging, on average. Another way to describe the health disparity between these classes is by looking at the differences between classes 5 and 4: The AL difference between classes 5 and 1 ($\beta = .64$) is more than double the AL difference between classes 5 and 4 ($\beta = .28$).

Table 5 also shows that, apart from the contributions that each demographic indicator brings to the configuration of the classes, AL differences exist independent from social stratification—most notably between age groups and to a lesser extent between the genders, but not between the races ($\beta = .07$, SE = .09). This finding suggests that the poor health status of blacks is better characterized by the econo-demographic composition of class 1—or by social stratification processes—than by other independent factors. Racial disparities in allostatic load are to an important degree socially constructed.

It is worth noting that predicted AL levels for classes 2, 3, and to some extent for class 4, are similar in spite of the distinct configuration of the classes. This indicates that different social stratification experiences can lead to similar levels of physiological dysregulation. This is a

crucial piece of the story that gets lost in traditional additive models of SES-gradients. Whether diverse environmental stressors/stimuli may take similar physiological tolls is a question that needs further exploration. These results imply, as well, that policymakers attempting to ameliorate negative health outcomes in the population at large should understand the underlying social stratification forces to which individuals are exposed to in the first place.

Discussion

Most of social stratification research focuses on how SES differs on the basis of age, race, and gender rather than how these indicators *jointly* conform to actual social structures. This traditional model of analysis has been widely replicated in studies of SES differences in health, including health as measured by allostatic load. Departing from, yet complementing to, this approach, this study shows that social stratification—an important source of unobservable heterogeneity—operates by non-randomly sorting individuals into a set of mutually exclusive econo-demographic classes. Results show that variation in multi-system physiological dysregulation, as measured by allostatic load, is strongly associated with social class membership.

These findings extend current research on SES-gradients and demographic differences in health because social stratification encompasses dimensions of social differentiation beyond SES, including historical, legal, and political processes that act selectively upon the demographic profile of individuals. Social stratification influences daily life experiences by delineating life opportunities, quality of life, and individual's relative power and privileges (Williams and Collins, 1995; Haas, 2006; Rodriguez, Bound and Geronimus, 2013; Rodriguez, Bound and Geronimus, 2014; Rodriguez *et al.*, 2015; Solís *et al.*, 2015; Cottrell *et al.*, 2018; Rodriguez, 2018). In this study, the posterior probability (PP) of *group* membership results from the interactive association between socioeconomic and demographic indicators at the *individual* level, allowing for the simultaneous integration of these two commonly separate aspects of social stratification. Accordingly, the resulting classes represent the complex, interactive socioeconomic circumstances of individuals—a key component of health variation scarcely captured through additive modeling of SES-gradients.

Findings are consistent with previous research on SES-gradients in health (Merkin *et al.*, 2009; Gruenewald et al., 2012; Hudson *et al.*, 2016) and on the association between SES and allostatic load in specific (Gruenewald *et al.*, 2009; Bird *et al.*, 2010; Slopen *et al.*, 2010; Geronimus *et al.*, 2015). For instance, privileged non-blacks (class 5) showed the lowest AL while low-SES non-blacks (class 2) showed a much higher level of AL (O'Brien, 2012). Findings also show strong evidence supporting previous findings that low SES is concentrated among blacks, females, and older adults (Geronimus et al., 2006; Juster, McEwen and Lupien, 2010; Upchurch *et al.*, 2015). Results suggest that variations in AL are linked to underlying socially-related interactions between SES and demographic indicators beyond non-social variation found in demographic indicators.

The AL index was operationalized using 23 biomarkers that measure both primary mediators of stress and its secondary effects (McEwen and Seeman, 1999). Using AL scores free of social and cultural components allowed to differentiate the variations in AL due to social stratification from those explicitly due to race, gender, or age. Accordingly, the findings indicate that AL variations in age are influenced by both social and non-social sources of variation, as are AL variations in gender—albeit to a lesser extent. AL differences between blacks and non-blacks, however, showed a high degree of association with the indicator composition of the classes, suggesting that racial disparities in AL are mostly determined by social stratification. In the analyses, blacks tended to inhabit an isolated social class with the lowest socioeconomic standing and the worst health. This indicates that racial variation in health is closely related to the degree to which the individual characteristics of blacks are grouped by forces of social differentiation, and that this process is neither necessarily nor entirely related to the independent variation of their individual characteristics. That the most disadvantaged class tends to be inhabited practically by blacks alone, reaffirms their unique historical and socio-political circumstances.

The findings also concur with research on “weathering” among black populations, which posits that blacks in general, and black females in particular, experience earlier and more significant multi-system physiological dysregulation than their white counterparts (Geronimus, 1992). Results showing that high AL scores were concentrated among black females also supports the “double jeopardy” effect hypothesis, or the view that black women suffer the dual negative effects of gender and racial discrimination (Geronimus et al., 2010).

The associations found between AL and the indicator composition of the 5 social classes represent the physiological expressions of social stratification. Since the LCA methodological approach permitted to disentangle the unobservable heterogeneity extant in the socioeconomic and demographic characteristics of individuals, the composition of the resulting classes may well represent the distribution of and access to health-relevant resources and other health-related circumstances beyond observable associations between these indicators. In this case, the value of the LCA approach lies in the exhibition of how these indicators “stick together” and how their latent interactions are associated with health —which needed not to be true. Consequently, the variation in AL found across classes may be less attributable to individual characteristics than to the structural forces that sort individuals into mutually exclusive classes in the first place.

Taken together, these findings present evidence that disparities in health emerge from group-differentiation processes to the degree that individuals are differentially exposed to the ecological, political, social, economic and historical contexts in which social stratification is ingrained. Considering that social stratification is to an important extent tempered by government-mediated processes, the diminishment of health disparities will require a deeper understanding of the historically entrenched institutional structures that outlive the individual and that prescribe and execute current policy. This study posits that these historical and existing aspects of the legal and political environments constitute the underlying social class-generating mechanism—more so than individual characteristics. Given that health policies and programs emanate from said legal and political environments, findings indicate that interventions should target the structural conditions that expose

different subgroups to different stress risks (Thoits, 2010). Results highlight the importance of developing innate political and legal mechanisms for self-monitoring and self-correcting the production and elimination of conditions that induce stress among vulnerable populations.

There are some limitations of this study worth mentioning. As noted earlier, the complete financial data in the Biomarker Project was limited to 1,190 participants, which could have led to some selection bias. Our analytic sample is also skewed toward higher socioeconomic status; however, there is enough variation in the sample to effectively identify five mutually exclusive classes, as demonstrated by our latent class analysis. A third limitation is that, although the overall characteristics of black participants in our sample resemble those in the general population, a majority of these back participants come from Milwaukee, reminding us that the statistical generalizability of findings should be done with caution. Even though our findings reinforce and expand research on socioeconomic and racial disparities in health, another limitation is that the uncovered relationships between social stratification and allostatic load are cross-sectional, which prevents us from making causal claims. That our analysis is cross-sectional, also precludes us to control for allostatic load at baseline, and therefore to understand how the evolution of social stratification at different life stages affect physiological pathways according to individual characteristics.

Results from our study confirm that the social stratification processes that distribute the psychosocial environment in which we grow up and grow old influence how we age, our health and functioning in later life, and our life expectancy. The deleterious consequences of social differentiation forces are thought to modify the dysregulation of the physiological systems associated with how we handle the challenges and stresses of life. Individuals exposed to greater life stresses therefore have more dysregulation; allostatic load is the biological summary of dysregulation across multiple physiological systems, and as such is greater in the groups that are more likely to experience life stresses intrinsically related to the degree to which certain individuals—as a group—are forcedly put in a disadvantaged status in society. The hierarchy/rank-ordering of allostatic load across the latent classes uncovered in this study is consistent with the hierarchy/ranking of social privilege in the United States. That there is persistent association of allostatic load with age even within the latent classes implies that in addition to the life stresses related to the social ranking/hierarchy (as captured by the classes), there is also an accumulation of effects of stress over time (with increasing age). That no substantive residual gender and racial differences in allostatic load are detected once the classes are accounted for, offers supporting evidence that gender and racial differences in allostatic load are mostly socially-constructed through social stratification processes.

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Table 1.

Descriptive statistics for socioeconomic and demographic indicators

	%	Mean	SD
Race			
Black	17.6		
Non-Black	82.4		
Gender			
Female	56.5		
Male	43.5		
Age		54	11.7
65 yrs +	21.1		
Age (by decade)			
34 to 44 yrs	23.2		
45 to 54 yrs	29.3		
55 to 64 yrs	26.5		
65 to 74 yrs	14.5		
75 to 84 yrs	6.6		
Education			
Less than high school (<HS) degree	5.6		
High school (HS) or General Equivalence Diploma (GED)	22.3		
Some college (SC) or Associate's (AA) degree	29.6		
Bachelor's (BA/BS) degree	20.5		
Graduate School and above (Grad +)	22.0		
HHPIR			
< 2.5	29.5		
2.5/5.0	32.0		
5.0/7.5	19.0		
7.5/10.0	9.9		
> 10.0	9.6		
Finances			
Worst	34.2		
Average	33.8		
Best	32.0		
Need			
Worst	23.8		
Average	49.2		
Best	27.0		
Bills			
Worst	30.5		
Average	33.4		
Best	36.1		

Table 2.

Descriptive Statistics for Individual Biomarkers of System Subscales, and Multi-system Physiological Risk Scale (Allostatic Load, n=1,190)

	N	Mean	SD	High-risk cutpoint	Clinical cutpoint
Sympathetic Nervous System (SNS) *					
Urine Epinephrine (μg/g creatine)	1168	1.96	1.26	≥2.46	
Urine Norepinephrine (μg/g creatine)	1176	27.24	12.92	≥32.96	
Parasympathetic Nervous System (PNS) **					
SDRR (msec)	1096	35.18	17.50	≤23.72	
RMSSD	1096	22.63	17.87	≤12.14	
Low frequency spectral power	1096	414.30	589.87	≤115.03	
High frequency spectral power	1096	315.71	782.94	≤58.85	
Hypothalamic Pituitary Adrenal Axis (HPA) *					
Urine Cortisol (μg/g creatine)	1185	15.37	15.43	≥19.00	
Blood DHEA-S (μg/dL)	1186	105.43	77.26	≤51.00	
Inflammation ***					
CRP (mg/L)	1179	2.93	4.23	≥3.66	>3
IL6 (pg/mL)	1190	3.03	2.99	≥3.47	
Fibrinogen (mg/dL)	1181	348.71	88.85	≥399.00	
sE-Selectin (ng/Ml)	1189	43.35	22.78	≥51.89	
sICAM-1 (ng/Ml)	1189	288.32	115.89	≥335.45	
Cardiovascular **					
Resting SBP (mmHg)	1190	131.38	18.17	≥143.00	≥140 (≥120)
Resting DBP (mmHg)	1190	75.66	10.69	≥82.00	≥90 (≥80)
Resting heart rate (bpm)	1189	70.94	11.07	≥78.00	>90 (>80)
Metabolic (Glucose Metabolism) **					
Glycosylated hemoglobin (HbA_{1c})	1181	6.09	1.17	≥6.24	≥7 (>6.4)
Fasting glucose (mg/dL)	1183	101.88	26.80	≥105	≥126 (>100)
Insulin resistance (HOMA-IR) +	1181	3.48	3.35	≥4.35	
Metabolic (Lipids Metabolism) ***					
BMI	1190	29.76	6.64	≥32.92	≥25, ≥30
WHR	1189	.90	.10	≥.97	>1 (>.9)
Triglycerides (mg/dL)	1188	128.19	79.51	≥155.25	≥200 (≥150)
HDL Cholesterol (mg/dL)	1188	55.40	18.02	≤42.38	<40
Cholesterol (mg/dL)	1188	105.59	35.30	≥127.28	≥160 (≥130)
Multi-system Physiological Risk Scale (AL)	1190	1.74	1.04		

* Scores computed for individuals with at least 1 item.

** Scores computed for individuals with at least 2 items.

*** Scores computed for individuals with at least 3 items.

+ *Note*: [HOMA-IR = fasting insulin (uIU/mL) * fasting glucose (mg/dL) * .00247] (Matthews et al., 1985).

Table 3.

LCA results for final 5-classes model (longitudinal sample)

	Pr(1)*	Pr(2)	Pr(3)	Pr(4)	Pr(5)
Race	*Non-Black*	*Black*			
Class 1	.435	.565			
Class 2	.914	.086			
Class 3	.865	.135			
Class 4	.895	.105			
Class 5	.962	.038			
Gender	*Male*	*Female*			
Class 1	.293	.707			
Class 2	.435	.565			
Class 3	.387	.613			
Class 4	.483	.517			
Class 5	.530	.470			
Age	*34–44 yrs*	*45–54 yrs*	*55–64 yrs*	*65–74 yrs*	*75–84 yrs*
Class 1	.303	.222	.219	.158	.098
Class 2	.267	.357	.256	.084	.036
Class 3	.077	.078	.235	.379	.232
Class 4	.300	.344	.233	.097	.026
Class 5	.160	.286	.324	.168	.063
Education	*<HS*	*HS/GED*	*SC/AA*	*BA/BS*	*Grad +*
Class 1	.286	.400	.286	.007	.024
Class 2	.019	.310	.305	.208	.157
Class 3	.125	.457	.310	.074	.035
Class 4	.012	.232	.328	.233	.195
Class 5	.008	.115	.222	.288	.367
HHPIR	*≤ 2.5*	*2.5/5.0*	*5.0/7.5*	*7.5/1.0*	*>1.0*
Class 1	.679	.204	.078	.031	.009
Class 2	.115	.373	.261	.156	.096
Class 3	.331	.347	.205	.114	.003
Class 4	.038	.152	.298	.302	.210
Class 5	.045	.046	.122	.271	.515
Financial	*Worst*	*Average*	*Best*		
Class 1	.784	.178	.039		
Class 2	.800	.196	.004		
Class 3	.171	.294	.535		
Class 4	.209	.644	.147		
Class 5	.008	.211	.781		

	Pr(1)*	Pr(2)	Pr(3)	Pr(4)	Pr(5)
	Pr(1)	**Pr(2)**	**Pr(3)**	**Pr(4)**	**Pr(5)**
Need	*Worst*	*Average*	*Best*		
Class 1	.752	.248	.000		
Class 2	.622	.378	.000		
Class 3	.010	.777	.214		
Class 4	.071	.844	.085		
Class 5	.005	.235	.760		
Bills	*Worst*	*Average*	*Best*		
Class 1	.797	.131	.072		
Class 2	.882	.111	.007		
Class 3	.021	.425	.554		
Class 4	.135	.708	.157		
Class 5	.004	.145	.851		
Observations		4332			
Estimated parameters		104			
Maximum log-likelihood		−35413.85			
AIC		71035.71			
BIC		71698.58			
Likelihood rat./dev. Stat)		659.03			
Chi-square		20025.86			

*Conditional item response probabilities (column (Pr)) by outcome variable for each class (row)

Table 4.

Predicted probability of class membership for prototypical individuals (n=1,190)

	Descriptive characteristics				
Race	Black	Non-Black	Non-Black	Non-Black	Non-Black
Gender	Female	Male/Female	Female	Male	Male
Age	34–55 yrs	34–55 yrs	65–84 yrs	34–55 yrs	55–64 yrs
Education	< HS	SC-BA	HS	SC	≥BA
HHPIR	≤ 2.5	2.5/5.0	≤5.0	5.0/7.5	≥7.5
Financial	Worst	Worst	Average/Best	Average	Best
Need	Worst	Worst	Average	Average	Best
Bills	Worst	Worst	Average	Average	Best
	Predicted probabilities of class membership				
Pr.Class 1	**.998**	.093	.001	.000	.000
Pr.Class 2	.002	**.903**	.000	.008	.000
Pr.Class 3	.000	.000	**.966**	.016	.000
Pr.Class 4	.000	.004	.030	**.972**	.001
Pr.Class 5	.000	.000	.002	.003	**.999**

Table 5.

Linear regression models parameter estimates

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Pr. Class 1 (Blacks)		.55**	.55**	.53**	.73**	.52**	.67**	.70**	.64**
		(.11)	(.14)	(.11)	(.11)	(.14)	(.13)	(.11)	(.13)
Pr. Class 2 (Low Class)		.24*	.24*	.24*	.38**	.24*	.39**	.37**	.38**
		(.10)	(.10)	(.10)	(.10)	(.10)	(.10)	(.10)	(.10)
Pr. Class 3 (Older adults)		.92**	.92**	.91**	.43**	.91**	.42**	.41**	.40**
		(.13)	(.13)	(.13)	(.13)	(.13)	(.14)	(.13)	(.14)
Pr. Class 4 (Middle Class)		.09	.09	.09	.28**	.09	.28**	.28**	.28**
		(.09)	(.09)	(.09)	(.09)	(.09)	(.09)	(.09)	(.09)
Race (1=Non-Black, 2=Black)	.31**		.01			.01	.07		.07
	(.08)		(.10)			(.10)	(.09)		(.09)
Gender (1=Male, 2=Female)	.14*			.08		.08		.10†	.10†
	(.06)			(.06)		(.06)		(.06)	(.06)
Age (45–54 yrs)	.33**				.37**		.37**	.37**	.37**
	(.08)				(.08)		(.08)	(.08)	(.08)
Age (55–64 yrs)	.71**				.77**		.77**	.77**	.77**
	(.08)				(.08)		(.08)	(.08)	(.08)
Age (65–74 yrs)	.84**				.87**		.87**	.87**	.87**
	(.10)				(.10)		(.10)	(.10)	(.10)
Age (75–84 yrs)	1.33**				1.27**		1.28**	1.29**	1.30**
	(.13)				(.14)		(.14)	(.14)	(.14)
Constant	.65**	1.47**	1.46**	1.36**	.91**	1.35**	.84**	.76**	.68**
	(.14)	(.06)	(.12)	(.11)	(.08)	(.15)	(.12)	(.12)	(.15)
Observations	1,190	1,190	1,190	1,190	1,190	1,190	1,190	1,190	1,190
R-squared	.138	.057	.057	.058	.159	.058	.160	.162	.162

Note: Robust standard errors in parentheses. Statistical significance code:

** p<.01,

* p<.05,

† p<.1.

The class of reference is Class 5 (the privileged class).