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Temporal Changes in Allostatic Load Patterns by Age, Race/Ethnicity, and Gender Among the US Adult Population; 1988-2018

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Abstract

The objective of this study is to provide an assessment of allostatic load (AL) burden among US adults across race/ethnicity, gender, and age groups over a 30-year time period. We analyzed data from 50,671 participants of the National Health and Nutrition Examination Survey (NHANES) years 1988 through 2018. AL score was defined as the sum total for abnormal measures of the following components: serum albumin, body mass index, serum C – reactive protein, serum creatinine, diastolic blood pressure, glycated hemoglobin, systolic blood pressure, total cholesterol, and serum triglycerides. We performed modified Poisson regression to estimate the adjusted Relative Risks (aRRs) of allostatic load, and generalized linear models to determine adjusted mean differences accounting for NHANES sampling weights. Among US adults aged 18 or older, the prevalence of high AL increased by more than 45% from 1988 – 1991 to 2015-2018, from 33.5% to 48.6%. By the latest period, 2015 – 2018, Non-Hispanic Black women (aRR: 1.292; 95% CI: 1.290 - 1.293) and Latina women (aRR: 1.266; 95% CI: 1.265 – 1.267) had higher risks of AL than non-Hispanic White women. Similar trends were observed among men. Age-adjusted mean AL score among NH-Black and Latino adults was higher than for NH-Whites of up to a decade older regardless of gender. From 1988 through 2018, Adults aged 40 years old and older had over 2-fold increased risks of high AL when compared to adults 18-29 years old. After 30-years of collective data, racial disparities in allostatic load persist for NH-Black and Latino adults.

Keywords

life-course; cumulative stress; psychosocial stress; race; disparities

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INTRODUCTION

Decades of research into the disproportionally higher morbidity and mortality burden among racial minorities, e.g. Non-Hispanic (NH) Blacks and Latinos, relative to NH-White Americans have failed to identify a satisfactory model to explain the disparity or produce effective strategies to address the problem[1,2]. In fact, for many conditions, disparities have widened in recent years[3–8]. The fundamental causes of health disparities in the United States (US) are multifactorial, multilevel, and multigenerational; socially marginalized groups endure prolonged psychosocial and physiological challenges that increase their risk of disease, lead to early onset of disease, and accelerate cellular aging[3,9–18].

The biological incorporation of the social and material environment in which humans live is termed 'embodiment' and serves as a model for re-conceptualizing health disparities not only as the result of differential distribution of health-related risk factors, but as the result of historically contingent and racially patterned exposures, leading to altered susceptibility to exogenous factors[11,15,16]. For instance, social determinants of health such as income, education and access to healthcare, in addition to exposure to racial discrimination, may directly or indirectly influence health related risk factors such as diet, exercise, smoking, obesity, psychosocial stress and comorbidities[3,19,20]. The biological consequences of such exposures include dysregulated immune, cardiovascular and metabolic systems, which are typically tightly regulated via the hypothalamic-pituitary-adrenal (HPA) axis in a state of allostasis[21], leading to increased risk of complex diseases.

Allostatic load (AL) represents a measure of biological wear and tear due to chronic over-activation of biological systems[1,22,23]. While allostatic load attempts to characterize the accelerated ageing of biological systems, it is more defined as the cost or the price the organ system pays for an overactive or inefficiently managed stressor [24]. Seeman and colleagues composed an AL score based on 10 biological parameters with the purpose of comprehensively characterizing physiological burden on the human body[21,25,26]. Previously, AL has been used to predict the morbidity of cardiovascular disease (CVD), diabetes mellitus, high BMI, cognitive function, and overall mortality[23,27,28]. Socioeconomic factors such as higher income or educational status may mitigate AL burden via benefits of resources and social capital, thus reducing biological stress[29,30]. In addition, male/female sex differences in AL burden have been observed through varying mechanisms including social integration, occupation, socioeconomic position, support groups, and higher self-perceived masculinity[31–34]. Furthermore, prior research on AL among racial/ethnic minority groups has shown elevated levels of AL among NH-Blacks compared to NH-Whites and among individuals perceiving racial or social discrimination[35–43]. Disparities in the burden of AL, therefore, provide a useful global measure of embodiment such that higher scores indicate greater exposure to adversity.

While recent studies have examined trends in AL [44], there is limited knowledge on comparisons in the prevalence of AL over the past few decades. The goal of the present analysis is to provide the most robust assessment of AL burden among a representative sample of US adults and to understand the differences attributed to race/ethnicity, gender, and age groups across a 30-year time period from 1988 through 2018. We hypothesize that

racial/ethnic minorities will have higher burden of AL, within gender subgroups, and these disparities will persist throughout age groups.

METHODS

Study Design and Participants:

We performed analyses using data from a representative sample of non-institutionalized US residents. The National Health and Nutrition Examination Survey (NHANES) is a nationally representative sample of US adults, where persons aged 60 and older, Latinos and NH-Blacks are oversampled, and weighted analysis generates generalizable estimates [45]. The NHANES weighted sample is considered to be representative of the U.S. civilian non-institutionalized population [46]. We examined trends in allostatic load over time by establishing multiple time periods; 1988 – 1991, 1991 – 1994, 1999 – 2002, 2003 – 2006, 2007 – 2010, 2011-2014, and 2015-2018 [47]. NHANES includes demographic, socioeconomic, dietary, and health-related questionnaires, and includes clinical measures of blood pressure, fasting blood glucose, triglycerides and HDL cholesterol, in addition to self-reported medication use for health conditions. We performed analysis among NHANES participants with data on biomarkers and within the fasting subsample. This analysis included all NH White, NH-Black, Latinos participants, as well as those who identified as mixed raced or other race, ages 18 and older: a total of 50,671 participants over the 30-year study period for the main analysis (Figure 1). The Institutional Review Boards considered this study exempt from review because of the use of publicly available, de-identified data.

Allostatic Load Definition:

AL has been defined using varying configurations, although most incorporate biomarker measures from three different categories of physiologic functioning: cardiovascular, metabolic, and immune systems[48]. While there is no consensus definition, we elected to define AL using the Geronimus et al (2006) and Mays et al (2018) taxonomies [38,49]. To determine the high-risk thresholds for each AL component, we examined the distribution of each component among the entire study sample with complete biomarker data. High-risk thresholds were determined by either being above the 75th percentile for body mass index (BMI), C – reactive protein (CRP), diastolic blood pressure (DBP), glycated hemoglobin, systolic blood pressure (SBP), total cholesterol, and serum triglycerides; or below the 25th percentile for serum albumin and serum creatinine. Therefore, each NHANES participant was scored as either 1 (high-risk) or 0 (low-risk) based on gender-specific cutoffs for each component (Supplemental Table 1**)**. We calculated total AL score by summing the individual components, and this score ranged from 0 to 9. We further categorized participants with AL score greater or equal to 3 as having high allostatic load [48,49].

Sociodemographic Characteristics:

To assess socio-demographic differences in the prevalence and trends of AL, we evaluated differences by age, race/ethnicity, education, and poverty to income ratio (PIR) (adjusted for inflation),. The NHANES education variable was categorized into: 1) less than high school education; 2) high school graduate/GED/ or equivalent; 3) some college; 4) college graduate or above; and 5) unknown/refused to answer. Poverty income ratio (PIR) was calculated

as the ratio of total family income to poverty threshold values (in dollars). Persons who reported having had no income were assigned a zero value for PIR. PIR values less than 1 are considered below the official poverty line, whereas PIR values greater than 1 are above the poverty level [50].

Health Behaviors and Comorbidities:

We evaluated health behaviors that may influence AL score in analysis, including self-reported smoking status. Participants that had not smoked 100 cigarettes in lifetime were categorized as never smokers, while participants with at least 100 cigarettes smoked in lifetime but no current smoke use were categorized as past smokers. Participants with at least 100 lifetime cigarettes used and current smoking use were categorized as current smokers[51]. We included any self-reported response to a physician-diagnosed history of cancer, as well as self-reported congestive heart failure and heart attack as comorbidities.

Statistical Analysis:

Analyses were performed using NHANES generated sampling statistical strata, clusters, and weights as designated and described in detail in the NHANES methodology handbook [45]. NHANES only measures biomarkers among a random sample of participants each survey period, and in turn created subsample weights to account for the probability of being selected into the subsample component, and additional non-response bias. As a result, our analysis focuses on all participants with the fasting subsample weight as we followed the National Center for Health Statistics guidelines for NHANES data, and applied the “least common denominator” approach when deciding the appropriate statistical weights [45]. With this approach, we checked the variables of interest in our study and selected the variable that was collected on the smallest number of persons (“least common denominator”) that were our biomarkers: C-reactive protein, albumin, creatinine, glycated hemoglobin, and triglycerides. When a sample is weighted in NHANES it is considered to be representative of the U.S. civilian non-institutionalized population. Only poverty-to-income ratio had missing values, and we categorized those missing as so when performing regression analyses.

Categorical variables were presented as weighted row percentages and continuous variables as mean and associated 95% confidence intervals. The primary outcome of interest was the prevalence of high AL and mean AL score, overall and by interaction terms containing the gender and race/ethnicity variables for the subgroup analyses. For all time periods, the prevalence of high AL and mean AL score stratified by gender-race/ethnicity were estimated. We performed modified Poisson regression models for estimating risk of high AL, stratified by gender-race/ethnicity interaction terms and adjusting for potential confounders including education, age groups, PIR, smoking status, any history of cancer, congestive heart failure, or heart attack[52]. Weighted generalized linear models associating the mean AL score by gender-race/ethnicity additionally adjusted for possible aforementioned confounders were also conducted. Estimates derived from modified Poisson regression are presented as relative risks (RRs) and associated 95% confidence intervals (CIs), and estimates derived from generalized linear models are presented as mean estimates and differences (from referent group) and associated 95% CIs.

In sensitivity analyses, we conducted regression analysis categorizing participants who reported current use of medications for hypertension, hypercholesterolemia, or pre-diabetes as high-risk for the corresponding AL biomarker [38]. Again, NHANES participants were scored one point (high-risk) for the respective condition if they indicated using medications for hypertension (we gave 0.5 points for both systolic and diastolic blood pressure if participant was on antihypertensive, summing to one total point for this medication), hypercholesterolemia, or pre-diabetes[38]. Prior research has assumed that degradation has already occurred among individuals taking medications, and given our analysis covers multiple time periods among multiple age groups, similar analytic adjustments were made. All statistical analyses were performed using SAS (version 9.4, SAS Institute, Inc.).

RESULTS

A total of 50,671 NHANES participants between 1988 and 2018 were included in this analysis (Table 1). Table 1 displays the demographic and personal level characteristics by NHANES periods. Over the 30-year observation period, there was an overall 35% increase in mean AL score from 1991 to 2018. The mean AL score was lowest in 1988-1991; remained steady throughout 1991-1994 (mean AL: 2.38), 1999-2002 (mean AL: 2.29), 2003-2006 (mean AL: 2.17), and 2007-2010 (mean AL: 2.44); and peaked in the latest period 2015-2018 (mean AL: 2.62). The distribution of age-adjusted mean AL score by race/ethnicity and time period among males and females is presented in Figure 2. Age-adjusted mean AL score increased among both NH-White males (from 2.18 to 2.55) and females (from 1.97 to 2.44) from 1988-1991 to 2015-2018, respectively. We observed the same elevated trend of age-adjusted mean AL scores among NH-Black males (from 2.75 to 2.86) and females (from 2.69 to 3.04) in the same time periods. Age-adjusted mean AL score was also higher among Latina females compared to males, and increased from 2.48 to 3.10. The age-adjusted mean AL score by race/ethnicity and time period stratified by age groups are presented in Figure 3. While mean AL score increased over time in all racial/ethnic groups, there were clear racial/ethnic differences observed as early as age 18-29 years for both men and women that persisted through ages 70+ years. The distributions of each individual AL component and mean AL score across time periods are presented in Supplemental Tables 1–2. Supplemental Table 2 shows allostatic load components such as obesity (BMI), chronic inflammation (C-reactive protein), creatinine, and elevated glycated hemoglobin all increased over time.

In multivariable adjusted models, NH-Black males were at 33% increased risk of high allostatic load (AL score greater or equal to 3) compared to NH-White males in 1988 – 1991 (aRR: 1.332, 95% CI: 1.331 – 1.334; Table 2), and nearly 11% increased risk (aRR: 1.106; 95% CI: 1.105 – 1.107) in 2015 – 2018. Latino males also had significantly higher risks of high AL compared to NH-White males in 1988 – 1991 (aRR: 1.228; 95% CI: 1.225 – 1.230), 2003 – 2006 (aRR: 1.297, 95% CI: 1.295 – 1.298) and 2015-2018 (aRR: 1.259; 95% CI: 1.258 - 1.260). Other & Mixed race males were at significantly increased risks of high AL in 1999-2002 (aRR: 1.527; 95% CI: 1.524 – 1.529), 2007–2010 (aRR: 1.146; 95% CI: 1.145 – 1.147), and 2015-2018 (aRR: 1.120; 95% CI: 1.119 – 1.121). Among females, NH-Blacks had higher risks for high AL compared to NH-Whites regardless of time period, with lowest risk observed at 1999 – 2002 (aRR: 1.198; 95% CI: 1.197 – 1.200) and peaking

at nearly 46% higher (aRR: 1.456; 95% CI: 1.455 – 1.458) in 2003 – 2006.. Latina females were also at increased risk of high AL compared to NH-White females regardless of time period, ranging from nearly 32% higher (aRR: 1.316; 95% CI: 1.313 – 1.318) in 1988 – 1991 to only 20% higher (aRR: 1.206; 95% CI: 1.205 – 1.207) in 2003 – 2006, to over 26% higher in 2015-2018 (aRR: 1.266; 95% CI: 1.265 – 1.267). In earliest period, 1988 - 1991, we observed that older age (50 and older compared to those aged 18 – 29), was associated with up to 7-fold higher risk of allostatic load (aRRs: 7.109 for 50 – 59 year olds; 8.052 for 60 – 69 year olds; and 8.196 for 70+ year olds). However, this increased risk slightly attenuated over time and by the 2015-2018 period, and older age (50 and older compared to those aged 18 – 29) was only associated with a 2.5-fold increased risk of high allostatic load (aRRs: 2.546 for 50 – 59 year olds; 2.545 for 60 – 69 year olds; and 2.539 for 70+ year olds). Similar trends were observed for absolute measures of mean AL scores across time periods (Table 3). Sensitivity analysis results when accounting for medications for hypertension, hypercholesterolemia, and pre-diabetes are presented in Supplemental tables (Supplemental Table 3 & 4**)**; results mirrored the main analysis for the effects seen in race-gender groups, and age, although there were larger observed differences.

DISCUSSION

In a diverse, nationally representative sample of US adults, we observed marked disparities in the burden of AL across race/ethnicity, gender and age groups over a 30-year period. The burden of high AL and mean AL score increased significantly over time; a relative increase of 45.1% and 35.1% from 1988 through 2018, respectively. By 2015-2018, nearly than one in two US adults met criteria for high AL, and mean AL score was highest among both NH-Black and Latina females, followed by Latino males, Other & Mixed race females, and finally NH-Black males. Racial differences in AL score were evident as early as 18 – 29 years old and persisted across age groups over the entire period examined. For instance, age-adjusted mean AL score among NH-Black and Latino males ages 30-39 years was higher than for NH-White males ages 40 – 49, and higher among NH-Black and Latino males ages 40 – 49 than among NH-White males ages 50 – 59 years. Similar patterns were observed among females; age-adjusted AL score among NH-Black and Latino females at age 40 – 49 years were significantly higher than for NH-White females at ages 50 – 59 years.

Higher AL has been associated with increased mortality risk in numerous studies[25,53–57]; there was an 88% (HR: 1.88, 95% CI: 1.56, 2.26) increased risk of all-cause mortality[53], and 55% (HR: 1.55, 95% CI: 1.04, 2.32) increased risk of cardiovascular or diabetes-related mortality observed in previous studies, with stronger associations among NH-Blacks [53]. Other studies have also documented a role of AL in health outcomes globally, including China [58], Denmark [59], and Germany [60], highlighting a critical role for AL in health outcomes across diverse population groups. Similar to our findings, other US studies have documented higher mean AL among NH-Blacks compared with Whites starting in early adulthood; Geronimus et al. reported that NH-Black women were consistently more likely than NH-Black men to have high AL score, and poor Whites were less likely than non-poor NH-Blacks to have high AL score[61]. Our analysis updates the previous results of nationally generalizable NHANES by including Latino adults in the US, a demographic group that

has experienced a significant increase from about 5% of the US population in 1988-1992 to almost 14% in 2007-2010. Similar to NH-Blacks, AL score among Latinos has increased over time and is consistently higher among females compared with males. We also observed that after adjusting for race/ethnicity, age and comorbidity status, there was significantly increased odds of high AL score among those with higher versus lower poverty-to-income ratio, and among those with lower versus higher education.

There are several proposed mechanisms that explain the association of high AL and poorer health outcomes. The 'weathering' hypothesis, proposed by Geronimus et al in 1992[61], describes the cumulative impact of socio-economic adversity and political marginalization on individual health, leading to early and disproportionate physiological deterioration. Persistent high-coping effort due to chronic stress has also been shown to result in cumulative wear and tear on the body's adaptive biological systems. This stress-related physiologic burden was conceptualized as AL by Seeman et al (1997) and McEwen & Seeman (1999) and has been well examined as a measure of weathering given the inclusion of subclinical measures of stress response across several biological systems [21,26]. In addition to socio-economic adversity, exposure to racial discrimination over the life-course such as de-facto racial segregation via Jim Crow laws, other forms of adversity such as conflict and social instability, can also induce chronic stress, leading to pathophysiology in cardiovascular, metabolic and immune systems, key components of AL. Consistent and significant associations have also been shown between exposure to structural racism, racial and economic segregation and biological dysregulation – specifically cardiovascular (e.g., hypertension[62–66]); metabolic (e.g., obesity[67,68], diabetes[69,70], metabolic syndrome[71,72]); and inflammatory (e.g., CRP, TNF-alpha and IL-6[19,20,73–78]) systems. In addition, research studies have documented a potential role for epigenetic dysregulation as a consequence of various forms of psychosocial stress (e.g., abuse as a child, active combat), with differential DNA methylation observed in loci associated with HPA axis regulation, hypertension, and immune response [79–81 82–85]. Individual responses to chronic stress may also further exacerbate physiological deterioration, for instance, health damaging behavior e.g. smoking, excessive alcohol use among lower SES groups as a coping mechanism for anxiety and stress, or as a consequence of targeted marketing of health damaging commodities, further increasing AL. Recent phenomena such as elevated rates of multiple chronic diseases (e.g. type 2 diabetes, congestive heart failure, hypertension) among US adults including the obesity epidemic[86], i.e. rates of adult obesity significantly increasing in approximately 30 years, could also explain the increases of AL over time in our sample of US adults.

These results should be interpreted with a few strengths and limitations. NHANES is a nationally representative, standardized survey on a multitude of health and nutritional related topics. It also utilizes a stratified, multistage sampling scheme, thus the results of this study are generalizable to other US residents and have high validity. Although NHANES collected a wealth of personal and health information on its study participants, there is possibility for residual confounding. For example, unmeasured factors that have been associated with accelerated health declines specifically in NH-Blacks are self-perceived stress, residential segregation, racial discrimination, and institutionalized racism [87]. Future work examining the roles of perceived racism and objective measures of racial segregation on the association between race/ethnicity and AL are warranted to further elucidate these observed racial

disparities in AL. We did not examine the potential role of acculturation, language proficiency, and country of birth on the race/ethnicity differences in AL burden. However, recent data suggests that age-related gradients in AL are steepest among foreign-born Blacks of both genders and foreign-born Latina women[44]. Future studies could also examine how AL may be associated with other social determinants of health (e.g. socioeconomic position), self-perceived health status, dietary patterns, or ecological phenomenon and disparities existing within them, such as the steady decline of life expectancy in the US. In addition, while there is no current consensus on which biomarkers are used to calculate AL, we operationalized an AL definition most consistent with prior literature using that NHANES sample[48], and which also allowed us to have the most consistent definition throughout the 30-year observation period.

In conclusion, this study examined temporal trends in AL among a large representative sample of US residents across race/ethnicity, gender and age group over a 30-year period. Results confirm that a disproportional burden of AL- a measure of pathophysiological distress- persists among NH-Blacks and Latino adults in the US, an observation that highlights the need for a paradigm shift in studies examining and addressing health disparities. Implementation of culturally-specific, community based participatory research (CBPR) programs[88,89] in communities of color can provide a holistic approach in combating these disparities. There is a need to consider the historical and social context in which minorities live, work, and play, as these will have significant and long-lasting impact on health-related risk factors, biological mechanisms and health outcomes.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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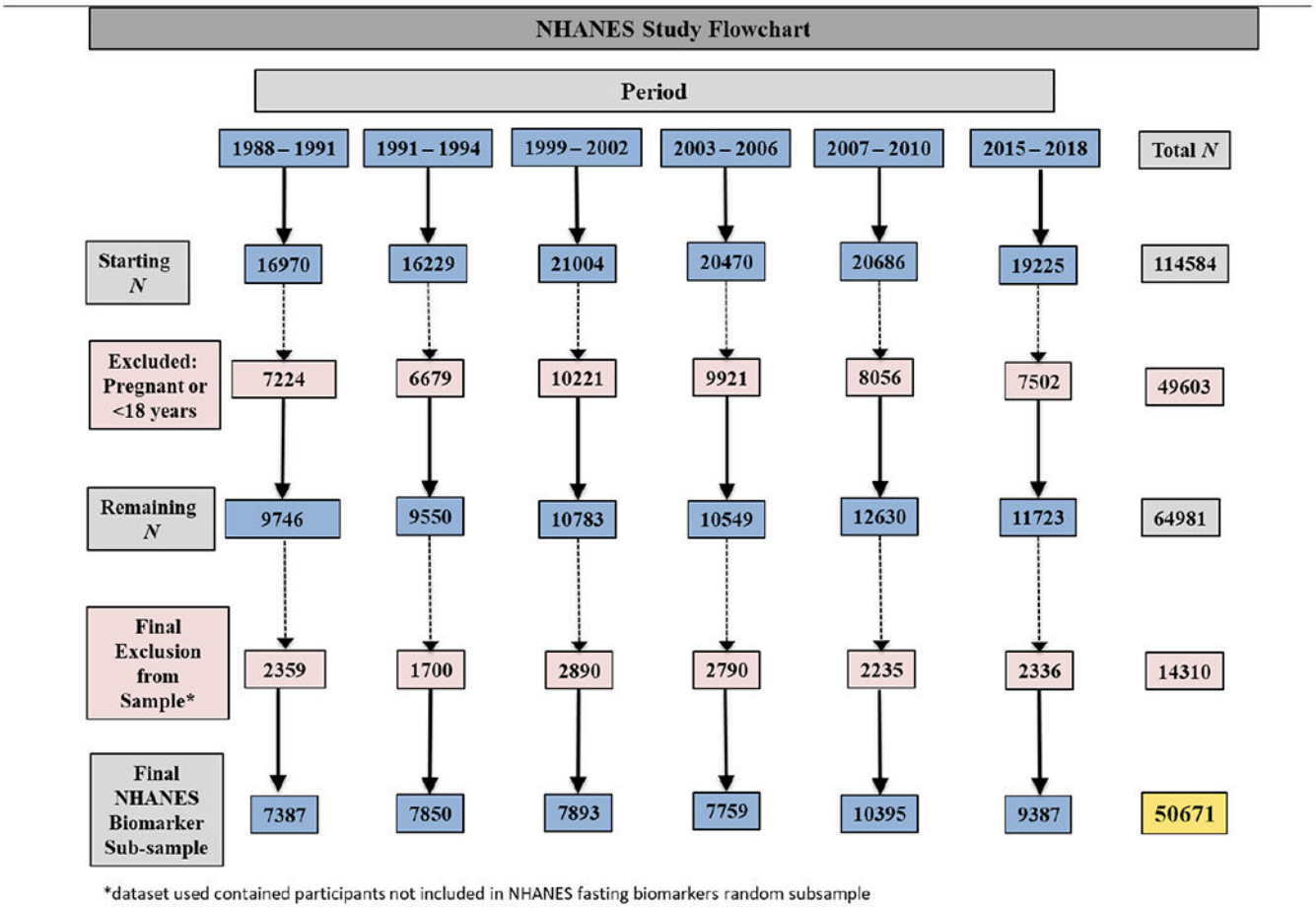
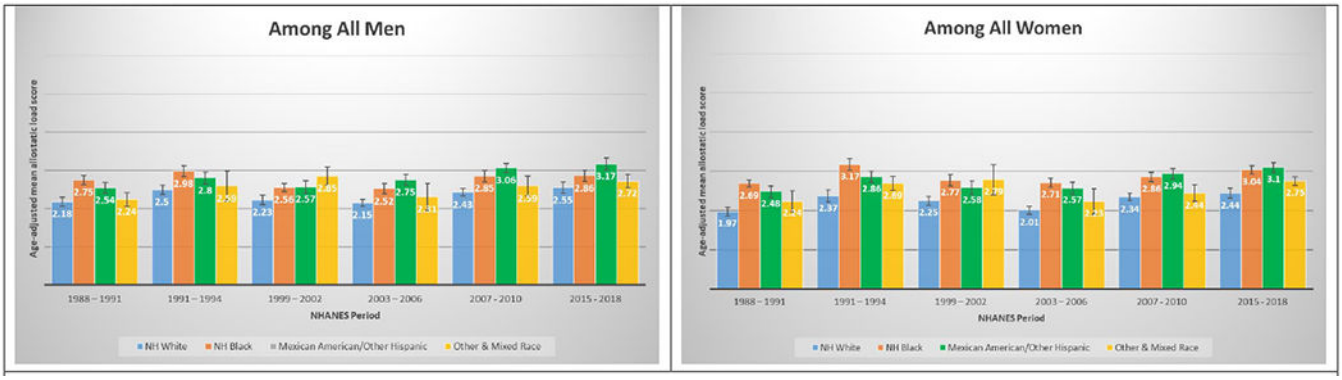
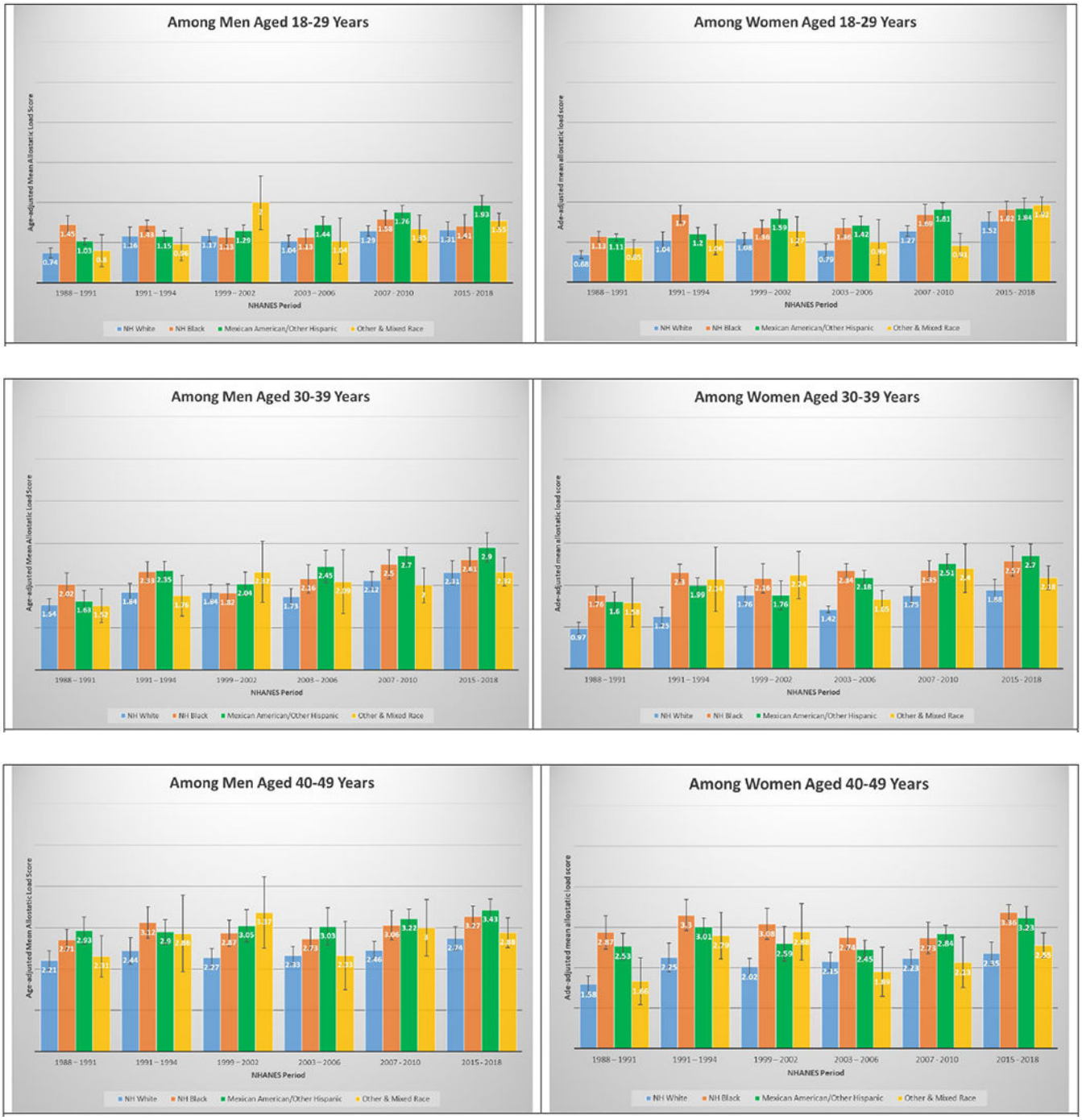


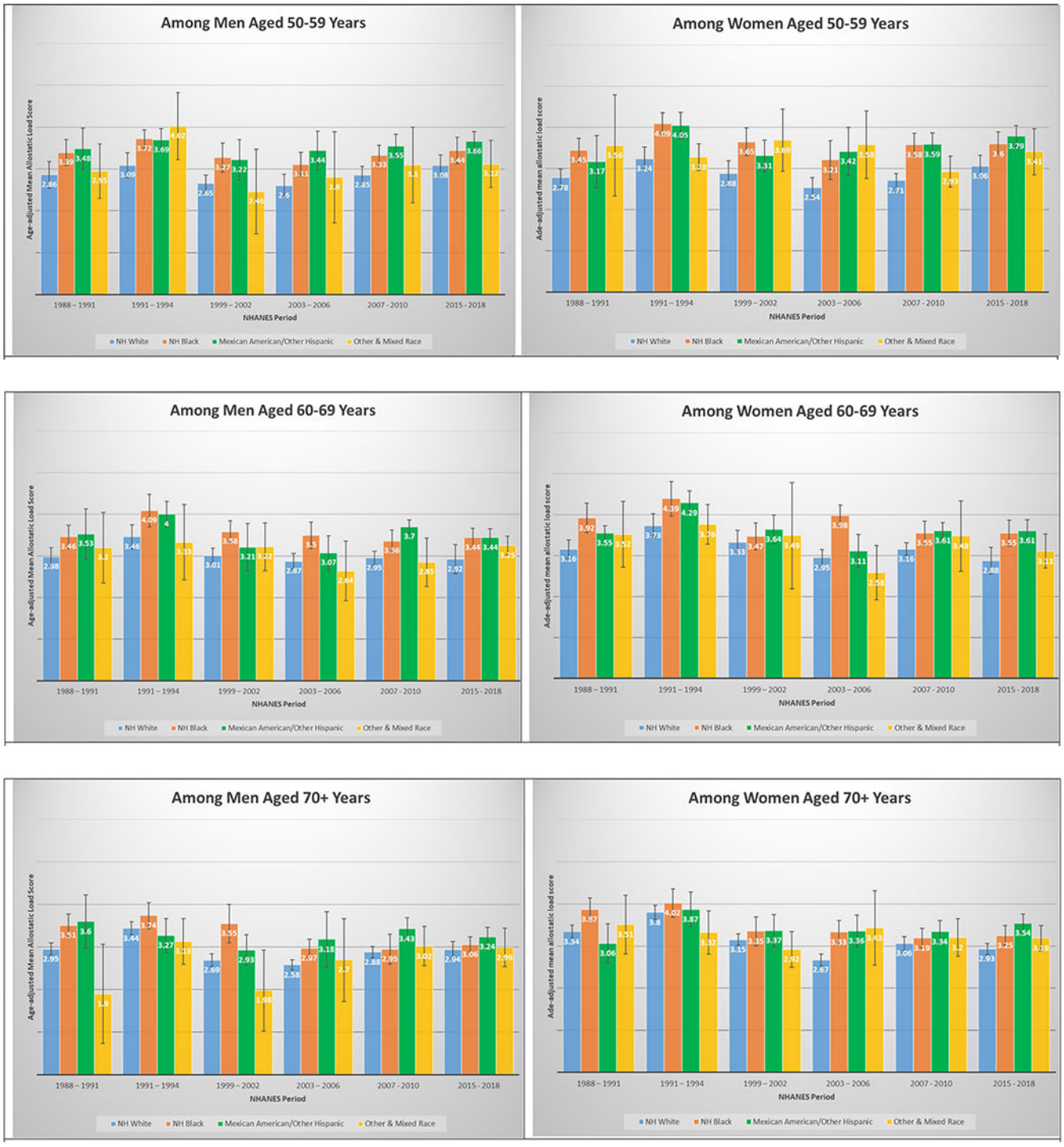
Figure 1:
Flowchart of exclusion criteria and final study population of NHANES participants by study periods.



Figures 2.

2A and 2B. Age-adjusted mean allostatic load scores among US adults, National Health and Nutrition Examination Survey (NHANES), 1988–2018. Panel A represents age-adjusted mean allostatic load scores among men, and panel B represents age-adjusted mean allostatic load scores among women. Based on the sum total of components with high-risk thresholds: albumin, BMI, C-reactive protein, creatinine clearance, diastolic blood pressure, glycated hemoglobin, systolic blood pressure, total cholesterol, triglycerides. Score range from 0 to 9 for all NHANES years.





Figures 3.

3A and 3B. Age-adjusted mean allostatic load scores among US adults aged 18 – 29, National Health and Nutrition Examination Survey (NHANES), 1988–2018. Panel A represents age-adjusted mean allostatic load scores among men, and panel B represents age-adjusted mean allostatic load scores among women. Based on the sum total of components with high-risk thresholds: albumin, BMI, C-reactive protein, creatinine clearance, diastolic

blood pressure, glycated hemoglobin, systolic blood pressure, total cholesterol, triglycerides. Score range from 0 to 9 for all NHANES years.

3C and 3D. Age-adjusted mean allostatic load scores among US adults aged 30 – 39, National Health and Nutrition Examination Survey (NHANES), 1988–2018. Panel A represents age-adjusted mean allostatic load scores among men, and panel B represents age-adjusted mean allostatic load scores among women.

3E and 3F. Age-adjusted mean allostatic load scores among US adults aged 40 – 49, National Health and Nutrition Examination Survey (NHANES), 1988–2010. Panel A represents age-adjusted mean allostatic load scores among men, and panel B represents age-adjusted mean allostatic load scores among women.

3G and 3H. Age-adjusted mean allostatic load scores among US adults aged 50 – 59, National Health and Nutrition Examination Survey (NHANES), 1988–2010. Panel A represents age-adjusted mean allostatic load scores among men, and panel B represents age-adjusted mean allostatic load scores among women.

3I and 3J. Age-adjusted mean allostatic load scores among US adults aged 60 – 69, National Health and Nutrition Examination Survey (NHANES), 1988–2010. Panel A represents age-adjusted mean allostatic load scores among men, and panel B represents age-adjusted mean allostatic load scores among women.

3K and 3L. Age-adjusted mean allostatic load scores among US adults aged 70+, National Health and Nutrition Examination Survey (NHANES), 1988–2010. Panel A represents age-adjusted mean allostatic load scores among men, and panel B represents age-adjusted mean allostatic load scores among women.

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Table 1:

Socio-demographic characteristics, personal health, and medical conditions by National Health Examination Survey (NHANES) study period. Among 50,671 participants an estimated 1,056,925,341 US residents.

	NHANES Period					
	1988 – 1991	**1991 – 1994**	**1999 – 2002**	**2003 – 2006**	**2007 – 2010**	**2015 – 2018**
Participants (N)	7,387	7,850	7,893	7,759	10,395	9,387
Estimated N[a] (%)[b]	155,959,992 (14.76)	165,955,459 (15.70)	165,937,416 (15.70)	168,342,341 (15.93)	192,397,679 (18.20)	208,332,545 (19.71)
	Presented as % (SE) or Mean (95% CI) [c]					
Allostatic Load Total Score [d]	1.94 (1.84 – 2.05)	2.38 (2.28 – 2.48)	2.29 (2.20 – 2.38)	2.17 (2.10 – 2.23)	2.44 (2.37 – 2.51)	2.62 (2.53 – 2.71)
% High Allostatic Load [e]	33.47 (1.37)	43.91 (1.32)	40.17 (1.07)	39.15 (0.88)	44.69 (0.99)	48.55 (1.22)
Male Sex	48.89 (0.63)	48.95 (0.86)	49.71 (0.54)	49.97 (0.54)	49.67 (0.45)	49.19 (0.66)
Mean Age in years	43.77 (42.81 – 44.73)	44.25 (43.03 – 45.46)	45.03 (44.35 – 45.70)	45.51 (44.46 – 46.56)	46.13 (45.50 – 46.76)	47.44 (46.52 - 48.36)
Age Group						
18 – 29	24.70 (1.24)	23.30 (0.90)	20.79 (0.86)	20.33 (0.90)	21.16 (0.68)	20.26 (0.86)
30 – 39	23.12 (1.07)	22.95 (0.83)	20.53 (0.85)	19.12 (0.78)	17.29 (0.51)	16.93 (0.63)
40 – 49	17.98 (0.79)	19.51 (1.08)	21.71 (0.78)	21.92 (0.83)	19.94 (0.60)	16.42 (0.58)
50 – 59	12.43 (0.41)	12.44 (0.64)	16.00 (0.59)	17.16 (0.67)	18.19 (0.52)	18.66 (0.78)
60 – 69	11.65 (0.66)	10.98 (0.80)	10.47 (0.56)	10.79 (0.56)	12.10 (0.50)	15.14 (0.77)
70+	10.12 (0.74)	10.82 (1.00)	10.50 (0.45)	10.69 (0.80)	11.31 (0.40)	12.59 (0.72)
Race/Ethnicity						
Non-Hispanic White	78.04 (2.44)	75.02 (2.27)	72.56 (1.82)	73.15 (2.18)	69.89 (2.41)	63.95 (2.40)
Non-Hispanic Black	10.25 (1.21)	10.74 (1.11)	9.60 (1.16)	10.50 (1.27)	10.24 (0.98)	10.46 (1.32)
Latino	4.87 (0.57)	5.26 (0.72)	13.58 (1.86)	11.21 (1.33)	13.72 (1.79)	15.80 (1.73)
Other & Mixed Race	6.84 (1.32)	8.98 (1.39)	4.25 (0.65)	5.15 (0.52)	6.15 (0.63)	9.79 (0.88)
Education						
< High school	25.97 (1.46)	24.21 (1.44)	21.31 (0.87)	17.82 (1.02)	19.92 (0.90)	12.11 (0.95)
High school/GED	34.34 (1.21)	33.73 (1.33)	27.15 (1.08)	27.22 (0.77)	25.07 (0.89)	23.32 (0.93)
Some college or Associates degree[f]	19.99 (0.75)	21.15 (1.24)	27.57 (0.89)	30.77 (0.85)	29.03 (0.65)	31.11 (1.08)

	NHANES Period					
	1988 – 1991	**1991 – 1994**	**1999 – 2002**	**2003 – 2006**	**2007 – 2010**	**2015 – 2018**
College graduate	19.22 (1.64)	20.63 (1.26)	23.82 (1.55)	24.15 (1.40)	25.88 (1.25)	30.63 (2.04)
Poverty to Income Ratio (PIR) Group						
1st quartile (0 – 1.11)	13.25 (0.74)	13.88 (1.64)	15.03 (1.10)	13.47 (0.83)	15.47 (0.79)	14.68 (0.77)
2nd quartile (1.11 – 2.08)	18.92 (1.17)	19.89 (1.02)	17.87 (1.18)	18.41 (0.81)	17.98 (0.74)	17.67 (0.77)
3rd quartile (2.08 – 3.77)	34.79 (1.25)	28.50 (1.08)	22.76 (0.77)	25.75 (0.90)	22.19 (0.98)	22.45 (0.95)
4th quartile (3.77 – 11.89)	25.92 (1.98)	32.01 (2.24)	36.08 (1.77)	37.84 (1.48)	37.27 (1.22)	36.20 (1.68)
Missing	7.11 (0.60)	5.72 (0.52)	8.26 (0.84)	4.53 (0.43)	7.09 (0.60)	9.01 (0.55)
Mean BMI, kg m^{-2}	26.11 (25.89 – 26.33)	26.73 (26.46 – 26.99)	27.78 (27.50 – 28.06)	28.18 (27.87 – 28.50)	28.46 (28.26 – 28.66)	29.45 (29.08 – 29.82)
Current Smoker Status	30.64 (1.24)	26.45 (1.20)	23.82 (0.93)	24.52 (0.85)	20.81 (0.81)	17.81 (0.79)
Any Cancer History [f]	7.63 (0.48)	7.44 (0.55)	7.55 (0.41)	7.95 (0.40)	9.04 (0.41)	10.61 (0.47)
Ever Congestive Heart Failure	2.29 (0.18)	1.93 (0.24)	1.97 (0.23)	2.12 (0.19)	2.01 (0.18)	2.17 (0.20)
Ever Heart Attack	3.33 (0.31)	3.37 (0.35)	3.20 (0.25)	3.41 (0.32)	3.20 (0.23)	3.37 (0.32)

[a] Estimated using sampling weights from National Health and Nutrition Examination Survey (NHANES).

[b] Presented as weighted row percentage, describes the total percentage of participants among all study periods.

[c] Presented as column proportion (standard error) or mean (95% confidence intervals) for continuous variables.

[d] Allostatic load total score was calculated as sum total of components based on high-risk thresholds: albumin, BMI, C-reactive protein, creatinine clearance, diastolic blood pressure, glycated hemoglobin, systolic blood pressure, total cholesterol, triglycerides. Score range from 0 to 9.

[e] High Allostatic load is defined as total Allostatic load score greater than or equal to 3 (presented as column percentages and standard errors).

[f] Defined as self-reported response to ever being diagnosed by a doctor or health professional of any cancer or malignancy.

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Table 2:

Multivariable modified Poisson regression presented as Relative Risks (RRs) for high[a] allostatic load in US adults stratified by race/ethnicity and sex, by National Health and Examination Survey (NHANES) study period. Among 50,671 participants an estimated 1,056,925,341 US residents.

	NHANES Period					
	1988 – 1991	**1991 – 1994**	**1999 – 2002**	**2003 – 2006**	**2007 – 2010**	**2015 – 2018**
Participants (N)	7,387	7,850	7,893	7,759	10,395	9,387
Estimated N[b] (%)[c]	155,959,992 (14.76)	165,955,459 (15.70)	165,937,416 (15.70)	168,342,341 (15.93)	192,397,679 (18.20)	208,332,545 (19.71)
	Presented as Adjusted Relative Risks (95% CI) [d]					
Race/Ethnicity and Male Sex						
Non-Hispanic White	1.000 (Referent)	1.000 (Referent)	1.000 (Referent)	1.000 (Referent)	1.000 (Referent)	1.000 (Referent)
Non-Hispanic Black	1.332 (1.331 – 1.334)	1.154 (1.153 – 1.155)	1.131 (1.130 – 1.133)	1.169 (1.168 – 1.171)	1.189 (1.188 – 1.190)	1.106 (1.105 – 1.107)
Latino	1.228 (1.225 – 1.230)	1.072 (1.070 – 1.073)	1.038 (1.037 – 1.039)	1.297 (1.295 – 1.298)	1.220 (1.219 – 1.221)	1.259 (1.258 – 1.260)
Other & Mixed-Race	0.932 (0.931 – 0.934)	0.909 (0.908 – 0.910)	1.527 (1.524 – 1.529)	1.105 (1.103 – 1.107)	1.146 (1.145 – 1.147)	1.120 (1.119 – 1.121)
Race/Ethnicity and Female Sex						
Non-Hispanic White	1.000 (Referent)	1.000 (Referent)	1.000 (Referent)	1.000 (Referent)	1.000 (Referent)	1.000 (Referent)
Non-Hispanic Black	1.399 (1.397 – 1.401)	1.380 (1.379 – 1.381)	1.198 (1.197 – 1.200)	1.456 (1.455 – 1.458)	1.223 (1.221 – 1.224)	1.292 (1.290 – 1.293)
Latina	1.316 (1.313 – 1.318)	1.255 (1.253 – 1.257)	1.048 (1.047 – 1.049)	1.206 (1.205 – 1.207)	1.191 (1.189 – 1.192)	1.266 (1.265 – 1.267)
Other & Mixed-Race	1.154 (1.152 – 1.155)	1.183 (1.182 – 1.184)	1.180 (1.178 – 1.182)	1.144 (1.142 – 1.146)	1.001 (0.999 – 1.002)	1.158 (1.157 – 1.160)
Age Groups, in years						
18 – 29	1.000 (Referent)	1.000 (Referent)	1.000 (Referent)	1.000 (Referent)	1.000 (Referent)	1.000 (Referent)
30 – 39	2.406 (2.403 – 2.409)	1.859 (1.857 – 1.861)	1.700 (1.698 – 1.701)	1.967 (1.965 – 1.969)	1.901 (1.900 – 1.903)	1.634 (1.632 – 1.635)
40 – 49	4.596 (4.590 – 4.602)	2.986 (2.983 – 2.989)	2.513 (2.511 – 2.516)	2.905 (2.902 – 2.908)	2.243 (2.241 – 2.245)	2.198 (2.196 – 2.199)
50 – 59	7.019 (7.010 – 7.028)	4.256 (4.252 – 4.260)	3.556 (3.552 – 3.560)	3.502 (3.498 – 3.506)	2.975 (2.973 – 2.978)	2.546 (2.544 – 2.548)
60 – 69	8.052 (8.042 – 8.062)	4.698 (4.694 – 4.703)	3.900 (3.895 – 3.904)	3.759 (3.754 – 3.763)	3.329 (3.325 – 3.332)	2.545 (2.543 – 2.547)
70+	8.196 (8.185 – 8.207)	4.934 (4.929 – 4.939)	3.451 (3.447 – 3.455	3.328 (3.324 – 3.332)	3.024 (3.021 – 3.027)	2.539 (2.537 – 2.542)

[a]High Allostatic load is defined as total Allostatic load score greater than or equal to 3 (presented as column percentages and standard errors). Allostatic load total score was calculated as sum total of components based on high-risk thresholds: albumin, BMI, C-reactive protein, creatinine clearance, diastolic blood pressure, glycated hemoglobin, systolic blood pressure, total cholesterol, and triglycerides.

[b]Estimated using sampling weights from National Health and Nutrition Examination Survey (NHANES).

[c]Presented as weighted row percentage, describes the total percentage of participants among all study periods.

[d] Presented as Relative Risks and 95% confidence intervals for high allostatic load estimated using modified Poisson regression with robust variance estimation and accounting for NHANES weighting. Adjusted for race, gender, education, age groups, poverty-to-income ratio, smoking status, cancer, congestive heart failure, and heart attack.

Table 3:

Multivariable association between mean allostatic load and participant characteristics in US adults stratified by race and sex, by National Health and Examination Survey (NHANES) study period. Presented as the absolute differences from referent groups. Among 50,671 participants an estimated 1,056,925,341 US residents.

	NHANES Period					
	1988 – 1991	**1991 – 1994**	**1999 – 2002**	**2003 – 2006**	**2007 – 2010**	**2015 – 2018**
Participants (N)	7,387	7,850	7,893	7,759	10,395	9,387
Estimated N[a] (%)[b]	155,959,992 (14.76)	165,955,459 (15.70)	165,937,416 (15.70)	168,342,341 (15.93)	192,397,679 (18.20)	208,332,545 (19.71)
	Presented as Adjusted Mean Differences, β (95% CI) [c]					
Race/Ethnicity and Male Sex						
Non-Hispanic White (Referent)	2.57 (2.35, 2.79)	3.12 (2.70, 3.53)	2.40 (2.12, 2.69)	2.25 (1.92, 2.58)	2.67 (2.51, 2.82)	2.97 (2.56, 3.38)
Non-Hispanic Black	+0.46 (0.30, 0.61)	+0.31 (0.11, 0.51)	+0.13 (−0.02, 0.27)	+0.25 (0.10, 0.40)	+0.27 (0.09, 0.44)	+0.17 (0.01, 0.33)
Latino	+0.20 (0.02, 0.38)	+0.09 (−0.11, 0.29)	+0.14 (−0.07, 0.36)	+0.42 (0.26, 0.59)	+0.42 (0.28, 0.56)	+0.44 (0.25, 0.64)
Other & Mixed-Race	+0.03 (−0.19, 0.25)	−0.06 (−0.45, 0.32)	+0.59 (0.39, 0.78)	+0.14 (−0.24, 0.52)	+0.23 (−0.01, 0.46)	+0.13 (−0.04, 0.31)
Race/Ethnicity and Female Sex						
Non-Hispanic White (Referent)	2.38 (2.15, 2.60)	3.01 (2.67, 3.35)	2.41 (2.12, 2.71)	2.11 (1.79, 2.44)	2.58 (2.43, 2.73)	2.90 (2.47, 3.33)
Non-Hispanic Black	+0.60 (0.47, 0.73)	+0.63 (0.41, 0.86)	+0.34 (0.18, 0.51)	+0.58 (0.44, 0.72)	+0.38 (0.22, 0.53)	+0.48 (0.33, 0.63)
Latina	+0.32 (0.16, 0.49)	+0.27 (0.03, 0.51)	+0.13 (−0.08, 0.34)	+0.36 (0.17, 0.56)	+0.41 (0.27, 0.55)	+0.50 (0.37, 0.64)
Other & Mixed-Race	+0.20 (−0.08, 0.48)	+0.20 (−0.09, 0.49)	+0.42 (0.11, 0.73)	+0.18 (−0.14, 0.50)	+0.11 (−0.07, 0.28)	+0.29 (0.12, 0.47)
Age Groups, in years						
18 – 29 (Referent)	1.24 (1.01, 1.48)	1.71 (1.23, 2.20)	1.45 (1.15, 1.76)	1.24 (0.89, 1.58)	1.73 (1.56, 1.91)	2.12 (1.71, 2.53)
30 – 39	+0.61 (0.47, 0.75)	+0.62 (0.40, 0.83)	+0.58 (0.47, 0.68)	+0.70 (0.59, 0.82)	+0.72 (0.57, 0.86)	+0.71 (0.53, 0.88)
40 – 49	+1.29 (1.13, 1.45)	+1.47 (1.25, 1.68)	+1.15 (1.00, 1.30)	+1.28 (1.14, 1.43)	+1.13 (0.96, 1.29)	+1.22 (1.06, 1.39)
50 – 59	+2.14 (1.94, 2.34)	+2.19 (1.99, 2.40)	+1.70 (1.54, 1.86)	+1.68 (1.50, 1.86)	+1.57 (1.40, 1.75)	+1.64 (1.49, 1.80)
60 – 69	+2.34 (2.17, 2.51)	+2.46 (2.12, 2.81)	+1.97 (1.84, 2.10)	+1.90 (1.74, 2.07)	+1.75 (1.62, 1.89)	+1.45 (1.29, 1.61)
70+	+2.35 (2.19, 2.53)	+2.43 (2.24, 2.62)	+1.66 (1.53, 1.78)	+1.55 (1.40, 1.70)	+1.52 (1.38, 1.65)	+1.36 (1.17, 1.54)

[a] Estimated using sampling weights from National Health and Nutrition Examination Survey (NHANES).

[b] Presented as weighted row percentage, describes the total percentage of participants among all study periods.

[c] Presented as multivariable adjusted estimates (mean difference from referent group) from generalized linear models. Adjusted for race, gender, education, age groups, poverty-to-income ratio, smoking status, cancer, congestive heart failure, and heart attack. For example, in 1988 – 1991, non-Hispanic white males are the referent and non-Hispanic black males had adjusted allostatic load scores 0.46 points higher (95% CI: 0.30 – 0.61) than non-Hispanic white males, adjusted for all abovementioned covariates.