Racial and ethnic profiles of allostatic load among adult women in the US: Findings from the National Health & Nutrition Examination Survey 1999-2004

BACKGROUND AND SIGNIFICANCE

An extensive body of literature has investigated the ways in which social and environmental factors influence health and are associated with differential morbidity and mortality rates across social groups (Taylor, Repetti, & Seeman, 1997). In particular, race and ethnicity are important sociodemographic and class indicators in the US and have been long established as major predictors of health status. To understand the biological processes underlying racial/ethnic and other social health disparities, past research has primarily focused on establishing links between sociodemographic characteristics and single biological parameters. More recently, however, researchers have used a multisystem perspective to examine possible biological pathways by which characteristics of the social context are translated to health outcomes (Crimmins & Seeman, 2004; Singer, Ryff, & Seeman, 2004). Several concepts have been developed based on this idea of cumulative biological risk, including the "weathering hypothesis" (Geronimus, 1992), "allostatic load" (McEwen & Stellar, 1993), and "cumulative physiological dysregulation" (Seplaki, Goldman, Weinstein, & Lin, 2006). These all point to potential mechanisms through which socioenvironmental factors "get under the skin", impact biological processes in multiple systems, and manifest as differential health risks by race/ethnicity, socioeconomic status (SES), and other sociodemographic factors (Taylor, Repetti, & Seeman, 1997). While allostatic load (AL) has been investigated in elderly populations and across racial/ethnic groups, a descriptive profile of AL specifically among adult women has yet to be examined.

Allostatic load was originally conceptualized by McEwen, Seeman, and colleagues as the cumulative impact of physiological stress responses that chronically exceed optimal operating ranges and result in wear and tear on the body's regulatory systems (McEwen, 2002; Seeman et al., 2004; Seeman, McEwen, Rowe, & Singer, 2001; Seeman, Singer, Rowe, Horwitz, & McEwen, 1997). Cumulative physiological dysregulation is the mechanism by which AL affects multiple physiological systems, including cardiovascular, immune, metabolic, neuroendocrine, and respiratory systems. When repeatedly exposed to environmental challenges over the life course, these systems may begin to operate outside their optimal ranges or have difficulty returning to optimal ranges (Geronimus, Hicken, Keene, & Bound, 2006; McEwen, 2002). The impact of AL on health is thought to not only result from large, clinically significant deviations from normal ranges, but also from more modest, subclinical dysregulation in multiple systems. The cumulative impact of modest dysregulation in multiple systems can be substantial, even if individual components have minimal, insignificant effects on health (McEwen & Stellar, 1993). The cumulative biological burden exacted on the body represents some of the earliest evidence of decline in health that may eventually lead to disease pathology and disability. Because men and women experience fundamentally different social conditions, it is also expected that underlying biological risk profiles to differ substantially by gender. Past research has shown gender differences in individual biomarkers as well as cumulative biological risk, suggesting the importance of examining AL separately for men and women (Goldman et al., 2004; Seplaki, Goldman, Weinstein, & Lin, 2004).

Prior research in elderly populations has shown higher AL to be associated with mortality, cardiovascular disease, and decreased physical and cognitive functioning (Karlamangla et al., 2002; Seeman et al., 2004; Seeman et al., 2001; Seplaki et al., 2004). Higher AL (indicator of poor health) has also generally been shown to be associated with older age, male gender, black race/ethnicity, lower SES, and US nativity (Crimmins, Johnston, Hayward, & Seeman, 2003; Crimmins et al., 2007; Geronimus et al., 2006). One socioenvironmental factor that has been found to be a particularly strong predictor of AL is race/ethnicity. One study found that racial disparities in AL persisted even after controlling for SES (measured as poverty income ratio), and that poor and non-poor black women had the highest risk of having high AL compared to their male and white counterparts (Geronimus et al., 2006).

Race and ethnicity are powerful determinants of social class and life experiences in the US and are reflected in racial/ethnic disparities in health and AL. African American women have higher morbidity and all-cause mortality rates across the lifespan and are worse off on virtually every major health index in comparison to white women (NCHS, 2006). This persistence of racial/ethnic disparities in AL and health may reflect the stress of living in a race-conscious society. Moreover, black women may experience both the stress of gender and racial discrimination (Geronimus et al., 2006). Hispanics are less clearly disadvantaged; studies have found that Hispanics in the US have better or similar health to non-Hispanic whites, despite having lower SES, a pattern called the Hispanic paradox (Franzini, Ribble, & Keddie, 2001; Palloni & Arias, 2004) However, certain Hispanic subgroups are at high risk for specific health conditions; Mexican women have sigificantly higher rates of obesity, diabetes, and heart disease, while Puerto Rican women suffer disproportionately from HIV/AIDS, asthma, and infant mortality (NCHS, 2006).

Socioeconomic differences account substantially for, but do not fully explain, racial/ethnic health disparities. Within each level of SES, African Americans generally have worse health than whites (Williams & Collins, 1995). One explanation for the persistence of racial differences despite adjustment of SES is that commonly used SES indicators do not fully capture economic status differences beyond income, such as inheritance of wealth, intergenerational transfers of wealth, and other financial assets. Persistent racial/ethnic health disparities are also attributable to racial discrimination, which can lead to unsafe living and working environments, reduced access to health-related resources, and psychological distress, which in turn negatively affect health (Lillie-Blanton, Martinez, Taylor, & Robinson, 1993). Moreover, SES indicators do not have equivalent meanings and yields across races, such that SES-health gradients may be stronger among whites compared to blacks and other disadvantaged racial/ethnic minority groups. Racial/ethnic minorities may have lower quality education and consequently bring fewer basic skills to the labor market; blacks and Hispanics also may receive lower income returns than whites for equivalent levels of education (Williams & Collins, 1995). Given the increasing racial and ethnic diversity of the US population and the persistence of race/ethnicity as strong predictors of health, research that expands beyond black-white differences in AL is warranted.

When examining racial/ethnic patterns in health and AL, nativity status is also important to consider. US-born Hispanic individuals generally tend to have worse health than their foreign-born counterparts, and increasing time of residence in the US has also been shown to be associated with worse health (Cho, Frisbie, Hummer, & Rogers, 2004). While some researchers have attributed the Hispanic paradox to health and lifestlye behaviors, family structure, and social buffering in Hispanic culture, others argue that the Hispanic health advantage is found predominantly in foreign-born Hispanics due to selective migration of healthy individuals from, and the return of sick individuals to, the country of origin (Palloni & Arias, 2004). In addition to examining black-white differences, this study will also focus on Hispanics of Mexican descent and their nativity status to provide important new information about this demographically significant group.

This study uses data from the National Health and Nutrition and Examination Survey (NHANES) 1999-2004 to examine allostatic load (AL) among women in the US, focusing on racial/ethnic variation by SES and nativity status. We will test the hypotheses that: 1) AL varies by race/ethnicity, such that black women have higher levels of AL, and Mexican American women have comparable levels, relative to white women, net of SES and other covariates; 2) the association between SES and AL is weaker among black women due to the effects of racial discrimination and unmeasured SES differences; and 3) the association between race/ethnicity on AL differs by nativity status, such that US-born Mexican American women are at greater risk of having higher AL scores than foreign-born Mexican American women due to migrant health selectivity. This paper is the first to provide a descriptive profile of AL specifically among adult women of all ages, focusing on racial/ethnic differences and identifying demographically important subgroups of women at high risk of having elevated cumulative biological risk.

DATA AND METHODS

Data description. This study uses data from the National Health and Nutrition Examination Survey (NHANES) 1999-2004. The NHANES survey is a cross-sectional study that uses a complex stratified, multistage probability sampling design to provide national estimates of health and nutritional status for the civilian, non-institutionalized US population. Beginning in 1999, NHANES IV has been collected on a continuous basis and released every two years. Eligible respondents are obtained with the use of household-based sampling procedures. Survey components include questionnaire interviews, clinical examination, and laboratory procedures (the clinical and laboratory components occur in Mobile Examination Centers (MEC)). Standardized procedures and protocols were developed and validated by the National Center for Health Statistics for all clinical exams and laboratory tests (CDC, NCHS, 1999-2004). The analytic sample for this study is all women ages 18 years and older who have valid data on all biomarkers used to create the AL score, are not pregnant, and completed the interview and MEC exam components (N=6,256). The extensive, nationally representative biomarker data in NHANES are conducive for studying AL and have been used in previous studies to develop AL measures. Another advantage of NHANES is that sample sizes are large enough to allow for meaningful racial/ethnic comparisons. In particular, Hispanics of Mexican descent were specifically sampled to allow for analysis of a more homogeneous Hispanic group.

Biomarker Measures. Biomarkers used for the creation of AL scores are selected based on representation of multiple systems, availability in the dataset, and prior research (Crimmins et al., 2003; Geronimus et al., 2006; Seeman et al., 2004). The following 10 biomarkers will be used: 1) Albumin (serum); 2) Diastolic blood pressure; 3) Systolic blood pressure; 4) Body mass index; 5) C-reactive protein; 6) Glycosylated hemoglobin; 7) High-density lipoprotein; 8) Homocysteine; 9) Pulse rate; and 10) Total cholesterol. These biomarkers represent functioning across a number of physiological systems, including cardiovascular, metabolic, and immune-inflammatory systems. In this analysis, AL scores are created using: 1) empirical cutpoints based on the distribution of the analytical sample; and 2) clinical cutpoints. Both versions are created using a count-based summation method. For the first version, we first establish cutoffs based on the highest risk quartile value based on the analytic sample distribution. Values above the 75th percentile are defined as high risk for all the biomarkers, with the exception of HDL and serum albumin, for which values below the 25th percentile are defined as high risk. The quartile cutpoint criteria are based on prior studies and are regarded as a standard approach (Seeman et al., 2004; Seeman et al., 2001). For the second version of AL score, we use national and professional standards for defining high-risk. This approach has also been employed in prior studies and has the advantage of being calibrated to meaningful clinical standards (Crimmins et al., 2001; Crimmins et al., 2007). Both versions of AL scores are then calculated by summing the number of biomarkers for which the subject falls into the highest-risk quartile. A higher AL score is an indicator of poorer health. Although there are other options for data reduction techniques, prior research suggests this count-based summation approach is quite robust (Seplaki et al., 2006).

Independent Variables. The key independent variables are race/ethnicity (non-Hispanic white; non-Hispanic black; Mexican American; and Other), SES (education, family income, and poverty income ratio), and nativity status (US- and foreign-born). Educational attainment is categorized as less than 12 years, high school graduate, and more than high school. Family income is categorized in $15,000 increments, and poverty income ratio will be included as a continuous variable. Other control variables include: 1) age (10-year intervals); and 2) marital status (never married; currently married/living together; and divorced/separated/widowed). Transformations for family income, poverty income ratio, and age will also be explored.

Analysis. The distributional qualities of each of the 10 individual biomarkers and summary AL scores will be examined (range, mean, median, quartiles). Bivariate cross-tabulations will then be conducted to assess associations between each individual biomarker and covariates, and summary AL scores and covariates. Mean AL scores will be determined by race/ethnicity and SES groups and compared using analysis of covariance models (ANCOVA). For multivariate regression analyses, we will explore a variety of functional forms of AL and utilize analytic techniques that are appropriate to these different

functional forms, including ordinary least squares (OLS), transformed OLS, and binomial and ordered logit models. In multivariate regression analyses, we will investigate interactive effects between race/ethnicity and SES, and race/ethnicity and nativity status, net of other confounders. Lastly, we will estimate predicted probabilities of AL scores for clinically important subgroups of women who are at high risk of having elevated AL. All analyses and estimates will be conducted using Stata 10 (StataCorp, 2005) and weighted using the NHANES individual-level sampling weights, which adjust for complex sample design, selection, and non-response.

PRELIMINARY RESULTS AND NEXT STEPS

Table 1 presents descriptive statistics, including range, mean, median, and quartiles, for the individual biomarkers that comprise AL in this study. Table 2 displays mean empirical AL scores by race/ethnicity, education, and nativity status. Overall, mean AL scores were lower (indicating better health) among women with higher education and foreign-born women. Black women had the highest mean AL scores across educational levels, followed by white women. Among women with a high school education or less, Mexican American women had the lowest mean AL score. Health gains by increasing educational status were smallest for black women; the difference in mean AL score between black women with more than a high school education and black women with less than 12 years of education was 0.24. In contrast, the difference in mean AL score between white women with more than high school education and the least educated white women was 0.96, suggesting that the association between education and AL is weaker among blacks than whites. The difference in mean AL score between Mexican American women with a high school education and Mexican American women with less than 12 years of education was 0.33; however, the difference was only 0.04 when Mexican American women in the two most extreme education categories were compared (differences not shown in table).

Comparison of AL scores by nativity status showed that foreign-born women across racial/ethnic groups were healthier than US-born women, however differences in mean AL score between nativity status varied by race/ethnicity. Foreign-born black women differed by 0.43 in mean AL score relative to their US counterparts, whereas the difference was only 0.09 between foreign-born and US-born white women. The difference between foreign-born and US-born Mexican Americans (0.33) was intermediate between black and white women nativity differences.

Further analyses will compare mean AL scores between groups using ANCOVA models. Interactive effects between race/ethnicity and SES variables, and race/ethnicity and nativity status on AL score will be tested in multivariate regression models. All analyses will be conducted for two versions of AL scores, the first based on empirically-based cutpoints and the second on clinical cutpoints. We expect the effects of race/ethnicity on AL to remain significant even after controlling for SES and other variables. It is also expected that the effect of SES on AL score will differ by race/ethnicity, such that SES has a weaker assocation with AL among black women compared to other racial/ethnic groups. These hypotheses are based on previous research and theory suggesting that racial disparities in health reflect effects of racial discrimination and unmeasured SES factors. We also expect the effect of race/ethnicity on AL score to differ by nativity status. In particular, US-born Mexican American will have significantly higher odds of having higher AL scores than their foreign-born counterparts, due to migrant health selectivity. Finally, predicted probabilities of AL scores will be estimated for demographically important subgroups of women who are at high risk of having elevated AL.

Table 1. Descriptive statistics of individual biomarkers among women, NHANES 1999-2004 (N=6285).

Biomarker	Range	Mean	25%	50%	75%	Clinical cutpoint
Albumin (g/dL)	2.7 – 5.3	4.24	4.00	4.20	4.40	<3.8
Blood pressure - systolic (mm Hg)	73 – 248	124.81	108.00	119.00	137.00	≥140
Blood pressure - diastolic (mm Hg)	0 – 122	69.45	63.00	70.00	77.00	≥90
Body mass index (kg/m^2)	14.42 – 66.44	28.29	23.37	27.15	32.05	≥25
CRP (mg/dL)	0.01 – 29.6	0.51	0.10	0.26	0.59	≥0.3
Glycosylated hemoglobin (%)	2.5 – 18.8	5.54	5.10	5.30	5.60	≥6.4
HDL (mg/dL)	8 – 160	56.48	45.00	54.00	66.00	<40
Homocysteine (µmol/L)	1.65 – 156.30	8.21	6.00	7.28	9.19	≥9.19
Pulse rate (bt/min)	38 - 134	73.39	66.00	72.00	80.00	≥90
Total cholesterol (mg/dL)	84 - 650	201.76	172.00	198.00	228.00	≥240

Note: For the empirically-based AL score, values ≥ 75% are defined as high risk for all individual biomarkers, with the exception of albumin and HDL, for which values ≤ 25% are defined as high risk.
Sample (N) consists of women 18 years and older who are not pregnant, completed both interview and MEC exam, and are not missing on any biomarkers.

Table 2. Mean empirical allostatic load (AL) scores by race/ethnicity, education, and nativity status among women, NHANES 1999-2004 (N=6285).

		Education				Nativity status		
Mean empirical AL score	**Total**	**<12 yrs**	**HS grad**	**>HS**	**Mean difference between >HS and <12 yrs**	**US-born**	**Foreign-born**	**Mean difference between US-born and foreign-born**
Race/ethnicity								
Non-Hispanic White	2.46	3.11	2.75	2.15	0.96	2.47	2.38	0.09
Non-Hispanic Black	3.09	3.24	3.12	3.00	0.24	3.13	2.70	0.43
Mexican American	2.23	2.40	2.07	2.03	0.37	2.41	2.08	0.33
Other	2.28	2.76	2.59	1.87	0.89	2.45	2.17	0.28
Total		2.95	2.74	2.20	0.75	2.54	2.18	0.36

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