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#### Article

# Nativity differences in allostatic load by age, sex, and Hispanic background from the Hispanic Community Health Study/Study of Latinos



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#### ARTICLE INFO

#### Article history: Received 2 December 2015 Received in revised form 9 May 2016 Accepted 9 May 2016

Keywords: Allostatic load Physiological dysregulation Hispanic ethnicity Nativity Age patterns

#### ABSTRACT

Allostatic load (AL), an index of biological "wear and tear" on the body from cumulative exposure to stress, has been little studied in US Hispanics/Latinos. We investigated AL accumulation patterns by age, sex, and nativity in the Hispanic Community Health Study/Study of Latinos. We studied 15,830 Hispanic/ Latinos of Mexican, Cuban, Dominican, Puerto Rican, Central and South American descent aged 18-74 years, 77% of whom were foreign-born. Consistent with the conceptualization of AL, we developed an index based upon 16 physiological markers that spanned the cardiometabolic, parasympathetic, and inflammatory systems. We computed mean adjusted AL scores using log-linear models across age-groups (18-44, 45-54, 55-74 years), by sex and nativity status. Among foreign-born individuals, differences in AL by duration of residence in the US ( < 10,  $\geq$  10 years) and age at migration ( < 24,  $\geq$  24 years) were also examined. In persons younger than 55 years old, after controlling for socioeconomic and behavioral factors, AL was highest among US-born individuals, intermediate in foreign-born Hispanics/Latinos with longer duration in the US ( $\geq$  10 years), and lowest among those with shorter duration in the US (< 10 years) (P < 0.0001 for increasing trend). Similarly, AL increased among the foreign-born with earlier age at immigration. These trends were less pronounced among individuals  $\geq 55$  years of age. Similar patterns were observed across all Hispanic/Latino heritage groups (P for interaction = 0.5). Our findings support both a "healthy immigrant" pattern and a loss of health advantage over time among US Hispanics/Latinos of diverse heritages.

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#### Introduction

Exposure to stressors over the life course is thought to accelerate biological aging by promoting physiological dysregulation and influencing disease trajectories (Masoro, 1997). Allostatic load (AL) is an index of physiological dysfunction from a failure to adapt to chronic and repeated exposure to stressors (Ben-Shlomo & Kuh, 2002). As a multisystem model of biological risk, AL has been a

useful construct in conceptualizing how chronic adversity imposes "wear and tear" on biological systems, increasing morbidity and mortality over the life course (McEwen & Seeman, 1999), and contributing to health disparities in the US (Geronimus, Hicken, Keene, & Bound, 2006). Studies suggest that AL increases with age (Crimmins, Johnston, Hayward, & Seeman, 2003) and can vary by sex (Goldman et al., 2004; Yang & Kozloski, 2011). While some available evidence links AL with cardiovascular disease (CVD) risk factors in Hispanics/Latinos in the US (Mattei, Demissie, Falcon, Ordovas, & Tucker, 2010), there has been a scarcity of studies examining patterns of AL accumulation by age and sex in this

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vulnerable population. A greater understanding of the accumulation of biological mediators of risk may help to explain the increased burden of disease among US Hispanics/Latinos.

Emerging evidence suggests that place of birth (nativity) has an influence on AL. Data from the National Health and Nutrition Examination Surveys (NHANES 1999-2002) showed that while Hispanics/Latinos tend to have CVD risk factor values at high risk levels than do non-Hispanic whites, US-born Hispanics/Latinos (who were predominantly of Mexican origin) had higher levels of AL than foreign-born Hispanics/Latinos (Crimmins, Kim, Alley, Karlamangla, & Seeman, 2007). Similar results from a cross-sectional study of Mexican adults residing in Texas City, TX, showed differences across groups that persisted after controlling for socioeconomic status, smoking, and physical activity (Peek et al., 2010). These findings may suggest an "unhealthy assimilation" effect where increased stress from discrimination (Paradies, 2006), worsening dietary habits (Akresh, 2007), physical inactivity (Ham, Yore, Kruger, Heath, & Moeti, 2007), and adoption of unhealthy behaviors such smoking and drinking (Eitle, Wahl, & Aranda, 2009; Leung, 2014) confer a physiological toll and a deterioration in health with time spent in the US (Antecol & Bedard, 2006). Because each major Hispanic/Latino group living in the US has a distinct history and culture, it is informative to investigate heterogeneity in the relationship between nativity, duration in the US, age at immigration and AL across Hispanic heritage backgrounds. Moreover, the few studies that have investigated these relationships have had limited age ranges and modest sample sizes, precluding the study of AL across age groups.

The objective of this study is to examine differences in AL by age and sex patterns of AL in a diverse, representative sample of Hispanic/Latino adults in the US, and to investigate the influence of nativity status and Hispanic heritage on these observed patterns.

#### Methods

#### Sample and procedures

The Hispanic Community Health Study/Study of Latinos is a community based prospective cohort study of 16,415 Hispanic/ Latino persons of diverse Hispanic heritages (Mexican, Puerto Rican, Cuban, Dominican, Central and South American) aged 18-74 recruited from four U.S. field centers (Chicago, IL; Miami, FL, Bronx, NY; San Diego, CA), with baseline measurements conducted during 2008-2011. Detailed information regarding the sampling design and cohort selection is available elsewhere (Lavange et al., 2010). Briefly, a stratified two-stage area probability sampling approach was used to select households in each of the four field centers. For the first stage, census block groups were randomly selected with stratification on the basis of Hispanic/Latino concentrations and proportions of high and low socioeconomic status. For the second stage, households were randomly selected with stratification on the basis of whether the occupant had a Hispanic surname from US Postal Service registries that covered the census block groups selected. At each stage, strata were oversampled to increase likelihood of selecting a Hispanic/Latino household. Additionally, Hispanic/Latino participants aged 45-74 were oversampled to facilitate an analyses of cardiovascular disease outcomes. The Institutional Review Boards at each participating institution approved this study and all subjects gave written informed consent.

#### Study visits

At the time of enrollment, all participants attended a clinical examination at a local field center. Fasting morning blood draw and two-hour oral glucose tolerance test was obtained with clinical chemistry panels conducted by a core study laboratory. Standardized questionnaires were administered by bilingual interviewers in English or Spanish according to the participant's preference (Sorlie et al., 2010). Other measurements included seated blood pressure, resting electrocardiogram, pulmonary function testing and anthropometry (Sorlie et al., 2010).

#### Allostatic load markers

We defined AL based upon values of 16 available biomarkers collected using standardized protocols during the baseline clinical examination. Measures that comprised the AL index were designed to capture (a) cardiometabolic risk: body mass index (BMI), waist-to-hip ratio (WHR), serum triglycerides, and fasting levels of high- and low-density lipoprotein cholesterol (HDL-c and LDL-c); (b) glucose metabolism: fasting plasma glucose (FPG), blood glycosylated hemoglobin (HbA1c), and homeostatic model assessment of insulin resistance (HOMA-IR); (c) cardiopulmonary functioning: systolic blood pressure (SBP), resting pulse pressure, resting heart rate, and lung function (% FEV<sub>1</sub>/FVC); (d) parasympathetic functioning using two ultra-short time domain measures of heart rate variability (HRV), including the square root of the mean squared difference of successive NN intervals and the standard deviation of NN intervals; and (e) inflammation: high-sensitivity C-reactive protein (hs-CRP) and total white blood cell count (WBC). These biomarkers span a wide selection of regulatory systems theorized to be involved in adaptive processes related to life stresses and linked to health outcomes later in life (Gruenewald et al., 2012; Juster, McEwen, & Lupien, 2010; Seeman, Epel, Gruenewald, Karlamangla, & McEwen, 2010). We excluded participants who had < 8 h of fasting prior to blood draw (n=294, < 2%) and those who had > 2 missing biomarkers of AL (n=230, < 2%).

Details of laboratory methods for AL markers in HCHS/SOL are described on the study website (www2.cscc.unc.edu/hchs/). Briefly, BMI was computed as weight in kilograms divided by height in meters squared. Plasma glucose was measured using a hexokinase enzymatic method (Roche Diagnostics). HbA1c was measured using a Tosoh G7 Automated HPLC Analyzer (Tosoh Bioscience). Fasting insulin was measured using two commercial immunoassays (ELISA, Mercodia AB, Uppsala, Sweden; and sandwich immunoassay on a Roche Elecsys 2010 Analyzer, Roche Diagnostics, Indianapolis, IN; early measures conducted with the Mercodia assay were calibrated, and values were equivalent to the Roche method (Qi et al., 2015). HOMA-IR was calculated using the following equation: fasting glucose × fasting insulin/405 (Matthews et al., 1985). The two measures of heart rate variability were assessed through ECG recordings read by the Central ECG Reading Center (EPICARE) using GEMSIT MAC1200 portable electrocardiograph while participants were in a fasting state. Serum hs-CRP was assayed in blood with a RocheModular P Chemistry Analyzer using an immunoturbidimetric method (Roche Diagnostics). Inter-assay coefficient of variation was < 2.5%, and intraassay coefficient of variation was < 4.7%. White blood counts were measured in EDTA whole blood using a Sysmex XE-2100 instrument, (Sysmex America, Inc., Mundelein, IL). White blood counts were measured in EDTA whole blood using a Sysmex XE-2100 instrument, (Sysmex America, Inc., Mundelein, IL).

#### Operationalization of allostatic load

We created a count-based summary measure of AL following the approach developed by Seeman and colleagues (Seeman, Singer, Rowe, Horwitz, & McEwen, 1997). Each marker was assigned a score of one if its value reached a high-risk quartile;

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