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Race/Ethnicity, Poverty, Urban Stressors and Telomere Length in a Detroit Community-Based Sample

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Abstract

Residents of distressed urban areas suffer early aging-related disease and excess mortality. Using a community-based participatory research approach in a collaboration between social researchers and cellular biologists, we collected a unique data set of 239 black, white, or Mexican adults from a stratified, multi-stage probability sample of three Detroit neighborhoods. We drew venous blood and measured Telomere Length (TL), an indicator of stress-mediated biological aging, linking respondents' TL to their community survey responses. We regressed TL on socioeconomic, psychosocial, neighborhood, and behavioral stressors, hypothesizing and finding an interaction between poverty and racial/ethnic group. Poor whites had shorter TL than nonpoor whites; poor and nonpoor blacks had equivalent TL; poor Mexicans had longer TL than nonpoor Mexicans. Findings suggest unobserved heterogeneity bias is an important threat to the validity of estimates of TL differences by race/ethnicity. They point to health impacts of social identity as contingent, the products of structurally-rooted biopsychosocial processes.

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Background

Determining how structurally rooted social processes work through biological mechanisms to impact health is fundamental to understanding racial, ethnic, and socioeconomic health inequality. Everyday challenges shaped by social disadvantage may trigger repeated activation of physiological stress processes (Geronimus 1992, Geronimus 2001, Geronimus et al. 2006, Geronimus et al. 2010, McEwen 1998a, Sapolsky, Romero and Munck 2000). Researchers posit that prolonged psychosocial or physical challenges to metabolic homeostasis can increase disease susceptibility and promote the early onset of chronic conditions (Geronimus and Thompson 2004, Geronimus et al. 2007, James 1994, Steptoe et al. 2002). The weathering hypothesis suggests that the cumulative biological impact of being chronically exposed to, and having to cope with, socially structured stressors can increase health vulnerability and accelerate aging in marginalized populations (Geronimus 1992; Geronimus et al. 2006, Geronimus, et al. 2010).

The weathering hypothesis emphasizes that population differences in the early onset of chronic disease result from the qualitatively different life experiences, exposure to stressors, and access to coping resources associated with salient social identities or assignments such as race or ethnicity, from conception through at least middle adulthood. It augments life course theories that highlight epigenetic programming for later life disease occurring in utero or during other developmental periods in youth, by emphasizing how structured life experiences in adulthood continue to impact health trajectories. (See Colen (2011) and Geronimus (2013a) for discussion of weathering and alternative life course theories in the context of marginalized groups). While initially applied to reproductive-age women and birth outcomes, the weathering hypothesis also has been studied in the context of population differences for men and women across the life-span (Geronimus et al. 1996, Geronimus 2001, Geronimus et al. 2006, Geronimus et al. 2007). Evidence that population differences in morbidity and mortality are most pronounced in young adulthood through middle-age (Adler et al. 2013, Geronimus 2001, Geronimus et al. 2006, Geronimus et al. 2007, House et al. 1994, Kim and Miech 2009) is consistent with the weathering hypothesis. Most empirical evidence of weathering pertains to African Americans; yet, populations subject to such health impacts are a broader and more variegated set than suggested by a black-white binary. Evidence of such health impacts also have been seen in the ethnic, religious, socioeconomic, sexual orientation, gender, geographic, or nativity divisions within population groups, including whites (Gee et al. 2006, Geronimus 2000, Geronimus and Thompson 2004, Geronimus and Snow 2013, Hatzenbuehler, Keyes and Hasin 2009, James 1993, Pearson and Geronimus 2011, Viruell-Fuentes 2007)

The concept of allostatic load (McEwen and Seeman 1999, Seeman et al. 1997) -- that overexposure to stress hormones can cause wear and tear on important body systems -- lends biological plausibility to the weathering hypothesis. Humans respond to stressors through the cooperative effects of the primary stress response systems – the sympathetic nervous system (SNS) and hypothalamic-pituitary-adrenal (HPA) axis (Sapolsky, Romero and Munck 2000). With repeated activation of the stress response systems, these mechanisms become inefficient, resulting in an allostatic load on the body’s systems (McEwen 1998b). Allostatic load may contribute to the development or progression of a broad range of clinical

and preclinical pathological processes, including cardiovascular disease, obesity, diabetes, susceptibility to infection, carcinogenesis, and accelerated aging (Geronimus and Thompson 2004, Geronimus et al. 2010, Khansari, Shakiba and Mahmoudi 2009, McEwen and Seeman 1999).

Algorithms to measure allostatic load generally account for the number of stress-related biomarker values for a subject that places him or her in a high-risk category – generating an allostatic load score. Studies using different algorithms find evidence that racial/ethnic or socioeconomic inequalities in allostatic load score increase across young through middle adulthood, consistent with weathering (Geronimus et al. 2006, Seeman et al. 2010). However, by necessity, biomarker selection for research is data driven. And while certain components are common to most algorithms, no standard score exists that can be compared across studies.[1]

Telomere Length as an indicator of weathering

An alternative biomeasure for studying weathering might be telomere length (TL) in a subset of leukocytes called peripheral blood mononuclear cells (PBMC)[2] (Geronimus et al. 2010). Telomeres, the stabilizing caps on chromosomes that protect them from deterioration, are made up of base pairs (DNA-protein complexes[3]). Telomeres shorten (lose base pairs) with cell division until a point at which the chromosomes are functionally impaired and exhibit genomic instability, resulting in cellular senescence or death (Blackburn, Greider and Szostak 2006). Because cell division is necessary to replenish damaged cells, senescence could theoretically pose a serious problem in tissues and organs when a critical number of cells are no longer dividing.[4]

TL in an individual is determined by many factors, among them genetics, health behavior, cell environment, and physical environment. TL differences in twins typically become more pronounced with age, illustrating the role of environmental factors in transforming a common genotype into different phenotypes (Cherkas et al. 2006, Fraga et al. 2005) and suggesting that adult life experiences have molecular impacts that affect aging. Shorter TL appears to signal higher risk of infectious and chronic disease onset (Zalli et al. 2014). Although some studies (e.g., Harris et al. (2006)) find no significant association between TL and mortality, several other large studies do, including one prospective study of 100,000 subjects (Bojesen 2013).

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[1]The first allostatic load algorithm, which had 10 components (Seeman et al 1997) has been modified on the basis of available data to encompass fewer or a greater number of components in other studies. As theory on ideal measurement of allostatic load is refined and potential components of score algorithms expanded, the suitability of any single data set for measuring the construct becomes more strained and findings from studies analyzing different data sets become less comparable to each other.

[2]Telomere length from different tissues and cell types of the same individual are highly correlated (Lin et al 2010; Friedrich et al. 2000), suggesting PBMC TL is a good representative of TL in other tissues from the same individual. PBMC TL is an average across different cell subpopulations: T cells, B cells, NK cells and monocytes. Thus, PBMC TL may not inform about the specificity of immune aging, which might vary across these cell types. However, PBMC TL is the metric that has been most commonly linked to morbidity and mortality in the human literature.

[3]Base pairs are the four unique building blocks of DNA that, once ordered into sequences, make up genes. At the caps of each chromosome, telomeres are made of a short, repeated sequence of DNA base pairs complexed with proteins.

[4]The evidence that short telomere length causes replicative senescence in human cells is well documented. Researchers suggest that cellular senescence may also be linked to inflammation and increased cancer risk, as senescent cells secrete pro-inflammatory cytokines and substances that trigger cell division and suppress cancer-prohibitive mechanisms of neighboring cells (Allsopp et al 1992, Campisi 2005, Chan and Blackburn, 2004, Effros et al 2005)

A rapidly growing set of studies finds that life stressors are associated with TL in diverse circumstances and populations (Damjanovic et al. 2007, Drury et al. 2012, Epel et al. 2004, Kananen et al. 2010, Tyrka et al. 2010). Some studies have linked PBMC TL to biological stress responses via activation of the sympathetic nervous system (SNS) and hypothalamic-pituitary axis (HPA) (Epel et al. 2006, Parks et al. 2009, Tomiyama et al. 2012).

Breaks in DNA structure due to oxidative stress are not easily repaired in telomeres. Because oxidative stress is an important mechanism linking aging, psychosocial stress, biological stress response, inflammation, and disease development, PBMC telomere length may serve as a powerful marker of overall biological age (Bauer, Jeckel and Luz 2009, Demissie et al. 2006, Harrison et al. 2003, Valdes et al. 2005).[5] However, whether TL is an aging determinant in itself or, instead, registers the cumulative effects of other aging determinants remains to be fully explicated (Aviv and Bogden 2010). In either case, if frequent activation of physiological stress mechanisms over time causes weathering, and if TL is negatively associated with biological stress activation, then population TL in young through middle adulthood may be a parsimonious indicator of population weathering.

Studies of racial/ethnic, socioeconomic, or residential differences in TL are few, yield conflicting results, and sometimes suffer from critical methodological and, we argue, paradigmatic weaknesses. Samples in most TL work are highly select, racially homogeneous, or convenience samples (Cherkas et al. 2006, Epel et al. 2004), with socioeconomic measures completely absent in some studies of racial variation (Hunt et al. 2008). Some multiethnic TL studies include only healthy individuals at baseline, excluding those who experienced health deterioration at an earlier age (Adler et al. 2013, Carroll et al. 2013, Diez Roux et al. 2009); and studies focusing on socioeconomic characteristics often have little racial/ethnic diversity (Steptoe et al. 2011). Some studies of TL are limited to children (Mitchell et al. 2014, Theall et al. 2013) or elderly populations (Adler et al. 2013), excluding the young-through-middle-adult ages when social inequalities in health are largest and when TL is most likely to indicate weathering.[6]

One recent study measuring socioeconomic characteristics used national samples and is thus more representative (Needham et al. 2013). Yet, the authors do not consider interactions of income with race/ethnic group, despite compelling evidence that the relationship between income and health varies in magnitude and sometimes direction by racial/ethnic group, nativity status, and, among immigrants, duration of residence in the U.S. (Kaestner et al. 2009, Pearson 2008, Viruell-Fuentes, Miranda and Abdulrahim 2012). Moreover, given the nature of national survey sampling, such surveys are underpowered for the study of the poorest blacks, whites or Latinos. In national samples, whites who are as poor as the poorest blacks or Latinos are scarce and disproportionately rural, making estimates of the size of

[5]Researchers consistently report a robust inverse relation between TL and chronological age (Benetos et al 2001, Frenck, Blackburn and Shannon 1998, Iwama et al 1998, Lindsey et al 1991) most likely due to telomere shortening that occurs as cells replicate, and possibly also to higher proportions of more differentiated, or memory, cells in the blood samples of older populations (Lin et al, 2010). Researchers also have reported accelerated telomere shortening as an underlying factor in conditions and diseases associated with an accelerated aging process (Allsopp et al 1992, Baird et al 2004, Metcalfe et al 1996, Mitchell, Wood, and Collins 1999, Vaziri et al 1993, Wynn et al 1998, Yankiwski et al 2000).

[6]Children are too young to confidently interpret TL as the culmination of a *long-term* process, especially as rapid telomere attrition is expected in children and may signal normal growth rather than any pathological or pre-pathological process (Kuh 2006). The elderly may show little population variation in TL owing to survivor bias (Geronimus et al 2010).

racial or ethnic differences in health subject to serious residual confounding (Kaufman, Cooper and McGee 1997). Additionally, if weathering is sensitive to life experience and environmental context, the assumption of national sampling -- that a randomly selected low-income white, black or Latino is representative of all comparably low-income whites, blacks, or Latinos -- cannot be taken as axiomatic across time and place.

Two recent studies provide suggestive evidence that TL is sensitive to aspects of residential place. Theall et al. (2013), who studied TL in a convenience sample of African American children, found that those living in high-poverty census tracts or “high disorder” environments as defined by caregiver reports had lower salivary TL than others. Needham et al. (2014) analyzed data on middle-aged to elderly adults from New York and Los Angeles who were selectively healthy and economically advantaged. Their data included respondent-reported measures of neighborhood characteristics. After controlling for sociodemographic characteristics, the researchers found that while a neighborhood’s perceived lower aesthetic quality, safety, and social cohesion were associated with shorter leukocyte TL, neighborhood disadvantage, defined at the census tract level, was not. The study did not examine the structurally-rooted experience of residential setting or any intersectionality between residential setting and local population dynamics across racial/ethnic or socioeconomic groups.

More broadly, previous social epidemiological investigations of TL are designed under a common biomedical and social epidemiological paradigm that implicitly views race/ethnicity or socioeconomic group as risk factors, with health disparities modeled to be determined by some combination of race and socioeconomic group that are either additive in their effects or potential confounders of each other. Neighborhood studies often rely on census administrative units or test for independent effects using a small number of setting characteristics, rather than viewing neighborhoods holistically as places whose meaning to residents may be structured by historical factors, ideologically informed expectations, and social ties. When taking this risk factor approach to the social determinants of health, statistical correlations to a measured aspect of a setting or a racial/ethnic/socioeconomic group may obscure the more complex underlying processes that shape the observed patterning of population health (Geronimus 2000, Pearson 2008, Reed and Chowkwanyun 2011).

Given the nature of quantitative analysis, we cannot escape this common paradigm completely. However, we modify it by using a problem-driven and theory-based, rather than methods driven approach (Shapiro 2004); by considering key interactions; and by applying the analysis in one setting – avoiding the assumption that race or socioeconomic group is experienced similarly everywhere and has a universal impact on TL.

We focus on a high-poverty urban area because residents of such areas have the steepest age-gradient increases in morbidity and mortality from young through middle adulthood (Geronimus, Bound and Waidmann 1999). We consider a specific setting – Detroit – allowing us to account for the particular race-conscious history, current political economy, population dynamics, and use of residential space as staging ground for enacting race and class. The racial/ethnic population dynamics of note include the influx of Mexican

immigrants, and the changing composition of blacks and whites over time in absolute number, in proportion to each other, and in socioeconomic composition.

The current study is an interdisciplinary collaborative effort between researchers in population studies and in cellular biology and biochemistry. We employ a community-based participatory research (CBPR) approach, allowing us some grounding to interpret findings through the understandings of the participants, rather than only through pervasive characterizations of demographic groups or their behavior in the "power" literature (Shapiro 2004, p.202). Primary data collection among residents of these neighborhoods allowed us to explore distinct aspects and experiences of "disadvantage" among residents of one locality, and to consider whether stressors may crystallize differently among them to pattern a biomarker of age, rather than viewing disadvantage as comprising distinct, universal, or reified risk factors.

The Setting

Detroit offers an important opportunity to examine the relationship between health and urban disinvestment, race-based residential segregation, and additional stressors characteristic of poor urban settings (Schulz et al. 2000, Schulz et al. 2005a, Sugrue 1996). Population out-migration and economic divestment over the past five decades have reduced Detroit's population from 1.8 million in 1950 to just 706,585 in 2011. Out-migration of middle- and high-socioeconomic status whites has been particularly dramatic, reflected in the proportionate growth of African American residents from about 30% in 1950 to about 85% today. A larger fraction of Detroit children (about 60%) live below the poverty threshold than in any other large US city (Annie E. Casey Foundation 2014). Also, the Mexican population, a significant presence in Southwest Detroit since the 1940s, grew substantially with the post-1965 upsurge in immigration, and especially during the 1990s (Waters, Ueda and Marrow 2007).

Detroit's worsening problems of poverty, disinvestment, and a shrinking tax base occasioned by macroeconomic restructuring and the loss of manufacturing jobs toward the end of the 20th century has been intensified in the wake of the 2008 banking crisis. "Austerity urbanism" (Peck 2012), or extreme local fiscal retrenchment, culminated in 2013 with the state governor declaring a financial crisis in the city and appointing an emergency manager, and the subsequent filing of the largest Chapter 9 municipal bankruptcy in U.S. history. Between 1990 and 2013, Detroit reduced its municipal workforce by nearly half. The state's decreased commitment to funding public services in Detroit is also manifest in Detroit's renegotiated pay and pensions for the current and former public service workforce, its approximately 78,000 abandoned buildings, scarcity of working residential street-lights, and the residential water shut-offs that were recently protested by the UN as a human rights violation (UN Office of the High Commissioner for Human Rights 2014).

Theoretical framework and Hypotheses

We theorize that structurally rooted biopsychosocial processes contribute importantly to racial/ethnic or socioeconomic health inequality (Gee and Payne-Sturges 2004, Geronimus and Thompson 2004, Geronimus et al. 2010, Montoya 2013, Schulz et al. 2005a). This

position assumes that all U.S. residents are racialized actors in a pervasive paradigm by which historical legacies and ongoing structural processes empower or protect some racial/ethnic populations and marginalize others, advantaging the former in access to economic resources, opportunities, healthy residential or work environments, coping options, and social identity safety in specific settings (Geronimus 2013b). On a concrete and material level, historical race-conscious beliefs, policies, and practices are fundamental causes of poor urban health because they act to segregate blacks into low-income communities and to spur sustained disinvestment in these areas that ultimately affects all residents (Geronimus 2000). Black, white, Mexican, or other residents of high-poverty urban areas with predominantly black populations are likely to experience limited educational and socioeconomic opportunities; overburdened social networks; physical environments marked by urban decay and weak infrastructure; high levels of psychosocial distress; concern for physical safety; and psychological responses that include anger or hopelessness – any of which may contribute to poor health.

Additionally, and based on accumulating evidence, we theorize that health is affected not only by material resources and physical exposures, but also by social psychological forces affecting residents' sense of belonging, cultural affirmation, and identity safety (Geronimus 2013b). For example, Pearson (2008) highlights that social epidemiological frameworks and models based on conventional SES measures may mask heterogeneity across groups by overestimating the health benefits of income or education, per se, by underestimating the psychosocial and physical health costs of resource acquisition or human capital investment for some groups who face increasingly frequent "othering" encounters as they work toward becoming socially mobile (Colen et al. 2006, Viruell-Fuentes 2007), and by overlooking the value of alternative cultural orientations (James 1993) that may be more easily maintained by immigrants or in ethnic enclaves and racially homogeneous neighborhoods than in integrated settings.

At the population level, all of these health inputs – the material, the physical environmental, and the social psychological – are patterned historically and by present-day context and circumstance. Moreover, they are maintained or experienced through dominant ideologies such as the American Creed, with its emphasis on self-sufficiency and rewards for hard work and good character (Geronimus and Thompson 2004) and by dominant cultural frameworks (James 1993) that shape common understandings regarding who is deserving and who is "othered" in response to their race/ethnicity; income poverty, lack of power and prestige; or place of residence (Geronimus and Thompson 2004, Keene and Padilla 2010). The negative health impacts of these social psychological inputs occur through repeated activation of physiological stress mechanisms related to threatening contingencies of social identity (Geronimus 2013b) and microaggressions; sustained cognitive or emotional engagement with adversity (James 1994); and/or the use of health-harmful stress management behaviors including smoking and unhealthful eating (Jackson, Knight and Rafferty 2010), any of which could affect TL.

Conversely, strong social ties and networks among stigmatized racial/ethnic group members may protect health by providing identity safety in the face of negative stereotypes and an affirming alternative cultural framework to the dominant, marginalizing one (Geronimus

2000, Geronimus and Thompson 2004, James 1993, Pearson and Geronimus 2011, Viruell-Fuentes 2007). James (1993) suggests such cultural affirmation may become "progressively more important to preserving the health of its members as the group's (economic) strengths … diminish"(James 1993, p.135; Pearson and Geronimus 2011).

Thus, we conceptualize race as intersectional with class and with structure, and do not assume that the socially patterned health implications of structural processes always disadvantage those of color relative to whites, or those with lower household incomes relative to those with higher income, or that either of these categorizations (race or class) is monolithic. In this perspective, associations between race/ethnicity and health are theoretically mutable across class, historical moment, geographic place, and throughout the life course and can be moderated by the strength and adaptability of autonomous protections (Geronimus 2000). They are contingent on socially situated experience and knowledge, on the degree to which interactions and cues are interpreted as either affirmative of or threatening to social identity, and on the degree to which specific encounters activate, prevent, or curtail harmful physiological processes in specific settings (Geronimus 2013a,b)

Following from this theoretical frame, we hypothesize that the effects of material, psychosocial, and environmental stressors induce weathering among Detroit residents, which is expressed in accelerated biological aging as gauged by telomere length. We expect that the pattern of weathering across racial/ethnic and socioeconomic groups will reflect both the environmental conditions faced by all residents and the variation among groups in experiences that either affirm and protect or stigmatize and threaten social identity (Becares, Nazroo and Stafford 2009, Geronimus 2013b, Halpern and Nazroo 1999, James 1994, Pearson 2008, Viruell-Fuentes 2007, Viruell-Fuentes, Miranda and Abdulrahim 2012). We hypothesize the importance of this social psychological dimension in addition to the material, such that:

> Hypothesis 1: Interaction effects between race/ethnicity and poverty status on TL will be significant.

We hypothesize such interactions because, in a weathering framework, poverty is not only a marker for household material disadvantage, but also a marker for how the history of poverty and segregation in Detroit structures current systems of risk pooling, opportunities for cultural affirmation, and exposures to "othering" encounters. For example, while blacks have suffered the longest history of poverty and marginalization of the three racial/ethnic groups examined here, they also may benefit from greater ethnic density, which acts to reduce othering encounters, to enhance cultural affirmation, and enables the establishment of autonomous risk-pooling protections and deeply rooted social ties.

> Hypothesis 2: Neighborhood stressors will be associated with TL in absolute terms and in accounting for differences in TL across race/ethnicity x poverty groups.

Because we theorize that population differences in TL are expressions of a structurally rooted biopsychosocial process that works through physiological stress-process activation to accelerate aging in marginalized groups, we expect that measured stressors will impact TL and contribute to explaining any race/ethnicity x poverty interactions we find. Consistent with our theoretical approach, our goal is to estimate whether the measured stressors, writ

large, account for TL within or across populations rather than to delineate the precise degree that a given measured stressor mediates Detroit population-variation in TL. This goal recognizes that our data is cross-sectional and measurement of stressors can be imprecise and incomplete; and also that the underlying weathering theory suggests that any given stressor may be a marker for variation in stress process activation across populations, rather than a measure of a particular or uniform impact of the labeled stressor, or "risk factor", itself. While a finding that a specific measured stressor statistically accounts for a share of population differences in TL will be suggestive for future research, the first-order goal here is to examine whether unobserved heterogeneity in life experience across populations in specific settings is an important threat to the validity of interpretations of observed racial/ethnic population differences in TL as static or essential.

Data and Methods

Overview

The Healthy Environments Partnership (HEP), a community-based participatory research (CBPR) partnership, began in 2008 to field the second wave of a community survey designed to examine and address aspects of Detroit's social and physical environment. The study used a stratified, multi-stage probability sample of residents aged 25 and older living in three low-to-moderate income neighborhoods (Schulz et al. 2005a): Eastside, a segregated high-poverty black community; Northwest, a relatively more affluent, largely black but less segregated community; and Southwest, a high-poverty community with a racial/ethnic mix of residents, including the majority of the city's Mexican population, US-born and immigrant. (Southwest residents were oversampled to allow meaningful comparisons on the basis of ethnicity.) We were able to add venous blood collection to this second wave of HEP, completing data collection in 2011 and conducting subsequent laboratory analyses for TL using well-established methods. We estimated a series of nested models to regress TL (base pairs) controlling for age and sex, on potential stress indicators: socioeconomic characteristics, psychosocial stressors, neighborhood satisfaction, psychological response styles, and health behavior variables. To avoid spurious inferences, we applied a multiple-testing procedure (Benjamini and Hochberg 1995) and conducted robustness checks on study findings using matching methods (Hausman 1978, Rubin 1973a, Rubin 1973b)

Data Collection

HEP participants who expressed interest in the telomere study were given information regarding the purpose of the study and participation requirements. After completing the informed consent process and an in-person interview, 239 (92% of those asked and 52.0% of the full wave 2 participants) enrolled in the telomere portion of the study. Of these, 227 (87% of those expressing interest; 49% of all HEP 2008 survey participants) provided blood samples for analysis; the other 11 individuals ultimately declined to give a blood sample or were unable to complete the blood draw for medical or physical reasons. We also excluded from the current analysis the 25 participants (10% of those expressing interest; 5% of all HEP 2008 survey participants) whose self-reported race/ethnicity was other than white, black, or Mexican (n = 3); or reported that their education was completed outside of the U.S. (n = 4). Multiple imputation was used to generate missing poverty-to-income ratios (PIR);

however, 7 of these participants had inconsistent imputation results and we, thus, also exclude 18 individuals who did not provide income data. The final sample for the current analysis included 202 participants. In terms of race/ethnicity, gender, education, and neighborhood of residence, participants in the TL portion of the study were not significantly different from individuals who were unable or declined to participate, but they were older (mean age 49.7 years vs. 43.7 years, $p = 0.04$) and less likely to have a household income of $70,000 or higher (10.0% vs. 28.6%, $p = 0.01$).

A phone call or home visit was made the evening before scheduled blood collection to remind participants of the protocols and answer participant questions. [7] All blood collection tubes and accompanying paperwork were labeled with identification numbers only and all lab personnel were blind to respondents' identities and sociodemographic characteristics. Blood for TL analysis was processed for shipment to the Blackburn Lab at the University of California, San Francisco. Specifically, peripheral blood mononuclear cells (PBMCs) were pelleted, washed, flash-frozen in liquid nitrogen, and stored at −80°C; then they were packed on dry ice for shipment to the lab using FedEx Overnight services and adhering to all requirements for the shipment of blood products.

Outcome Variable: Leukocyte Telomere Length (TL)

All measurements were made in the Blackburn Lab using the telomere length measurement assay that was adapted from the published original method by Cawthon (Cawthon 2002, Lin et al. 2010). The Blackburn Lab has shown excellent reliability in measuring TL, expressed here in numbers of base pairs, with correlations of $r = 0.999$ in duplicate samples. Further, the lab has shown excellent reproducibility, with an inter-assay coefficient of variability of 6.1. (See appendix for details of the telomere thermal cycling profile, primers used, and controls for inter-assay variation.)

Explanatory Variables

Age was measured in years based on the respondent's date of birth and the date of the study interview. *Race/ethnicity* and *Sex* were self-reported. *Poverty-to-Income Ratio (PIR)* was calculated based on the respondent's household income relative to the federal poverty level designated for the participant's household size, with a PIR value of >1 for incomes greater than the household size-specified federal poverty level, and <1 for incomes below it. *Highest level of education* included four categories: less than a high school degree; high school degree or general equivalency degree; some college or an associate degree; and bachelor's degree or higher. *Safety stress* was assessed using three questions regarding how often

[7]Critical to the success of the project was developing data collection techniques appropriate for a largely low income multi-ethnic urban population. The project director, a Spanish speaking Black American male with training and experience in Community Based Participatory Methods, directly supervised the collection of every data point. The project protocol required a phone call the evening before the blood draw to remind participants of the scheduled data collection, the fasting protocol and the importance of not drinking alcohol or smoking cigarettes 12 hours before the collection. To facilitate adherence to the fasting protocol all blood collection was scheduled between 8 and 10:30 a.m. and a light breakfast was provided after the blood draw. A health assessment data collection survey at the time of the blood draw appointment directly asked the participants if they had eaten in the past 12 hours. Those who had eaten too recently were rescheduled for the blood draw on another morning. During the initial 6 weeks of data collection approximately 15 percent of participants did not meet the fasting protocol, and, thus, could not complete blood collection at the scheduled time. The project director then instituted a practice of home visits the night before scheduled blood collection to remind participants of the protocols and answer any questions participants might have. Implementation of these home visits reduced protocol non-adherence and resultant rescheduling to approximately 5 percent.

respondents worry about safety in their home or neighborhood. Responses ranged from 1 (never) to 5 (always). *Perceived unfair treatment* was measured using 5 items from the *everyday unfair treatment* scale (e.g., treated with less courtesy than others, treated as if you're not as good as others), modified from the Detroit Area Study (Jackson and Williams 2002, Williams et al. 1997). Responses for frequency of perceived unfair ranged (never) to 5 (always).

A series of 7 questions using a 5-point agreement scale assessed respondents' perceptions of their *Neighborhood physical environment* – both negative physical features of their neighborhood (e.g., heavy car or truck traffic; air pollution; contaminated land; vacant homes and lots in the neighborhood; noise pollution) and positive physical features (well-maintained homes; clean streets, sidewalks, and vacant lots) (Israel et al. 2006). Higher scores on this scale reflect better perceived neighborhood physical environment, with more positive and less negative features.

The *Negative social interactions* scale, adapted from Schuster, Kessler, and Aseltine (1990), quantified the frequency with which friends and family members either 1) made too many demands on the respondent or 2) criticized the respondent or the respondent's behavior. Responses ranged from 1 (never) to 5 (always).

Neighborhood satisfaction was coded using a 5-point Likert scale characterizing level of agreement to a single item: "I would move out of this neighborhood if I could." The item was reverse-coded such that in the Tables, "strongly agree" implies high levels of neighborhood satisfaction.

Consistent with our orientation toward lived experience, the neighborhood-level questions (neighborhood physical environment, neighborhood satisfaction) were in a section of the HEP survey that did not define "neighborhood" explicitly. Participants responded according to their own definition.

Anger out was measured using a 4-item scale (Spielberger et al. 1985) regarding how often a person argues with others, strikes out, says nasty things, or loses his or her temper while feeling angry or mad. *Hopelessness* was measured using level of agreement to two items from the Beck Hopelessness scale (Beck et al. 1974): "The future seems to be hopeless, and I can't believe things are changing for the better" and "I feel it is impossible for me to reach the goals I strive for." Higher scores for anger or hopelessness reflect more frequent expressions of anger or stronger agreement with hopelessness statements, respectively.

Using survey items derived from previous studies (Frazier, Franks, and Sanderson 1992, Gentry et al. 1985), respondents who reported never regularly smoking tobacco products were the reference category relative to current regular smokers and to former regular smokers. During the interview, weight was measured using an electronic scale; height was measured with a tape measure. Adiposity was measured using *body mass index* (BMI), calculated using the standard procedure of dividing weight in pounds by height in inches squared, then multiplying by a conversion factor of 703. According to standard conventions (National Institutes of Health 1998), BMIs were categorized as "obese" (BMI ≥ 30.0),

"overweight" (BMI = 25.0–29.9), and a referent group of all others (BMI ≤ 24.9, including four "underweight" participants with BMIs ≤ 18.5)[8].

Statistical Analyses

We regressed TL (base pairs) on the above set of variables in a series of nested models. All models included respondent race/ethnicity, age, and sex. We also controlled for continuous PIR for comparison to other studies. However, given earlier evidence of interactions between race/ethnicity and poverty status, we stratified each racial/ethnic group by PIR and sequentially added education, psychosocial stressors, neighborhood satisfaction, response styles, smoking, and BMI.[9]

Because the HEP data do not represent a simple random sample, we accounted for stratification and clustering by employing the survey data commands built into STATA. Weights were created to ensure appropriate representation of racial and ethnic groups across SES in the sample, and were applied to adjust for probabilities of selection within socioeconomic strata and non-response bias, and to match the sample to Census 2000 population distributions of the study communities (Schulz, et al., 2005a). Although the proportion of missing data was low, we used multiple imputation procedures derived from Bayesian models (Barnard, Rubin and Schenker 2001) to impute missing values via the %IMPUTE routine (Imputation and Variance Estimation software, Ann Arbor MI) in SAS 9.1 (SAS Institute Inc, Cary NC, 2002–2003). Multiple imputations allowed us to use the complete case approach and thus obtain robust standard error estimates (Rubin 1996, Schafer 1997).

Results

Sample description

As shown in Table 1, the household income distribution is far more comparable across blacks, whites, and Mexicans in the Detroit sample than in the nation as a whole.

As shown in Table 2, poverty rates among study participants aged 25 to 64 years are more than double the national rate for blacks and Mexicans, and six times the national rate for whites. Poverty rates in Detroit are roughly comparable across race/ethnic groups, at 55.6% for whites, 50.0% for blacks, and 52.3% for Mexicans, while nationwide twice as many blacks and Mexicans are in poverty than whites. Greater percentages of study participants also report having less than a high school education than nationally, with the difference especially stark among white participants, who are almost four times more likely to have less than a high school education than their counterparts nationwide. Also, compared to national averages, the study sample has smaller percentages of the youngest and oldest adults – results consistent with younger adults moving out of the financially-strapped city in

[8]As robustness checks, we alternated this measure of BMI with a continuous BMI measure, with a categorical BMI variable that separated out the four underweight individuals, and also with a continuous variable measuring waist circumference.

[9]We initially performed models separately by sex including estradiol in models for women. As inclusion of that variable did not affect the findings, we dropped it and report models including men and women together.

search of better jobs or better resourced residential areas (Geronimus, Bound and Ro 2014) and with older adults experiencing excess mortality (Geronimus, Bound and Colen 2011).

The mean telomere length in the study sample, adjusted for age and sex, was 5,624 base pairs, which is lower than, but within one standard deviation of, the mean TL found in a study by Needham, et al. (2013) that used a national sample with a similar age distribution, and also conducted TL measurements in the Blackburn Lab.

Regression Results

Tables 3 and 4 show coefficient estimates from the nested regression models. Consistent with expectations based on prior research, TL decreased with age (−16.54 base pairs, p<.00) and was longer among females than males (199.76 base pairs, P<.01). In the baseline (Model 1) – and as illustrated in Figure 1 (Panel A for women, D for men) we found no statistically significant differences in estimated TL among whites, blacks, and Mexicans in the Detroit sample.

When we entered PIR as a continuous variable (Table 3, Model 2'A), we found that having a higher PIR was associated with longer TL (15.19 base pairs, p<0.01). To test the proposition that the experience of being in poverty and coping options may differ across racial/ethnic populations, we added interaction terms for race/ethnicity x PIR as our income variable (Model 2'B). We found that the positive effect of PIR was less for blacks than for whites (15 increased based pairs per unit PIR for blacks versus 44 for whites); and became a negative effect for Mexicans, among whom increasing PIR was associated with *shorter* telomeres. When we tested to see whether the three interactions of race/ethnicity x PIR were jointly statistically significantly different from each other, they were marginally (p<.10).

These differences by race suggest that the effect of income varies by race/ethnicity and may not be linear. For subsequent specifications (Models 2–8 in Table 4) we maintained a race/ethnicity x PIR interaction term. We chose a categorical PIR measure (PIR< v. >1), rather than using alternative functional forms of the continuous PIR variable for theoretical and practical reasons, and for ease of presentation.[10] The results shown in Model 2'B, which include continuous PIR x race/ethnicity terms, suggest that finding a significant interaction between PIR and race/ethnicity is not an artifact of turning to a dichotomous PIR measure.

Model 2 estimates show that living below the poverty level has a large interactive effect for whites (−327.25 base pairs, p<.01); an insignificant but negative interactive effect for blacks; and a positive interactive effect for Mexicans that approached statistical significance (194.34 base pairs, p<.14). Model 3, which includes education measures net of age, sex, and race/ethnicity x poverty status, indicates that TL is associated with education. Those with less than a high school education have shorter TL than college graduates (−208.11 base pairs, p<.08), while those with a high school degree, GED, or some college have shorter TL than college graduates but longer than high school drop outs. Taking education into account

[10]Theoretically, because we hypothesize that the effects of social identity on TL vary with context, a flexible nonparametric specification is appropriate. It is also practical given the sample sizes available. Because of the relative homogeneity in the distribution of PIR across the three racial/ethnic groups there is little concern that taking a nonparametric approach biases results as it might very well if national data were being analyzed, where the distribution of and percent PIR varies substantially by race/ethnicity.

also has suggestive implications for the associations between PIR and TL by race/ethnicity. Among whites or blacks, their educational distribution is estimated to reduce the association between PIR and TL by about one-third; while for Mexicans accounting for education strengthens the association between poverty and longer TL (235.55 base pairs, p <.09). The differences between the models within race/ethnicity are not statistically significant and for Mexicans, the joint test for differences between the three race/ethnicity x poverty interaction terms remains statistically significant (p <.04).

Psychosocial stress measures are added in Model 4. We found opposite and significant effects on TL for negative social interactions and safety stress: an increase of 67.13 base pairs (p<07) for safety stress and a decrease of 55.74 base pairs (p<.06) for negative social interactions. Neither negative physical environment nor perceived unfair treatment showed an independent effect on TL in this sample, net of the already measured variables. The three race/ethnicity x PIR interaction terms remain jointly statistically significantly different (p<. 04) in this model.

In Model 5 we see that those expressing greater neighborhood satisfaction have longer TL than those reporting less neighborhood satisfaction. The estimated association between TL and reporting the highest level of neighborhood satisfaction is an increase of 215.39 base pairs (p<.02) relative to having low neighborhood satisfaction. Controlling for neighborhood satisfaction brings the relationship between poverty and TL closer among the three racial/ ethnic groups, though they remain marginally statistically distinct (p<.10).

As shown in Model 6, the independent association of a participant's angry response style and TL was in the expected direction, but was not statistically significant. Hopelessness was associated with a TL reduction of about 40 base pairs (p<.06). Moreover, inclusion of these variables accounted for part of the effect of poverty on TL and reduced the differences in that effect by racial/ethnic group to insignificance. Note one implication of these findings is that poor Mexicans are less prone to report feeling anger or hopelessness than nonpoor Mexicans.

Net of other variables, being a current or former smoker was estimated to reduce TL, but not significantly (Model 7). Nor did it significantly alter the relationship between TL and poverty across the race/ethnic groups. In Model 8, we found obesity was independently and negatively associated with TL (−145.22 base pairs, p<.05).

In the final model (Model 8), the variables that remained statistically significantly associated with TL were age, and, in declining magnitude, white PIR<1 (shorter TL), strong neighborhood satisfaction (longer TL), female sex (longer TL), obesity (shorter TL), safety stress (longer TL), negative social interactions (shorter TL), and hopelessness (shorter TL). To assess whether the measured stressors as a group account for the race/ethnicity x poverty interaction, we tested the difference between the estimated effect of poverty status on TL within race/ethnicity between Models 2 and 8. The results are illustrated in Figure 2. Compared to estimates in Model 2, the measured variables in Model 8 account for a substantial part, but not all, of the difference in TL between poor and non-poor whites (about one-third) (p<.09); fully account for the smaller initial difference between poor and non-

poor blacks (<.04); and increase the advantage of poor Mexicans compared to non-poor Mexicans by about 20 percent (p<.02). Here, poor Mexicans have longer TL on average than non-poor whites and, therefore, net of covariates, the longest TL of any group studied.

Using matching methods (Rubin 1973a, Rubin 1973b) to separately estimate the mean TLs for black and Mexican participants using the same age and gender distribution as our white participants produced similar estimated mean TLs for blacks and Mexicans, suggesting that results are not driven by functional form and are applicable to differences across groups in the full distribution of TL, not only the means.

Discussion

Within and across racial/ethnic groups in our Detroit sample, TL varied according to PIR, education, safety stress, negative social interactions, neighborhood satisfaction, hopelessness, and obesity. Net of other variables, we found no independent association with TL for smoking, being overweight (but not obese), or perceiving everyday unfair treatment. Most important, as hypothesized, race/ethnicity interacted with PIR to affect TL differently across racial/ethnic groups.

Based on a few studies with highly select samples that included few to no social covariates, some concluded that blacks have longer telomeres than whites and turned to biological speculations to account for their findings, rather than consider possible effects of selection bias or residual confounding (Adler, et al. 2013; Aviv et al. 2009; Hunt et al. 2008; Needham et al. 2013; Zhu et al. 2011).[11] This study shows that telomere lengths of racial/ethnic groups in Detroit are not consistently ranked and vary depending on context and experiences not accounted for in other research. Once we stratified each racial/ethnic group by PIR, blacks' average TL was shorter (but not statistically significantly) than the TL for non-poor whites, and the greatest variation was between poor and non-poor whites (see Table 3 or Figure 1). More generally, we found that TL for all racial/ethnic groups was sensitive to the addition of socioeconomic, psychosocial, coping, and biobehavioral variables, suggesting some earlier investigators may have too quickly assumed that estimates of racial differences in their studies are universal (Hunt et al. 2008). Given the sensitivity of TL to the measured covariates, unmeasured differences likely account for the remaining disadvantage of poor whites and the increased advantage of poor Mexicans in Detroit, as well.

Several studies find an association between education and TL. In this study, the association between having less than a high school education and TL was strong, and it intensified in models including psychosocial and neighborhood stress variables. However, once hopelessness and obesity were controlled, the association declined, suggesting that, in Detroit, these factors partially account for the association between TL and having less than a high school education. This constellation of factors (not completing high school, being obese, feeling hopeless) may characterize those who are among the most marginal

[11]For example, they argued that since the TL measure used is an average of telomere length across all leukocyte cell types, future research should assess TL in single cell types to consider the possibility that race-based heterogeneity in cell-type TL explains the longer telomeres in Blacks than Whites they described.

community members or are struggling the most. Notably, net of poverty status, we found the negative association between having less than a high school education and TL was strongest for whites (−465.37 base pairs, p<.01). One possibility is that, at least in this context, poor whites may lack the collective strategies for pooling risk that buffer the negative health effects of material deprivation and stigma for other low-income groups.

Some studies that found an association between education and TL found no association between TL and income. Adler et al. (2013) and Steptoe et al. (2011) studied older adults – ages at which income differences are smaller than they are earlier in adult life and when education better represents socioeconomic position than current income. Needham et al. (2013) used a continuous poverty-to-income variable to gauge main income effects. This approach may have obscured a threshold effect on TL of a very high level of material hardship relative to less impoverished circumstances, and also may have masked the racial/ethnic diversity in the PIR-TL association that we identified. While we estimated a positive effect when we entered PIR as a continuous variable, the estimated PIR coefficient masked important interactions between PIR and race/ethnicity that vary in magnitude and direction.

We found no evidence that perceived unfair treatment was independently associated with TL. Hypersegregation in the Detroit metropolitan area, and particularly in our survey areas, means (1) that most blacks in our sample live almost exclusively with other blacks (97% of Eastside Detroit residents are black), or are the majority group in integrated neighborhoods (e.g. 70% of Northwest Detroit residents are black); (2) that whites are a clear minority in all of our Detroit areas (ranging from 2% to 21% of residents); and (3) that Mexicans are concentrated in Southwest Detroit, where they comprise 60% of the residents. This high racial/ethnic density for Detroit Blacks and Mexicans may reduce their encounters with overt discrimination and provide greater identity safety and social support – which may blunt the impact of unfair treatment on health (Becares, Nazroo and Stafford 2009, Bhugra and Becker 2005). Our findings suggest that studies of population health disparities using measures of perceived unfair treatment to represent psychosocial stress or exposure to racism may not: (1) adequately encompass the many psychosocial stressors that differentially impact health by race/ethnicity, or (2) measure the most salient indicators of racism in circumstances where structural rather than interpersonal racism has a predominant influence on population health (Gee 2002, Jones 2000).

Taken together, these study findings point out the limitations of conventional risk factor approaches to examining racial/ethnic differences in TL, and suggest that future research should construe race/ethnicity as a contextually fluctuating conceptual variable (Brubaker 2004); rather than as a bounded and reified entity as is more typical in the “disparitarian” literature (Reed and Chowkwanyun 2011). The weathering hypothesis interprets TL as a marker of accelerated aging that is biomechanistically impacted by repeated or chronic physiological stress-process activation. In the Detroit context, these stress processes are conceptualized as having been initiated by: physical environmental threats and material hardship attributable to a history of race-conscious ghettoization and urban disinvestment; a current political economy guided by austerity urbanism; and interpersonal encounters or cues that are experienced as threats to identity safety. The stress potential of these circumstances is heightened when they are interpreted through racial stigma, cultural

oppression, or acceptance of the American Creed, and reduced by the availability of cultural affirmation, identity safety, and collective networks where material resources and risks are pooled (Geronimus and Thompson 2004). How might we understand the race/ethnicity x PIR interactions through this analytic prism?

Only whites showed results consistent with pervasive social epidemiological understandings – that is, the poor had shorter TL than the nonpoor – and this disparity was significantly reduced in models including the measured covariates. What might explain this? Perhaps, with the exodus of most whites and many jobs from Detroit, the shrinking benefits of labor union membership and public pensions, and the overall reduction in taxation-based city services, the poor whites who remain are particularly adversely affected by the social and ecological consequences of austerity urbanism. Lacking the financial resources, social networks, and identity affirmation of the past, remaining Detroit whites may have less to protect them from the health effects of poverty, stigma, anxiety, or hopelessness in this setting (Geronimus 2000; Pearson 2008). To the extent that whites accept the American Creed ideology, they may be acutely sensitive to their perceived socioeconomic failures or, possibly, experience a version of status incongruity between expectations of white privilege and current circumstance that may be health harmful. Systematic exploration of such speculations may be a fruitful avenue for continued research.

Among blacks, we saw less differentiation in TL by PIR and found evidence of a different experience of their neighborhood compared to whites or Mexicans. Much research suggests the separation between poor and nonpoor blacks in everyday life is less marked than between poor and nonpoor whites (Geronimus and Thompson 2004, Helflin and Pattillo 2006). Not only do blacks tend to have greater residential proximity owing to residential segregation, but often poor and the nonpoor blacks are members of the same families and social networks, practice reciprocal obligations, or have similar experiences of cycling between low and moderate incomes. Income instability among middle class blacks reflects job insecurity (Pattillo-McCoy 1999), a relative lack of conventional assets or wealth to tide them over in rough times (Shapiro 2004), or a network level division of labor, whereby some are expected to contribute to family economies through income generating work; others contribute by seeing to the family caretaking needs that facilitate the employment of others; and still others provide important services and skills as barter exchange (Stack 1974; Geronimus 1987; Burton and Whitfield 2003; Hicks-Bartlett 2000). Given deep cross-class affective ties, a strong collective ethos, elastic household boundaries, and shared resources, PIR measured at the individual household level may not be an apt way to represent or categorize differences in material hardship or life stressors among Detroit blacks.

The associations between TL and perceptions of neighborhood physical environment and neighborhood satisfaction were strongest for blacks, with more positive perceptions associated with an increase of 78.30 base pairs ($p<.03$) and high satisfaction associated with 387.63 base pairs ($p<.00$). Also, improved neighborhood physical environment was positively associated with TL in blacks (78.30 base pairs, $p <.03$). When we disaggregated our findings by race/ethnicity in exploratory analyses, we found the counterintuitive positive association between safety stress and TL (97.09 base pairs, $p<.00$) solely pertained to blacks. Perhaps safety stress, physical environment, and neighborhood satisfaction tap into a more

global construct of how black participants experience Detroit neighborhoods, which on balance may be more positively than for white or Mexican participants. Previous research in Detroit found that black residents report significantly lower levels of social and physical environment stress than do white residents living within the same neighborhoods (Schulz et al. 2008).

Mexicans reported the highest levels of safety stress, which was marginally associated with shorter TL (−97.78 base pairs, $p<.11$). This may, in part, reflect that data collection coincided with a period of heightened surveillance by immigration officials in Southwest Detroit. This is an example of a contingency of social identity that is population specific and may have health impacts.

Our small sample size precluded disaggregating the Mexican population by nativity for statistical analyses, but we note that Mexicans in the non-poor group were disproportionately U.S. born, while those in the poor group were disproportionately foreign born. Moreover, 80% of all poor Mexicans reported that Spanish was the most commonly spoken language in their homes, regardless of nativity. Spanish speaking in the home may signal some protection from marginalization by offering an affirming cultural framework (James 1993; Pearson 2008). As James (1993) first suggested, Mexicans who are better able to maintain an alternative cultural framework to the dominant U.S. one that marginalizes them – in this case, as poor Mexicans in Detroit may have done to a larger extent than non-poor Mexicans – gain some protection from the health impacts of psychosocial and neighborhood stressors (Geronimus 2013b; Pearson 2008; Viruell-Fuentes 2007; Viruell-Fuentes, Miranda, and Abdulrahim 2012). The finding that poor Mexicans reported feeling less anger or hopelessness than nonpoor Mexicans might be consistent with this finding.

Studies document that poor Mexican immigrants often have better health than other poor people in the U.S. (Markides and Eschbach 2005). However, Mexican immigrants' health advantage is reduced with years of residence in the U.S. and disappears altogether in the next generation (Collins et al. 2001, Kaestner et al. 2009) perhaps as integration heightens the exposure to and impact of othering experiences (Viruell-Fuentes 2007). As new immigrants reside in the United States longer, or as the progeny of immigrants are raised in the United States, they become aware of and attuned to US racial hierarchies and ideologies, and are vulnerable to the physiological impacts of racialized contingencies of social identity, such as the common presumption that they are not legal residents of the US, or truly American (Garcia 2004). Navigating such prejudices and stereotypes in daily interactions in integrated settings may activate physiological stress processes.

In interviews with first- and second-generation Mexican women in Detroit, Viruell-Fuentes (2007) found consistent evidence that the second generation (US-born) Latinas reported more experiences of discrimination and othering than did their immigrant parents. She attributed this in part to the protective effects of residence in ethnic enclaves for the immigrant generation, but also to her finding that the second generation lived with "a frequent, cumulative, and ongoing burden of exposure to 'othering'" (Viruell-Fuentes, 2007 p. 1531), noting that: "the long-term labor of constructing an ethnic identity under a

stigmatizing racial structure and the accumulation of ‘othering’ experiences over the life course might take a toll on the health of the second and later generations”.

Trade-offs & Limitations

Consistent with our theoretical objectives, we traded off analyzing a large or nationally representative sample for a primary data collection effort that offered the opportunity to study a specific place; to include comparably economically disadvantaged whites, blacks and Mexicans; and to include a broad array of measures to better reflect a complex theoretical model of racial/ethnic and socioeconomic health inequality. Although this choice limits the generalizeability of our findings, it is the only way to study a sample of blacks, whites, and Mexicans who are comparable on critical dimensions.

We considered the alternative of analyzing an existing nationally representative sample, and re-weighting the population to have an income distribution similar to the one we study here and comparable across whites, blacks, and Mexicans. It is worth noting that to do so, the effective sample size for whites in the national data set would be reduced by 85%. Moreover, the remaining whites would be dispersed across the country, and in large part regionally separate from economically comparable blacks or Mexicans, increasing the threats of unobserved heterogeneity and residual confounding.

The comparability of the income distribution and poverty rates across blacks, whites, and Mexicans in our sample increased the efficiency of the sample for testing our hypotheses and allowed us to consider the most disadvantaged urban whites, a demographic group missing from most health research. It also allowed us to consider whether estimated race effects in TL equations are likely to reflect residual confounding. By estimating nested models, we eliminated important shares of the TL differences observed across and within racial/ethnic groups – an important contribution that poses a serious empirical challenge to those who interpret race-associated TL differences as essentially racial. However, we do not claim to have fully accounted for all of the unobserved heterogeneity across or within racial/ethnic, socioeconomic, or geographic groups.

The survey areas do not include very affluent communities or suburban or rural areas, contrasts that would have allowed us to explore our hypotheses more fully. The sample size for the current study was also too small for extensive analyses separately by race, poverty group, nativity or gender. As with most TL research to date, this study used cross-sectional data. A more revealing test of the weathering hypothesis would be to follow cohorts from early life through middle age.

Our findings suggest that unobserved heterogeneity bias is a major threat to the validity of causal interpretations of associations between race/ethnicity, socioioeconomic characteristics, place of residence and TL. Investigators of TL and health disparities must continue to wrestle with the ways varying social histories and current social realities contribute to racial/ethnic differences in health, ideally in interdisciplinary research teams and/or community-based research partnerships (James 1994; Geronimus 2013a,b; Geronimus and Thompson 2004; Kaestner et al. 2009; Pearson 2008; Schulz et al. 2008; Viruell- Fuentes 2007). Though more needs to be learned about what underlies the social

patterning of TL across and within racial/ethnic groups in Detroit, our findings are consistent with a conceptual understanding of race/ethnicity as contingent, and of health implications as context dependent and fluid (Geronimus 2013b). Recent decades have witnessed growing income inequality, large waves of immigration, newly emergent or intensified xenophobia, tensions around whether our vision for a post-racial society should be race-blind or multicultural, and policy disagreements over whether to respond to severe economic crisis with austerity measures or with stimulus and infrastructure investment. In this context, it may be particularly necessary for those hoping to eliminate health inequality to go beyond reliance on static and binary conceptions of the interrelation of race and health – to acknowledge that marginalization of any identified social group may have population health repercussions, to broaden the theories of how such marginalization is enacted, and to view marginalization and its consequences as dynamic and relational.

Appendix A: Laboratory Procedures used for Measuring Telomere Length

Regarding the PBMC purification, we used the BD Vacutainer® CPT™ Cell Preparation Tube with Sodium Citrate. Specifically, we spun the cells in a horizontal rotor (swing-out head) for 30 minutes at 1500 to 1800 RCF (Relative Centrifugal Force) according the manufacturer's instruction. According the manufacturer, this should yield mononuclear cells (https://www.bd.com/vacutainer/products/molecular/citrate/).

We used the telomere length measurement assay that was adapted from the published original method by Cawthon (Cawthon 2002, Lin et al. 2010). The telomere thermal cycling profile consisted of: *Cycling for T (telomic) PCR: denature at 96°C for 1 second, anneal/ extend at 54°C for 60 seconds, with fluorescence data collection, 30 cycles. Cycling for S (single copy gene) PCR: denature at 95°C for 15 seconds, anneal at 58°C for 1 second, extend at 72°C for 20 seconds, 8 cycles; followed by denature at 96°C for 1 second, anneal at 58°C for 1 second, extend at 72°C for 20 seconds, hold at 83°C for 5 seconds with data collection, 35 cycles*.

The primers for the telomere PCR are *tel1b* [5'-CGGTTT(GTTTGG)5GTT-3'], used at a final concentration of 100 nM, and *tel2b* [5'-GGCTTG(CCTTAC)5CCT-3'], used at a final concentration of 900 nM. The primers for the single-copy gene (human beta-globin) PCR are *hbg1* [5' GCTTCTGACACAACTGTGTTCACTAGC-3'], used at a final concentration of 300 nM, and *hbg2* [5'-CACCAACTTCATCCACGTTCACC-3'], used at a final concentration of 700 nM. The final reaction mix contains 20 mM Tris-HCl, pH 8.4; 50 mM KCl; 200 M each dNTP; 1% DMSO; 0.4x Syber Green I; 22 ng E. coli DNA per reaction; 0.4 Units of Platinum Taq DNA polymerase (Invitrogen Inc.) per 11 microliter reaction; 0.5 – 10 ng of genomic DNA. Tubes containing 26, 8.75, 2.9, 0.97, 0.324 and 0.108ng of a reference DNA (from Hela cancer cells) are included in each PCR run so that the quantity of targeted templates in each research sample can be determined relative to the reference DNA sample by the standard curve method. The same reference DNA was used for all PCR runs.

To control for inter-assay variability, 8 control DNA samples are included in each run. In each batch, the T/S ratio of each control DNA is divided by the average T/S for the same DNA from 10 runs to get a normalizing factor. This is done for all 8 samples and the

average normalizing factor for all 8 samples is used to correct the participant DNA samples to get the final T/S ratio. The T/S ratio for each sample will be measured twice. When the duplicate T/S value and the initial value vary by more than 7%, the sample is run the third time and the two closest values are reported. To estimate the LTLs in terms of base pairs, a standard conversion formula of 3274+2413*(T/S) was established by examining a set of control DNA and comparing its T/S ratio to its number of telomere base pairs measured via Southern blot.

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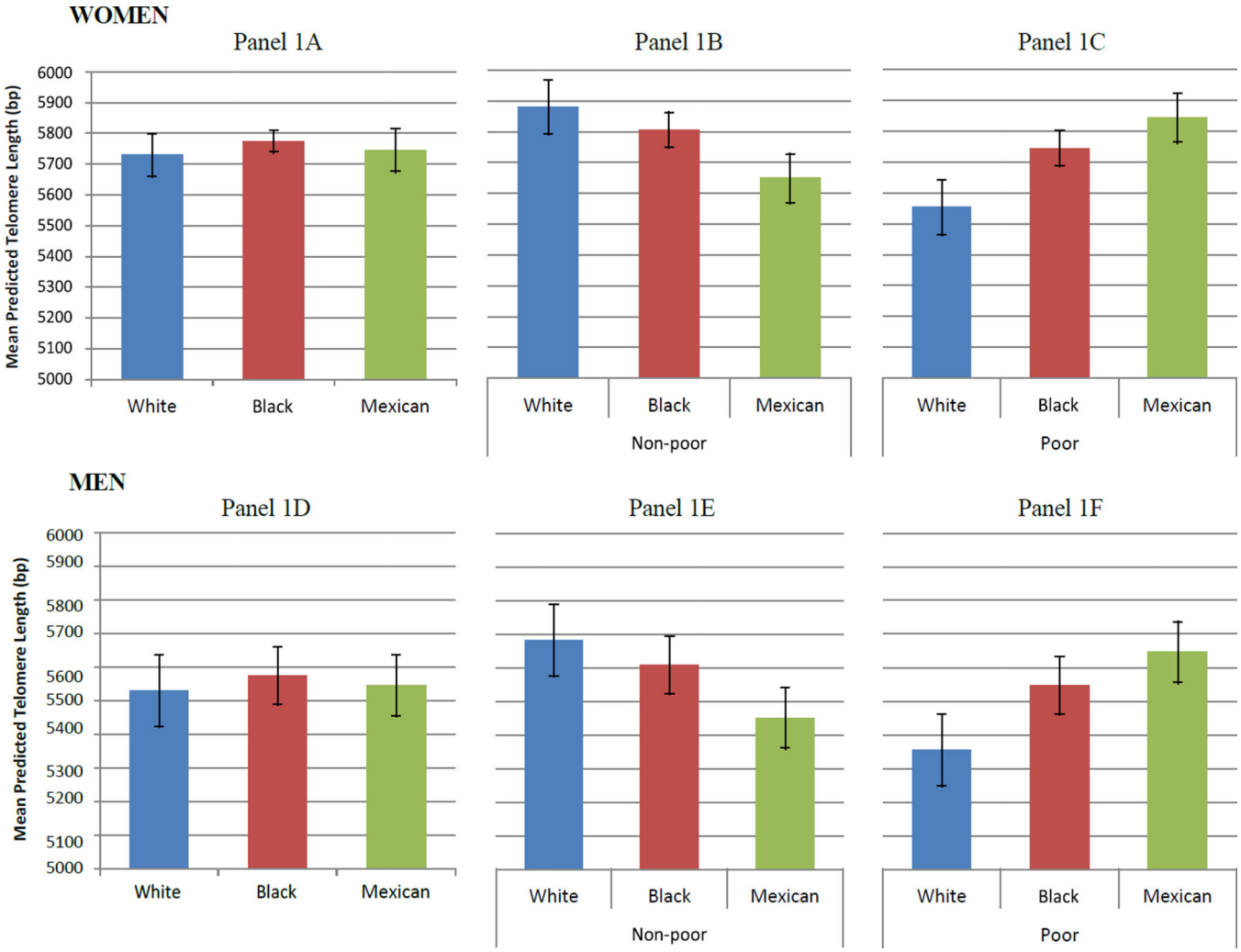

Figure 1.

Predicted telomere length for 45-year-old participants adjusting for race/ethnicity (left), and also stratifying by poverty status (center; right)

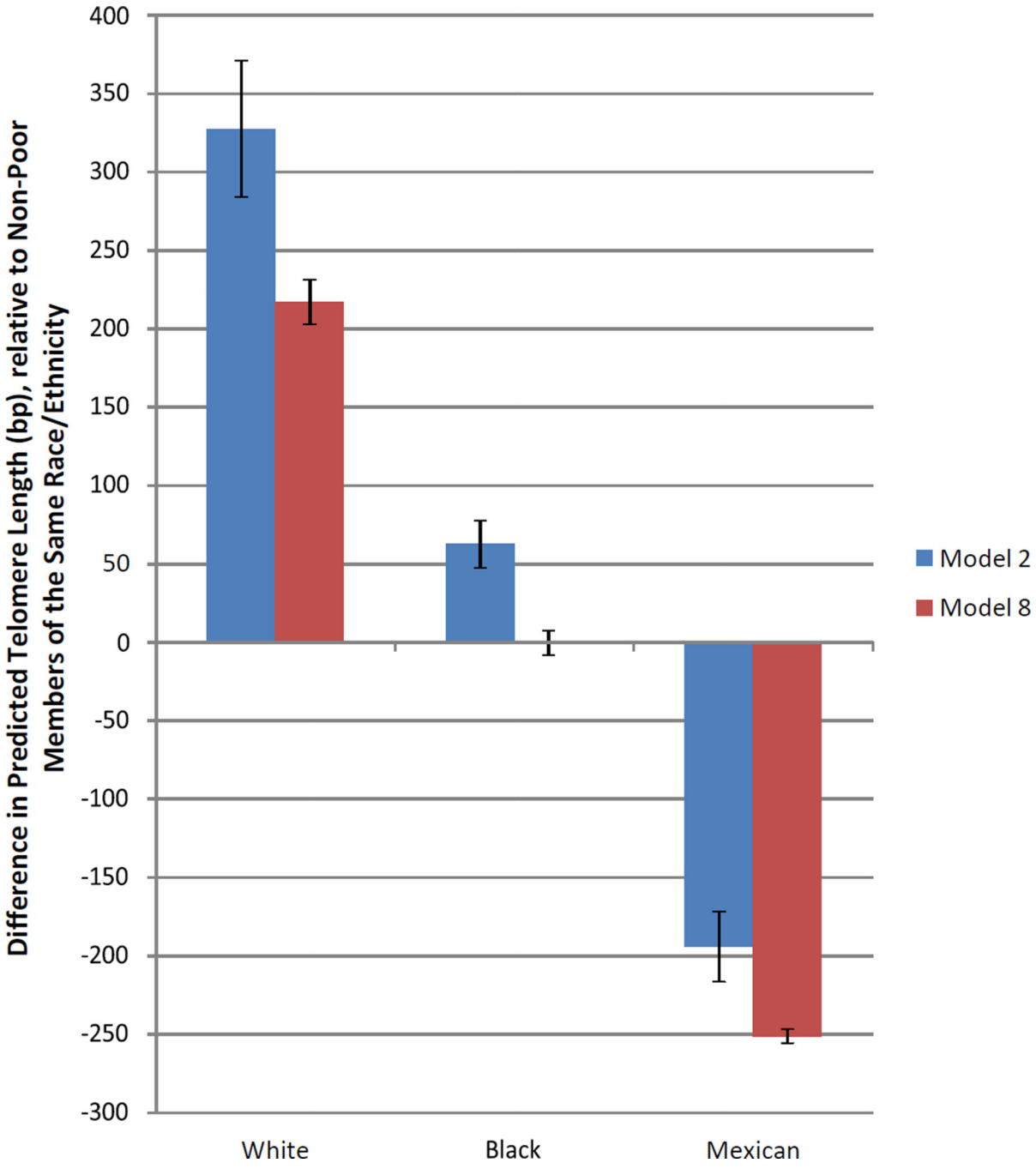

Figure 2.

Predicted telomere length differences among the poor compared to the non-poor within each race/ethnicity group, in Model 2 and Model 8

Notes: Model 2 controls for age and sex; Model 8 adjusts for all measured covariates.

Table 1

Household income distributions for Whites, Blacks, and Mexicans; National American Community Survey (ACS) estimates, 2008-2010, compared to Detroit Telomere participants

	National ACS Estimates			Detroit Telomere Study		
% of Households	Non-Mexican White	Non-Mexican Black	Mexican	Non-Mexican White	Non-Mexican Black	Mexican
<$10,000	6%	15%	9%	39%	37%	34%
$10,000-$24,999	15%	23%	21%	30%	32%	38%
$25,000-$49,999	24%	27%	30%	20%	22%	26%
$50,000-$99,999	32%	25%	28%	5%	9%	2%
$100,000+	23%	10%	12%	7%	1%	0%

Table 2

Demographic profiles: National American Community Survey (ACS) Estimates, 2008-2010, and the Detroit Telomere Study

	National ACS Estimates			Detroit Telomere Study		
	Non-Mexican White	Non-Mexican Black	Mexican	Non-Mexican White	Non-Mexican Black	Mexican
Age distribution						
25 to 34 years	17.0%	22.6%	31.3%	6.8%	7.2%	27.7%
35 to 44 years	18.6%	23.1%	27.2%	20.5%	27.0%	36.2%
45 to 54 years	22.5%	23.4%	19.9%	20.5%	29.7%	14.9%
55 to 64 years	19.1%	16.2%	11.5%	34.1%	24.3%	14.9%
65 to 74 years 75	11.9%	8.6%	6.0%	13.6%	6.3%	6.4%
years and over	10.9%	6.1%	4.1%	4.5%	5.4%	0.0%
Poverty rates					[As measured by PIR < 1]	
25 to 64 years 65	9.0%	23.0%	19.8%	55.6%	50.0%	52.3%
years and over	10.6%	22.7%	20.7%	0.0%	33.3%	33.3%
Educational Attainment (Ages 25+)						
Less than high school diploma	9.6%	18.4%	38.4%	34.1%	20.7%	46.8%
High school graduate (includes GED)	29.3%	31.6%	26.2%	13.6%	28.8%	25.5%
Some college or associate's degree	30.0%	32.2%	22.4%	29.6%	42.3%	23.4%
Bachelor's degree	19.5%	11.6%	8.9%	13.6%	6.3%	2.1%
Graduate or professional degree	11.6%	6.2%	4.1%	9.1%	1.8%	2.1%

Table 3

Estimated telomere length coefficients

	Model 1		Model 2'A		Model 2'B	
	Coefficient	**Std. Error**	**Coefficient**	**Std. Error**	**Coefficient**	**Std. Error**
Age (in years)	−16.54 ***	1.84	−16.39 ***	1.82	−16.04 ***	1.79
Race: [ref = White]						
Black	45.31	73.50	48.63	71.01	131.07	80.37
Mexican	16.53	93.48	27.24	90.95	172.69	114.89
Female	199.76 **	74.43	206.39 **	73.37	203.69 **	75.22
PIR (continuous)			15.19 **	5.11	15.00 ***	2.77
Race * PIR interactions:						
White * continuous PIR					38.56	39.64
Mexican * continuousPIR					−41.52 *	18.12
[constant]	6274.26 ***	131.10	6230.41 ***	123.82	6132.88 ***	123.19

* Two-tailed tests significant at the p < 0.05 level

** Two-tailed tests significant at the p < 0.01 level

*** Two-tailed tests significant at the p < 0.001 level

Table 4

Estimated telomere length coefficients* in a series of nested regression models.

	Model 1			Model 2			Model 3		
	Coef.	**Std. Err.**	**P>|t**	**Coef.**	**Std. Err.**	**P>|t**	**Coef.**	**Std. Err.**	**P>|t**
Age (in years)	**−16.54**	**1.84**	**0.00**	**−16.92**	**1.89**	**0.00**	**−16.87**	**1.83**	**0.00**
Race: [ref = White]									
Black	45.31	73.50	0.54	−73.68	100.38	0.47	−32.40	109.15	0.77
Mexican	16.53	93.48	0.86	**−231.42**	**117.28**	**0.06**	−182.39	120.79	0.14
Female	**199.76**	**74.43**	**0.01**	**197.97**	**75.11**	**0.01**	**195.77**	**71.88**	**0.01**
Race * PIR interactions:									
White * PIR < 1				**−327.25**	**107.80**	**0.01**	**−255.73**	**116.83**	**0.04**
Black * PIR < 1				−62.40	83.37	0.46	−33.11	92.31	0.72
Mexican * PIR < 1				194.34	128.10	0.14	**235.55**	**133.20**	**0.09**
Education: [ref = college]									
< high school							**−208.11**	**114.07**	**0.08**
HS degree/GED							−127.76	99.20	0.21
Some college							−124.60	103.38	0.24
Psychosocial stress:									
Safety stress									
Everyday unfair treatment									
Physical environment									
Negative social									
Neighborhood									
Strongly agree									
Somewhat agree									
Neither agree or disagree									
Somewhat disagree									
Strongly disagree [ref.]									
Response type:									
Anger out									
Hopelessness									

	Model 1			Model 2			Model 3		
	Coef.	**Std. Err.**	**P>|t**	**Coef.**	**Std. Err.**	**P>|t**	**Coef.**	**Std. Err.**	**P>|t**
Smoking status: [ref = 0]									
Current smoker									
Former smoker									
BMI: [ref = normal/under]									
Overweight									
Obese									
[constant]	**6274.26**	**131.10**	**0.00**	**6444.44**	**163.07**	**0.00**	**6527.36**	**167.42**	**0.00**
	Model 4			**Model 5**			**Model 6**		
	Coef.	Std. Err.	P>|t	Coef.	Std. Err.	P>|t|	Coef.	Std. Err.	P>|t|
Age (in years)	**−16.34**	**1.92**	**0.00**	**−16.85**	**1.78**	**0.00**	**−17.00**	**1.65**	**0.00**
Race: [ref = White]									
Black	−6.38	116.25	0.96	19.02	111.45	0.87	−24.14	106.27	0.82
Mexican	−205.09	135.71	0.14	−183.75	133.05	0.18	−151.49	134.16	0.27
Female	**199.15**	**66.18**	**0.01**	**196.56**	**66.29**	**0.01**	**190.48**	**65.23**	**0.01**
Race * PIR interactions:									
White * PIR < 1	**−252.27**	**108.02**	**0.03**	**−233.42**	**106.54**	**0.04**	**−219.04**	**108.04**	**0.05**
Black * PIR < 1	−27.75	88.07	0.76	−36.45	85.53	0.67	−7.03	82.88	0.93
Mexican * PIR < 1	**227.38**	**136.10**	**0.10**	192.33	150.78	0.21	158.36	153.54	0.31
Education: [ref = college]									
< high school	**−228.66**	**118.78**	**0.06**	**−241.53**	**109.66**	**0.04**	**−202.07**	**113.86**	**0.09**
HS degree/GED	−143.72	97.02	0.15	−137.23	91.96	0.15	−118.26	90.47	0.20
Some college	−147.71	108.40	0.18	−143.13	104.68	0.18	−139.91	103.27	0.19
Psychosocial stress:									
Safety stress	**67.13**	**35.42**	**0.07**	**80.29**	**34.02**	**0.02**	**78.98**	**33.25**	**0.02**
Everyday unfair treatment	−4.51	41.45	0.91	8.00	44.55	0.86	37.01	45.81	0.43
Physical environment	36.47	38.81	0.35	56.35	41.31	0.18	59.23	39.15	0.14
Negative social interactions	**−55.74**	**28.20**	**0.06**	**−51.53**	**29.38**	**0.09**	−47.66	29.20	0.11
Neighborhood Satisfaction:									
Strongly agree				**215.39**	**84.78**	**0.02**	**201.31**	**88.33**	**0.03**

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	Model 1			Model 2			Model 3		
	Coef.	**Std. Err.**	**P>|t**	**Coef.**	**Std. Err.**	**P>|t**	**Coef.**	**Std. Err.**	**P>|t**
Somewhat agree				103.17	131.41	0.44	100.81	121.00	0.41
Neither agree or disagree				56.39	97.40	0.57	53.12	97.52	0.59
Somewhat disagree				41.47	78.20	0.60	32.20	77.23	0.68
Strongly disagree [ref.]									
Response type:									
Anger out							−15.19	10.95	0.18
Hopelessness							**−39.89**	**20.10**	**0.06**
Smoking status: [ref = 0]									
Current smoker									
Former smoker									
BMI: [ref = normal/under]									
Overweight									
Obese									
[constant]	**6355.34**	**215.98**	**0.00**	**6174.28**	**211.49**	**0.00**	**6322.28**	**203.98**	**0.00**

	Model 7			Model 8			Model 2		
	Coef.	Std. Err.	P>|t|	Coef.	Std. Err.	P>|t|	Coef.	Std. Err.	P>|t|
Age (in years)	**−16.70**	**1.79**	**0.00**	**−16.25**	**1.75**	**0.00**	**−16.92**	**1.89**	**0.00**
Race: [ref = White]									
Black	−32.39	105.54	0.76	−39.52	103.81	0.71	−73.68	100.38	0.47
Mexican	−168.86	131.98	0.21	−157.28	134.32	0.25	**−231.42**	**117.28**	**0.06**
Female	**177.43**	**61.68**	**0.01**	**180.68**	**60.42**	**0.01**	**197.97**	**75.11**	**0.01**
Race * PIR interactions:									
White * PIR < 1	**−215.44**	**111.00**	**0.06**	**−215.63**	**110.16**	**0.06**	**−327.25**	**107.80**	**0.01**
Black * PIR < 1	0.06	81.81	1.00	−9.04	82.67	0.91	−62.40	83.37	0.46
Mexican * PIR < 1	163.48	152.14	0.29	170.25	145.75	0.25	194.34	128.10	0.14
Education: [ref = college]									
< high school	−189.63	114.51	0.11	−140.11	120.88	0.26			
HS degree/GED	−114.61	96.92	0.25	−55.19	108.23	0.61			
Some college	−138.84	104.31	0.19	−104.49	113.35	0.36			

	Model 1			Model 2			Model 3		
	Coef.	**Std. Err.**	**P>|t**	**Coef.**	**Std. Err.**	**P>|t**	**Coef.**	**Std. Err.**	**P>|t**
Psychosocial stress:									
Safety stress	**81.11**	**31.15**	**0.01**	**77.80**	**31.14**	**0.02**			
Everyday unfair treatment	39.92	49.31	0.42	41.55	49.33	0.41			
Physical environment	56.08	38.60	0.16	54.46	36.41	0.14			
Negative social interactions	−47.97	29.02	0.11	**−51.50**	**28.63**	**0.08**			
Neighborhood Satisfaction:									
Strongly agree	**187.99**	**89.72**	**0.04**	**195.09**	**86.73**	**0.03**			
Somewhat agree	96.27	122.79	0.44	90.55	119.90	0.46			
Neither agree or disagree	69.75	95.74	0.47	93.43	102.70	0.37			
Somewhat disagree	36.13	77.97	0.65	32.89	77.21	0.67			
Strongly disagree [ref.]									
Response type:									
Anger out	−12.78	11.66	0.28	−15.46	10.95	0.17			
Hopelessness	**−39.02**	**20.20**	**0.06**	**−39.27**	**18.65**	**0.04**			
Smoking status: [ref = 0]									
Current smoker	−51.44	71.15	0.48	−71.80	72.17	0.33			
Former smoker	−79.02	92.05	0.40	−80.19	92.30	0.39			
BMI: [ref = normal/under]									
Overweight				−46.09	80.17	0.57			
Obese				**−145.22**	**69.82**	**0.05**			
[constant]	**6337.24**	**210.23**	**0.00**	**6408.79**	**202.72**	**0.00**	**6444.44**	**163.07**	**0.00**

*Bolded values are statistically significant (p<.05) or marginally