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Carmen Veronica Mosley

Candidate

Anthropology

Department

This dissertation is approved, and it is acceptable in quality and form for publication:

Approved by the Dissertation Committee:

Heather J.H. Edgar, Chairperson

Keith Hunley

Melissa Emery-Thompson

Michael L. Trujillo

**A BIOCULTURAL EXAMINATION OF HEALTH RISK
AMONG NEW MEXICANS OF SPANISH-SPEAKING
DESCENT**

by

CARMEN VERONICA MOSLEY

B.A., Anthropology, University of California, Santa Cruz
M.S., Anthropology, University of New Mexico

DISSERTATION

Submitted in Partial Fulfillment of the
Requirements for the Degree of

**Doctor of Philosophy
Anthropology**

The University of New Mexico
Albuquerque, New Mexico

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DEDICATION

For my parents, Jerome and Lourdes Mosley, Andrew, Rocco, and Baby Bolla #2.

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A BIOCULTURAL EXAMINATION OF HEALTH RISK AMONG NEW MEXICANS OF SPANISH-SPEAKING DESCENT

**by
CARMEN VERONICA MOSLEY**

B.A., Anthropology, University of California, Santa Cruz, 2006

M.S., Anthropology, University of New Mexico, 2008

Ph.D., Anthropology, University of New Mexico, 2019

ABSTRACT

Individuals of Hispanic, Latino, or Spanish (HLS) origin suffer disproportionately from higher poverty rates, less education, less access to health care, and greater risk factors for and prevalence of chronic diseases compared to their White counterparts. How health disparities emerge over the life course remains unclear. Allostatic load (AL) provides an approach in health research that utilizes a life course perspective and multi-system view of cumulative physiological, or health risk. AL is used to identify sociodemographic and biological factors that contribute to racial differences in health risk. However, AL is not widely used to explore causes of poorer health outcomes in HLS populations. New Mexico provides a unique opportunity to address gaps in our understanding of HLS health for three reasons. First, New Mexico has the largest percentage of self-identified HLS residents in the U.S. Second, some of the ethnic terms that New Mexican HLS use to describe themselves depart from the Office of Management and Budget nomenclature. Third, health disparities are particularly striking in New Mexico where performance measures in health care and health care coverage have long ranked near the lowest in the country.

This dissertation addressed three aims. First, I tested whether social and biological factors that contribute to disparities in health differ between 1) New Mexicans of Spanish-speaking descent (NMS) and other U.S. census groups, and 2) different ethnic groups of NMS. Second, I examined patterns of AL and investigated the sociodemographic and biological correlates of AL in NMS. Third, I tested whether AL is associated with six chronic disease outcomes. Data included sociocultural, biological, and anthropometric measures from 507 self-identified NMS.

I found that education, household income, skin color, and continental ancestry differ between NMS and other U.S. census groups, demonstrating that NMS are not socially or biologically homogeneous. In NMS, mean AL scores increased with age. Comparisons of AL biomarkers between NMS and other U.S. groups showed that NMS had significantly higher biomarker measures. Further, higher proportions of Native American ancestry were significantly associated with higher AL scores. Finally, I found that AL was significantly associated with only gallbladder disease and abdominal obesity.

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Chapter 1. Introduction

This dissertation integrates anthropological and epidemiological perspectives and methods to examine the biological and sociocultural factors associated with health risk and health outcomes in New Mexicans of Spanish-speaking descent (NMS). This introductory chapter will provide 1) an overview of health disparities and Hispanic health in the U.S., 2) a background of allostasis and allostatic load, the theoretical framework employed in this dissertation, 3) a brief literature review of racial and ethnic differences in allostatic load, 4) a description of New Mexico as a case study for this dissertation, and 5) outline the research design. This chapter concludes with a guide to the dissertation.

Health Disparities in the U.S.

According to the U.S. Department of Health and Human Services' health prevention initiative *Healthy People 2020*, a health disparity is a "particular type of health difference that is closely linked with social, economic, and/or environmental disadvantage" (p. 28). Health disparities adversely affect groups of people who have systematically experienced greater social or economic obstacles to health based on racial or ethnic origin, socioeconomic status (SES), gender, cognitive or physical disability, sexual orientation, and/or other characteristics that have historically been linked to discrimination or exclusion (USHHS, 2008). Substantial health and healthcare disparities among racial and ethnic groups has long been documented in the U.S. (AHRQ, 2009, 2016; Fiscella, Franks, Gold, & Clancy, 2000). However, only since 1999, with the publication of *Healthy People 2010*, has addressing racial and ethnic disparities in health become a national public health priority (USHHS, 2000; Warnecke et al., 2008). Rates of

major causes of illness and death, including cardiovascular disease, diabetes, hypertension, and HIV/AIDS are significantly higher in African Americans, Hispanics, American Indians, and Native Hawaiians and Pacific Islanders compared to the rest of the U.S. population (AHRQ, 2009, 2016). As these racial and ethnic minority groups continue to grow in their proportion of the nation's population (Aponte, 2009; Morales, Lara, Kington, Valdez, & Escarce, 2002), there is a pressing need to understand the causes of these disparities.

Hispanic Health in the U.S.

Individuals of Hispanic, Latino or Spanish origin¹ (hereafter HLS), represent the largest and fastest-growing minority group in the U.S. (Ennis, Rios-Vargas, & Albert, 2011; Velasco-Mondragon, Jimenez, Palladino-Davis, Davis, & Escamilla-Cejudo, 2016). In 2017, HLS comprised 18.1% (58.9 million) of the nation's total population; a percentage that is expected to increase to 28% (111.2 million) by 2060 (Census Bureau, 2018). Research has shown that health disparities in HLS are related to their SES, cultural background(s), education level, citizenship status, and discrimination (Vega, Rodriguez, & Gruskin, 2009; Velasco-Mondragon et al., 2016). Higher poverty rates, less education, and less access to health care contributes to HLS having greater risks factors for and prevalence of obesity, diabetes, hypertension, and diseases of the stomach, gallbladder, liver, and kidneys compared with non-HLS Whites (Aponte, 2009; Braveman, Egerter, &

¹This dissertation uses the U.S. Census Bureau's definition of the phrase "of Hispanic, Latino or Spanish origin" to describe "a person of Mexican, Puerto Rican, Cuban, Central or South American or other Spanish culture or origin, regardless of race" (Office of Management and Budget 1997; Humes, Jones, & Ramirez, 2011).

Williams, 2011; Flegal, Carroll, Ogden, & Johnson, 2002; Morales et al., 2002; NCHS, 2016; Peralta et al., 2006)

Though research in social sciences and public health has made important contributions to our understanding of health risks and disease prevalence in U.S. HLS populations, several issues remain to be addressed. First, a large proportion of HLS health research in the U.S. focuses on the implications and causes of the “Hispanic health paradox.” This paradox refers to the epidemiological finding that despite disadvantages in education, occupation, income and healthcare access, some HLS populations—specifically Mexicans and Mexican Americans—experience similar or better health outcomes compared to their non-HLS White counterparts (Markides & Coreil, 1986). Despite extensive research over the past two decades, the mechanisms of this advantage and its distribution across ethnic subgroups, age, nativity status, and sex remains unclear (Borrell & Lancet, 2012; Ruiz, Steffen, & Smith, 2013). Second, and seemingly contradictory, many studies document a particular profile of poor health in HLS (Daviglius et al., 2012; Flegal, Carroll, Ogden, & Curtin, 2010; Samet, Coultas, Howard, Skipper, & Hanis, 1988). These poor outcomes are generally attributed to ethnicity-specific genetic factors as well as to social factors, including socioeconomic status, education, and discrimination and racism (Bertoni et al., 2003). Third, our understanding of HLS health in the U.S. is largely based on the most populous HLS subgroups in the U.S.: Mexican/Mexican Americans, Puerto Ricans, and Cubans, (Ennis et al., 2011). This focus ignores less sizeable populations and discounts heterogeneity in culture, biology and identity within groups (e.g., Spanish, Chicano, etc.) (Hajat, Lucas, & Kington, 2000).

While a large body of literature has documented links between sociodemographic factors and health in HLS populations, there is need to better understand how health disparities may emerge over the life course (Adler & Rehkopf, 2008; Chyu & Upchurch, 2011; S. E. Taylor, Repetti, & Seeman, 1997).

Allostasis and Allostatic Load

In trying to examine underlying causes of racial and ethnic disparities in health, research has often assumed genetic attributes, an increased tendency to engage in risky behaviors, or the use of race or ethnicity as a proxy for measures for poor socioeconomic factors (Carlson & Chamberlain, 2005; Kneipp & Drevdahl, 2003). Allostasis and allostatic load (AL) provide an alternative approach in health research, which instead utilizes a life course perspective and multi-system view of adaptive physiological responses (Carlson & Chamberlain, 2005). Allostasis refers to the process whereby adaptive adjustments are made to the body's physiological systems in response to external and internal stressors. When the body experiences a real or perceived stressful event, the sympathetic nervous system and the hypothalamic-pituitary-adrenal axis (HPA axis) activate numerous physiological systems, including the cardiovascular, metabolic and inflammatory systems (Juster, McEwen, & Lupien, 2010; B. McEwen, 1998; Sterling & Eyer, 1988). Normal functioning requires continual fluctuation, or adaptation, in physiological systems in response to stressful events, but these fluctuations are only adaptive when they are short-term, and the stressors are acute. When exposed to repeated stressors over the life course, however, these physiological systems may begin to experience impaired physiological functioning, or physiological "wear and tear" (B. McEwen, 1998). This cumulative burden, AL, is the cost of allostasis, and may

contribute to the development of complex and chronic conditions such as hypertension, diabetes, obesity, and cardiovascular disease (B. McEwen, 1998; T. E. Seeman, Singer, Rowe, Horwitz, & McEwen, 1997; Sterling & Eyer, 1988). The allostatic load framework measures primary mediators in combination with secondary outcomes across multiple systems to predict individuals at risk of developing particular outcomes (e.g., disorder, disease, death). Primary mediators are stress hormones, such as cortisol and epinephrine, which mediate the body's response to stressors through their effects on soft tissues and organs. Primary mediators interact with each other to produce secondary outcomes, whereby metabolic (e.g., total cholesterol, glycosylated hemoglobin, body mass index), cardiovascular (e.g., systolic and diastolic blood pressure), inflammatory (C-reactive protein, interleukin-6) and immune (e.g., Epstein Barr virus antibody) parameters reach sub-clinical levels (Juster et al., 2010). These and various other biomarkers are commonly used in calculating allostatic load indices (Duong, Bingham, Aldana, Chung, & Sumner, 2017; Edes & Crews, 2017). Figure 1.1 presents a conceptual model of contributors, components and outcomes of allostatic load.

Comparisons across populations and among studies are particularly challenging with regard to allostatic load for three main reasons. First, not all studies use the same biomarkers in their calculations of allostatic load, due to convenience, availability, and data collection limitations (Beckie, 2012; Carlson & Chamberlain, 2005; Duong et al., 2017; Edes & Crews, 2017; Mosley, Edgar, Hunley, & Healy, in review; Stewart, 2006). Second, various methods for calculating allostatic load indices have been proposed, making it difficult to meaningfully compare allostatic load scores and effects across samples and across populations (Beckie, 2012; Duong et al., 2017; Edes & Crews, 2017;

McDade, 2008). AL indices are calculated by turning each biomarker measure into a dichotomous variable; 1 point is given if the biomarker falls within high risk range and 0 if not. However, there are various methods for assessing high risk and aggregating biomarkers into indices. Among these methods are utilizing high risk cut-points defined by quartiles, quintiles, deciles, clinical criteria, or a combination of clinical criteria and quartiles (Duong et al., 2017; Edes & Crews, 2017). Third, evidence suggests the relationship between various biomarkers and health outcomes differs by race, ethnicity, and socioeconomic status (Beckie, 2012; McDade, 2008). Despite these limitations, Edes and Crews (2017) make a strong case for the potential utility of allostasis via AL in biological anthropology. They argue that the use of AL in anthropological studies can be informative about life-history evolution, evolutionary trade-offs, health and well-being over the life course, and human variation in response to extrinsic stressors (Edes & Crews, 2017).

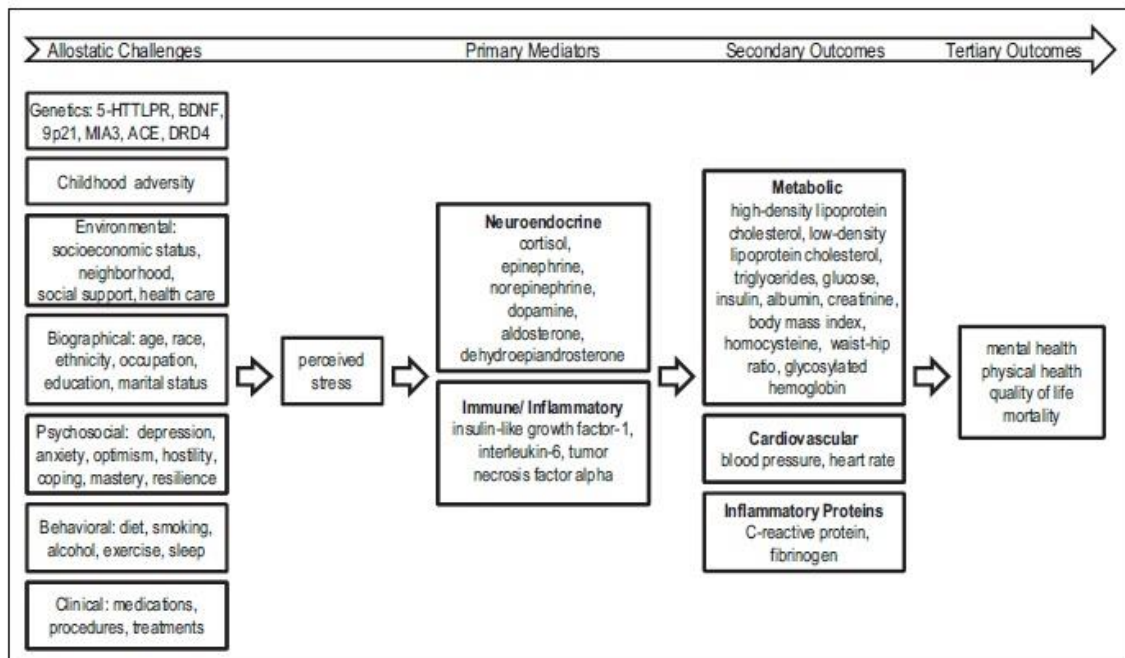


Figure 1.1 Conceptual model of contributors, components, and outcomes of allostatic load. From Beckie (2012).

Racial and ethnic differences in allostatic load

Recent research has provided evidence linking socioeconomic (i.e., socioeconomic status, education level, household income) and sociodemographic factors (i.e., nativity status), race, and ethnicity to allostatic load scores (Carlson & Chamberlain, 2005; Howard & Sparks, 2015; Upchurch et al., 2015). With these findings, a clearer picture is beginning to form regarding how sociodemographic conditions are embodied via physiological and biological pathways that ultimately affect health and health outcomes. Geronimus and colleagues (2006) found that U.S. Black men and women had higher AL scores compared to their White counterparts in all age groups, regardless of SES. In the same study, it was also found that Black women had disproportionately higher AL relative to Black men and White women (Geronimus, Hicken, Keene, & Bound, 2006). In addition, AL has been shown to be lower among Mexicans not born in the U.S. compared to U.S.-born self-described Mexicans and is lower among those who have resided in the U.S. for a shorter period of time (Crimmins, Kim, Alley, Karlamangla, & Seeman, 2007; Peek et al., 2010). Research examining AL in adolescents further support these findings. For example, Rainisch and Upchurch (2013) found that Black adolescents had the highest mean AL score compared to Mexican American and White adolescents. Furthermore, U.S.-born adolescents had significantly higher mean AL scores than foreign-born adolescents, suggesting differences in AL begin early in the life course, and that the relative advantage of Whites and Mexican Americans over Blacks declines over adolescence (Rainisch & Upchurch, 2013).

While significant, these studies only present differences between racial and ethnic categories that are recognized by the Office of Management and Budget (OMB).

However, there are HLS subpopulations in the U.S., including NMS, which self-identify with distinctive ethnic subgroups within those larger OMB categories. These subgroup identities often have region-specific meanings tied to local histories (Doan & Stephan, 2006; Duany, 1998; Gonzales, 1993; Healy, Edgar, Mosley, & Hunley, 2018; Hunley et al., 2017). Additionally, many studies examining AL in HLS populations focus on individuals who are foreign-born or have recently immigrated to the U.S. Studies that look for patterned differences in AL among region-specific U.S. subgroups, such as the one reported here, are missing from the literature.

New Mexico: A Case Study

New Mexico has a long and complex history of human settlement that began more than 11,000 years ago when Native Americans first arrived in the American Southwest (Huckell, 2014). In the 16th century, Spanish colonizers began to arrive in New Mexico. Initially, the predominantly male settlers intermingled with Native groups, lodging in the native pueblos, adopting material goods and farming practices from the Puebloan Indians, and marrying Native American women (Nostrand, 1992).

By the 18th century, a hierarchical social system began to emerge. At the top of the hierarchy were the governing Spanish nobility, who claimed pure Spanish descent. Next were the peasants, who recognized their mixed ancestry but considered themselves to be culturally Spanish. The third group, termed the *genizaro*, consisted of Native Americans that took on Spanish surnames and cultural practices (Nostrand, 1992). Finally, at the bottom of the Spanish hierarchy were un-aculturated Native Americans from surrounding pueblos (Gutierrez, 1991; Nieto-Phillips, 1996). Until about 1760,

marriage was largely endogamous within these groups (Gutierrez, 1991). Around that time, intermarriage between these groups became more prevalent, with many Spanish men marrying women from other groups, resulting in a social classification system based on the perceived amount of Spanish vs. Native American ancestry.

As migration from Mexico increased, many New Mexicans asserted their European, particularly Spanish, heritage (Gonzales, 1993). This, in part, gave rise to the unique ethnic nomenclature (e.g. Nuevomexicano, Hispano, Spanish) that is still used among NMS (Healy et al., 2018; Hunley et al., 2017; Lomelí, Sorell, & Padilla, 2002; Nieto-Phillips, 1996; Nostrand, 1992). Today, there is a tendency among New Mexicans to distill this complex history into a simple, dichotomous model of ethnic identity. On one hand are people who consider themselves to be direct descendants of the earliest Spanish colonists. On the other hand, are the more recent immigrants from Latin America, primarily Mexico. Scholars have argued that the first group has developed and maintained a distinctive “Hispano” subculture, despite continued migration of peoples of diverse ancestry to the region (Gonzales, 1993; Nieto-Phillips, 1996; Nostrand, 1992; Trujillo, 2010). Others have argued that acculturation has blurred the distinction between any ethnic subgroups that might have once existed (Bustamante, 1991). Figure 1.2 shows the geographic distribution of HLS who further self-identify as Mexican (left) and Spanish (right) on the (Census Bureau, 2018). The north-south gradient in both plots captures the historical pattern of migration, beginning with the 16th century migration into the central-northern portion of what would become New Mexico and the continued northward displacement of their descendants caused by labor-related migrations from Mexico that accelerated during the 18th century (Nostrand, 1992; Swadesh, 1974; Zeleny,

1974). The gradient is consistent with the proposed Spanish/non-Spanish ethnic subdivision.

The HLS population of New Mexico provides a unique opportunity to address gaps in our understanding of HLS health in the U.S. for three reasons. First, New Mexico has the largest proportion of self-identified HLS citizens in the U.S. (48% compared to the national average of 18.1%; See Figure 1.3) (Census Bureau, 2018). Second, the unique history of the state and its isolated position in the American Southwest have led to the development of a distinctive New Mexican HLS cultural landscape (Gonzales, 1993; Healy et al., 2018; Hunley et al., 2017; Nieto-Phillips, 1996; Nostrand, 1992; Swadesh, 1974; Zeleny, 1974). Third, New Mexico is one of the poorest states in the U.S., ranking fourth highest in the percent of the population below the poverty level (17.8%) (Census Bureau, 2018), and ranks well below average in performance measures of health care, rating the state as weak or very weak in treatment for diabetes, cancer, heart disease, respiratory disease, and maternal and child health (AHRQ, 2009, 2016).

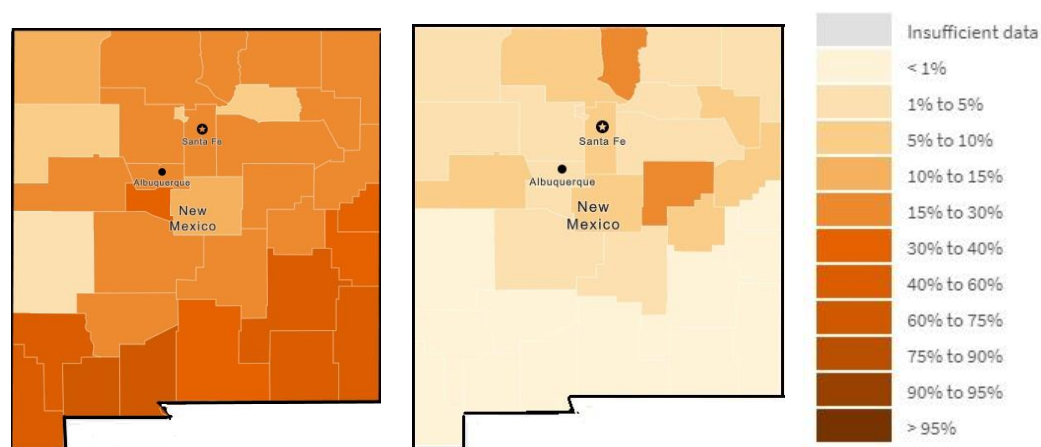


Figure 1.2. Percent of Hispanics of Mexican (left) and Spanish origin (right). Source: Social Explorer; ACS 2017, 5-year estimates, U.S. Census Bureau.

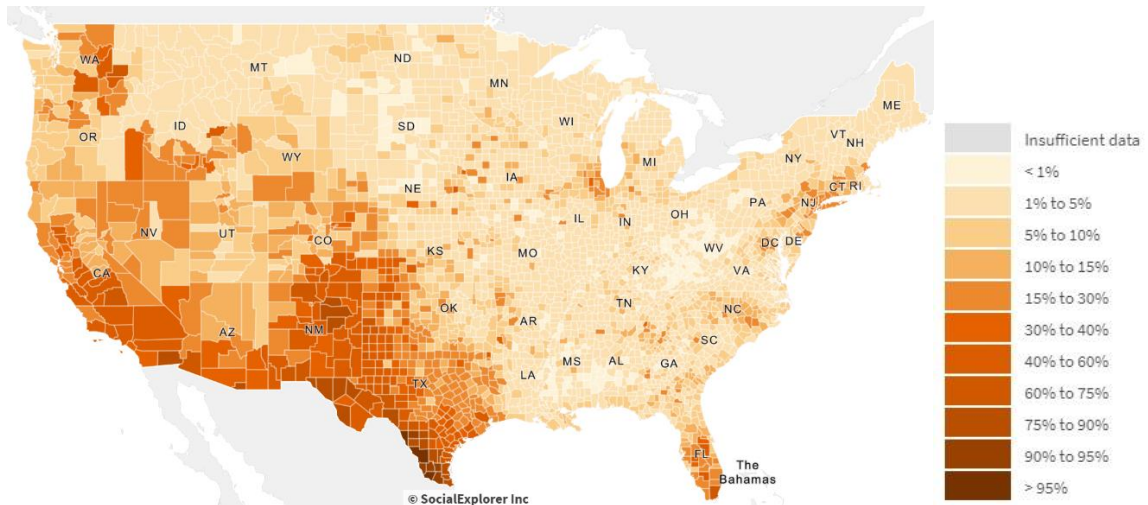


Figure 1.3. Percentage of residents in a county identifying as Hispanic or Latino. Source: Social Explorer; ACS 2017, 5-year estimates U.S. Census Bureau.

In spite of the great deal of research conducted on Hispanic health, there is a tendency to assume that the findings of any one study apply to all Hispanics, even with the evidence of regional biological and sociocultural variation among the different subgroups (Doan & Stephan, 2006; Healy et al., 2018; Hunley et al., 2017; Lomeli et al., 2002). This dissertation integrates anthropological and epidemiological perspectives and methods to examine the biological and sociocultural factors associated with health risk—measured as allostatic load—and health outcomes in NMS.

Research Design

Between August 2010 and June 2012, data were collected from 507 individuals who were 18 years of age or older and self-identified as NMS. Study participants were initially recruited using a convenience-based sampling strategy through personal contacts, advertisements in school bulletins and email LISTSERVs, public libraries, community centers, and non-profit organizations. The University of New Mexico Main Campus

Institutional Review Board approved the research protocol (HRPO #09-412, 10-310). Informed consent was obtained from all participants prior to data collection.

A 129-question survey, developed based on the results of a 2008 pilot study examining ethnic identity in New Mexico (Hunley et al., 2017), was administered to study participants during 1.5-2 hour in-person interviews. A description of the study sampling design and recruitment strategy is described in Hunley et al. (2017). The complete study questionnaire can be found on our study website:

<http://heritagenm.unm.edu/research-design-methods> (See also Appendix B). Additional details of the sample and data collected are provided in each chapter.

Guide to the Dissertation

This dissertation follows a hybrid format, organized in three chapters written for peer-review publication. Chapter 2 tests whether key social and biological features that contribute to disparities in health differ between 1) NMS and other U.S. racial and ethnic groups, and 2) different ethnic groups of NMS. Chapter 3 examines patterns of AL and investigates the sociodemographic and biological correlates of AL in the sample. This chapter is currently under review with the *American Journal of Human Biology*. Chapter 4 tests whether AL is associated with six chronic disease outcomes (abdominal obesity, hypertension, diabetes, cardiovascular disease, cancer, and gallbladder disease). Chapter 4 will be submitted as an original research article to the *Journal of Racial and Ethnic Health Disparities*. A summary of key findings and conclusions from each chapter is presented in Chapter 5.

Chapter 2. Relationships among socioeconomic status, continental ancestry, skin color, and self-identified ethnicity among New Mexicans of Spanish-speaking descent: implications for health disparities research

Introduction

In recent years, epidemiological and social science research has increasingly focused on the role of race and ethnicity in health disparities in the U.S. (Dorsey et al., 2014; Gravlee & Sweet, 2008; Lee & Choi, 2009). As it stands, this focus has several limitations, including: 1) the use of ill-defined measures of race and ethnicity (Baer et al., 2013; Gravlee & Sweet, 2008), 2) the reliance on broad Office of Management and Budget (OMB, 1997) categories that do not reflect variation in the sociocultural or geographic characteristics of the groups under study (Bradby, 2003; Hunley et al., 2017; P. Taylor, Lopez, Martínez, & Velasco, 2012), 3) the imprecise use of race and/or ethnicity as proxies for socioeconomic status (SES), or some other correlate(s) of health, and 4) the assumption of relative sociocultural and biological homogeneity within racial and ethnic groups. These limitations make it difficult for researchers to assess the role of race and ethnicity in health disparities.

This issue is particularly salient for individuals of Hispanic, Spanish or Latino (HLS) origin in the U.S. Due to their long and complex history of migration to the Americas and their subsequent interactions with culturally diverse people of wide-ranging continental ancestry, this group is highly biologically, culturally, and sociodemographically diverse today (Bonilla et al., 2004; Duany, 1998; Healy et al., 2018; Vega et al., 2009). This biological and cultural diversity is readily apparent at the national level, but it is also significant at the regional level. In New Mexico, for example, the history of the state and its isolated position in the American Southwest have led to the development of a uniquely New Mexican Hispanic cultural landscape (Gonzales, 1993;

Nieto-Phillips, 1996; Nostrand, 1992; Swadesh, 1974; Zeleny, 1974). This landscape includes multiple ethnic identities that are the product of initial migration of Spanish-speaking peoples, relative isolation of the descendants of this migration in northern-central regions of the state, and generations of subsequent migrations from diverse locations through the world. Despite this complexity, all New Mexicans of Spanish-speaking descent (NMS) are often classified using a single pan-ethnic label, Hispanic or Latino, on the U.S. Census and other national health surveys, such as the National Health Interview Survey, National Health and Nutrition Examination Survey, and Current Population Survey. This pan-ethnic label fails to capture the complex history of interactions between diverse peoples within the state and the potential health consequences of this history.

The health of HLS populations in the U.S. is shaped by factors such as language and cultural barriers, lack of preventive care access, lack of health insurance, and undocumented status (OMH, 2019; Velasco-Mondragon et al., 2016). This puts HLS populations at greater risk for teen pregnancy, obesity, tobacco and alcohol abuse, poor health screening rates, occupational hazards; and contributes to the disproportionately high rates of obesity, chronic liver disease, and kidney disease in HLS populations (OMH, 2019; CDC, 2015). Additionally, disparities in health risk and health outcomes exist among HLS subgroups. For example, Puerto Ricans have higher rates of asthma, HIV/AIDS, and infant mortality. Mexican Americans have higher prevalence rates of diabetes (OMH, 2019).

Given this variation in ethnic identity and health among HLS populations in the U.S., researchers have stressed the importance of examining variation within census-

based racial and ethnic categories at state and regional levels (Bilheimer & Sisk, 2008; Gold, Dodd, & Neuman, 2008). To this end, in this study we ask: Do social and biological factors that contribute to differences in health differ 1) between NMS vs. other U.S. racial and ethnic groups, and 2) between different ethnic groups within NMS?

The HLS population of New Mexico provides a unique opportunity to address these issues for three reasons. First, New Mexico has the largest proportions of self-identified Hispanic residents in the U.S. (48% compared to the national average of 18.1%; (Census Bureau, 2018)). Second, the unique history of the state and its isolated position in the American Southwest have led to the development of a uniquely New Mexican Hispanic cultural landscape (Gonzales, 1993; Nieto-Phillips, 1996; Nostrand, 1992; Swadesh, 1974; Zeleny, 1974). This landscape includes at least two Hispanic subgroups, one consisting of people that consider themselves to be direct descendants of the earliest Spanish colonists and the other consisting of more recent immigrants from Latin America, primarily Mexico. Third, the U.S. Department of Health and Human Services ranks New Mexico well below average in performance measures of health care, rating the state as weak or very weak in treatment for diabetes, cancer, heart disease, respiratory disease, and maternal and child health (AHRQ, 2009, 2016). The Commonwealth Fund has ranked New Mexico high in the percent of uninsured adults and children, and in overall inequity of health care (Radley, McCarthy, & Hayes, 2018; Radley, McCarthy, Lippa, Hayes, & Schoen, 2014).

Social factors include socioeconomic status (SES) and skin color and continental ancestry, variables frequently implicated in health disparities. Continental ancestry is often used as a proxy for racial variation in the genetic factors underlying multifactorial

disease (Burchard et al., 2003; Risch, Burchard, Ziv, & Tang, 2002; Tang, Coram, Wang, Zhu, & Risch, 2006), but, to the extent that it is correlated with phenotypes, it might more accurately reflect social risk factors (Florez et al., 2011; Gravlee, Non, & Mulligan, 2009; Non, Gravlee, & Mulligan, 2012).

Materials and Methods

Study Sample and Procedures

The study sample consisted of 507 adults (18 or older) who self-identified as NMS. For a full description of our sampling design, recruitment strategy, and interview procedures not discussed here, see Hunley et al. (2017) and Mosley et al. (in review). The complete study questionnaire can be found on our study website:

<http://heritagenm.unm.edu/research-design-methods> (See also Appendix B).

Ethnicity

Ethnicity was based on participant responses to a list of seven ethnic terms identified during semi-structured interviews (Hunley et al. 2017). These terms (Table 2.1) were specifically identified by NMS to be culturally and regionally relevant. Of the list of terms, participants were asked to identify their top three choices for which they most identified with. In this study, participants' first-choice responses are used for analysis.

Socioeconomic status

We focused on four indicators of SES: household income, education, maternal education, and paternal education. Household income and education reflected current SES characteristics, while parental education reflected lifelong SES characteristics

(Braveman et al., 2005; Crimmins et al., 2007). Household income was measured by asking participants which of nine income ranges described their total household income before taxes in the last twelve months. Household income was coded as a categorical variable (<\$5,000, \$5,000-\$24,999, \$25,000-\$34,999, \$35,000-\$49,999, \$50,000-\$74,999, \$75,000-\$99,999, \$100,000⁺). Education was measured using five categories of highest completed level of education (some high-school or less, high-school graduate, some college/vocational/technical, college graduate, some postgraduate/professional school).

Skin color

Skin pigmentation has been used in recent anthropological studies as a proxy for UV light exposure, continental ancestry, and social stress exposure (Gravlee & Dressler, 2005; Klimentidis, Miller, & Shriver, 2009; Shriver & Parra, 2000). Facial pigmentation in particular may be used by others to assign membership to particular racial and ethnic groups (Brown, 1998; Gravlee, 2005; Gravlee & Dressler, 2005; Harris, 1970). While skin color is a phenotype, it is categorized here as a social factor because it has been shown to be correlated with perceived racism and discrimination (Araújo & Borrell, 2006; Espino & Franz, 2002; Golash-Boza & Darity Jr, 2008; Klonoff & Landrine, 2000; Krieger, Sidney, & Coakley, 1998), and has been shown to be associated with poor health outcomes in African Americans and HLS individuals (Dressler, 1991; Gravlee & Dressler, 2005; Montalvo, 2005; Montalvo & Codina, 2001; Perreira & Telles, 2014). The literature on the social and health correlates of skin color is sparse for HLS populations (Codina & Montalvo, 1994; Gómez, 2000); the current study will be one of the first to assess how it varies within and among ethnic subgroups in a Hispanic

population. Here, we measured the CIELab lightness metric (*L) on the forehead using a handheld reflectometer (DSM II ColorMeter; Cortex Technology, Hadsund, Denmark). Higher values of *L correspond to lighter skin pigmentation.

Continental ancestry

DNA was extracted from mouthwash samples and extracts were genotyped with the Illumina HumanCytoSNP-12 DNA Analysis BeadChip Kit (Illumina, Inc). Continental ancestry was estimated using polymorphic loci from individuals from the Human Genome Diversity Panel-CEPH (Cann et al., 2002; Consortium, 2005). Ancestry estimation methods have been described elsewhere (Hunley et al., 2017).

Comparative data

We compared household income, education, and continental ancestry for NMS to that of African American/Black, Asian, and non-Hispanic White populations in the U.S. We used national household income data from the 2011 Annual Social and Economic Supplement of the Current Population Survey (CPS). The CPS provides detailed information on income, employment, and healthcare coverage. National educational attainment data were taken from the 2010-2012 U.S. Census American Community Survey. We used continental ancestry data from four native North American (Chipewyan, Cree, Ojibwa, and Pima) (Wang et al., 2008) and four African American/Black populations (Chicago, Pittsburgh, North Carolina, and Baltimore) (Tishkoff et al., 2009). We also compared the continental ancestry estimates from this study those from three

other genetic studies of other U.S. Hispanic Groups (Bonilla et al., 2004; Lisabeth, Morgenstern, Burke, Sun, & Long, 2011; Wang et al., 2008).

Statistical Analysis

Twelve study participants were excluded from analysis because they self-identified as non-NMS (e.g. Puerto Rican, Cuban, Panamanian, Filipino American, Anglo). The final sample consisted of 495 individuals. To assess the relationship between race and household income and educational attainment, Pearson's chi-square tests were conducted. To assess differences in continental ancestry between NMS and other racial and ethnic groups, two-sample t-tests assuming unequal variances were used to compare unadjusted means. We evaluated differences in these measures between NMS subgroups using chi-square of independence for categorical variables and one-way ANOVAs for continuous variables. The Tukey-Kramer method was performed post-hoc to perform pairwise comparisons and identify subgroup means that were significantly different from each other (Kramer, 1956). Analyses were conducted in Stata 11 (StataCorp, College Station, TX) and Minitab 17 (State College, PA, 2010).

Results

Descriptions of the NMS subgroups, summarized from participant responses, are listed in Table 2.1. Demographic and socioeconomic statistics of the full sample and the seven ethnic subgroups are summarized in Table 2.2. Almost half the sample self-identified as Hispanic (45.4%). Mean age was 47.9 (± 17.6), mean education level was 15.2 years (equivalent to completion of some college/tech/vocational school), and mean household income was \$64,262 ($\pm 30,523$). Females made up 55.3% of the sample. The

mean forehead skin pigmentation was 25.7 (± 4.2). The average European, Native American, and African genetic ancestry proportions for the sample were 73.6% (± 10.1), 21.6% (± 8.2), and 4.8% (± 4.0), respectively.

Table 2.1. Study participant descriptions of NMS subgroups.

Ethnic Subgroup	Description
<i>Chicano/a</i>	Indicating a mixture of Spanish and indigenous Mexican ancestry. This term is a politicized term that, for many, conveys pride in one's mixed ancestry.
<i>Hispanic</i>	Indicating a historical relationship to Spain and Spanish ancestry. For some this is a general term that encapsulates all people of the Spanish-speaking ancestry regardless of geographic origin; for others, this term is specific to individuals who are born in and have deep ties to New Mexico and are of Spanish and Native American ancestry. Almost half of our sample (44%) used this term to identify themselves.
<i>Latino/a</i>	Indicating a recent historical and/or cultural relationship to Latin America, south of Mexico.
<i>Mexican</i>	Indicating a relationship to Mexico. This term is often used by participants who were born in Mexico and have emigrated to the U.S.
<i>Mexican American</i>	Indicating a relationship to Mexico. This term is often used by participants who: 1) were born in Mexico and emigrated to the U.S. as young children, or 2) are first-generation Americans whose parents and grandparents were born in Mexico.
<i>Nuevomexicano/a</i>	Indicating a historical relationship to New Mexico. This term describes participants who are of Spanish and Native American ancestry, were born in New Mexico, and whose families have been in New Mexico for many generations.
<i>Spanish</i>	Indicating a historical relationship to Spain and Spanish ancestry. The majority of individuals who identify as Spanish in our sample specify that they are either not of Native American ancestry or that any Native American ancestry is of a negligible amount.

Table 2.2. Means (\pm SD) or percentages for study variables.

	Total Sample (N=495)	Chicano (n=52)	Hispanic (n=225)	Latino (n=15)	Mexican (n=17)	Mexican American (n=40)	Nuevomexicano (n=75)	Spanish (n=71)
Household income, \$	64,262 (30,523)	59,636 (32,150)	65,187 (29,294)	63,106 (36,602)	59,066 (33,618)	61,674 (28,547)	64,788 (31,117)	67,108 (32,317)
Self-reported Education, years	15.2 (2.2)	14.9 (2.0)	15.1 (2.1)	16.1 (2.5)	15.8 (1.9)	15.7 (2.2)	15.5 (2.1)	14.7 (2.3)
Maternal education, years	12.4 (2.4)	12.2 (2.3)	12.5 (2.4)	12.5 (2.2)	11.4 (1.8)	12.3 (2.6)	13.3 (2.5)	11.7 (2.2)
Paternal education, years*	12.6 (2.6)	12.6 (2.7)	12.6 (2.5)	12.4 (2.2)	10.8 (1.6)	12.6 (3.0)	13.2 (2.7)	12.4 (2.6)
European ancestry, % *	73.6 (10.1)	73.2 (8.3)	74.4 (9.9)	72.4 (9.9)	64.5 (9.9)	63.4 (11.4)	76.7 (8.1)	75.7 (8.3)
Native American ancestry, % *	21.6 (8.2)	22.2 (7.1)	20.7 (7.9)	22.6 (9.1)	28.0 (8.7)	29.8 (9.0)	19.3 (6.4)	19.8 (6.9)
African ancestry, % *	4.8 (4.0)	4.5 (3.0)	4.8 (4.6)	4.9 (3.7)	7.5 (2.7)	6.7 (3.6)	4.0 (2.8)	4.4 (3.1)
Forehead skin pigmentation (*L)	25.7 (4.2)	25.5 (4.1)	27.1 (5.4)	27.5 (3.7)	24.4 (3.9)	26.2 (3.3)	27.8 (4.3)	28.0 (4.4)
Age*	47.9 (17.6)	47.7 (16.4)	48.3 (17.5)	50.3 (13.8)	42.1 (12.7)	42.5 (18.2)	43.3 (17.9)	55.8 (17.2)
Sex (% ♀)	55.3	42.3	58.7	73.3	29.4	55	52	60.6

*Tests of significance by ANOVA using Tukey-Kramer adjustment for continuous variables and by chi-square for categorical variables. * $p < 0.05$.

NMS compared to other U.S. groups

We found significant differences in education, household income, and continental ancestry between NMS and other U.S. racial and ethnic groups (See also Appendix A for statistical analysis output). Figure 2.1 shows the distribution of educational attainment in NMS, Whites, Blacks, Asians, and Hispanic across the U.S. Fewer NMS had high school diplomas and college degrees in contrast to Asians and Whites; more NMS had some college and postgraduate school than was expected in Hispanics and Blacks (chi-square, $p < 0.001$). Figure 2.2 shows the distribution of household income in NMS, Whites, African-American/Blacks, Asians, and Hispanics across the U.S. More NMS had a

household income of \$100,000+ than the expected household income for Hispanics and Blacks; fewer NMS had a household income of <\$5,000 than expected (chi-square, $p < 0.001$).

Table 2.3 illustrates the mean proportion of Native American, European, and African ancestry among NMS, African-Americans, and native North Americans. Compared to the four Native North American populations, NMS had higher European and African ancestry, and lower Native American ancestry. Compared to the four African American populations, NMS had higher European and Native American ancestry, and lower African ancestry. Genetic studies on nearby regional populations show that Native American ancestry is lower in the American Southwest than in other regions of the U.S. and Mexico, while European ancestry is higher, and African ancestry is low (Healy et al. 2018).

Figure 2.1. Comparison of educational attainment, by race and ethnic group, U.S., 2011.

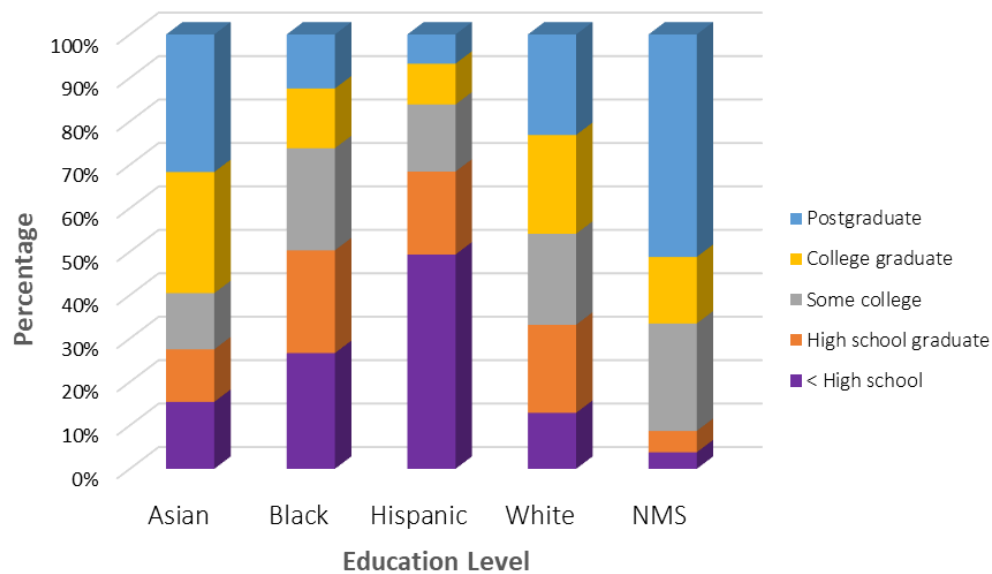
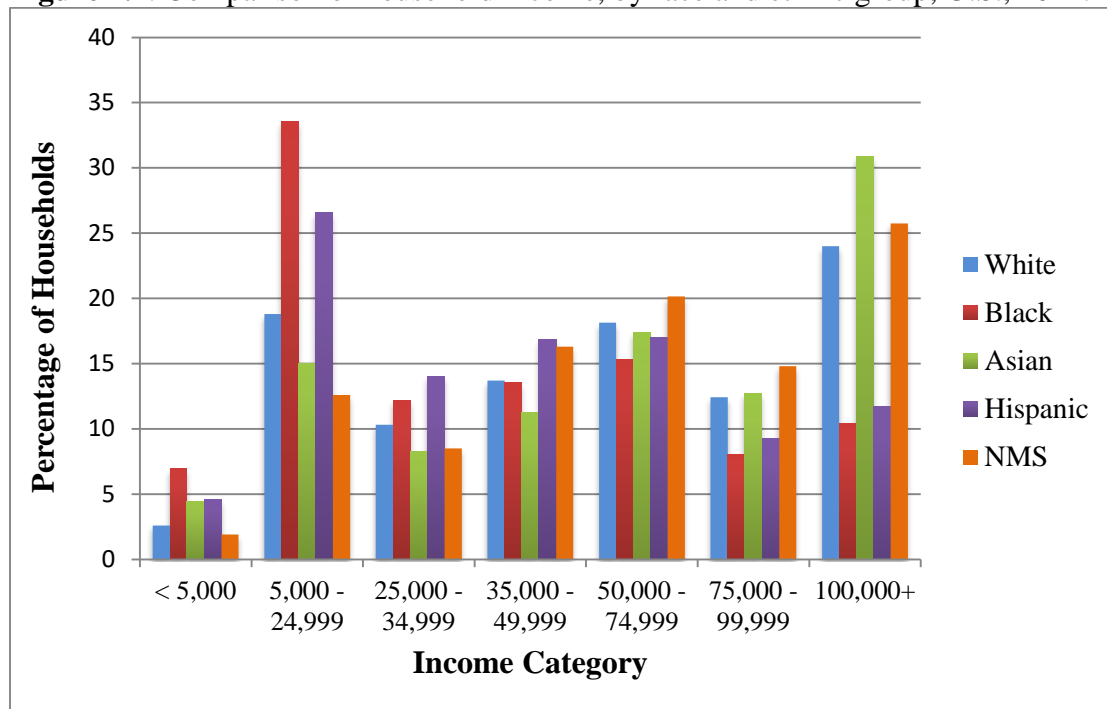


Figure 2.2. Comparison of household income, by race and ethnic group, U.S., 2011.

Source: U.S. Census Bureau, Current Population Survey, 2012 Annual Social and Economic Supplement.

Table 2.3. Distribution of continental ancestry in NMS and comparative samples [proportion (standard deviation or range)].

Study sample	Native American	European	African	Author(s), year
Albuquerque, New Mexico	0.22 (0.08)	0.73 (0.10)	0.05 (0.04)	Current study
Colorado	0.34 (0.02)	0.63 (0.02)	0.03 (0.02)	Bonilla et al. 2004
Mexico City	0.40 (0.07)	0.57 (0.07)	0.03 (0.01)	Wang et al. 2008
SE Texas	0.409±0.014	0.591±0.014	NA	Lisabeth et al. 2011
Native North American	0.0671±0.020	0.322±0.020	0.007±0.002	Tishkoff et al. 2009
U.S. African American	0.013±0.002	0.192±0.012	0.794±0.002	Tishkoff et al. 2009

Comparisons among ethnic subgroups within NMS

Table 2.4 shows pairwise comparisons of household income, self-reported education, parental education, continental ancestry, skin pigmentation, and demographic variables between the ethnic subgroups of NMS. There were no significant differences in socioeconomic factors between the subgroups. NMS individuals that self-identified as Mexican had significantly higher Native American and significantly lower European

continental ancestry compared to the NMS individuals that self-identified as Hispanic, Spanish and Nuevomexicano ($p < 0.05$). They also had significantly darker skin color than NMS individuals that self-identified as Hispanic, Spanish, and Nuevomexicano ($p=0.0484$, $p=0.0029$, $p=0.0035$, respectively). Self-identified Mexican-American NMS had significantly lower European ancestry compared to those who self-identified as Hispanic, Spanish, Nuevomexicano, Chicano, and Latino; and significantly darker skin color than self-identified Spanish and Nuevomexicano ($p=0.0264$ and $p=0.0374$, respectively). There were significant differences in age between Hispanic and Spanish, Spanish and Nuevomexicano, Spanish and Mexican, and Spanish and Mexican-American subgroups.

Table 2.4. Pairwise comparisons of self-reported ethnicity, SES, genetic ancestry, skin pigmentation, and control variables*.

Self-reported ethnicity	Hispanic	Spanish	Nuevo-mexicano	Mexican	Mexican American	Latino	Chicano
Hispanic	--						
Spanish	Age	--					
Nuevo-mexicano		Age	--				
Mexican	Eur. ancestry African ancestry Skin color	Eur. ancestry African ancestry Skin color Age	Eur. ancestry African ancestry Skin color	--			
Mexican American	Eur. ancestry	Eur. ancestry Skin color Age	Eur. ancestry Skin color		--	Eur. ancestry	Eur. ancestry
Latino					Eur. ancestry	--	
Chicano					Eur. ancestry		--

*Only significant differences (Tukey-Kramer, $p < 0.05$) presented in table.

Discussion

We identified significant differences in education, household income, skin color, and continental ancestry between NMS and other U.S. groups, illustrating that people of Spanish-speaking descent in NM differ in important factors that may contribute to health.

Furthermore, when asked to self-report their race, 46% of our participants selected “Some Other Race alone” from the same racial categories used in the 2000 U.S. Census. This is a larger proportion than state (27.7%) and national (36.7%) level responses to the same question on the 2010 U.S. Census, and suggests that people of Spanish-speaking descent cannot easily be subsumed into existing race categories because many do not identify with any of the categories (López, 2008).

To our knowledge, this is the first study to systematically explore self-identity in a U.S. HLS, population and measure how social and biological variables vary within and among self-identified ethnic subgroups. In contrast to a single national ethnic label, Hispanic/Latino, and also in contrast to the simplistic dichotomous model of Hispanic ethnicity in New Mexico, we show that NMS identified with seven distinctive ethnic subgroups. We found that there were no significant differences in socioeconomic factors between the subgroups. However, we observed patterns in the distribution of forehead skin pigmentation and European ancestry by subgroup. NMS individuals that self-identified as Mexican were also significantly different in continental ancestry and skin color compared to the NMS individuals that self-identified as Hispanic, Spanish and Nuevomexicano. Self-identified Mexican-American NMS showed significant differences in European ancestry and skin color compared to those who self-identified as Hispanic, Spanish, Nuevomexicano, Chicano, and Latino. These results are expected given the cultural history and ethnic variation of New Mexico, and further support the Spanish/non-Spanish ethnic subdivision in New Mexico (Bustamante, 1991; Trujillo, 2010). These results may suggest that there may be two commonly used ethnic terms (Hispanic and Mexican) that are the ends of a continuum; and within that continuum are several other

ethnic terms that are regionally, culturally, and temporally specific (Hunley et al., 2017; Healy et al., 2018).

These findings indicate that racial-genetic models that use continental ancestry as a proxy for racial and ethnic variation in the genetic factors underlying multifactorial disease is unwarranted. People of Spanish-speaking descent are not homogeneous; they describe themselves using a variety of ethnic subgroup names, and these groups are culturally and biologically diverse.

With regard to implications for health disparities research, these findings illustrate the general need to carefully define social and biological factors, such as ethnicity and socioeconomic status and explain: 1) how these variables are measured, and 2) how relevant those indicators are to the particular health outcome of interest *and* to the population under study. As with ethnic identity, socioeconomic characteristics vary by contextual factors such as age, situation, religion, place of residence, and country of origin. Therefore, determining the adequate data to collect on the ethnic and socioeconomic factors that contribute to health disparities will depend on knowledge of the regional and micro-level relevance of particular ethnic subgroups and socioeconomic variables (Gold et al., 2008).

Further, this study provides a subgroup-specific framework by which researchers can follow when measuring SES in health disparities research. First, identify racial/ethnic subgroups that may cause hidden heterogeneity in the study sample. Second, measure as much relevant socioeconomic information as possible. Third, systematically evaluate the associations between the socioeconomic measures and consider how relevant the measures are within the study sample. Fourth, specify the particular socioeconomic

factors measured and how the socioeconomic factors will be used in analysis (i.e., control variable or variable of main interest?). Finally, assess the relationships between the socioeconomic variables and other independent variables, such as ethnic subgroups *before* analyzing their predictive role in health status and health risk. This framework follows recommendations by social scientists and health disparities researchers alike to link ethnographic methods and data to health research, improve the quality of data on race or ethnicity and socioeconomic status, and increase the samples sizes for specific subgroups at state and regional levels (Braveman et al., 2005; Gold et al., 2008; Gravlee & Sweet, 2008; Kaplan & Bennett, 2003). It should be noted that this regional subgroup-level approach is not without limitations. Our attempt to understand the relationship between ethnicity and SES at the macro (examining our sample as one large “Hispanic” group) and micro level (examining our sample as composed of seven ethnic subgroups) did not reveal any apparent patterns related to income, education, skin color, or genetic ancestry that might be associated with health disparity. We question the use of standard socioeconomic variables in health disparities research and call for careful examination of the relationships between socioeconomic variables and ethnicity in health disparities research.

Chapter 3. Allostatic load and biomarkers among New Mexicans of Spanish-speaking descent and as compared to other U.S. groups

*This manuscript is currently under review with the *American Journal of Human Biology*

Introduction

Following recommendations from the Office of Management and Budget (OMB), the U.S. Census uses the phrase “of Hispanic, Latino or Spanish origin” (hereafter HLS), to refer to persons “of Cuban, Mexican, Puerto Rican, South or Central American, or other Spanish culture or origin regardless of race” (Humes, Jones, & Ramirez, 2011; OMB, 1997). HLS represent the largest and fastest-growing minority group in the U.S. (Census Bureau, 2018; Ennis et al., 2011). They have higher poverty rates, less education and less access to health care than non-Hispanic Whites (Braveman et al., 2011; Morales et al., 2002). Compared with non-HLS whites, the risk factors and prevalence of obesity, diabetes, hypertension, and chronic diseases of the stomach, liver, and kidneys are greater in HLS populations (Aponte, 2009; Braveman et al., 2011; Flegal et al., 2002; NCHS, 2017; Peralta et al., 2006). While a large body of literature has documented links between sociodemographic factors and health outcomes, there is need to better understand how health disparities may emerge over the life course (Adler & Rehkopf, 2008; Chyu & Upchurch, 2011; S. E. Taylor et al., 1997).

Allostatic load (AL) is a cumulative index of physiological dysfunction from a failure to adapt to chronic and prolonged exposure to stressors (Ben-Shlomo & Kuh, 2002). Exposure to stressors over the life course is believed to accelerate biological aging by promoting physiological dysregulations and increasing risk for certain chronic diseases (Masoro, 1997). As a multi-physiological system model of biological risk, AL is

useful for conceptualizing how chronic extrinsic and intrinsic stressors impose wear and tear on physiological systems, increasing morbidity and mortality over the life course (B. S. McEwen & Seeman, 1999) and contributing to health disparities in the U.S. (Geronimus et al., 2006). In some studies, AL is used to predict health outcomes, in others AL is used as an indicator of physiological stress. There is evidence linking AL with cardiovascular disease (Mattei, Demissie, Falcon, Ordovas, & Tucker, 2010) and nativity status in HLS, particularly Mexican immigrants, in the U.S. ((Crimmins et al., 2007; Kaestner, Pearson, Keene, & Geronimus, 2009; Peek et al., 2010; Salazar et al., 2016). However, there is a dearth of studies examining AL in HLS individuals who are U.S.-born and who self-identify with various HLS groups (e.g. Cuban, Mexican, Puerto Rican, South or Central American). In this study, we use AL as an indicator of physiological stress in New Mexicans of Spanish-speaking descent (NMS).

Three factors make New Mexico a good location for studying AL and its sociodemographic and biological correlates. First, New Mexico has the largest percentage of self-identified HLS residents in the U.S. (48% compared to the national average of 18.1%; (U.S. Census Bureau, 2018)). Second, due to New Mexico's specific regional history, a large proportion of its HLS residents are New Mexico-born and identify strongly with their Spanish ancestry and heritage, while another portion identifies strongly with recent Mexican ancestry and heritage (Hunley et al., 2017; Nostrand, 1992). Third, the U.S. Department of Health and Human Services ranks New Mexico well below average in performance measures of health care, rating the state as weak or very weak in treatment for diabetes, cancer, heart disease, respiratory disease, and maternal and child health (AHRQ, 2009, 2016). A 2018 report by the Commonwealth Fund ranked New

Mexico 35th and 36th for the percent of uninsured adults and children, respectively; and 42nd and 44th for overall performance in prevention and treatment, and healthcare access and affordability, respectively (Radley et al., 2018).

There is evidence suggesting that some HLS subgroups in New Mexico have a unique biological and cultural history (Doan & Stephan, 2006; Healy et al., 2018; Hunley et al., 2017; Salgado, 2018); here, we investigate variation in AL and its relationship to sociodemographic and biological factors between ethnic groups recognized by NMS. This work will elucidate the utility of AL for detecting variation in exposure to repeated stressors in a population, its predictive value for health risk, and the extent to which stress and risk vary within HLS subgroups and among racial and HLS groups in other U.S. regions (Salazar et al., 2016). To this end we ask: 1) Does AL differ among ethnic subgroups of New Mexicans of Spanish-speaking descent (NMS), and 2) Does AL differ between NMS and other HLS and racial groups in the U.S.?

Allostasis and allostatic load

Allostasis refers to the process whereby adaptive adjustments are made to the body's physiological systems in response to external and internal stressors. Normal functioning requires continual fluctuation, or adaptation, in physiological systems in response to stressful events, but these fluctuations are only adaptive when they are short-term, and the stressors are acute (Juster et al., 2010). When exposed to repeated stressors over the life course, however, these physiological systems may begin to experience impaired physiological functioning, or physiological "wear and tear" (B. McEwen, 1998). This cumulative burden, allostatic load, is the cost of allostasis, and may contribute to the

development of complex and chronic conditions such as hypertension, diabetes, obesity, and cardiovascular disease (B. McEwen, 1998; T. E. Seeman et al., 1997; Sterling & Eyer, 1988).

Comparisons across populations and among studies are particularly challenging with regard to allostatic load for three main reasons. First, not all studies use the same biomarkers in their calculations of allostatic load, due to convenience, availability, and data collection limitations (Beckie, 2012; Carlson & Chamberlain, 2005; Duong et al., 2017; Edes & Crews, 2017; Stewart, 2006). Second, there is no agreement about which equation to use when calculation allostatic load scores, making it difficult to meaningfully compare allostatic load scores and effects across samples and across populations (Beckie, 2012; Duong et al., 2017; McDade, 2008). Third, evidence suggests the relationship between various biomarkers and health outcomes differs by race, ethnicity, and socioeconomic status (Beckie, 2012; McDade, 2008). Despite these limitations, Edes and Crews (2017) make a strong case for the potential utility of allostasis via AL in biological anthropology. They argue that the use of AL in anthropological and other studies can be informative about life-history evolution, evolutionary trade-offs, health and well-being over the lifecourse, and human variation in response to extrinsic stressors (Edes & Crews, 2017).

Racial and ethnic differences in allostatic load

Recent research has provided evidence linking socioeconomic (i.e., socioeconomic status, education level, household income) and sociodemographic factors (i.e., nativity status), race, and ethnicity to allostatic load scores (Carlson & Chamberlain,

2005; Chyu & Upchurch, 2011; Howard & Sparks, 2015). With these findings, a clearer picture is beginning to form regarding how sociodemographic conditions are embodied via physiological and biological pathways that ultimately affect health and health outcomes.

These studies only present differences between racial and ethnic categories that are recognized by the Office of Management and Budget (OMB). However, there are HLS subpopulations in the U.S., including NMS, which self-identify with multiple distinctive ethnic subgroups within those larger OMB categories. These subgroup identities often have region-specific meanings tied to local histories (Doan & Stephan, 2006; Duany, 1998; Gonzales, 1993; Healy et al., 2018; Hunley et al., 2017; Salgado, 2018). Additionally, many studies examining AL in HLS populations focus on individuals who are foreign-born or have recently immigrated to the U.S. Studies that look for patterned differences among region-specific subgroups, such as the one reported here, are missing from the literature.

Materials and Methods

Study Objectives

The objectives of this study are to examine the patterns of AL and assess sociodemographic and biological differences in AL in a sample of New Mexicans of Spanish-speaking descent (NMS). The sociodemographic covariates include household income, education level, birthplace, age, and perceived discrimination. The biological covariates include continental ancestry and skin pigmentation. Continental ancestry is sometimes used as a proxy for geographic variation in the genetic factors underlying

multifactorial disease (Burchard et al., 2003; Risch, Burchard, Ziv, & Tang, 2002; Tang, Coram, Wang, Zhu, & Risch, 2006), but it also reflects social risk factors (Florez et al., 2011; Gravlee, Non, & Mulligan, 2009; Non, Gravlee, & Mulligan, 2012). We measured skin pigmentation because it may lead to discrimination that affects health (Klonoff & Landrine, 2000), noting that the literature on the relationships between skin pigmentation, discrimination, and health is sparse in HLS populations (Golash-Boza & Darity Jr, 2008). We looked at the relationship between these sociodemographic and biological factors and AL in our overall sample of NMS, then also examined differences in AL and its relationship to sociodemographic and biological factors between self-identified ethnic subgroups of NMS. We then compared our findings on AL in NMS to findings from previous research on AL and biomarkers in other U.S. subpopulations.

Study Sample and Procedures

We recruited 507 NMS and matched them by age, sex, and SES with the larger New Mexico HLS population (Hunley et al., 2017). Twelve study participants were excluded from analysis because they self-identified as a unique ethnic subgroup (e.g. Puerto Rican, Cuban, Panamanian, Filipino American, Anglo). A description of our sampling design, including participant self-identification of ethnic subgroup and birthplace, recruitment strategy, and estimation of continental ancestry is described in (Hunley et al., 2017). The complete study questionnaire can be found on our study website: <http://heritagenm.unm.edu/>.

Demographic factors

Sex was coded as a dichotomous variable equal to 1 for males and 2 for females. Age was collected as a continuous variable but used as a categorical variable (18-29, 30-39, 40-49, 50-59, 60-69, 70-70, and 80+ years) in regression models to examine patterns of increasing AL by age cohorts (Chyu & Upchurch, 2011; Pasta, 2009; Salazar et al., 2016). Household income was measured by asking participants which of eight income ranges described their total household income before taxes in the last twelve months, then coded as a categorical variable. Education was measured using five categories to represent the highest completed level of education (some high school or less, high-school graduate/GED, some college/vocational/technical, college graduate or, some postgraduate/professional school). Birthplace was dichotomized as U.S.-born/foreign-born.

Skin pigmentation

We measured the CIELab lightness metric (*L) on the forehead using a handheld reflectometer (DSM II ColorMeter; Cortex Technology, Hadsund, Denmark). Higher values of *L correspond to lighter skin pigmentation.

Continental ancestry

DNA was extracted from mouthwash samples and extracts were genotyped with the Illumina HumanCytoSNP-12 DNA Analysis BeadChip Kit (Illumina, Inc). Continental ancestry was estimated using polymorphic loci from individuals from the International HapMap Project and the Human Genome Diversity Panel-CEPH (Cann et

al., 2002; Consortium, 2005). Ancestry estimation methods have been described elsewhere (Hunley et al., 2017).

Perceived Discrimination

Discrimination may account for racial differences in health outcomes (Golash-Boza & Darity Jr, 2008; Williams & Mohammed, 2009). We asked participants six questions from the Everyday Discrimination Scale (Williams, Yu, Jackson, & Anderson, 1997) that measured participants' experience of different types of discrimination in the last six months. For each question, participants were given four choices: never, occasionally, sometimes and often. Answers were converted to a 4-point ordinal scale and summed for the six questions.

Allostatic Load

AL was measured using nine biomarkers that represent various physiological systems relevant to disease risk (Crimmins et al., 2007; T. E. Seeman et al., 2004; T. E. Seeman et al., 1997). Cardiovascular markers included diastolic and systolic blood pressure and pulse rate. Metabolic markers included body mass index (BMI), waist-to-hip ratio (WHR), glycosylated hemoglobin (HbA1c), and hemoglobin (Hb). C-reactive protein (CRP) was used as a marker of inflammation, and Epstein-Barr virus antibody (EBV) was used as a measure of immune response.

Blood pressure and resting pulse rate measurements were taken with an automated blood pressure monitor (Omron, Model # BP710N). Three measurements were taken while participants were seated at the beginning, middle, and end of the

interview (Perloff et al., 1993). The average of the three measurements was used in analyses. BMI was calculated as weight in kilograms divided by height in meters squared. Waist and hip circumferences were taken in centimeters for WHR.

HbA1c, Hb, CRP and EBV were obtained through dried blood spot samples. Each participant's finger was cleaned with an isopropyl alcohol wipe, then pricked with a disposable sterile lancet. The first drop of whole blood was wiped away with cotton and five subsequent blood drops were collected onto filter paper (Whatman #903). Samples were dried overnight, then each was sealed individually with desiccant in a resealable plastic bag and stored in a plastic container in a laboratory-grade freezer (-25°C) until analysis. All biomarkers except HbA1c were assayed in the Hominoid Reproductive Ecology Laboratory in the Department of Anthropology at the University of New Mexico. HbA1c dried blood spot samples were assayed by Healthpoint Diagnostix, Inc. (See Appendix C for full DBS protocol).

For each of the nine biomarkers, empirical cut-points were determined by the 75th percentile, operationalized as high-risk, with the exception of Hb, for which the high-risk cut-point was defined as below the 25th percentile. This is the most common method for determining cut-points in individual biomarkers (Edes & Crews, 2017). AL was calculated by summing the number of biomarkers identified as high-risk, following Seeman et al. (1997). For a composite score with nine biomarkers, the possible range of AL scores was 0-9, with higher AL scores indicating greater physiological dysregulation, and thus a greater accumulation of biological risk. Participants on medication were not differentiated from those not on medication (Chyu & Upchurch, 2011; T. E. Seeman, Singer, Ryff, Love, & Levy-Storms, 2002).

Comparative data

We used data from four AL studies (Gay et al., 2015; Howard & Sparks, 2015; Mattei et al., 2010; Salazar et al., 2016) to compare biomarker measures in NMS to those in other racial and HLS ethnic subgroups in the U.S. Given that not all studies use the same biomarkers in their calculations of allostatic load, there is incomplete overlap of data among the comparative studies and our NMS study.

Salazar et al. (2016) investigated AL accumulation patterns by age, sex and nativity status in a sample of 15,830 adults from the Hispanic Community Health Study/Study of Latinos. The study included HLS of Mexican, Cuban, Dominican, Puerto Rican, and Central and South American descent aged 18-74 years. AL was determined using an index based on 16 biomarkers spanning cardiometabolic, parasympathetic, and inflammatory systems. High-risk cut-off points were determined using the sample's lowest or highest 25th percentile, depending on the biomarker.

Gay et al. (2015) examined whether physical activity is associated with lower AL and inflammation in a sample of 330 Mexican American adults aged 18 years and older from the Cameron County Hispanic Cohort. The study included 10 biomarkers used to estimate AL scores based on clinical high-risk criteria.

Howard & Sparks (2015) assessed whether racial and ethnic differences in AL persist across levels of educational attainment using data from four waves (2005; 2007; 2009; and 2011) of the National Health and Nutrition Survey (NHANES). The study included 6,990 individuals aged 35 years and older who self-identified as either non-Hispanic White, non-Hispanic black, or Mexican American. AL was calculated using 10 biomarkers with clinically determined high-risk cut-off points.

Mattei et al. (2010) determined whether AL was associated with six chronic conditions in a sample of 1,116 Puerto Ricans ages 45-75 years from the Boston Puerto Rican Health Study. The outcome variables in the study were self-reported cardiovascular disease (CVD), hypertension, diabetes, abdominal obesity, cancer, and arthritis. The authors used clinical cut-off values to create a summary measure of AL from 10 biomarkers.

Statistical Analysis

We excluded 56 participants from analysis because they were missing one or more biomarker measures used to calculate AL. The final sample consisted of 439 individuals. We assessed differences in means of biomarker measures and sociodemographic variables between NMS groups using Fisher's Monte Carlo simulations and one-way ANOVAs for categorical variables and Pearson's correlation for continuous variables. We used the Tukey-Kramer method post-hoc for pairwise comparisons and to identify subgroup means that were significantly different from each other (Kramer, 1956). Because AL was operationalized as a count outcome and followed a non-normal distribution (see Figure 3.1), multivariate analyses were conducted using negative binomial regression models to investigate effects of covariates on AL (Ismail & Jemain, 2007).

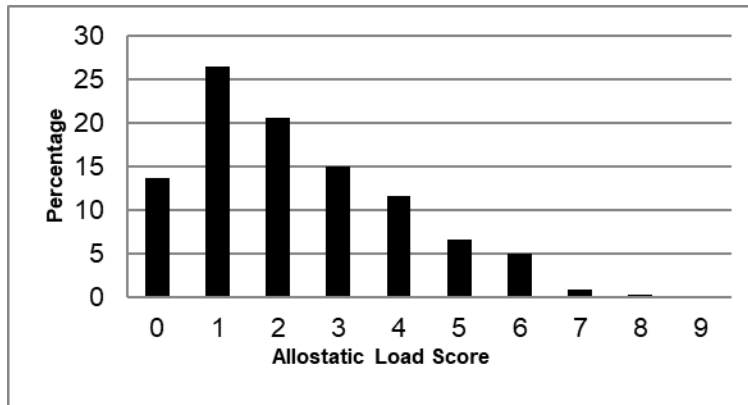


Figure 3.1. Percent distribution of allostatic load in NMS.

To test whether Native American, European, or African continental ancestry was associated with AL, we added each ancestry measure individually into separate models, as well as all combinations of ancestries. We present only the statistically significant models as results.

To assess differences in biomarker measures among NMS and the four studies described above, we conducted one-sample t-tests. We adjusted p-values for multiple tests using the Bonferroni correction method. Analyses were conducted in Stata 11 (StataCorp, 2009).

Results

Table 3.1 presents descriptive statistics of the nine biomarkers used to calculate AL, including range, mean, standard deviation, and quartiles. Figure 1 shows the distribution of AL. AL scores within NMS ranged from 0-8, with a mean of 2.30 and a standard deviation of 1.74.

Table 3.1. Distribution of individual allostatic load biomarkers and high-risk cutoffs.

				<i>Quartiles</i>			
<i>Biomarker</i>	<i>Range</i>	<i>Mean</i>	<i>Standard Deviation</i>	<i>25%</i>	<i>50%</i>	<i>75%</i>	<i>Clinical high-risk cutoff</i>
Cardiovascular markers							
Systolic blood pressure (mm Hg)	89.33-192.33	128.39	19.22	113	126.83	141.67	≥140 ^b
Diastolic blood pressure (mm Hg)	56-118.33	85.11	11.67	76.33	84.67	93.67	≥90 ^b
Pulse rate (beats/min)	40-123	71.54	11.24	64	71	77	≥90 ^c
Metabolic markers							
Body mass index (kg/m ²)	17.39-52	28.64	5.60	24.82	27.91	31.60	≥30 ^d
Waist-hip ratio (cm)	0.6526-1.329	0.8686	0.0936	0.8018	0.8693	0.9363	--
Females	0.6526-1.329	0.8196	0.0801	0.7706	0.8153	0.8686	≥0.85 ^e
Males	0.75-1.1523	0.9293	0.0707	0.8928	0.9313	0.9696	≥0.90 ^e
Glycosylated hemoglobin (HbA1c, %)	4.6-11.3	6.07	0.7782	5.6	5.9	6.3	≥6.5 ^f
Hemoglobin (g/dL)	9.25-21.89	15.99	1.90	14.62	15.93	17.26	≤13.5 ^g
Inflammatory markers							
C-reactive protein (mg/L)	0.00-9.4	1.96	1.91	0.63	1.3	2.43	≥3.0 ^h
Immune response biomarkers							
Epstein-barr virus antibody (ELISA units)	10.65-334.51	130.99	69.59	77.55	123.72	178.12	N/A
Allostatic load score ^a	0-8	2.30	1.74	--	--	--	--

Table 3.2 shows the sociodemographic characteristics and mean AL. Mean AL scores increased with each subsequent age category, except for the 80+ category (Figure 3.2A). Mean AL scores for individuals 18-29 were significantly lower than the mean AL scores for 40-49, 50-59, 60-69, and 70-79 age categories (TK-test, $p \leq 0.05$). AL scores do not differ significantly between males and females or by household income. Average AL values for individuals who self-identified as Latino and Spanish were higher than others

at 2.67 and 2.61, respectively, while individuals who self-identified as Mexican and Nuevomexicano showed lower AL scores at 2.00 and 2.06, respectively. However, there were no significant differences in AL between ethnic subgroups, when controlled for age. Individuals with high school diplomas/GEDs had significantly higher mean AL scores (3.15) than individuals with some college (2.29) and individuals with some postgraduate/professional college (2.03) (TK-test, $p \leq 0.05$; Figure 3.2B), even when controlled for age.

Table 3.3 presents the results from the negative binomial regression model. AL significantly increased with each subsequent age category. NMS individuals in the following age categories: 50-59, 60-69, 70-79, and 80+, had AL scores more than two times higher than individuals 18-29 years. Compared to individuals 18-29, NMS ages 70-79 were expected to have a rate 2.24 times greater for allostatic load score ($p \leq 0.001$). Native American ancestry was significantly positively associated with AL scores ($p = 0.014$). For each one percent increase in Native American ancestry, the rate for AL score would increase by a factor of 3.08. The correlation between AL and European ancestry is the inverse of AL and Native American ancestry. There were no significant differences associated with sex, education levels, perceived discrimination, or skin color among ethnic subgroups in AL scores.

Table 3.2. Distribution of sociodemographic characteristics and mean AL score among NMS.

Characteristic	Total (n = 439)		% Distribution 100.00	Allostatic load Mean
	Mean or %	SD		
Age*	47.90	17.55		
18-29			19.13	1.34
30-39			17.31	1.88
40-49			14.80	2.34
50-59			16.62	2.68
60-69			22.00	2.77
70-79			7.97	3.17
80+			2.05	3.00
Sex, %				
Female	55.35		55.35	2.32
Male	44.65		44.65	2.28
Self-reported ethnicity				
Chicano			10.71	2.30
Hispanic			46.01	2.29
Latino			3.42	2.67
Mexican			3.64	2.00
Mexican American			7.29	2.22
Nuevomexicano			14.12	2.06
Spanish			14.81	2.61
Nativity Status, %				
U.S. born	96.58		96.58	2.33
Mexico born	3.42		3.42	1.47
Education*				
Less than high school			2.94	2.23
High school/GED			9.05	3.15
Some college			42.37	2.29
College degree			15.49	2.38
Postgraduate			29.84	2.03
No response			0.22	1.00
Household income				
< \$5,000			1.35	1.67
\$5,000-24,999			10.93	2.12
\$25,000-34,999			7.28	2.47
\$35,000-49,999			15.03	2.35
\$50,000-74,999			20.95	2.47
> \$75,000			40.77	2.33
Skin color, forehead	27.20	4.51		
European ancestry, %	73.57	0.10		
Native American ancestry, %	21.64	0.08		

* $p \leq 0.05$

Figure 3.2. Heat barplots of allostatic load by age (A), and education (B). Each bar shows the proportion of individuals with allostatic load between 0-8.

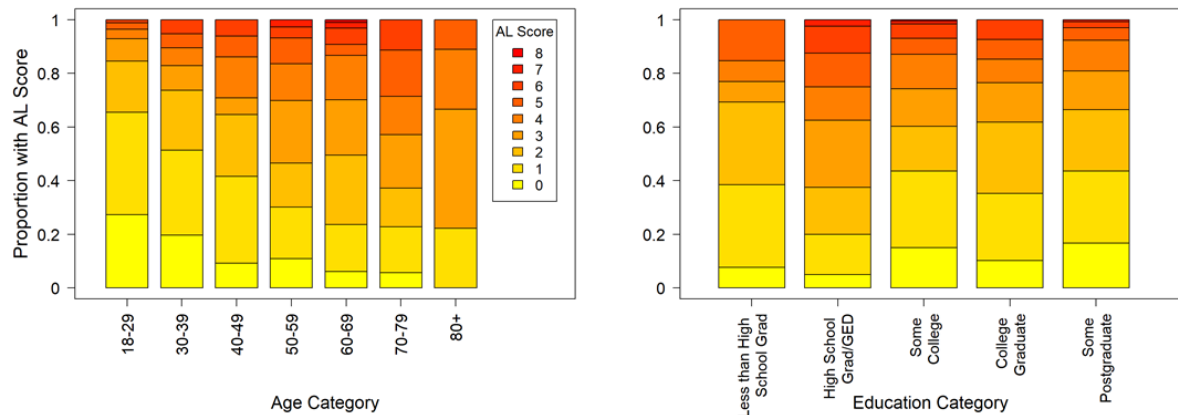


Table 3.3. Negative binomial regression results for allostatic load score in NMS.

<i>Sociodemographic characteristics (reference group)</i>	<i>Incidence rate ratios (95% confidence intervals)</i>
Age (18-29)	
30-39	1.37 (1.05, 1.78)*
40-49	1.76 (1.35, 2.28) ***
50-59	2.02 (1.57, 2.59)***
60-69	2.06 (1.62, 2.62)***
70-79	2.24 (1.67, 2.99)***
80+	2.18 (1.37, 3.47)**
Sex (Male)	
Female	0.98 (0.85, 1.12)
Self-reported Ethnicity (Hispanic)	
Spanish	1.02 (0.84, 1.25)
Nuevomexicano	0.97 (0.79, 1.20)
Mexican	0.84 (0.56, 1.24)
Mexican American	0.98 (0.73, 1.31)
Latino	1.11 (0.78, 1.60)
Chicano	0.99 (0.78, 1.24)
Education (Less than high school)	
High school/GED	1.21 (0.78, 1.88)
Some college	1.31 (0.75, 1.69)
College degree	1.05 (0.69, 1.61)
Postgraduate	0.94 (0.62, 1.42)
Skin color	1.00 (0.99, 1.01)
Native American ancestry	3.08 (1.25, 7.61)*
Perceived discrimination	1.01 (0.99, 1.03)

Variable in parenthesis indicates reference group. * $p \leq 0.05$, ** $p \leq 0.01$, *** $p \leq 0.001$.

Table 3.4 (pg. 45) shows comparisons of individual AL biomarkers between NMS and other U.S. AL study samples. The table highlights differences in individual biomarkers across the studies. We ran 29 one-sample t-tests to compare the means of individual biomarkers in the current research to those previously published and found that 82.76% of comparisons were significant. The majority (62.5%) of those comparisons demonstrated that NMS had significantly higher biomarker values. NMS men and women had significantly higher mean SBP compared to men and women in The Hispanic Community Health Study/Study of Latinos (HCHS/SOL) (Salazar et al., 2016), while NMS women had significantly lower mean SBP compared to Puerto Rican women from the Boston Puerto Rican Health Study (Mattei et al., 2010). Mean SBP was significantly higher in NMS compared to Mexican American adults in the Cameron County Hispanic Cohort (Gay et al., 2015), and compared to mean SBP in non-Hispanic Whites, non-Hispanic Blacks, and Mexican Americans from the National Health and Nutrition Survey (NHANES) (Howard & Sparks, 2015). NMS men and women had significantly higher DBP than Puerto Rican men and women from Boston. Mean DBP was significantly higher in NMS compared to Mexican American adults from The Cameron County Hispanic Cohort, and compared to non-Hispanic Whites, non-Hispanic Blacks, and Mexican Americans from NHANES. NMS men and women had significantly higher heart rates compared to men and women from the HCHS/SOL sample; and the mean heart rate in NMS was significantly than the mean heart rate in Mexican American adults from the Cameron County Hispanic Cohort. NMS women had significantly lower mean BMI compared to women from HCHS/SOL. Mean BMI was significantly lower in NMS compared to adults from the Cameron County Hispanic Cohort. The only significant

difference in WHR was found between NMS women and women from HCHS/SOL. HbA1c was significantly higher in NMS men and women compared to men and women from HCHS/SOL. Mean HbA1c in NMS was also significantly higher compared to the mean HbA1c in non-Hispanic Whites, non-Hispanic Blacks, and Mexican Americans from NHANES. However, HbA1c was significantly lower in NMS men and women compared to men and women in the Boston Puerto Rican Health Study; and mean HbA1c in NMS was also significantly lower than in Mexican Americans in the Cameron County Hispanic Cohort.

Discussion

To our knowledge, this study provides the first profile of the sociodemographic and biological correlates of AL among NMS adults. This study demonstrates the explanatory power of AL to examine health inequities which may result from exposure to extrinsic stressors throughout the lifecourse. Since AL is an indicator of accumulated physiological responses to stressors through the life course, we expected to see AL increase with age, and we did find this in our sample. We found no differences in AL scores between men and women, a novel finding among U.S. Hispanic/Spanish-speaking descent populations (Mattei et al., 2010; Salazar et al., 2016). Despite perceived differences among NMS subgroups, our results do not provide evidence of subgroup differences in allostatic load. To further test the utility of AL as a measure of health risk in HLS populations in the U.S., future research should focus on examining differences in region-specific ethnic subgroups. While we find no significant differences in health risk (AL) among NMS subgroups, there may still be differences in health outcomes among

Table 3.4. Data comparison of individual allostatic load biomarkers

Author(s), year	Sample (type: racial/ethnic subgroups)	N	Age (mean \pm SD; range)	Sex (% ♀)	# of AL biomarkers	Biomarkers	Biomarker mean (M) from comparison study		Biomarker mean (M) from NMS sample		One-sample t-test p-value ^a
Salazar et al. (2016)	Hispanic Community Health Study/Study of Latinos: Dominican, Puerto Rican, Cuban, Mexican, Central American, South American, Other/more than 1	15,830	18-74	60%	16	BMI*, WHR*, Trig, HDL, LDL, Gluc, HbA1c*, HOMA-IR, SBP*, RPP, HR*, %FEV ₁ /FVC, HRV ¹ , HRV ² , CRP, WBC	BMI, ♀	29.8	BMI, ♀	27.8	<0.0001**
							BMI, ♂	28.9	BMI, ♂	29.7	0.0397
							WHR, ♀	0.89	WHR, ♀	0.82	<0.0001**
							WHR, ♂	0.94	WHR, ♂	0.93	0.0377
							SBP, ♀	117	SBP, ♀	122	<0.0001**
							SBP, ♂	123	SBP, ♂	136	<0.0001**
							HR, ♀	67	HR, ♀	72	<0.0001**
							HR, ♂	64	HR, ♂	71	<0.0001**
							HbA1c, ♀	5.7	HbA1c, ♀	6.0	<0.0001**
							HbA1c, ♂	5.7	HbA1c, ♂	6.1	<0.0001**
Gay et al. (2015)	Cameron County Hispanic Cohort (CCHC): Mexican American	330	18 and older	67.9%	10	SBP*, DBP*, HR*, TC, HDL, BMI*, HbA1c*, CRP*, TNF- α , IL-6	SBP	117	SBP	128	<0.0001**
							DBP	72	DBP	85	<0.0001**
							HR	51	HR	72	<0.0001**
							BMI	30.8	BMI	28.6	<0.0001**
							HbA1c	6.7	HbA1c	6.1	<0.0001**
Howard & Sparks (2015)	National Health Nutrition Examination Survey (NHANES) 2003-2010: Non-Hispanic Black, Non-Hispanic White, Mexican American	6,990	25 and older	52.01%	10	SBP*, DBP*, HR*, TC, HDL, Trig, HbA1c*, BMI*, Alb, CRP*	SBP	122	SBP	128	<0.0001**
							DBP	70	DBP	85	<0.0001**
							HR	71	HR	72	0.3170
							HbA1c	5.6	HbA1c	6.1	<0.0001**
							BMI	28.9	BMI	28.6	0.2937
							CRP	0.43	CRP	1.96	<0.0001**
Mattei et al. (2010)	Boston Puerto Rican Health Study: Puerto Rican	1,116	45-75	72.04%	10	DHEA-S, Cort, NE, E, SBP*, DBP*, HDL, TC, HbA1c*, WC*	SBP, ♀	135	SBP, ♀	122	<0.0001**
							SBP, ♂	138	SBP, ♂	136	0.0786
							DBP, ♀	80	DBP, ♀	83	<0.0001**
							DBP, ♂	83	DBP, ♂	87	<0.0001**
							HbA1c, ♀	7.0	HbA1c, ♀	6.0	<0.0001**
							HbA1c, ♂	7.0	HbA1c, ♂	6.1	<0.0001**
							WC, ♀	101.1	WC, ♀	87.9	<0.0001**
							WC, ♂	101.9	WC, ♂	99.8	0.0614

Biomarker abbreviations: Alb: albumin, BMI: body mass index, Cort: cortisol, CRP: c-reactive protein, DHEA-S: dehydroepiandrosterone-sulphate, DBP: diastolic blood pressure, E: epinephrine, %FEV₁/FVC: Tiffeneau-Pinelli index (lung function), Gluc: glucose, HbA1c: glycosylated hemoglobin, HDL: high density lipoprotein cholesterol, HOMA-IR: homeostatic model assessment of insulin resistance, HR: heart rate, HRV₁: heart rate variability (square root of mean squared differences of NN intervals), HRV₂: heart rate variability (standard deviation of NN intervals), IL-6: interleukin-6, LDL: low density lipoprotein, NE: norepinephrine, RPP: resting pulse pressure SBP: systolic blood pressure, TC: total cholesterol, TNF- α : tumor necrosis factor-alpha, Trig: triglycerides, , WBC: total white blood count, WC: waist circumference, WHR: waist-to-hip ratio,

^a Bonferroni correction made to p-values to account for multiple one-sample comparisons. *Biomarker overlap with NMS sample. **p<0.05/n where n=number of comparisons

NMS subgroups. For nativity, we observed lower AL scores in Mexican-born NMS individuals in our sample; however, the difference was not statistically significant ($p = 0.0589$). Our data therefore do not support the healthy migrant effect, in which recent immigrants, specifically those born in Mexico, demonstrate health advantages over U.S.-born counterparts (Crimmins et al., 2007; Kaestner et al., 2009; Peek et al., 2010; Salazar et al., 2016).

Our findings show that while education level is not predictive of allostatic load score in a linear fashion, there is a significant association between certain levels of education and allostatic load. Specifically, controlled for age, NMS individuals with only high school diplomas/GEDs had significantly higher mean AL scores than NMS individuals with some college and individuals with some postgraduate/professional college. Significant associations between education level and allostatic load have been found in other studies that propose a dose response effect—the benefit of education increases as the level of education increases (Howard & Sparks, 2015). However, our findings suggest that the education-allostatic load relationship is more complex.

Perhaps most surprisingly, we found that continental ancestry was significantly associated with AL scores. In our sample, AL score decreased with increased proportions of European ancestry, and increased with increased proportions of Native American ancestry. To our knowledge, this is the first study to examine differences in AL by continental ancestry and report significant findings. There are several potential explanations for this result. It is possible that specific alleles more likely to be inherited from European ancestors are protective against health risk. Richman et al. (2012) found European ancestry to be protective against the development of renal disease in lupus patients from across the U.S. Alternatively, specific aspects of Native American ancestry may lead to greater health risk. However, given that much

of health disparities such as obesity and low birth weight, as well as preventative measures like colorectal screening and mammography are associated with socioeconomic variation (Berkowitz et al., 2015; Braveman et al., 2015; Frederick, Snellman, & Putnam, 2014; Vichare, 2016), it is perhaps more likely that continental ancestry is correlated with sociocultural factors not identified in the present study (Campbell et al., 2012; Florez et al., 2011; Gravlee et al., 2009; Richman et al., 2012). While we examined survey data on household income, perceived discrimination, and many other factors, no study can be comprehensive. Our results may be confounded by nongenetic factors that are, at this time, too complex or nuanced to measure. Additional avenues for future work exploring this relationship might include the mediating effects of rural and urban environments, neighborhood effects, environmental toxin exposure, parental health outcomes, or other variables important specific to the population histories and regions where the research takes place (Florez et al., 2011; Richman et al., 2012).

Our comparisons with other AL studies highlight the frequent use of metabolic and cardiovascular biomarkers such as SBP, DBP, HR, and HbA1c in calculations of AL. We observed significant differences in overlapping biomarkers that spanned different physiological systems. Given this finding, we might expect that a complete overlap in biomarkers would yield a similar trend in the way of significant differences in AL scores between NMS and the other samples containing individuals from different racial and ethnic groups. To this end, the inclusion of additional biomarkers in future studies might be warranted. We also found that most of the comparisons to other AL study samples demonstrated that NMS had significantly higher indicators of health risk through their biomarker measures. While the ultimate cause of this remains unknown, it cannot go ignored that New Mexico ranks below average in performance measures of health care and treatment, maternal and child health, number of insured adults and

children, and overall health care equity (AHRQ, 2009, 2016; Radley et al., 2018; Radley et al., 2014). Even with this consideration in mind, there is a large amount of variation in individual biomarkers and AL scores among NMS subgroups, highlighting that the complexity in interpreting AL reflects the complexity among HLS populations.

Several limitations to the current research should be noted. Our initial convenience-based sampling strategy led to biases in participant enrollment with regard to income and education, such that participants in our sample had higher completed levels of education and lower median income compared to the entire population of Albuquerque and the state of New Mexico (Healy et al., 2018). Participant interviews were conducted in English only. This may be one of the contributing factors to the small sample sizes of individuals who self-reported as Mexican and Mexican American. We did not have neuroendocrine or anti-inflammatory biomarkers such as, cortisol, epinephrine, or interleukin-6 available for analyses, thereby hampering our ability to compare our findings with other AL studies that include different sets of biomarkers, particularly primary mediators. Finally, while our findings do not support the healthy migrant hypothesis, our sample size of foreign-born individuals is small. To further confound the matter, a high proportion of our foreign-born participants were younger adults, who according to the aging trend of AL, are expected to have lower AL scores. A larger sample size of foreign-born individuals, likely to be recruited with the implementation of Spanish-speaking interviews, would help in determining whether a health migrant effect exists in our sample.

Chapter 4. Allostatic load is associated with gallbladder disease and abdominal obesity in New Mexicans of Spanish-speaking descent

Introduction:

Understanding causes of racial and ethnic health disparities and eliminating such disparities has been an enduring challenge and public health priority in the U.S. (Mattei et al., 2010; NCHS, 2015, 2019; Warnecke et al., 2008). As the largest minority group in the U.S., populations of Hispanic, Latino, or Spanish origin (HLS) have been shown to have higher prevalence of chronic diseases compared to other racial and ethnic groups (Cuevas, Dawson, & Williams, 2016; Hajat et al., 2000; Velasco-Mondragon et al., 2016; Zsembik & Fennell, 2005). Compared with non-HLS Whites, the risk factors and prevalence of obesity, diabetes, hypertension, and chronic diseases of the heart, stomach, liver, kidneys, and gallbladder are greater in HLS populations (Aponte, 2009; Flegal et al., 2010; Jaruvongvanich, Yang, Peeraphatdit, & Roberts, 2017; Peralta et al., 2006; Velasco-Mondragon et al., 2016). HLS are also disproportionately affected by poor quality/conditions of daily life that are shaped by adverse structural and social factors such as ethnic discrimination, income, education, occupation, language, cultural values and behaviors, and health care access and quality (Velasco-Mondragon et al., 2016; Hunley et al., 2017). These disparities in social factors and health are particularly striking in New Mexico, the state with the largest proportion (46%) of self-identified HLS U.S. Census 2010), and where performance measures in health care, health care coverage, chronic disease treatment, and maternal and child health and well-being have long ranked near the lowest in the nation ((AHRQ, 2009, 2016; Radley et al., 2018; Radley et al., 2014)).

Allostatic load (AL) has been proposed as a theoretical framework to understand how health disparities in minoritized groups emerge (Carlson & Chamberlain, 2005; Salazar et al., 2016; Tucker, 2005). Whereas previous public health research has focused on health disparities

as a genetic attribute, an increased tendency to engage in risky behaviors, or with the use of race or ethnicity as a proxy for measures for poor socioeconomic factors (Carlson & Chamberlain, 2005; Kneipp & Drevdahl, 2003), allostasis theory instead utilizes a life course perspective and multi-system view of adaptive physiological responses (Carlson & Chamberlain, 2005).

Allostasis refers to the process of adaptation to ever-changing internal and external challenges through adjustments made in multiple physiological systems, including the hypothalamic-pituitary-adrenal axis, autonomic nervous system, and metabolic and immune systems (B. McEwen, 1998; Steptoe et al., 2014). Allostasis is essential for maintaining homeostasis, but chronic insults from intrinsic and extrinsic stressors leads to allostatic load, the cumulative wear and tear that results from the inability to maintain regulatory processes (B. McEwen, 1998; B. McEwen & Stellar, 1993). When a stressor is triggered, primary responders/mediators (HPA axis and sympathetic nervous system) are activated and signal changes in secondary physiological systems (e.g., metabolic, cardiovascular, inflammatory) as part of a generalized stress response (McEwen, 1998). With each new or sustained stressor, allostatic load accumulates, producing an allostatic *overload*; and tertiary outcomes such as cardiovascular disease, hypertension, diabetes, and mortality may emerge as stress-related diseases among individuals and as health disparities at the population level (McEwen, 1998). Although this framework has gained momentum in studies of racial and ethnic health disparities, until now there have been few studies on HLS populations (Mattei et al., 2010; Mosley et al., in review).

Stress may be triggered by various external and internal factors and events, as well as the individual's adaptive responses to those events. In New Mexico for example, HLS populations experience high poverty levels, higher unemployment rates, greater health care inequities, negative life events, and real or perceived ethnic discrimination than non-HLS Whites (Atkeson,

Bryant, Hall, Saunders, & Alvarez, 2010; Monforti & Sanchez, 2010; NMDH, 2019).

Additionally, individual biological variation and harmful behavioral patterns may influence the ways in which a person responds physiologically to stress(ors) (Edes & Crews, 2017; B. McEwen, 1998; T. E. Seeman, McEwen, Rowe, & Singer, 2001). Some harmful behavioral patterns seen in New Mexican HLS include alcohol and substance abuse, tobacco use, low physical activity, and poor dietary/nutritional intake (NMDH, 2019). Thus, external stressors and individual factors likely influence the observed disparities of chronic diseases experienced by HLS individuals in New Mexico. The link between external and internal stressors, allostatic load, and chronic disease(s) is not fully understood in HLS individuals, particularly those who are U.S.-born and self-identify with various HLS subgroups.

The aim of this study is to determine the association of AL with six chronic health disease outcomes (abdominal obesity, hypertension, diabetes, cardiovascular disease, cancer, and gallbladder disease) in a sample of New Mexican HLS (hereafter referred to as New Mexicans of Spanish-speaking descent [NMS]) to distinguish study participants from the larger HLS population in New Mexico and the U.S). Previous research has shown that higher allostatic load is associated with physical and cognitive decline (T. E. Seeman et al., 1997), ischemic heart disease (Sabbah, Watt, Sheiham, & Tsakos, 2008), periodontal disease (Sabbah, Watt, Sheiham, & Tsakos, 2008), cardiovascular disease (Mattei et al., 2010), diabetes (Mattei et al., 2010), arthritis (Mattei et al., 2010), and increased mortality risks (Borrell, Dallo, & Nguyen, 2010; Duru, Harawa, Kermah, & Norris, 2012; Karlamangla, Singer, & Seeman, 2006). However, to our knowledge, this is the first study to examine whether AL is associated with chronic conditions in a sample of New Mexican HLS (hereafter referred to as New Mexicans of Spanish-speaking descent [NMS]) to distinguish study participants from the larger HLS population in New

Mexico and the U.S.). These conditions are the leading causes of morbidity and mortality in the U.S., and disproportionately affect HLS populations (CDC 2013). Previous work (Mosley et al., in review) has shown that older NMS have higher AL than do younger NMS. We predict higher AL is associated with an increased likelihood of chronic diseases when this relationship is controlled for age. While this relationship may seem intuitive, to date it has only been explicitly tested in one prior study (Mattei et al., 2010).

Materials and Methods

Study sample and procedures

The study sample consisted of 507 adults (18 or older) who self-identified as NMS. For a full description of our sampling design, recruitment strategy, and interview procedures not discussed here, see Hunley et al. (2017) and Mosley et al. (in review).

Allostatic Load

A summary measure of AL was calculated from nine biomarkers of biological functioning across a range of physiological systems. Cardiovascular markers included diastolic and systolic blood pressure and pulse rate. Metabolic markers included body mass index (BMI), waist-to-hip ratio (WHR), glycosylated hemoglobin (HbA1c), and hemoglobin (Hb).

Inflammatory markers included C-reactive protein (CRP), and immune response biomarkers included Epstein-Barr virus antibody (EBV). HbA1c, Hb, CRP and EBV were obtained through dried blood spot samples. For details regarding collection of the biomarkers, including dried blood spot samples and assay protocols, see Mosley et al. (in review; Chapter 3). The AL score was constructed by obtaining a sum of the number of biomarkers for which a participant fell into

the highest risk group. Cutoff thresholds were defined using established clinical criteria [Table 4.2; (ADA, 2017; Jensen et al., 2014; Pearson et al., 2003; Whelton et al., 2018)]. One point was assigned for each type of medication used (i.e., hypertension and diabetes), if the respective AL biomarker was within the clinical cutoff. Other covariates adjusted for in the regression models included age, sex, and smoking status (current smoker, non-smoker).

Chronic disease definitions

Abdominal obesity was defined as having waist circumference (WC) ≥ 88 cm in women or ≥ 102 cm in men (Jensen et al., 2014). Hypertension was determined by self-reported medical diagnosis or taking hypertension medicine. Glycosylated hemoglobin (HbA1c) $\geq 6.5\%$ or use of anti-diabetes medication indicated a diagnosis of diabetes (ADA, 2017). Self-reported medical diagnosis of heart attack, heart disease or stroke was used to define cardiovascular disease (CVD). Diagnoses of cancer and gallbladder disease were self-reported. Disease variables were dichotomized as having or not having the disease. When examining a disease that included one of the AL biomarkers in its definition or determination, that biomarker was excluded from the AL index (e.g., exclusion of WHR when assessing abdominal obesity, of HbA1c for diabetes, and of SBP and DBP for hypertension).

Statistical Analyses

Pearson's chi-square test statistic was used to determine significant differences in categorical variables. We used T-tests used to compare means in continuous variables. Using simple logistic regression models, the associations of each individual AL biomarker with each chronic condition were assessed, both unadjusted and adjusted for the effect of the covariates

mentioned above. To evaluate the association between each dichotomous health condition and AL, logistic regression models, fitted to estimate odds ratios. Then, we evaluated the association between each dichotomous health condition with AL using logistic regression models fitted to estimate odds ratios (OR) and 95% confidence intervals (CI), controlling for age, sex, and current smoking status.

Additional logistic regression models were run to test the consistency of results and to adjust for other potential confounders (household income, health insurance status, education, nativity status, and perceived discrimination). These models did not alter the association between allostatic load and disease outcome, and so are not presented here. Analyses were conducted using STATA 11 (StataCorp 2009).

Results

Forty-seven percent of participants had an AL score of 0 or 1. Also, relatively few participants had AL scores greater than or equal to 5. Thus, participants with 0 and 1 scores were grouped together and served as the reference group in logistic regression models, and participants with AL scores of 5 and higher were grouped together, resulting in a total of five AL categories (0+1, 2, 3, 4, and ≥ 5).

Table 4.1 presents descriptive statistics for the independent and dependent variables. The mean age of our participants was 47.9 years (SD 17.55). Men had significantly greater prevalence of hypertension and cardiovascular disease than women, while women had significantly higher prevalence of gallbladder disease than men. Other covariate measures and outcome variables did not differ significantly by sex.

Table 4.1. Descriptive characteristics for NMS, presented as a mean (SD) or percent.

	Women (n = 243)	Men (n = 196)	All participants (n = 439)
Age (years), %	47.7 (17.47)	48.2 (17.71)	47.90 (17.55)
Age Category, %			
18-29	20.58	17.35	19.13
30-39	15.23	19.90	17.31
40-49	13.17	16.84	14.80
50-59	20.16	12.24	16.62
60-69	23.04	20.92	22.00
70-79	6.17	10.20	7.97
80+	1.65	2.55	2.05
Household Income, %			
< \$5,000	2.06	0.51	1.35
\$5,000-24,999	10.7	11.22	10.93
\$25,000-34,999	8.64	5.61	7.28
\$35,000-49,999	14.81	15.31	15.03
\$50,000-74,999	21.0	20.92	20.95
> \$75,000	38.68	42.86	40.77
No response	4.11	3.57	3.87
Current smoker, %	7.0	10.71	8.66
Abdominal obesity^a	45.7	40.8	43.5
Hypertension^b	19.3	27.6*	23.0
Diabetes^c	21.0	19.9	20.5
Cardiovascular disease^d	0.82	3.6*	2.05
Cancer^e	6.6	5.1	5.9
Gallbladder disease^e	11.5*	2.0	7.3

* $p < 0.05$.

^a WC ≥ 102 cm in men or ≥ 88 cm in women.

^b Taking hypertension medicine.

^c HbA1c $\geq 6.5\%$ or taking diabetes medication.

^d Self-reported heart disease, heart attack or stroke.

^e Self-reported.

Table 4.2 presents mean and standard deviation for AL scores and each of the nine biomarkers used to calculate AL. The mean AL score was 1.79 (1.5) for all participants. There was no significant difference in AL score between men and women. Men had significantly higher systolic blood pressure, diastolic blood pressure, BMI, and waist-to-hip ratio than women. Women had significantly lower hemoglobin levels than men, and significantly higher C-reactive protein and Epstein-Barr virus antibodies compared to men. Controlling for age, there were no statistically significant differences in pulse rate and glycosylated hemoglobin between men and women.

Table 4.2. Allostatic load, associated biomarkers, and percentage of NMS exceeding clinical cutoffs by sex.

Parameter	Mean \pm (SD) or %			Clinical cutoff points	%		
	Men	Women	All participants		Men	Women	All participants
Allostatic load	1.87 (1.49)	1.73 (1.49)	1.79 (1.5)	—	—	—	—
Systolic blood pressure (mmHg)***	135.85 (17.06)	122.38 (18.79)	128.39 (19.22)	$\geq 140^a$	39.28	18.52	27.79
Diastolic blood pressure (mmHg)***	87.47 (11.63)	83.19 (11.38)	85.11 (11.67)	$\geq 90^a$	43.88	27.57	34.85
Pulse rate (beats/min)	71.42 (12.94)	71.63 (9.69)	71.54 (11.24)	$\geq 90^b$	10.71	4.11	7.06
Body mass index (kg/m ²)***	29.67 (5.23)	27.80 (5.76)	28.64 (5.60)	$\geq 30^c$	37.76	30.86	33.94
Waist-hip ratio (cm)***	0.9293 (0.0707)	0.8196 (0.0801)	0.869 (0.094)	Men, $\geq 0.85^d$ Women, $\geq 0.9^d$	84.7	13.99	45.55
Glycosylated hemoglobin (HbA1c, %)	6.12 (0.74)	6.03 (0.80)	6.07 (0.78)	$\geq 6.5^e$	18.37	18.52	18.45
Hemoglobin (g/dL)***	17.01 (1.86)	15.17 (1.49)	15.99 (1.90)	Men, $\leq 13.5^f$ Women, $\leq 12^f$	3.06	1.23	2.05
C-reactive protein (mg/L)**	1.63 (1.67)	2.23 (2.05)	1.96 (1.91)	$\geq 3.0^g$	13.27	25.92	25.06
Epstein-barr virus antibody (ELISA units)***	113.9 (66.41)	144.78 (69.17)	131.0 (69.6)	$\geq 178.12^a$	17.35	31.27	20.27
Total medication use for diabetes, %	19.39	2.06	2.96	Medication use for diabetes and HbA1c ≥ 6.5	19.9	20.99	20.50
Total medication use for hypertension, %	4.08	14.40	16.63	Medication use for hypertension and SBP ≥ 140 and DBP ≥ 90	41.84	24.28	32.12

^aWhelton et al. (2018).^bAmerican Heart Association^cCenter for Disease Control and Prevention^dWorld Health Organization^eADA (2017).^fMayo Clinic^gPearson et al. (2003).

EBV= 75% quartile

Significantly different by sex at * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 4.3 shows the association between the health outcomes and the nine biomarkers of allostatic load. Five of the nine biomarkers were significantly associated with higher probability of chronic disease both in unadjusted models and after adjusting for the effect of age, sex, and current smoking status. Specifically, individuals with higher BMI were 1.12 times more likely to have gallbladder disease (95% CI 1.06, 1.19). Individuals with higher CRP levels were 1.29

times more likely to have abdominal obesity (95% CI 1.15, 1.44), and 1.14 times more likely to have hypertension (95% CI 1.01, 1.23). Individuals with higher hemoglobin levels had 28.8% lower odds of having gallbladder disease (95% CI .560, .907).

Table 4.4 presents logistic regression model results. When controlling for age, sex and current smoking status, the three highest AL categories were significantly associated with gallbladder disease (OR (95% CI) = 4.17 (1.38, 12.63), 3.45 (0.975, 12.23), and 8.42 (2.11, 33.57) for categories 3, 4, and ≥ 5 , respectively) compared to participants with 0 or 1 AL scores (See Figure 4.1). NMS with AL scores of 2, 3, 4 and ≥ 5 were significantly more likely to have abdominal obesity than those in the reference group (2.24 (1.34, 3.73), 3.13 (1.71, 5.74), 5.19 (2.57, 10.50) and 4.60 (1.71, 12.34), respectively (See Figure 4.2). AL score was not significantly associated with any other chronic conditions.

Table 4.4. Odds ratio (OR) and 95% confidence interval (CI) for having each chronic disease by allostatic load category in NMS.

Chronic Disease	Allostatic Load Category	Odds Ratio (CI)	p-value
Abdominal obesity	2	2.24 (1.34, 3.73)	0.002
	3	3.13 (1.71, 5.74)	<0.001
	4	5.19 (2.57, 10.50)	<0.001
	≥ 5	4.60 (1.71, 12.34)	0.002
Hypertension	2	1.16 (0.66, 2.04)	0.607
	3	1.23 (0.55, 2.76)	0.620
	4	2.48 (0.71, 8.58)	0.153
	≥ 5	2.69 (0.16, 46.00)	0.494
Diabetes	2	0.79 (0.39, 1.57)	0.499
	3	1.83 (0.94, 3.57)	0.075
	4	1.19 (0.51, 2.80)	0.687
	≥ 5	1.95 (0.52, 7.42)	0.328
Cardiovascular disease	2	5.01 (0.94, 26.68)	0.059
	3	4.58 (0.73, 28.60)	0.103
	4	4.05 (0.54, 30.35)	0.173
	≥ 5	--	--
Gallbladder disease	2	1.87 (0.62, 5.71)	0.268
	3	4.17 (1.38, 12.63)	0.012
	4	3.45 (0.98, 12.23)	0.055
	≥ 5	8.42 (2.11, 33.57)	0.003
Cancer	2	0.98 (0.31, 3.04)	0.970
	3	2.40 (0.84, 6.86)	0.103
	4	1.29 (0.33, 5.08)	0.711
	≥ 5	0.75 (0.09, 6.50)	0.798

Figure 4.1. Odds ratios and confidence intervals for gallbladder disease by allostatic load, controlling for age, sex, and smoking status.

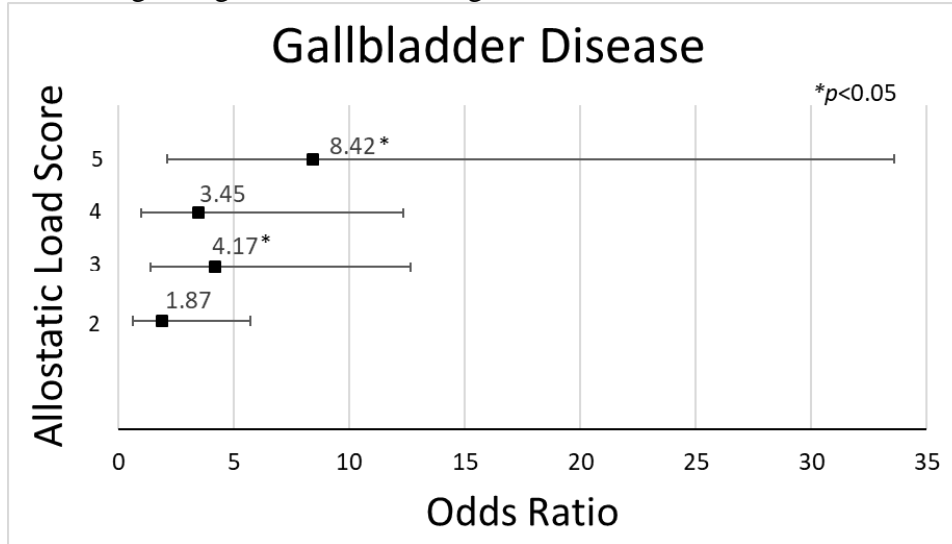
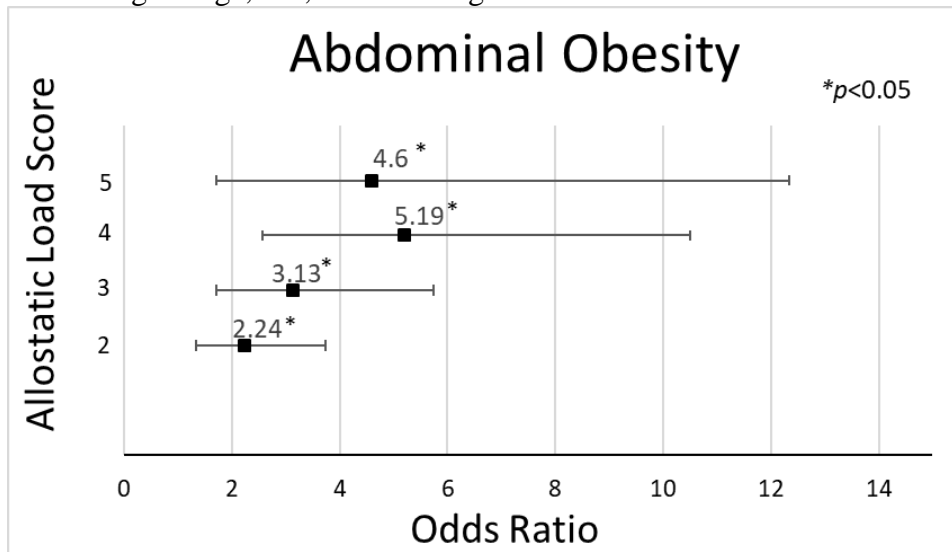


Figure 4.2. Odds ratios and confidence intervals for abdominal obesity by allostatic load, controlling for age, sex, and smoking status.



Discussion

Given AL is an indicator of accumulated physiological responses to stressors through the life course, we expected to see AL increase with age, and found this trend in our sample. We found no differences in AL scores between men and women, a novel finding considering

previous research has found sex differences in AL in other U.S. Hispanic samples (Mattei et al., 2010; Salazar et al., 2016).

We examined nine biomarkers of allostatic load, measuring their individual and cumulative effects on disease outcomes. Our findings show that five biomarkers were significantly associated with chronic diseases in NMS. We found C-reactive protein to be significantly associated with abdominal obesity and hypertension. C-reactive protein has also been linked to increased risk for cardiovascular disease (ERFC, 2010; Sabbah et al., 2008). However, we did not find a significant association between CRP and cardiovascular disease in our sample. Hemoglobin was significantly associated with lower odds of developing gallbladder disease, supporting research showing a correlation between iron deficiency and gallbladder disease (Aslam et al., 2013; Sahu, Jain, Prakash, Bahl, & Sachan, 2007).

Increasing categories of AL were significantly associated with higher odds of abdominal obesity and gallbladder disease, but not with cancer, cardiovascular disease, or hypertension. Previous research has shown the prevalence of gallbladder disease to be higher in HLS populations compared to any other ethnic or racial groups in the U.S. (Maurer et al., 1989), and higher in individuals with Native American ancestry (Henley, Weir, Jim, Watson, & Richardson, 2015; Miquel et al., 1998).

These findings point toward a recommendation that NMS should aim to have two or less biomarkers at high-risk levels, as the odds for chronic disease were greatly reduced under that value. These findings highlight the potential utility of AL in implementing targeted early interventions at the local and community level. In New Mexico, this could be smoking cessation, stress-reduction, stress-reduction programs, low-fat dietary guidance, and/or the promotion of increased physical activity.

There is a large body of evidence linking higher allostatic load levels with increased morbidity and mortality regardless of race and ethnicity (Borrell et al., 2010; Karlamangla et al., 2006; T. E. Seeman et al., 2001). Additionally, the existence of racial and ethnic disparities in AL has been established (Crimmins & Saito, 2001; Geronimus et al., 2006; Kaestner et al., 2009; Mattei et al., 2010; Peek et al., 2010; T. Seeman, Epel, Gruenewald, Karlamangla, & McEwen, 2010). However, there is a dearth of evidence linking AL to specific chronic diseases, let alone in a racial or ethnic minority population. There are potentially significant implications for medical treatments and intervention initiatives. Thus, we recommend that future AL studies focus on examining direct relationships between AL and chronic diseases in other populations.

Limitations

Our study under sampled some HLS subgroups, particularly Mexican and Mexican American (Hunley et al., 2017). Individuals from racial and ethnic minority populations, including individuals who identify as Mexican or Mexican American may be less inclined to participate in research studies (George, Duran, & Norris, 2014; Yancey, Ortega, & Kumanyika, 2006). Additionally, we conducted our interviews in English only, which may contribute to our low sample sizes for those two ethnic groups that may higher proportions of Spanish-speakers. The allostatic load biomarkers included in the estimation of AL did not include primary indicators such as norepinephrine, epinephrine, cortisol, and DHEA-s because these were not feasible within the parameters of our data collection. We did not find a significant association between any of the individual biomarkers or allostatic load and cardiovascular disease. Considering only 2% of our sample were diagnosed with cardiovascular disease, we did not have the statistical power to detect the effect of CRP on cardiovascular disease.

Table 4.3. Association between biomarkers of allostatic load with chronic diseases. Presented as odds ratio (OR) and 95% confidence interval (CI).

	Systolic BP	Diastolic BP	Pulse rate	BMI	HbA1c	Hemoglobin	CRP	EBV
Abdominal Obesity								
Unadjusted OR	1.01* (1.00, 1.02)	1.01 (0.995, 1.03)	1.02* (1.00, 1.03)	---	1.34* (1.04, 1.73)	0.925 (0.837, 1.02)	1.29* (1.16, 1.45)	1.00* (1.00, 1.01)
Adjusted OR	1.01* (1.00, 1.03)	1.01 (0.990, 1.02)	1.02* (1.00, 1.04)	---	1.24 (0.951, 1.62)	0.946 (0.842, 1.06)	1.29* (1.15, 1.44)	1.00 (0.999, 1.00)
Cancer								
Unadjusted OR	1.02* (1.00, 1.05)	1.00 (0.971, 1.04)	0.98 (0.945, 1.02)	0.983 (0.913, 1.06)	1.25 (0.821, 1.89)	0.844 (0.681, 1.05)	0.959 (0.769, 1.19)	1.00 (0.998, 1.01)
Adjusted OR	1.03* (1.00, 1.05)	1.00 (0.966, 1.04)	0.975 (0.936, 1.06)	0.985 (0.912, 1.06)	1.15 (0.742, 1.80)	0.820 (0.639, 1.05)	0.949 (0.748, 1.21)	1.00 (0.996, 1.01)
Cardiovascular Disease								
Unadjusted OR	1.02 (0.997, 1.05)	1.02 (0.970, 1.07)	0.936* (0.880, 0.997)	1.07 (0.937, 1.17)	1.44 (0.859, 2.41)	0.963 (0.710, 1.31)	0.590 (0.315, 1.11)	0.994 (0.984, 1.00)
Adjusted OR	1.01 (0.983, 1.05)	1.00 (0.958, 1.06)	0.949 (0.897, 1.00)	1.05 (0.952, 1.16)	1.42 (0.814, 2.47)	0.767 (0.549, 1.07)	0.633 (0.337, 1.19)	0.997 (0.987, 1.01)
Diabetes								
Unadjusted OR	1.02* (1.01, 1.04)	1.03* (1.01, 1.05)	1.00 (0.985, 1.03)	1.08 * (1.04, 1.12)	---	0.880* (0.776, 0.997)	1.11 (0.996, 1.25)	1.00* (1.00, 1.00)
Adjusted OR	1.00 (0.989, 1.02)	1.01 (0.988, 1.03)	1.01 (0.991, 1.03)	1.08* (1.01, 1.13)	---	0.904 (0.789, 1.04)	1.12 (0.989, 1.27)	1.00 (0.998, 1.00)
Gallbladder Disease								
Unadjusted OR	0.999 (0.981, 1.02)	1.00 (0.970, 1.03)	1.00 (0.972, 1.03)	1.10* (1.04, 1.17)	1.48* (1.05, 2.08)	0.653* (0.527, 0.809)	1.17 (0.995, 1.37)	1.00* (1.00, 1.01)
Adjusted OR	0.999 (0.977, 1.02)	0.999 (0.964, 1.03)	1.00 (0.969, 1.04)	1.12* (1.06, 1.19)	1.41 (0.971, 2.05)	0.712* (0.560, 0.907)	1.12 (0.943, 1.34)	1.00 (0.997, 1.01)
Hypertension								
Unadjusted OR	---	---	1.02 (0.998, 1.04)	1.08* (1.04, 1.13)	1.44* (1.11, 1.87)	1.07 (0.955, 1.21)	1.12* (1.00, 1.25)	0.999 (0.997, 1.00)
Adjusted OR	---	---	1.02* (1.00, 1.04)	1.07* (1.03, 1.12)	1.20 (0.908, 1.59)	1.06 (0.921, 1.21)	1.14* (1.01, 1.23)	0.999 (0.996, 1.00)

OR: odds ratio; BP: blood pressure; BMI: body mass index; W-H Ratio: waist-to-hip ratio; HbA1c: glycosylated hemoglobin; CRP: C-reactive protein; EBV: Epstein-barr virus antibodies

* $p < 0.05$

Adjusted model controls for age, sex, and current smoking status.

Chapter 5. Summary and Conclusions

Summary of Findings

Using sociocultural, biological, and genetic data, this dissertation presents a biocultural examination of health risk among New Mexicans of Spanish-speaking descent (NMS). Previous epidemiological research has combined individuals of Spanish-speaking descent into one ethnic group, supposedly homogenous culturally and biologically, and has focused mainly on examining predictors of allostatic load (AL) rather than examining AL as a predictor of health outcome. This dissertation 1) provides evidence of biological and ethnic subgroup variation among NMS, 2) examines patterns of AL among NMS, and 3) demonstrates a direct relationship between AL and chronic disease.

Chapter 2 explored self-reported ethnicity in NMS and measured how social and biological variables contributing to health disparities varied within and among ethnic subgroups. Findings show that education, household income, skin color, and continental ancestry differ between NMS and other U.S. census racial groups. The variation observed in self-reported ethnicity in NMS results from the unique history of New Mexico (Healy et al., 2018; Hunley et al., 2017). While no significant differences in household income and education between the NMS subgroups were found, skin color and European ancestry vary by subgroup. These findings demonstrate that people of Spanish-speaking descent are not socially or biologically homogenous within a single U.S. state, and demonstrate that conventional race and ethnic categories are likely too broad to capture important heterogeneity in social and biological determinants of health in the U.S.

Chapter 3 examined patterns of AL, investigated sociodemographic and biological correlates of AL in NMS, and compared individual AL biomarkers between NMS and

other U.S. study samples. In NMS, mean AL scores increased with age. Given the cumulative nature of AL, this pattern was expected. While education level is not predictive of AL, there is a significant association between certain levels of education and AL; controlling for age, individuals with only high school diplomas/GEDs had significantly higher mean AL scores than individuals with some postgraduate/professional college. This suggests that an education-AL relationship more complex than a simple dose-response effect. Comparisons of AL biomarkers between NMS and other U.S. groups showed that NMS had significantly higher biomarker measures. While the ultimate cause of this is unknown, it cannot go ignored that New Mexico ranks low in health care treatment, maternal and child health, and in the number of insured children and adults (AHRQ, 2009, 2016; Radley, McCarthy, & Hayes, 2018). Findings further demonstrate that higher proportions of Native American ancestry were significantly associated with higher AL scores. Given that disparities in health are associated with disparities in socioeconomic status, these findings reveal that continental ancestry may be correlated with complex, non-genetic sociocultural factors. This study provides the first profile of the sociodemographic and biological correlates of AL among NMS and presents trends in AL that may be useful for identifying demographic groups likely to experience greater cumulative biological risk.

Chapter 4 examined whether AL is associated with six specific chronic diseases (abdominal obesity, hypertension, diabetes, cardiovascular disease, cancer, and gallbladder disease) that contribute to leading causes of death and mortality in the U.S., particularly in Hispanic, Latino, or Spanish-origin (HLS) populations. When controlling for age, sex, and smoking status, increasing AL scores were significantly associated with

gallbladder disease and abdominal obesity. AL was not significantly associated with the other chronic diseases. These findings point toward a recommendation that New Mexicans of Spanish-descent should aim to have two or less biomarkers at high-risk levels, as the odds for chronic disease were greatly reduced for allostatic load scores less than 2.

Significance

This dissertation demonstrates that subtle variation in sociodemographic and biological factors that contribute to health disparities is measurable. The differences in health risk between NMS and other U.S. racial groups provides further evidence that HLS populations are at greater risk for poor health outcomes and highlights the need for continued research examining health risk in other HLS populations around the U.S. Though subtle, NMS vary by continental ancestry and skin color both of which have been shown to have significant associations with health risk and outcomes. This finding suggests a complex and nuanced relationship between ethnicity, continental ancestry and health in NMS.

Future Directions

Findings from this dissertation calls attention to three important priorities for future research. First, epidemiological research should use region-specific ethnic nomenclature and explore the consequences the ethnic terms researchers use may have on health and health outcomes, particularly in HLS populations. Previous research has shown NMS identify with some ethnic terms do not appear as stand-alone options on the U.S. Census (e.g., Nuevomexicano and Spanish), and some of the ethnic terms they

identify with are combined with other terms to form one category (e.g., Mexican/Mexican American/Chicano) (Hunley et al., 2017). This provides an example of how the creation and use of ethnic terminology meant to clarify health disparities may actually be masking racial and ethnic disparities in health. An anthropological approach to public health research can be informative in community-based research and involving local communities to determine what ethnic descriptors are relevant to them. Second, in order to identify key risk factors contributing to health disparities in HLS populations, more comparative work is needed to evaluate determinants of AL patterns among HLS. A larger body of comparative research can help identify HLS subgroups that may be at an increased health risk compared to other groups and can inform intervention programs targeted specific HLS subgroups. Third, it is well established that racial and ethnic minorities have greater AL scores than their White counterparts. Although one of only a handful of studies to examine the direct relationship between AL and chronic disease, this dissertation demonstrates that future research needs to focus on AL as a predictor of health outcomes so that early interventions can be developed and implemented. The unique data set utilized in this dissertation gave resolution to subtle distinctions in ethnic terminology, health, continental ancestry, skin color, and biological and sociodemographic measures that have significant consequences on peoples' lives. Future researchers should consider more broad data collection if they strive to understand underlying causes of health disparities.

Appendix A. Supplementary Tables for Chapter 2

Supplementary Table A.1. Statistical output for Pearson's chi-square tests between U.S. racial groups and NMS, and educational attainment.

Key						
<i>frequency</i>						
<i>expected frequency</i>						
Racial	College g	High scho	Education Less than	Postgradu	Some coll	Total
Asian	3,189 2,064.6	2,308 3,450.9	1,296 1,518.4	1,926 1,099.8	2,309 2,894.2	11,028 11,028.0
Black	3,291 5,208.4	9,460 8,705.6	4,690 3,830.5	1,584 2,774.4	8,795 7,301.2	27,820 27,820.0
Hispanic	2,895 4,817.7	9,825 8,052.5	11,175 3,543.1	1,101 2,566.3	737 6,753.5	25,733 25,733.0
NMS	82 92.5	44 154.6	15 68.0	146 49.3	207 129.6	494 494.0
White	32,065 29,338.8	47,765 49,038.4	13,361 21,577.0	17,361 15,628.3	46,158 41,127.5	156,710 156,710.0
Total	41,522 41,522.0	69,402 69,402.0	30,537 30,537.0	22,118 22,118.0	58,206 58,206.0	221,785 221,785.0

Pearson chi2(16) = 3.2e+04 Pr = 0.000

Supplementary Table A.2. Statistical output for Pearson's chi-square tests between U.S. racial groups and NMS, and household income.

Key								
<i>frequency</i>								
<i>expected frequency</i>								
Racial Group	100,000+	25,000 -	35,000 -4	5,000 - 2	50,000 -	75,000 -	< 5,000	Total
Asian	1,658 1,131.5	444 586.4	604 748.9	809 1,154.7	934 946.3	686 616.5	237 187.8	5,372 5,372.0
Black	1,625 3,281.9	1,896 1,700.9	2,110 2,172.3	5,229 3,349.4	2,387 2,744.7	1,246 1,788.1	1,089 544.6	15,582 15,582.0
Hispanic	1,757 3,146.7	2,091 1,630.8	2,513 2,082.8	3,969 3,211.4	2,543 2,631.7	1,379 1,714.4	688 522.2	14,940 14,940.0
NMS	106 86.8	35 45.0	67 57.4	52 88.6	83 72.6	61 47.3	8 14.4	412 412.0
White	20,104 17,603.1	8,620 9,122.9	11,419 11,651.5	15,710 17,964.9	15,170 14,721.8	10,385 9,590.7	2,168 2,921.1	83,576 83,576.0
Total	25,250 25,250.0	13,086 13,086.0	16,713 16,713.0	25,769 25,769.0	21,117 21,117.0	13,757 13,757.0	4,190 4,190.0	119,882 119,882.0

Pearson chi2(24) = 5.2e+03 Pr = 0.000

Appendix B. NMS Study Questionnaire

Date and time: Month____ Day____ Year_____ Time_____

Location: _____

Interviewer: _____

(Data to be entered into study Access Database at time of interview)

Section I: Sociocultural Information

(Show NM map with counties to get county for questions #1-7. If answer is Bernalillo County, specify area using Albuquerque map.)

1. Where were you born?
 - a. city: _____
 - b. county: _____
 - c. state: _____
 - d. country: _____

2. Where did you grow up?
 - a. city: _____
 - b. county: _____
 - c. state: _____
 - d. country: _____

3. Where do you live now?
 - a. city: _____
 - b. county: _____
 - c. state: _____
 - d. country: _____
 - e. zip code: _____

4. Where was your **mother** born?
 - a. city: _____
 - b. county: _____
 - c. state: _____
 - d. country: _____

5. Where was your **father** born?
 - a. city: _____

- b. county: _____
- c. state: _____
- d. country: _____

6. Where were your **mother's** parents born?

- a. Grandmother:
 - 1. city: _____
 - 2. county: _____
 - 3. state: _____
 - 4. country: _____
- b. Grandfather:
 - 1. city: _____
 - 2. county: _____
 - 3. state: _____
 - 4. country: _____

7. Where were your **father's** parents born?

- a. Grandmother:
 - 1. city: _____
 - 2. county: _____
 - 3. state: _____
 - 4. country: _____
- b. Grandfather:
 - 1. city: _____
 - 2. county: _____
 - 3. state: _____
 - 4. country: _____

8. How old are you? _____

9. What is your sex? _____

10. Does your **father** belong to an old New Mexico family (land grants)?

11. Does your **mother** belong to an old New Mexico family (land grants)?
12. Were any of your ancestors colonists from Spain? If so, who and when (e.g., number generations)?
13. Do you have any ancestors from Mexico? If so, who and when (e.g., number generations)?
14. Do you have ancestors from anywhere else? If so, where else, who and when (e.g., number generations)?

(Interviewer: describe observations of how NMS divide themselves into at least two distinct groups to contextualize the next set of questions.)

15. What are the groups (*within NMS*)?
16. Which group do you belong to?
17. Which group do you think your **mother** belongs to?
18. Which group do you think your **father** belongs to?
19. Are you married? ☐ yes ☐ no

(If yes) Which group do you think your **spouse** belongs to?

20. Which of the other groups you identified is most similar to yours?

21. Which of the other groups you identified is most different from yours?

22. Please describe how the members of _____ (*insert name of group identified as most different from subject's; answer to #21*) tend to differ from members of _____ (*insert name of subject's self-identified group; answer to #16*) in these features:

a. Skin color

1. ☐ same
2. ☐ different. How? _____
3. ☐ don't know
4. ☐ refuse

b. Hair

1. ☐ same
2. ☐ different. How? _____
3. ☐ don't know
4. ☐ refuse

c. Face

1. ☐ same
2. ☐ different. How? _____
3. ☐ don't know
4. ☐ refuse

d. Other physical difference

1. What feature? _____ Different how? _____
2. What feature? _____ Different how? _____
3. ☐ don't know
4. ☐ refuse

e. Amount of Spanish use

1. ☐ same
2. ☐ different. How? _____
3. ☐ don't know
4. ☐ refuse

f. Accent when speaking English

1. ☐ same

2. ☐ different. How? _____
3. ☐ don't know
4. ☐ refuse

g. Accent when speaking Spanish

1. ☐ same
2. ☐ different. How? _____
3. ☐ don't know
4. ☐ refuse

h. Other language difference

1. What feature? _____ Different how? _____
2. What feature? _____ Different how? _____
3. ☐ don't know
4. ☐ refuse

i. Food (what people eat)

1. ☐ same
2. ☐ different. How? _____
3. ☐ don't know
4. ☐ refuse

j. Clothing (what people wear)

1. ☐ same
2. ☐ different. How? _____
3. ☐ don't know
4. ☐ refuse

k. Make-up

1. ☐ same
2. ☐ different. How? _____
3. ☐ don't know
4. ☐ refuse

l. Other cultural difference

1. What feature? _____ Different how? _____
2. What feature? _____ Different how? _____
3. ☐ don't know

4. ☐ refuse

23. Between the members of the group you belong to and those of the group most different from yours, does either one:

a. Experience more discrimination?

1. ☐ yes, my group
2. ☐ yes, other group
3. ☐ neither
4. ☐ don't know
5. ☐ refuse

b. Have more wealth?

1. ☐ yes, my group
2. ☐ yes, other group
3. ☐ neither
4. ☐ don't know
5. ☐ refuse

c. Have more political influence?

1. ☐ yes, my group
2. ☐ yes, other group
3. ☐ neither
4. ☐ don't know
5. ☐ refuse

d. Have more education?

1. ☐ yes, my group
2. ☐ yes, other group
3. ☐ neither
4. ☐ don't know
5. ☐ refuse

("The last set of questions has asked you to define what groups exist in NMS and tell us about the differences between them. Because we know that participants will give us different groups, we now will ask you the same questions using a set of group names that we've gathered from other participants so that we can compare everyone's

opinions about these terms. The groups are listed in alphabetical order.” For questions 23-26, ask for first choice and mark as #1. Then ask if there is a second-best choice and mark as #2. Then ask if any of the other terms also describe the person of interest, mark as #3, etc.)

24. With which of these groups do you identify?

- a. ☐ Chicano/a
- b. ☐ Hispanic
- c. ☐ Latino/a
- d. ☐ Mexican
- e. ☐ Mexican American
- f. ☐ Nuevomexicano/a
- g. ☐ Spanish
- h. ☐ Other _____

25. Which of these groups do you think other New Mexicans of Spanish-speaking descent would think that you belong to?

- a. ☐ Chicano/a
- b. ☐ Hispanic
- c. ☐ Latino/a
- d. ☐ Mexican
- e. ☐ Mexican American
- f. ☐ Nuevomexicano/a
- g. ☐ Spanish
- h. ☐ Other _____

26. Which of these groups do you think your **mother** would identify with?

- a. ☐ Chicano/a
- b. ☐ Hispanic
- c. ☐ Latino/a
- d. ☐ Mexican
- e. ☐ Mexican American
- f. ☐ Nuevomexicano/a
- g. ☐ Spanish
- h. ☐ Other _____

27. Which of these groups do you think your **father** would identify with?

- a. ☐ Chicano/a

- b. ☐ Hispanic
- c. ☐ Latino/a
- d. ☐ Mexican
- e. ☐ Mexican American
- f. ☐ Nuevomexicano/a
- g. ☐ Spanish
- h. ☐ Other _____

28. (if yes to #19) Which of these groups do you think your **spouse** would identify with?

- a. ☐ Chicano/a
- b. ☐ Hispanic
- c. ☐ Latino/a
- d. ☐ Mexican
- e. ☐ Mexican American
- f. ☐ Nuevomexicano/a
- g. ☐ Spanish
- h. ☐ Other _____

29. Which of the other groups on the list is most similar to your first choice?

- a. ☐ Chicano/a
- b. ☐ Hispanic
- c. ☐ Latino/a
- d. ☐ Mexican
- e. ☐ Mexican American
- f. ☐ Nuevomexicano/a
- g. ☐ Spanish
- h. ☐ Other _____

30. Which of the other groups you identified is most different from your first choice?

- a. ☐ Chicano/a
- b. ☐ Hispanic
- c. ☐ Latino/a
- d. ☐ Mexican
- e. ☐ Mexican American
- f. ☐ Nuevomexicano/a
- g. ☐ Spanish

h. ☐ Other _____

31. Please describe how the members of _____ (*insert name of group from the list named as most different from subject's; answer to #30*) tend to differ from _____ (*insert name of group from the list that subject most identified with; answer to #24*) in these features:

a. Skin color

1. ☐ same
2. ☐ different. How? _____
3. ☐ don't know
4. ☐ refuse

b. Hair

1. ☐ same
2. ☐ different. How? _____
3. ☐ don't know
4. ☐ refuse

c. Face

1. ☐ same
2. ☐ different. How? _____
3. ☐ don't know
4. ☐ refuse

d. Other physical difference

1. What feature? _____ Different how? _____
2. What feature? _____ Different how? _____
3. ☐ don't know
4. ☐ refuse

e. Amount of Spanish use

1. ☐ same
2. ☐ different. How? _____
3. ☐ don't know
4. ☐ refuse

f. Accent when speaking English

1. ☐ same

2. ☐ different. How? _____
3. ☐ don't know
4. ☐ refuse

g. Accent when speaking Spanish

1. ☐ same
2. ☐ different. How? _____
3. ☐ don't know
4. ☐ refuse

h. Other language difference

1. What feature? _____ Different how? _____
2. What feature? _____ Different how? _____
3. ☐ don't know
4. ☐ refuse

i. Food

1. ☐ same
2. ☐ different. How? _____
3. ☐ don't know
4. ☐ refuse

j. Clothing

1. ☐ same
2. ☐ different. How? _____
3. ☐ don't know
4. ☐ refuse

k. Make-up

1. ☐ same
2. ☐ different. How? _____
3. ☐ don't know
4. ☐ refuse

l. Other cultural difference

1. What feature? _____ Different how? _____
2. What feature? _____ Different how? _____
3. ☐ don't know

4. ☐ refuse

32. Between the members of the group you chose from the list and those of the most different group from yours on the list, does either one:

a. Experience more discrimination?

1. ☐ yes, my group
2. ☐ yes, other group
3. ☐ neither
4. ☐ don't know
5. ☐ refuse

b. Have more wealth?

1. ☐ yes, my group
2. ☐ yes, other group
3. ☐ neither
4. ☐ don't know
5. ☐ refuse

c. Have more political influence?

1. ☐ yes, my group
2. ☐ yes, other group
3. ☐ neither
4. ☐ don't know
5. ☐ refuse

d. Have more education?

1. ☐ yes, my group
2. ☐ yes, other group
3. ☐ neither
4. ☐ don't know
5. ☐ refuse

33. What religion did your parents raise you?

34. What is your current religion?

35. Have you

- a. *(if female)* Had a quinceñera?
 - 1. ☐ Yes
 - 2. ☐ No
 - 3. ☐ Don't know
 - 4. ☐ No response
- b. Participated in a pilgrimage to Chimayó?
 - 1. ☐ Yes
 - 2. ☐ No
 - 3. ☐ Don't know
 - 4. ☐ No response
- c. Attended or taken part in the dance of the Matachines?
 - 1. ☐ Yes
 - 2. ☐ No
 - 3. ☐ Don't know
 - 4. ☐ No response
- d. Participated in Las Posadas festivities?
 - 1. ☐ Yes
 - 2. ☐ No
 - 3. ☐ Don't know
 - 4. ☐ No response

36. Do you have any Jewish ancestors?

- a. ☐ Yes
- b. ☐ No
- c. ☐ Don't know
- d. ☐ No response

37. On a scale of 0% to 100%, with 0% being no European ancestry and 100% being pure European, what percentage of European ancestry do you think you have?

38. Of your European ancestry, what percentage of Spanish ancestry do you think you have, with 0% being no Spanish ancestry and 100% being pure Spanish ancestry?

39. On a scale of 0% to 100%, with 0% being no Native American/indigenous ancestry and 100% being pure Native American/indigenous, what percentage of Native American/indigenous ancestry do you think you have?

40. *(if 37+39 doesn't add to 100%)* What do you think makes up the rest of your ancestry?

41. We've asked about your ancestors, and now we'd also like to ask about your appearance. For these features, please tell us how you appear on a scale from completely European to completely Native Americans/indigenous:

a. Skin color

European											Native American
----------	--	--	--	--	--	--	--	--	--	--	-----------------

b. Hair

European											Native American
----------	--	--	--	--	--	--	--	--	--	--	-----------------

c. Face

European											Native American
----------	--	--	--	--	--	--	--	--	--	--	-----------------

d. Other physical feature: _____

European											Native American
----------	--	--	--	--	--	--	--	--	--	--	-----------------

42. The 2000 U.S. Census used the following categories for race. Which of these apply to you?

- a. ☐ American Indian or Alaska Native
- b. ☐ Asian
- c. ☐ White
- d. ☐ Black or African American
- e. ☐ Native Hawaiian or other Pacific Islander
- f. ☐ Some other race _____

43. Which of these racial categories do you think most New Mexicans would use to describe you?

- a. ☐ American Indian or Alaska Native
- b. ☐ Asian

- c. ☐ White
- d. ☐ Black or African American
- e. ☐ Native Hawaiian or other Pacific Islander
- f. ☐ Some other race _____

44. When you were growing up, were you expected to finish high school?

45. When you were growing up, were you expected to go to college?

46. What is **your** highest completed level of education?

- a. ☐ Some high school or less
- b. ☐ High school graduate/GED
- c. ☐ Some college or technical/vocational school/Associates degree
- d. ☐ College graduate (4-year college degree)
- e. ☐ Some postgraduate courses/Advanced or Professional degree
- f. ☐ unknown

47. Are you still in school? _____

48. What is your **mother's** highest completed level of education?

- a. ☐ Some high school or less
- b. ☐ High school graduate/GED
- c. ☐ Some college or technical/vocational school/Associates degree
- d. ☐ College graduate (4-year college degree)
- e. ☐ Some postgraduate courses/Advanced or Professional degree
- f. ☐ unknown

49. What is your **father's** highest completed level of education?

- a. ☐ Some high school or less
- b. ☐ High school graduate/GED
- c. ☐ Some college or technical/vocational school/Associates degree
- d. ☐ College graduate (4-year college degree)
- e. ☐ Some postgraduate courses/Advanced or Professional degree
- f. ☐ unknown

50. What are your **mother's parents'** highest completed levels of education?

a. Grandmother:

1. ☐ Some high school or less
2. ☐ High school graduate/GED
3. ☐ Some college or technical/vocational school/Associates degree
4. ☐ College graduate (4-year college degree)
5. ☐ Some postgraduate courses/Advanced or Professional degree
6. ☐ unknown

b. Grandfather:

1. ☐ Some high school or less
2. ☐ High school graduate/GED
3. ☐ Some college or technical/vocational school/Associates degree
4. ☐ College graduate (4-year college degree)
5. ☐ Some postgraduate courses/Advanced or Professional degree
6. ☐ unknown

51. What are your **father's parents'** highest completed levels of education?

a. Grandmother:

1. ☐ Some high school or less
2. ☐ High school graduate/GED
3. ☐ Some college or technical/vocational school/Associates degree
4. ☐ College graduate (4-year college degree)
5. ☐ Some postgraduate courses/Advanced or Professional degree
6. ☐ unknown

b. Grandfather:

1. ☐ Some high school or less
2. ☐ High school graduate/GED
3. ☐ Some college or technical/vocational school/Associates degree
4. ☐ College graduate (4-year college degree)
5. ☐ Some postgraduate courses/Advanced or Professional degree
6. ☐ unknown

52. What is **your** current occupation?

(If student) What occupation do you expect to have after finishing school?

53. What is/was your **mother's** occupation?

54. What is/was your **father's** occupation?

55. What were/are your **mother's parents'** occupations?

a. Grandmother _____

b. Grandfather _____

56. What were/are your **father's parents'** occupations?

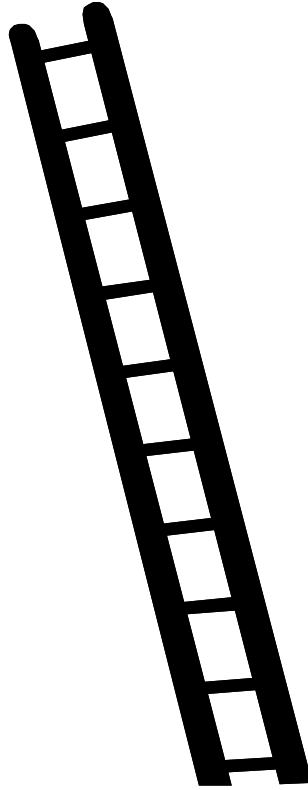
a. Grandmother _____

b. Grandfather _____

57. *Think of this ladder as representing where people stand in New Mexico. At the top of the ladder are the people who are the best off- those who have the most money, the most education and the most-respected jobs. At the bottom are the people who are the worst off- who have the least money, least education and the least-respected jobs or no job. The higher up you are on this ladder, the closer you are to the people at the very top; the lower you are, the closer you are to the people at the very bottom.*

Where would you place yourself on this ladder?

"Please place a large "X" on the rung where you think you stand at this time in your life, relative to other people in New Mexico."



Rung # from bottom: _____

58. Choose all of the following that describe your current daily activities and/or responsibilities:

- a. ☐ Working full time
- b. ☐ Working part-time
- c. ☐ Full-time student
- d. ☐ Unemployed or laid off
- e. ☐ Looking for work
- f. ☐ Keeping house or raising children full-time
- g. ☐ Retired

59. How much did you earn, before taxes and deductions, during the past 12 months?

- a. ☐ Less than \$5,000
- b. ☐ \$5,000 through \$11,999
- c. ☐ \$12,000 through \$15,999
- d. ☐ \$16,000 through \$24,999
- e. ☐ \$25,000 through \$34,999

- f. ☐ \$35,000 through \$49,999
- g. ☐ \$50,000 through \$74,999
- h. ☐ \$75,000 through \$99,999
- i. ☐ \$100,000 and greater
- j. ☐ Don't know
- k. ☐ No response

60. Does one or both of your parents still claim you as a dependent on their taxes?

- a. ☐ Yes
- b. ☐ No
- c. ☐ Don't know
- d. ☐ No response

For questions 61-68, subject should respond for family household that claims subject if response was "Yes" to question 60. If response was "No", subject should respond for current household.

61. How many people are currently living in your household, including yourself? *Your parents' number of family members in 2009-2010. Include in your parents' household: (1) your parents and yourself, even if you don't live with your parents, (2) your parents' other children if your parents will provide more than half of their support between July 1, 2009, and June 30, 2010, or and (3) other people only if they live with your parents, your parents provide more than half of their support and your parents will continue to provide more than half of their support between July 1, 2009, and June 30, 2010.*

- a. _____ Number of people
- b. _____ Of these people, how many are children?
- c. _____ Of these people, how many are adults?
- d. _____ Of the adults, how many bring income into the household?

62. Which best describes the building in which you/your family lives? (Include all apartments, flats, etc., even if vacant.)

- a. ☐ A mobile home
- b. ☐ A house detached from any other house
- c. ☐ A house attached to one or more houses
- d. ☐ A building with 2 apartments
- e. ☐ A building with 3 or 4 apartments
- f. ☐ A building with 5 or more apartments

- g. ☐ Boat, RV, van, etc.

63. Is your/your family's residence:

- a. ☐ Owned or being bought by you (or someone in the household)?
 b. ☐ Rented for money?
 c. ☐ Other (specify)_____

64. Do you or your family own land?

- a. ☐ Yes
 b. ☐ No
 c. ☐ Don't know
 d. ☐ No response

65. (*If household size > 1*) Which of these categories best describes your total combined family income for the past 12 months? This should include income (before taxes) from all sources, wages, rent from properties, social security, disability and/or veteran's benefits, unemployment benefits, workman's compensation, help from relatives (including child payments and alimony), etc.

- a. ☐ Less than \$5,000
 b. ☐ \$5,000 through \$11,999
 c. ☐ \$12,000 through \$15,999
 d. ☐ \$16,000 through \$24,999
 e. ☐ \$25,000 through \$34,999
 f. ☐ \$35,000 through \$49,999
 g. ☐ \$50,000 through \$74,999
 h. ☐ \$75,000 through \$99,999
 i. ☐ \$100,000 and greater
 j. ☐ Don't know
 k. ☐ No response

66. Beyond what your employer provides, do you have any financial investments?

- a. ☐ Yes
 b. ☐ No
 c. ☐ Don't know
 d. ☐ No response

67. Do you have at least one car?

- a. ☐ Yes (Make:_____ Model: _____ Year: _____ *If subject has more than one car, ask to describe primary car he/she drives*)
 b. ☐ No

- c. ☐ Don't know
- d. ☐ No response

68. Do you own a computer?

- a. ☐ Yes (#PC desktops:____#Mac desktops:____#PC laptops:____ #Mac laptops:____)
- b. ☐ No
- c. ☐ Don't know
- d. ☐ No response

69. Choose one:

- a. ☐ I speak Spanish better than I do English
- b. ☐ I speak Spanish and English equally well
- c. ☐ I speak English better than I do Spanish
- d. ☐ I do not speak Spanish

70. Choose one:

- a. ☐ My mother does not speak English
- b. ☐ My mother speaks Spanish better than English
- c. ☐ My mother speaks Spanish and English equally well
- d. ☐ My mother speaks English better than Spanish
- e. ☐ My mother does not speak Spanish

71. Choose one:

- a. ☐ My father does not speak English
- b. ☐ My father speaks Spanish better than English
- c. ☐ My father speaks Spanish and English equally well
- d. ☐ My father speaks English better than Spanish
- e. ☐ My father does not speak Spanish

72. What language did you speak in your household growing up?

73. How many full siblings do you have?

74. At what store/s do you do the most of your grocery shopping?

75. How often do you listen to English-speaking radio stations?

- a. ☐ Never
- b. ☐ Occasionally
- c. ☐ Sometimes
- d. ☐ Often

76. How often do you listen to Spanish-speaking radio stations?

- a. ☐ Never
- b. ☐ Occasionally
- c. ☐ Sometimes
- d. ☐ Often

77. What radio station/s do you listen to most?

78. How often do you watch television and movies in English?

- a. ☐ Never
- b. ☐ Occasionally
- c. ☐ Sometimes
- d. ☐ Often

79. How often do you watch television and movies in Spanish?

- a. ☐ Never
- b. ☐ Occasionally
- c. ☐ Sometimes
- d. ☐ Often

80. What television station/s do you watch most?

81. What sport do you and/or your family members enjoy watching most?

82. Which are you more likely to eat with a meal made at home?

- a. ☐ Rice
- b. ☐ Potatoes
- c. ☐ Both equally

d. ☐ Neither

83. Which are you more likely to eat with a meal made at home?

- a. ☐ Corn tortillas
- b. ☐ Flour tortillas
- c. ☐ Both equally
- d. ☐ Neither

Section II. Medical History

For questions #84-90, I am going to ask about your family history of several diseases. Please tell me whether you, your parents, or close relatives have/had any of these:

84. Cancer

- a. You: ☐ Yes* ☐ No ☐ Don't know *What type? _____
- b. Mother: ☐ Yes* ☐ No ☐ Don't know *What type? _____
- c. Father: ☐ Yes* ☐ No ☐ Don't know *What type? _____
- d. Other relatives:
Who: _____ What type? _____

85. Diabetes

- a. You: ☐ Yes* ☐ No ☐ Don't know *What type? _____
- b. Mother: ☐ Yes* ☐ No ☐ Don't know *What type? _____
- c. Father: ☐ Yes* ☐ No ☐ Don't know *What type? _____
- d. Other relatives:
Who: _____ What type? _____

86. Hypertension (high blood pressure)

- a. You: ☐ Yes* ☐ No ☐ Don't know *What type? _____
- b. Mother: ☐ Yes* ☐ No ☐ Don't know *What type? _____
- c. Father: ☐ Yes* ☐ No ☐ Don't know *What type? _____
- d. Other relatives:
Who: _____ What type? _____

87. Heart Attack

- a. You: ☐ Yes* ☐ No ☐ Don't know *What type? _____
- b. Mother: ☐ Yes* ☐ No ☐ Don't know *What type? _____
- c. Father: ☐ Yes* ☐ No ☐ Don't know *What type? _____
- d. Other relatives:
Who: _____ What type? _____

88. Gall Bladder disease

- a. You: ☐Yes* ☐No ☐Don't know *What type? _____
- b. Mother: ☐Yes* ☐No ☐Don't know *What type? _____
- c. Father: ☐Yes* ☐No ☐Don't know *What type? _____
- d. Other relatives:
Who: _____ What type? _____

89. Oculopharyngeal Muscular dystrophy (OPMD)

- a. You: ☐Yes* ☐No ☐Don't know *What type? _____
- b. Mother: ☐Yes* ☐No ☐Don't know *What type? _____
- c. Father: ☐Yes* ☐No ☐Don't know *What type? _____
- d. Other relatives:
Who: _____ What type? _____

90. Cavernous angioma/cerebral cavernous malformation/CCM

- a. You: ☐Yes* ☐No ☐Don't know *What type? _____
- b. Mother: ☐Yes* ☐No ☐Don't know *What type? _____
- c. Father: ☐Yes* ☐No ☐Don't know *What type? _____
- d. Other relatives:
Who: _____ What type? _____

91. Do you smoke cigarettes? ☐Yes ☐No92. Do you use any other form of tobacco (pipe/cigars/chewing)? ☐Yes ☐No93. (If yes to either 91 or 92) What is your **best estimate** of the number of days you smoked part or all of a cigarette or used another tobacco product during the past 30 days?

- a. ☐ 1 or 2 days
- b. ☐ 3 to 5 days
- c. ☐ 6 to 9 days
- d. ☐ 10 to 19 days
- e. ☐ 20 to 29 days
- f. ☐ All 30 days

94. (If yes to 91) On the days you smoked cigarettes during the past 30 days, how many cigarettes did you smoke per day, on average?

- a. ☐ Less than one cigarette per day/ 1 cigarette per day
- b. ☐ Less than half a pack a day (2 to 5 cigarettes per day)
- c. ☐ 6 to 15 cigarettes per day (about ½ pack)
- d. ☐ 16 to 25 cigarettes per day (about 1 pack)

- e. ☐ More than a pack a day

95. (If yes to 92) On the days that you used other forms of tobacco, how much?

96. (If yes to 91 or 92) How old were you when you first started using tobacco?
AGE: _____

97. Did you used to smoke cigarettes? ☐ Yes ☐ No

98. Did you used to use any other form of tobacco? ☐ Yes ☐ No

99. (If yes to 97 or 98) How many days per month did you smoke cigarettes or use tobacco?

- a. ☐ 1 or 2 days
- b. ☐ 3 to 5 days
- c. ☐ 6 to 9 days
- d. ☐ 10 to 19 days
- e. ☐ 20 to 29 days
- f. ☐ All 30 days

100. (If yes to 97) On the days that you smoked cigarettes, how many did you smoke per day, on average?

- a. Less than one cigarette per day/ 1 cigarette per day
- b. Less than half a pack a day (2 to 5 cigarettes per day)
- c. 6 to 15 cigarettes per day (about ½ pack)
- d. 16 to 25 cigarettes per day (about 1 pack)
- e. More than a pack a day

101. (If yes to 98) On the days that you used other forms of tobacco, how much?

102. (If yes to 97 or 98) For how many years did you smoke or use tobacco? ____

103. Do you drink alcohol? _____

104. (If yes to 103) On average, on how many days per week do you drink alcohol?

105. (If yes to 103) On days that you do drink, about how many drinks do you have, on average? ____

106. How often do you see a doctor?

107. For what reason/s would your parents have taken you to the doctor growing up?

- a. ☐ Regular checkup/exam
- b. ☐ Feeling sick
- c. ☐ Emergency (injury or severe illness)
- d. ☐ Treatment for condition doctor discovered earlier
- e. ☐ Other

108. Do you have health insurance? _____

(If student or under 25) Are you on your parents' health insurance plan? _____

109. Which of the following best describes **your** current health status?

- a. ☐ excellent
- b. ☐ good
- c. ☐ fair
- d. ☐ poor

110. Which of the following best describes your **mother's** current health status?

- a. ☐ excellent
- b. ☐ good
- c. ☐ fair
- d. ☐ poor
- e. ☐ N/A (not living or unknown)

111. Which of the following best describes your **father's** current health status?

- a. ☐ excellent
- b. ☐ good
- c. ☐ fair
- d. ☐ poor
- e. ☐ N/A (not living or unknown)

112. How often do you go to the dentist?

- a. ☐ At least once a year
- b. ☐ Every 2 years
- c. ☐ Less often than every 2 years
- d. ☐ Whenever needed - no regular schedule
- e. ☐ Other

113. What was the main reason for your last visit for dental care?

- a. ☐ Went in for checkup/exam/cleaning
- b. ☐ Something wrong/hurting/bothering
- c. ☐ Treatment for condition dentist discovered earlier
- d. ☐ Check/adjust appliance/orthodontia
- e. ☐ Other

114. What would you do if you had dental pain? _____

115. How would you describe the condition of your teeth and gums? Would you say:

- a. ☐ excellent
- b. ☐ good
- c. ☐ fair
- d. ☐ poor

These questions are about how you feel and how things have been with you. For each question, please give the one answer that comes closest to the way you have been feeling:

- 0. never
- 1. occasionally
- 2. sometimes
- 3. often

116. In the last month, how often have you felt that you were unable to control the important things in your life?

- ☐0 ☐1 ☐2 ☐3

117. In the last month, how often have you felt nervous and “stressed”?

- ☐0 ☐1 ☐2 ☐3

118. In the last month, how often have you felt that you were effectively coping with irritating life hassles?

- ☐0 ☐1 ☐2 ☐3

119. In the last month, how often have you been angered because of things that happened that were outside of your control?

- ☐0 ☐1 ☐2 ☐3

120. In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?

- ☐0 ☐1 ☐2 ☐3

121. In the last month, how often have you felt nervous and “stressed”?

- ☐0 ☐1 ☐2 ☐3

122. In the last month, how often have you felt that you were effectively coping with irritating life hassles?

☐0 ☐1 ☐2 ☐3

123. In the last month, how often have you been angered because of things that happened that were outside of your control?

☐0 ☐1 ☐2 ☐3

124. In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?

☐0 ☐1 ☐2 ☐3

The next set of questions is about how you are treated by other people. We're coming back to the groups of NMS that we talked about earlier, and asking about discrimination you may have experienced as a member of your group. The answers are the same as above; please give the one that comes closest to how often you receive the type of treatment described:

125. As a _____ (insert self-identified category from #16), how often are you treated with less courtesy than other people?

☐0 ☐1 ☐2 ☐3

126. As a _____ (insert self-identified category from #16), how often do you receive poorer service than other people in restaurants or stores?

☐0 ☐1 ☐2 ☐3

127. As a _____ (insert self-identified category from #16), how often do people treat you as if they are better than you?

☐0 ☐1 ☐2 ☐3

128. As a _____ (insert self-identified category from #16), how often do people act as if they are smarter than you?

☐0 ☐1 ☐2 ☐3

129. As a _____ (insert self-identified category from #16), how often do you think that discrimination makes it more difficult for you to accomplish your life goals?

☐0 ☐1 ☐2 ☐3

130. How often do you think that discrimination makes it more difficult for other _____ (insert self-identified category from #16) to accomplish their life goals?

☐0 ☐1 ☐2 ☐3

Section III. Photograph responses

I am now going to show you some photographs of other participants in this study and ask you two questions about the person in each photograph.

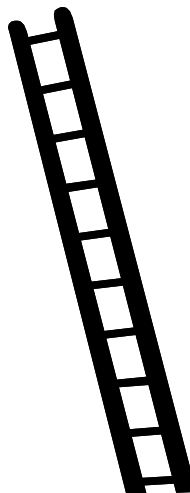
131. Of the ethnicity terms you listed at the outset (remind them), which term would you use to describe this person?

132. Which of these terms would you use?

- a. ☐ Chicano/a
- b. ☐ Hispanic
- c. ☐ Latino/a
- d. ☐ Mexican
- e. ☐ Mexican American
- f. ☐ Nuevomexicano/a
- g. ☐ Spanish
- h. ☐ Other _____

133. Earlier, I asked you where you fall on a ladder relative to other people in New Mexico. Where would you place this person on this ladder?

Please place an "X" on the rung where you think this person stands at this time in his/her life, relative to other people in New Mexico.



Rung # from bottom: _____

Appendix C. NMS Study Protocol for Dried Blood Spots (DBS)

Modified from SAGE Laboratory Training Manual for Dried Blood Spots, Version 2.0 (2010). Authors: Sharon R Williams & J. Josh Snodgrass

NMS 09-412 DBS Collection Protocol

1. Label Whatman #903 filter paper with participant's NMS participant ID number. (e.g., NMS ____)
2. Lay out materials needed for blood spot collection:
 - a. Labeled filter paper
 - b. Cotton ball
 - c. Alcohol swab
 - d. Lancet
 - e. Bandaid
 - f. Gloves
3. Have participant sit in chair at low table.
4. Put on gloves. Have participant gently shake their dominant hand.
5. Disinfect the puncture site (fingertip of ring finger) with the alcohol swab. Have participant lower arm and hand while swab dries. Do not make contact with finger while it is air-drying.
6. Twist off cap of lancet. Massage selected hand in downward motion to promote blood flow. Gently apply pressure to medial side of fingertip to pool blood.
8. Have participant stand to promote blood flow. Wipe away first blood drop with cotton ball.
9. Turn participant's palm down and place punctured finger over circles on filter paper. Allow each blood drop to form and fall into the middle of each circle. Continue to do this until all five blood spots are collected.
10. Once all blood spots are collected, apply pressure to puncture site with cotton ball to stop blood flow. If needed, apply a band aid to puncture site.
11. Discard used lancet in sharps container.
12. Allow used filter paper to air dry, without flap over the spots. Once dried, gently fold filter paper and place in Ziploc baggie with a desiccant packet. Place in freezer in Molecular Genetics Lab.

Dried Blood Spot Assays

General assay instructions

- All analytes except Hb should be analyzed using ELISA. Hb is a photometric assay.
- All samples should be run in duplicate.
- Samples should be reanalyzed if the coefficient of variation (CV) is greater than 10%.

- Plates should be reanalyzed if the controls are out of range, standards are not incomplete or the standard curve is problematic.
- Blotted, overlapping, doubled, or contaminated spots should not be analyzed.

Selection of spots

- The third spot (from the left) on each filter paper card has been treated with a modified citrate buffer solution (semicarbazide-aniline solution) for use in HbA1c analysis. This spot is only to be used for HbA1c analysis.

Punching of spots

- All spots for analysis should be punched with a small (3.2mm) hole punches.
- Ensure the front and back of each blood spot on the filter paper card is completely covered with dried blood. If any white filter paper is visible, discard the punched spot.
- Do not punch the spot directly into the tube because it will be hard to tell whether the disc is completely saturated with blood. Instead, punch the spots onto a clean surface (e.g., kimwipe, lab bench protectors, paper towel) and use tweezers or forceps to transfer the spot to the tube.
- Each 50 µl spot should yield 8 punches from the 3.2 mm punch.

Assay Protocols

Protocols for C-reactive protein (CRP), Epstein-Barr virus (EBV) antibodies, and hemoglobin (Hb) are provided below. Analysis of glycosylated hemoglobin (HbA1c) was conducted by Geonostics, Inc (Lincolnshire, Illinois). Preparation of dried blood spots for Geonostics is reported below.

High-sensitivity blood spot C-reactive Protein (CRP) Protocol

Method: Sandwich ELISA. Eluted dried blood spot samples are incubated with mouse anti-human CRP previously bound to the surface of the microtiter wells. Biotinylated mouse anti-human CRP and streptavidin-HRP conjugate binds to the CRP-antibody complexes in each well. Color forms with the addition of chromogenic substrate and the absorbance of the solution is read at 40nm. The quantity of CRP in each sample is determined based on comparison with a 4-parameter logistic (4PL) standard curve.

Reference: McDade, T.W., Burhop, J., and J. Dohnal (2004). High sensitivity enzyme immunoassay for C-reactive protein in dried blood spots. *Clinical Chemistry* 50: 652-654.

Coating plates with antibody

1. Dilute the coating antibody to 2 µg/mL
2. Add 100 µL of the diluted coating antibody to each well. Cover and rotate gently at 350rpm for 15 minutes.
3. Cover with plastic and incubate overnight at 4°C. *Plates can be stored for several months if wrapped securely in plastic, maintained at 4°C, and not allowed to dry out.*

Biotinylating the CRP detection antibody

1. Weight out 2.2mg of Biotin and dilute 400 μ L ultrapure or distilled H₂O. *Note: Bring Biotin to room temperature before opening, and recap quickly.*
2. Spin down the contents of the tube containing the antibody with a brief, high speed centrifugation to ensure that tube contents are collected at the bottom of the tube.
3. Add 13.5 μ L Biotin solution to 1mg of antibody.
4. Incubate at room temperature for 60 minutes.
5. Add PBS to the biotin-antibody solution to bring the total volume to 1mL.
6. Recover biotinylated antibody with the spin columns
7. Aliquot the purified biotinylated antibody in 50 μ L units and store at -80°C. Avoid repeated freeze/thaw of antibody.

CRP assay protocol

1. Punch out one 3.2mm disc of blood spot standards, samples, and controls. Elute overnight at 4°C in 250 μ L Assay Buffer (not more than 12 hours).
2. Next day: Remove samples from refrigerator. Rotate at 300rpm for 1 hour at RT.
3. Remove a coated microtiter plate and wash 4 times with Assay Buffer, leaving 350 μ L Assay Buffer in the wells.
4. Soak for 30 minutes to block the plate and then remove the buffer.
5. Add 100 μ L eluate from blood spot standards, controls, and samples. Cover the plate and rotate at 250 rpm at RT for 90 minutes.
6. Wash the wells 4 times with Assay Buffer.
7. Dilute the biotinylated detection antibody 1:20,000 to 5ng/mL:
 - a. Pre-dilute by adding 10 μ L antibody to 4mL Assay Buffer and mix (1:400 dilution).
 - b. Transfer 24 μ L of the 1:400 dilution to 12mL Assay Buffer and mix (1:500 dilution).
8. Add 100 μ L diluted detection antibody to each well.
9. Cover and rotate at 250 rpm at room temperature for 90 minutes.
10. Wash 4 times with Assay Buffer.
11. Dilute the streptavidin-HRP 1:7500.
 - a. Pre-dilute by adding 10 μ L strep-HRP to 5mL Assay Buffer and mix (1:500 dilution).
 - b. Transfer 750 μ L of the 1:500 dilution to 10.5mL Assay Buffer and mix (1:15 dilution).
12. Add 100 μ L diluted streptavidin-HRP to each well.
13. Cover and rotate at 250 rpm for 30 minutes.
 - a. During the incubation, prepare the chromogenic substrate
 - i. Place OPD tablet vial at RT for 10 minutes prior to opening.
 - ii. Open vial briefly and remove 4 tablets and transfer to a light impermeable container, avoiding skin contact with the tablets.
 - iii. Dissolve tablets in 12mL deionized H₂O at RT.
 - iv. Add 5 μ L of 30% H₂O₂ and mix. Protect from light and use within 1 hour.
14. Wash 4 times with Assay Buffer.
15. Add 100 μ L chromogenic substrate to each well.

16. Cover plate, protecting from the light, and incubate for 30 minutes at RT.
17. Add 100 μ L stop solution to each well and incubate 5 minutes at RT.
18. Read the absorbance at 490nm. Use a 4PL fitted curve to calculate unknown CRP concentrations.

Epstein-Barr Virus (EBV) Antibody Titer

Method: ELISA. This protocol is adapted from a commercially available EBV plasma assay kit (DiaSorrin #P001606A).

EBV assay protocol

1. Elute 1 disc for each sample in 250 μ L sample diluent overnight (12-18 hrs) at room temperature.
2. Prepare wash buffer: 25X wash buffer in one liter container. Fill with deionized water to one liter.
3. Dilute each control 1:1010. Add 5 μ L control and 0.05 mL sample diluent.
4. Pipette 100 μ L of each calibrator, diluted control, and eluted sample into identified wells. *Reverse pipette to avoid bubble formation. Do not touch the side of the elution tube and quantity should be sufficient. Leave blank wells empty.*
5. Incubate at 37°C for 60 minutes.
6. Dilute tracer: 0.3 mL tracer and 15 L diluent for full run. Mix in clean glass container.
7. Wash plate: 4X
8. Pipette 100 μ L diluted tracer. Leave blank wells empty.
9. Incubate at 37°C for 60 minutes.
10. Wash plate: 4X
11. Pipette 100 μ L chromogenic/substrate into all wells. Start 30 minute incubations after addition to first well.
12. Incubate at room temperature away from light for 30 minutes. *Wrap in aluminum foil to keep away samples away from light.*
13. Add 200 μ L stop solution to all wells. Wait 15 minutes.
14. Read plate at 450 nm
15. If a sample is beyond the range of the plate reader, use endpoint dilution: remove 150 μ L of solution, and add 150 μ L stop solution. Read again. Multiply result by 2.

Hemoglobin

Method: This protocol modifies the widely-used Drabkin's solution protocol for use with dried blood spots.

1. Elute two 3.2 mm blood discs for each sample in 450 μ L Drabkin's solution.
2. Vortex each sample briefly and let elute at room temperature on a plate shaker for 2 hours. *Cover with parafilm.*
3. Vortex each sample briefly before loading samples.
4. Load 200 μ L of each sample into each well. *Each sample should be run in duplicate.*
5. Read plate at 540 nm (520-500 nm) with an ELISA spectrophotometer.
6. Calculate sample concentrations from standard curve by plotting a linear curve of standard absorbance vs known absorbance.

Glycosolated Hemoglobin**Preparation of dried blood spots for Geonostics, Inc**

1. Cut out entire treated spot (third spot from left) from filter paper card. If treated spot isn't sufficient to yield a 3mm punch, cut out another dried blood spot. *Note: Geonostics has validated protocol for untreated spots.*
2. Label with participant's NMS participant ID (e.g., NMS ____)
3. Once all spots are cut out, put in shipping box with cooler packs and send off to Geonostics for analysis.

References

- ADA. (2017). Standards of medical care in diabetes—2017 abridged for primary care providers. *Clinical diabetes: a publication of the American Diabetes Association*, 35(1), 5.
- Adler, N. E., & Rehkopf, D. H. (2008). U.S. disparities in health: descriptions, causes, and mechanisms. *Annu Rev Public Health*, 29(29), 235-252. Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/18031225>. doi:10.1146/annurev.publhealth.29.020907.090852
- AHRQ. (2009). *2009 National Healthcare Quality and Disparities Report*: Department of Health and Human Services, Agency for Healthcare Research and Quality.
- AHRQ. (2016). *2016 National Healthcare Disparities Report*: Department of Health and Human Services, Agency for Healthcare Research and Quality.
- Aponte, J. (2009). Diabetes-related risk factors across Hispanic subgroups in the Hispanic health and nutritional examination survey (1982-1984). *Public Health Nursing*, 26(1), 23-38. Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/19154190>. doi:10.1111/j.1525-1446.2008.00741.x
- Araújo, B. Y., & Borrell, L. N. (2006). Understanding the link between discrimination, mental health outcomes, and life chances among Latinos. *Hispanic Journal of Behavioral Sciences*, 28(2), 245-266.
- Aslam, H. M., Saleem, S., Edhi, M. M., Shaikh, H. A., Hafiz, M., & Saleem, M. (2013). Assessment of gallstone predictor: comparative analysis of ultrasonographic and biochemical parameters. *International archives of medicine*, 6(1), 17.
- Atkeson, L. R., Bryant, L. A., Hall, T. E., Saunders, K., & Alvarez, M. (2010). A new barrier to participation: Heterogeneous application of voter identification policies. *Electoral Studies*, 29(1), 66-73.
- Baer, R. D., Arteaga, E., Dyer, K., Eden, A., Gross, R., Helmy, H., . . . Reeser, D. (2013). Concepts of race and ethnicity among health researchers: patterns and implications. *Ethnicity & health*, 18(2), 211-225.
- Beckie, T. M. (2012). A systematic review of allostatic load, health, and health disparities. *Biological Research for Nursing*, 14(4), 311-346. Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/23007870>. doi:10.1177/1099800412455688
- Ben-Shlomo, Y., & Kuh, D. (2002). A life course approach to chronic disease epidemiology: conceptual models, empirical challenges and interdisciplinary perspectives. *International Journal of Epidemiology*(31), 285-293.
- Berkowitz, S. A., Percac-Lima, S., Ashburner, J. M., Chang, Y., Zai, A. H., He, W., . . . Atlas, S. J. (2015). Building equity improvement into quality improvement: reducing socioeconomic disparities in colorectal cancer screening as part of population health management. *Journal of General Internal Medicine*, 30(7), 942-949.
- Bilheimer, L. T., & Sisk, J. E. (2008). Collecting adequate data on racial and ethnic disparities in health: The challenges continue. *Health Affairs*, 27(2), 383-391.

- Bonilla, C., Parra, E. J., Pfaff, C. L., Dios, S., Marshall, J. A., Hamman, R. F., . . . Shriver, M. (2004). Admixture in the Hispanics of the San Luis Valley, Colorado, and its implications for complex trait gene mapping. *Annals of human genetics*, 68(2), 139-153.
- Borrell, L. N., Dallo, F. J., & Nguyen, N. (2010). Racial/ethnic disparities in all-cause mortality in US adults: the effect of allostatic load. *Public Health Reports*, 125(6), 810-816.
- Borrell, L. N., & Lancet, E. A. (2012). Race/ethnicity and all-cause mortality in US adults: revisiting the Hispanic paradox. *American Journal of Public Health*, 102(5), 836-843.
- Bradby, H. (2003). Describing ethnicity in health research. *Ethnicity and Health*, 8(1), 5-13.
- Braveman, P., Cubbin, C., Egerter, S., Chideya, S., Marchi, K. S., Metzler, M., & Posner, S. (2005). Socioeconomic status in health research: one size does not fit all. *JAMA*, 294(22), 2879-2888.
- Braveman, P., Egerter, S., & Williams, D. R. (2011). The Social Determinants of Health: Coming of Age. *Annual Review of Public Health*, 32(1), 381-398. doi:10.1146/annurev-publhealth-031210-101218
- Braveman, P., Heck, K., Egerter, S., Marchi, K. S., Dominguez, T. P., Cubbin, C., . . . Curtis, M. (2015). The role of socioeconomic factors in black–white disparities in preterm birth. *American Journal of Public Health*, 105(4), 694-702.
- Brown, K. T. (1998). Consequences of skin tone bias for African Americans: Resource attainment and psychological/social functioning. *African American Research Perspectives*, 4(1), 55-60.
- Burchard, E. G., Ziv, E., Coyle, N., Gomez, S. L., Tang, H., Karter, A. J., . . . Risch, N. (2003). The importance of race and ethnic background in biomedical research and clinical practice. *New England Journal of Medicine*, 348(12), 1170-1175. Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/12646676>. doi:10.1056/NEJMs025007
- Bustamante, A. (1991). "The Matter Was Never Resolved": The "Casta" System in Colonial New Mexico, 1693-1823. *New Mexico Historical Review*, 66(2), 143.
- Campbell, D. D., Parra, M. V., Duque, C., Gallego, N., Franco, L., Tandon, A., . . . Bedoya, G. (2012). Amerind ancestry, socioeconomic status and the genetics of type 2 diabetes in a Colombian population. *PLOS ONE*, 7(4), e33570.
- Cann, H. M., De Toma, C., Cazes, L., Legrand, M.-F., Morel, V., Piouffre, L., . . . Cambon-Thomsen, A. (2002). A human genome diversity cell line panel. *Science*, 296(5566), 261-262.
- Carlson, E., & Chamberlain, R. (2005). Allostatic load and health disparities: a theoretical orientation. *Research in Nursing and Health*, 28(4), 306-315.
- Census Bureau, U. S. (2018). Annual estimates of the resident population by sex, age, race, and Hispanic origin for the United States and states: April 1, 2010 to July 1, 2017. In.
- Chyu, L., & Upchurch, D. M. (2011). Racial and ethnic patterns of allostatic load among adult women in the United States: findings from the National Health and Nutrition Examination Survey 1999–2004. *Journal of Women's Health*, 20(4), 575-583.

- Codina, G. E., & Montalvo, F. F. (1994). Chicano phenotype and depression. *Hispanic Journal of Behavioral Sciences*, 16(3), 296-306.
- Consortium, I. H. (2005). A haplotype map of the human genome. *Nature*, 437(7063), 1299.
- Crimmins, E. M., Kim, J. K., Alley, D. E., Karlamangla, A., & Seeman, T. (2007). Hispanic paradox in biological risk profiles. *American Journal of Public Health*, 97(7), 1305-1310.
- Crimmins, E. M., & Saito, Y. (2001). Trends in healthy life expectancy in the United States, 1970–1990: gender, racial, and educational differences. *Social Science and Medicine*, 52(11), 1629-1641.
- Cuevas, A. G., Dawson, B. A., & Williams, D. R. (2016). Race and skin color in Latino health: An analytic review. *American Journal of Public Health*, 106(12), 2131-2136.
- Daviglus, M. L., Talavera, G. A., Avilés-Santa, M. L., Allison, M., Cai, J., Criqui, M. H., . . . Kaplan, R. C. (2012). Prevalence of major cardiovascular risk factors and cardiovascular diseases among Hispanic/Latino individuals of diverse backgrounds in the United States. *JAMA*, 308(17), 1775-1784.
- Doan, G. O., & Stephan, C. W. (2006). The functions of ethnic identity: A New Mexico Hispanic example. *International Journal of Intercultural Relations*, 30(2), 229-241. doi:10.1016/j.ijintrel.2005.07.009
- Dorsey, R., Graham, G., Glied, S., Meyers, D., Clancy, C., & Koh, H. (2014). Implementing health reform: improved data collection and the monitoring of health disparities. *Annual Review of Public Health*, 35, 123-138.
- Dressler, W. W. (1991). Social support, lifestyle incongruity, and arterial blood pressure in a southern black community. *Psychosomatic Medicine*, 53(6), 608-620.
- Duany, J. (1998). Reconstructing racial identity: Ethnicity, color, and class among Dominicans in the United States and Puerto Rico. *Latin American Perspectives*, 25(3), 147-172.
- Duong, M. T., Bingham, B. A., Aldana, P. C., Chung, S. T., & Sumner, A. E. (2017). Variation in the calculation of allostatic load score: 21 examples from NHANES. *Journal of Racial and Ethnic Health Disparities*, 4(3), 455-461.
- Duru, O. K., Harawa, N. T., Kermah, D., & Norris, K. C. (2012). Allostatic load burden and racial disparities in mortality. *Journal of the National Medical Association*, 104(1-2), 89-95.
- Edes, A. N., & Crews, D. E. (2017). Allostatic load and biological anthropology. *American Journal of Physical Anthropology*, 162, 44-70.
- Ennis, S., Rios-Vargas, M., & Albert, N. (2011). The Hispanic Population: 2010. 2010 Census Briefs. 2011. In.
- ERFC, E. R. F. C. (2010). Diabetes mellitus, fasting blood glucose concentration, and risk of vascular disease: a collaborative meta-analysis of 102 prospective studies. *The Lancet*, 375(9733), 2215-2222.
- Espino, R., & Franz, M. M. (2002). Latino phenotypic discrimination revisited: The impact of skin color on occupational status. *Social Science Quarterly*, 83(2), 612-623.

- Fiscella, K., Franks, P., Gold, M. R., & Clancy, C. M. (2000). Inequality in quality: addressing socioeconomic, racial, and ethnic disparities in health care. *Journal of the American Medical Association*, 283(19), 2579-2584.
- Flegal, K. M., Carroll, M. D., Ogden, C. L., & Curtin, L. R. (2010). Prevalence and trends in obesity among US adults, 1999-2008. *JAMA*, 303(3), 235-241.
- Flegal, K. M., Carroll, M. D., Ogden, C. L., & Johnson, C. L. (2002). Prevalence and trends in obesity among US adults, 1999-2000. *Journal of the American Medical Association*, 288(14), 1723-1727.
- Florez, J., Price, A., Campbell, D., Riba, L., Parra, M., Yu, F., . . . Tello-Ruiz, M. (2011). Strong association of socioeconomic status and genetic ancestry in Latinos: Implications for admixture studies of type 2 diabetes. In *Racial Identities, Genetic Ancestry, and Health in South America* (pp. 137-153): Springer.
- Frederick, C. B., Snellman, K., & Putnam, R. D. (2014). Increasing socioeconomic disparities in adolescent obesity. *Proceedings of the National Academy of Sciences*, 111(4), 1338-1342.
- Gay, J. L., Salinas, J. J., Buchner, D. M., Mirza, S., Kohl, H. W., Fisher-Hoch, S. P., & McCormick, J. B. (2015). Meeting physical activity guidelines is associated with lower allostatic load and inflammation in Mexican Americans. *Journal of Immigrant Minority Health*, 17(2), 574-581.
- George, S., Duran, N., & Norris, K. (2014). A systematic review of barriers and facilitators to minority research participation among African Americans, Latinos, Asian Americans, and Pacific Islanders. *American Journal of Public Health*, 104(2), e16-e31.
- Geronimus, A. T., Hicken, M., Keene, D., & Bound, J. (2006). "Weathering" and age patterns of allostatic load scores among blacks and whites in the United States. *American Journal of Public Health*, 96(5), 826-833.
- Golash-Boza, T., & Darity Jr, W. (2008). Latino racial choices: the effects of skin colour and discrimination on Latinos' and Latinas' racial self-identifications. *Ethnic Racial Studies*, 31(5), 899-934.
- Gold, M., Dodd, A. H., & Neuman, M. (2008). Availability of data to measure disparities in leading health indicators at the state and local levels. *Journal of public health management and practice*, 14(6), S36-S44.
- Gómez, C. (2000). The continual significance of skin color: An exploratory study of Latinos in the Northeast. *Hispanic Journal of Behavioral Sciences*, 22(1), 94-103.
- Gonzales, P. B. (1993). The political construction of Latino nomenclatures in twentieth-century New Mexico. *Journal of the Southwest*, 158-185.
- Gravlee, C. C. (2005). Ethnic classification in southeastern Puerto Rico: The cultural model of "color". *Social Forces*, 83(3), 949-970.
- Gravlee, C. C., & Dressler, W. W. (2005). Skin pigmentation, self-perceived color, and arterial blood pressure in Puerto Rico. *American Journal of Human Biology*, 17(2), 195-206. Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/15736179>. doi:10.1002/ajhb.20111
- Gravlee, C. C., Non, A. L., & Mulligan, C. J. (2009). Genetic ancestry, social classification, and racial inequalities in blood pressure in Southeastern Puerto Rico. *PLOS ONE*, 4(9), e6821.

- Gravlee, C. C., & Sweet, E. (2008). Race, ethnicity, and racism in medical anthropology, 1977–2002. *Medical anthropology quarterly*, 22(1), 27-51.
- Gutierrez, R. A. (1991). When Jesus Came, the Corn Mothers Went Away. In. Stanford, CA: Stanford University Press.
- Hajat, A., Lucas, J., & Kington, R. (2000). Health outcomes among Hispanic subgroups: United States, 1992–95. *Advance Data*, 310.
- Harris, M. (1970). Referential ambiguity in the calculus of Brazilian racial identity. *Southwestern Journal of Anthropology*, 26(1), 1-14.
- Healy, M., Edgar, H., Mosley, C., & Hunley, K. (2018). Associations between ethnic identity, regional history, and genomic ancestry in New Mexicans of Spanish-speaking descent. *Biodemography and Social Biology*, 64(2), 152-170.
- Henley, S. J., Weir, H. K., Jim, M. A., Watson, M., & Richardson, L. C. (2015). Gallbladder cancer incidence and mortality, United States 1999–2011. *Cancer Epidemiology and Prevention Biomarkers*, 24(9), 1319-1326.
- Howard, J. T., & Sparks, P. J. (2015). The role of education in explaining racial/ethnic allostatic load differentials in the United States. *Biodemography and Social Biology*, 61(1), 18-39.
- Huckell, B. (2014). West of the Plains: Paleoindians in the Southwest. In N. J. Parezo & J. C. Janetski (Eds.), *Archaeology in the Great Basin and SOUTHWEST: Papers in Honor of Don D. Fowler* (pp. 17-34). Salt Lake City: University of Utah Press.
- Humes, K. R., Jones, N. A., & Ramirez, R. R. (2011). Overview of race and Hispanic origin: 2010.
- Hunley, K., Edgar, H., Healy, M., Mosley, C., Cabana, G. S., & West, F. (2017). Social Identity in New Mexicans of Spanish-Speaking Descent Highlights Limitations of Using Standardized Ethnic Terminology in Research. *Human Biology*, 89(3), 217-228.
- Ismail, N., & Jemain, A. A. (2007). *Handling overdispersion with negative binomial and generalized Poisson regression models*. Paper presented at the Casualty Actuarial Society Forum.
- Jaruvongvanich, V., Yang, J. D., Peeraphatdit, T., & Roberts, L. R. J. G. (2017). Racial and Socioeconomic Disparities in the Incidence Rates and Survival of Gallbladder Cancer in the United States. *152*(5), S1172-S1173.
- Jensen, M. D., Ryan, D. H., Donato, K. A., Apovian, C. M., Ard, J. D., Comuzzie, A. G., . . . Jakicic, J. M. (2014). Executive summary: guidelines (2013) for the management of overweight and obesity in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the Obesity Society published by the Obesity Society and American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Based on a systematic review from the The Obesity Expert Panel, 2013. *Obesity*, 22(S2), S5-S39.
- Juster, R.-P., McEwen, B. S., & Lupien, S. J. (2010). Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neurosci Biobehav Rev*, 35(1), 2-16. Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/19822172>. doi:10.1016/j.neubiorev.2009.10.002

- Kaestner, R., Pearson, J. A., Keene, D., & Geronimus, A. T. (2009). Stress, allostatic load, and health of Mexican immigrants. *Social Science Quarterly*, 90(5), 1089-1111.
- Kaplan, J. B., & Bennett, T. (2003). Use of race and ethnicity in biomedical publication. *JAMA*, 289(20), 2709-2716.
- Karlamangla, A. S., Singer, B. H., & Seeman, T. E. (2006). Reduction in allostatic load in older adults is associated with lower all-cause mortality risk: MacArthur studies of successful aging. *Psychosomatic Medicine*, 68(3), 500-507.
- Klimentidis, Y. C., Miller, G. F., & Shriver, M. D. (2009). Genetic admixture, self-reported ethnicity, self-estimated admixture, and skin pigmentation among Hispanics and Native Americans. *American Journal of Physical Anthropology*, 138(4), 375-383.
- Klonoff, E. A., & Landrine, H. (2000). Is skin color a marker for racial discrimination? Explaining the skin color-hypertension relationship. *Journal of Behavioral Medicine*, 23(4), 329-338.
- Kneipp, S. M., & Drevdahl, D. J. (2003). Problems with parsimony in research on socioeconomic determinants of health. *Advances in Nursing Science*, 26(3), 162-172.
- Kramer, C. Y. (1956). Extension of multiple range tests to group means with unequal numbers of replications. *Biometrics*, 12(3), 307-310.
- Krieger, N., Sidney, S., & Coakley, E. (1998). Racial discrimination and skin color in the CARDIA study: implications for public health research. Coronary Artery Risk Development in Young Adults. *American Journal of Public Health*, 88(9), 1308-1313.
- Lee, S., & Choi, S. (2009). Disparities in access to health care among non-citizens in the United States. *Health Sociology Review*, 18(3), 307-320.
- Lisabeth, L. D., Morgenstern, L. B., Burke, D. T., Sun, Y. V., & Long, J. C. (2011). Ancestral heterogeneity in a biethnic stroke population. *Annals of human genetics*, 75(4), 508-515.
- Lomelí, F. A., Sorell, V. A., & Padilla, G. M. (2002). *Nuevomexicano Cultural Legacy: Forms, Agencies, and Discourse*: UNM Press.
- López, I. (2008). " But you don't look Puerto Rican": The moderating effect of ethnic identity on the relation between skin color and self-esteem among Puerto Rican women. *Cultural Diversity and Ethnic Minority Psychology*, 14(2), 102.
- Markides, K. S., & Coreil, J. (1986). The health of Hispanics in the southwestern United States: an epidemiologic paradox. *Public Health Reports*, 101(3), 253.
- Masoro, E. (1997). Theories of aging: a pathophysiological perspective. *Aging (Milan, Italy)*, 9(6), 428-429.
- Mattei, J., Demissie, S., Falcon, L. M., Ordovas, J. M., & Tucker, K. L. (2010). Allostatic load is associated with chronic conditions in the Boston Puerto Rican Health Study. *Social Science and Medicine*, 70(12), 1988-1996. Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/20381934>. doi:10.1016/j.socscimed.2010.02.024
- Maurer, K. R., Everhart, J. E., Ezzati, T. M., Johannes, R. S., Knowler, W. C., Larson, D. L., . . . Roth, H. P. (1989). Prevalence of gallstone disease in Hispanic populations in the United States. *Gastroenterology*, 96(2), 487-492.

- McDade, T. W. (2008). Challenges and opportunities for integrative health research in the context of culture: A commentary on Gersten. *Social Science and Medicine*, 66(3), 520-524.
- McEwen, B. (1998). Stress, adaptation, and disease: Allostasis and allostatic load. *Annals of the New York Academy of Sciences*, 840(1), 33-44.
- McEwen, B., & Stellar, E. (1993). Stress and the individual: mechanisms leading to disease. *Archives of Internal Medicine*, 153(18), 2093-2101.
- McEwen, B. S., & Seeman, T. (1999). Protective and damaging effects of mediators of stress: elaborating and testing the concepts of allostasis and allostatic load. *Annals of the New York Academy of Sciences*, 896(1), 30-47.
- Miquel, J. F., Covarrubias, C., Villaroel, L., Mingrone, G., Greco, A. V., Puglielli, L., . . . Nervi, F. (1998). Genetic epidemiology of cholesterol cholelithiasis among Chilean Hispanics, Amerindians, and Maoris. *Gastroenterology*, 115(4), 937-946.
- Monforti, J. L., & Sanchez, G. R. (2010). The politics of perception: An investigation of the presence and sources of perceptions of internal discrimination among Latinos. *Social Science Quarterly*, 91(1), 245-265.
- Montalvo, F. F. (2005). Surviving race: Skin color and the socialization and acculturation of Latinas. *Journal of Ethnic and Cultural Diversity in Social Work*, 13(3), 25-43.
- Montalvo, F. F., & Codina, G. E. (2001). Skin color and Latinos in the United States. *Ethnicities*, 1(3), 321-341.
- Morales, L. S., Lara, M., Kington, R. S., Valdez, R. O., & Escarce, J. J. (2002). Socioeconomic, cultural, and behavioral factors affecting Hispanic health outcomes. *Journal of Health Care for the Poor and Underserved*, 13(4), 477.
- Mosley, C., Edgar, H., Hunley, K., & Healy, M. (in review). Allostatic load and biomarkers among New Mexicans of Spanish-speaking descent and as compared to other United States groups. . *American Journal of Human Biology*.
- NCHS. (2015, 11/6/2015). CDC Healthy People 2010. Retrieved from https://www.cdc.gov/nchs/healthy_people/hp2010.htm
- NCHS. (2016). *Health, United States, 2015: with special feature on racial and ethnic health disparities*.
- NCHS. (2017). *Health, United States, 2016: with chartbook on long-term trends in health*. Retrieved from
- NCHS. (2019). CDC Healthy People 2020. Retrieved from https://www.cdc.gov/nchs/healthy_people/hp2020.htm
- Nieto-Phillips, J. M. (1996). *"No Other Blood": History, Language, and Spanish American Ethnic Identity in New Mexico, 1880s-1920s*. University of California, Los Angeles,
- NMDH. (2019). Indicator-Based Information System for Public Health.
- Non, A. L., Gravlee, C. C., & Mulligan, C. J. (2012). Education, genetic ancestry, and blood pressure in African Americans and Whites. *American Journal of Public Health*, 102(8), 1559-1565.
- Nostrand, R. L. (1992). *The Hispano Homeland*: University of Oklahoma Press.
- OMB. (1997). *Revisions to the standards for the classification of federal data on race and ethnicity*. Retrieved from
- OMH, O. o. M. H. (2019, 2/11/2019). Hispanic/Latinop Population Profile.

- Pasta, D. J. (2009). *Learning when to be discrete: continuous vs. categorical predictors*. Paper presented at the SAS Global Forum.
- Pearson, T. A., Mensah, G. A., Alexander, R. W., Anderson, J. L., Cannon III, R. O., Criqui, M., . . . Myers, G. L. (2003). Markers of inflammation and cardiovascular disease: application to clinical and public health practice: a statement for healthcare professionals from the Centers for Disease Control and Prevention and the American Heart Association. *Circulation*, 107(3), 499-511.
- Peek, M. K., Cutchin, M. P., Salinas, J. J., Sheffield, K. M., Eschbach, K., Stowe, R. P., & Goodwin, J. S. (2010). Allostatic load among non-Hispanic Whites, non-Hispanic Blacks, and people of Mexican origin: effects of ethnicity, nativity, and acculturation. *American Journal of Public Health*, 100(5), 940-946.
- Peralta, C. A., Ziv, E., Katz, R., Reiner, A., Burchard, E. G., Fried, L., . . . Shlipak, M. (2006). African ancestry, socioeconomic status, and kidney function in elderly African Americans: a genetic admixture analysis. *Journal of the American Society of Nephrology*, 17(12), 3491-3496. Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/17082243>. doi:10.1681/ASN.2006050493
- Perloff, D., Grim, C., Flack, J., Frohlich, E. D., Hill, M., McDonald, M., & Morgenstern, B. Z. (1993). Human blood pressure determination by sphygmomanometry. *Circulation*, 88(5), 2460-2470.
- Perreira, K. M., & Telles, E. E. (2014). The color of health: Skin color, ethnoracial classification, and discrimination in the health of Latin Americans. *Social science & medicine*, 116, 241-250.
- Radley, D. C., McCarthy, D., & Hayes, S. L. (2018). *2018 Scorecard on State Health System*. Retrieved from
- Radley, D. C., McCarthy, D., Lippa, J., Hayes, S., & Schoen, C. (2014). *Aiming Higher: Results from a Scorecard on State Health System Performance, 2014*. Retrieved from
- Rainisch, B. K. W., & Upchurch, D. M. (2013). Sociodemographic correlates of allostatic load among a national sample of adolescents: findings from the National Health and Nutrition Examination Survey, 1999–2008. *Journal of Adolescent Health*, 53(4), 506-511.
- Richman, I. B., Taylor, K. E., Chung, S. A., Trupin, L., Petri, M., Yelin, E., . . . Gregersen, P. K. (2012). European genetic ancestry is associated with a decreased risk of lupus nephritis. *Arthritis and Rheumatism*, 64(10), 3374-3382.
- Risch, N., Burchard, E., Ziv, E., & Tang, H. (2002). Categorization of humans in biomedical research: genes, race and disease. *Genome Biology*, 3(7), comment2007.2001-2007.2012.
- Ruiz, J. M., Steffen, P., & Smith, T. B. (2013). Hispanic mortality paradox: a systematic review and meta-analysis of the longitudinal literature. *American Journal of Public Health*, 103(3), e52-e60.
- Sabbah, W., Watt, R., Sheiham, A., & Tsakos, G. (2008). Effects of allostatic load on the social gradient in ischaemic heart disease and periodontal disease: evidence from the Third National Health and Nutrition Examination Survey. *Journal of Epidemiology & Community Health*, 62(5), 415-420.

- Sahu, S., Jain, R., Prakash, A., Bahl, D., & Sachan, P. (2007). Correlation of gallstone disease with iron-deficiency anaemia: a prospective study. *Internet Journal of Surgery*, 14.
- Salazar, C. R., Strizich, G., Seeman, T. E., Isasi, C. R., Gallo, L. C., Avilés-Santa, L. M., . . . Sanders, A. E. (2016). Nativity differences in allostatic load by age, sex, and Hispanic background from the Hispanic Community Health Study/Study of Latinos. *Social Science and Medicine-Population Health*, 2, 416-424.
- Salgado, C. D. (2018). Mexican American Identity: Regional Differentiation in New Mexico. *Sociology of Race and Ethnicity*, 2332649218795193.
- Samet, J. M., Coultas, D. B., Howard, C. A., Skipper, B. J., & Hanis, C. L. (1988). Diabetes, gallbladder disease, obesity, and hypertension among Hispanics in New Mexico. *American Journal of Epidemiology*, 128(6), 1302-1311.
- Seeman, T., Epel, E., Gruenewald, T., Karlamangla, A., & McEwen, B. S. (2010). Socio-economic differentials in peripheral biology: Cumulative allostatic load. *Annals of the New York Academy of Sciences*, 1186(1), 223-239.
- Seeman, T. E., Crimmins, E., Huang, M.-H., Singer, B., Bucur, A., Gruenewald, T., . . . Reuben, D. B. (2004). Cumulative biological risk and socio-economic differences in mortality: MacArthur studies of successful aging. *Social Science and Medicine*, 58(10), 1985-1997.
- Seeman, T. E., McEwen, B. S., Rowe, J. W., & Singer, B. H. (2001). Allostatic load as a marker of cumulative biological risk: MacArthur studies of successful aging. *Proceedings of the National Academy of Sciences*, 98(8), 4770-4775.
- Seeman, T. E., Singer, B. H., Rowe, J. W., Horwitz, R. I., & McEwen, B. S. (1997). Price of adaptation—allostatic load and its health consequences: MacArthur studies of successful aging. *Archives of Internal Medicine*, 157(19), 2259-2268.
- Seeman, T. E., Singer, B. H., Ryff, C. D., Love, G. D., & Levy-Storms, L. (2002). Social relationships, gender, and allostatic load across two age cohorts. *Psychosomatic Medicine*, 64(3), 395-406.
- Shriver, M. D., & Parra, E. J. (2000). Comparison of narrow-band reflectance spectroscopy and tristimulus colorimetry for measurements of skin and hair color in persons of different biological ancestry. *American Journal of Physical Anthropology*, 112(1), 17-27.
- Stata statistical software, version 11. (2009). StataCorp, L. [Mobile application software]
- Steptoe, A., Hackett, R. A., Lazzarino, A. I., Bostock, S., La Marca, R., Carvalho, L. A., & Hamer, M. (2014). Disruption of multisystem responses to stress in type 2 diabetes: investigating the dynamics of allostatic load. *Proceedings of the National Academy of Sciences*, 111(44), 15693-15698.
- Sterling, P., & Eyer, J. (1988). Allostasis: A new paradigm to explain arousal pathology. In S. Fisher & J. Reason (Eds.), *Handbook of Life Stress, Cognition and Health*. New York: John Wiley and Sons.
- Stewart, J. A. (2006). The detrimental effects of allostasis: allostatic load as a measure of cumulative stress. *Journal of Physiological Anthropology*, 25(1), 133-145.
- Swadesh, F. L. (1974). *Hispanic Americans of the Ute Frontier*: University of Notre Dame Press.

- Tang, H., Coram, M., Wang, P., Zhu, X., & Risch, N. (2006). Reconstructing genetic ancestry blocks in admixed individuals. *The American Journal of Human Genetics*, 79(1), 1-12.
- Taylor, P., Lopez, M. H., Martínez, J. H., & Velasco, G. (2012). *When labels don't fit: Hispanics and their views of identity*. Retrieved from
- Taylor, S. E., Repetti, R. L., & Seeman, T. (1997). Health psychology: what is an unhealthy environment and how does it get under the skin? *Annual Review of Psychology*, 48(1), 411-447.
- Tishkoff, S. A., Reed, F. A., Friedlaender, F. R., Ehret, C., Ranciaro, A., Froment, A., . . . Doumbo, O. (2009). The genetic structure and history of Africans and African Americans. *Science*, 324(5930), 1035-1044.
- Trujillo, M. L. (2010). *Land of disenchantment: Latina/o identities and transformations in northern New Mexico*: UNM Press.
- Tucker, K. L. (2005). Stress and nutrition in relation to excess development of chronic disease in Puerto Rican adults living in the Northeastern USA. *The Journal of Medical Investigation*, 52(Supplement), 252-258.
- Upchurch, D. M., Stein, J., Greendale, G. A., Chyu, L., Tseng, C.-H., Huang, M.-H., . . . Seeman, T. (2015). A longitudinal investigation of race, socioeconomic status, and psychosocial mediators of allostatic load in midlife women: findings from the Study of Women's Health Across the Nation. *Psychosomatic Medicine*, 77(4), 402.
- USHHS. (2000). *Healthy People 2010: Understanding and Improving Health*. Washington, D.C.
- USHHS. (2008). The Secretary's Advisory Committee on National Health Promotion and Disease Prevention Objectives for 2020. Phase I report: Recommendations for the framework and format of Healthy People 2020. Section IV. Advisory Committee findings and recommendations. In.
- Vega, W. A., Rodriguez, M. A., & Gruskin, E. (2009). Health disparities in the Latino population. *Epidemiologic Reviews*, 31(1), 99-112.
- Velasco-Mondragon, E., Jimenez, A., Palladino-Davis, A. G., Davis, D., & Escamilla-Cejudo, J. A. (2016). Hispanic health in the USA: a scoping review of the literature. *Public Health Reviews*, 37(1), 31.
- Vichare, A. (2016). Disparities in mammogram screening among women with access to care: The role of socioeconomic status and education [abstract]. *Cancer Epidemiology, Biomarkers and Prevention*, 25(3 Supplement).
- Wang, S., Ray, N., Rojas, W., Parra, M. V., Bedoya, G., Gallo, C., . . . Hurtado, A. M. (2008). Geographic patterns of genome admixture in Latin American Mestizos. *PLoS genetics*, 4(3), e1000037.
- Warnecke, R. B., Oh, A., Breen, N., Gehlert, S., Paskett, E., Tucker, K. L., . . . Flack, J. (2008). Approaching health disparities from a population perspective: the National Institutes of Health Centers for Population Health and Health Disparities. *American Journal of Public Health*, 98(9), 1608-1615.
- Whelton, P. K., Carey, R. M., Aronow, W. S., Casey, D. E., Collins, K. J., Himmelfarb, C. D., . . . Jones, D. W. (2018). 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure

- in adults: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Journal of the American College of Cardiology*, 71(19), e127-e248.
- Williams, D. R., & Mohammed, S. A. (2009). Discrimination and racial disparities in health: evidence and needed research. *Journal of Behavioral Medicine*, 32(1), 20-47.
- Williams, D. R., Yu, Y., Jackson, J. S., & Anderson, N. B. (1997). Racial differences in physical and mental health: Socio-economic status, stress and discrimination. *Journal of Health Psychology*, 2(3), 335-351.
- Yancey, A. K., Ortega, A. N., & Kumanyika, S. K. (2006). Effective recruitment and retention of minority research participants. *Annu. Rev. Public Health*, 27, 1-28.
- Zeleny, C. (1974). *Relations between the Spanish-Americans and Anglo-Americans in New Mexico*: Arno Press.
- Zsembik, B. A., & Fennell, D. (2005). Ethnic variation in health and the determinants of health among Latinos. *Social Science and Medicine*, 61(1), 53-63.