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DISSERTATION

Selection, Wear, and Tear

The Health of Hispanics and Hispanic Immigrants in the United States

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This document was submitted as a dissertation in May 2009 in partial fulfillment of the requirements of the doctoral degree in public policy analysis at the Pardee RAND Graduate School. The faculty committee that supervised and approved the dissertation consisted of Jose Escarce (Chair), Emma Aguila, and Krishna Kumar.

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ABSTRACT

The health of Hispanics in the United States is a complex issue that is still not well understood. Among the factors that complicate the study of Hispanic health are data artifacts and cultural differences that originate from different degrees of assimilation. These problems may lead to biased estimations of the actual health profile of Hispanics relative to other ethnic groups.

In this work, I seek to provide a better understanding of the issues surrounding the health of Hispanics in general, and of Hispanic immigrants in particular. First, in Chapter 2, I provide a brief review of the literature on Hispanic health, and I discuss the hypotheses that have been proposed to explain three important results in that literature: (1) the apparent health advantage of Hispanics over other ethnic groups, despite a relatively low socioeconomic status; (2) the decline in the health status of Hispanic immigrants as their length of residence in the United States increases; and (3) a weak or even flat association between health and socioeconomic status among Hispanics.

In Chapter 3, I examine differences in health status between non-Hispanic Whites, Mexican Americans, and Mexican immigrants. I propose an index of biological risk composed by eight biomarkers that can be split into three subcomponents: inflammatory, metabolic, and cardiovascular. The index gives more weight to biomarkers that have stronger associations with mortality, and accounts for nonlinearities in those relationships. A separate set of analyses uses the Framingham risk score, a widely used indicator of risk of coronary heart disease (CHD). In addition, I explore the application of propensity score methods for the study of health disparities as an alternative to traditional regression analysis. Propensity score methods are more robust than regression to systematic differences in the distribution of characteristics between the groups being compared, and allow for simple assessment of the degree of overlap of those characteristics.

To construct the health index, I use data from the Third National Health Examination and Nutrition Survey (NHANES-III, 1988-1994) with linked mortality through 2000;

the propensity score analyses use data from NHANES-III and the 1999-2004 NHANES. Results with allostatic load as the outcome indicate that there is no general health advantage of Hispanics over Whites: Mexican Americans show higher (worse) scores for the general index and all three subcomponents. Mexican immigrants, on the other hand, have lower (better) inflammatory and cardiovascular scores, but higher metabolic scores, than Whites. Conversely, results using Framingham risk as the outcome suggest a general Mexican health advantage over Whites. Both US-born Mexicans and Mexican immigrants have lower 10-year risk of CHD than Whites; and Mexican immigrants enjoy an advantage in CHD risk over both Whites and US-born Mexicans. The discrepancies between the analyses that use allostatic load and those that use the Framingham score may be explained by the inclusion of smoking as a risk factor in the Framingham score. The qualitative results do not change when regression analysis is used, but the differences between the coefficients estimated using regression and propensity score methods are largest for the comparison of Mexican immigrants with Whites, indicating that these two groups have the largest differences in observed covariates and thus benefit the most from using propensity score methods.

In Chapter 4, I explore the Health-Age and Health-SES trajectories of Mexican immigrants using semiparametric methods. I assess the evidence supporting several of the hypotheses discussed in Chapter 2. I find indirect evidence supporting the "healthy migrant" hypothesis, which states that emigrants are positively selected in their health status from the population of their countries of origin. My results are also consistent with an apparent decline in immigrant health as the length of residence in the United States increases, a common result in the literature. However, unlike several recent studies, I find that the Health-SES gradient is similar for Whites, recent immigrants, and immigrants who have lived in the US for more than 15 years. Only immigrants who have lived between 5 and 15 years in the US appear to have a weaker gradient. Moreover, I do not find support for the "acculturation hypothesis", which states that the decline in immigrant health with increased duration of residence is a result of assimilation into US culture. In addition, my results suggest that this health decline is not likely to be due to better average health among recent immigrant cohorts when compared to ear-

lier immigrant cohorts. Two hypotheses to explain the decline in immigrant health remain consistent with my results: (1) the "life-course" hypothesis, which states that the deterioration of immigrant health status is a result of the cumulative negative effect of the adversities associated with the process of migration, and (2) the "regression to the mean" hypothesis, which maintains that immigrants self-select on health at the time of migration, but over time their health converges to the average health levels in their home countries. Finally, in Chapter 5, I summarize the main findings and I discuss the implications of this work for future research and public policy.

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Chapter 1.

Introduction and Research Questions

1.1 Introduction

It is a well established fact that individuals of higher socioeconomic status live longer and healthier lives.[1] Since there are important differences in socioeconomic levels between racial and ethnic groups in the United States, it is not surprising to find that significant racial/ethnic disparities exist in mortality and other health outcomes (National Center for Health Statistics 2007; Keppel 2007; Cooper et al. 2000; Hahn and Eberhardt 1995; Vega and Amaro 1994). However, socioeconomic differences between racial or ethnic groups are only part of the story regarding health disparities, as analyses of health disparities that account for socioeconomic measures, such as income or education, do not explain them entirely (LaVeist 2005; Williams and Collins 1995). As a result, a large number of studies have been conducted in recent years trying to identify factors that may account for these disparities in health outcomes, and policies that may be used to reduce them.

In particular, many questions remain unanswered regarding the health of Hispanics. Several studies have found that Hispanics enjoy better health and lower mortality than other racial/ethnic groups, including non-Hispanic Whites. This phenomenon has commonly been called the Hispanic Paradox because Hispanics have low socioeconomic profiles, more similar to those of non-Hispanic Blacks than those of non-Hispanic Whites, and thus we would expect them to have worse health outcomes than other ethnic groups with

[1] Goldman (2001) provides a comprehensive review of the literature on social inequalities in health.

higher levels of socioeconomic status (SES).[2] Over the last two decades, several studies have analyzed this phenomenon, using different outcome variables to compare the health of Hispanics with non-Hispanics. The results of these studies have provided mixed support for the existence of the paradox (*see* Chapter 2 for a review of the literature), but the general perception in the health literature is that Hispanics enjoy better health than expected given their socioeconomic status. In addition, a few recent studies suggest an additional puzzling finding regarding Hispanic health: the association between SES and health may be weak or even positive among Hispanics (Goldman et al. 2006; Turra and Goldman 2007; Zsembik and Fennell 2005).

A distinctive characteristic of the Hispanic population in the United States is the large proportion of immigrants among its numbers. In particular, Mexican immigrants account for over 25 percent of all Hispanics in the country. As is the case with Hispanics, available statistics indicate that Hispanic immigrants—Mexicans in particular—enjoy a better health status than other population groups in the United States. Estimates from the 2006 National Health Interview Survey (NHIS) reflect a lower prevalence of cardiovascular disease, cancer, and asthma among Mexican immigrants than other immigrants and the native White population (CONAPO 2008).

In fact, Hispanic immigration could be an important factor to explain the puzzling results described above for at least three reasons: (1) Mexican immigrants are significantly younger than the rest of the US population, and thus should be less likely to suffer from several health conditions; (2) a large number of the Mexican-born are recent immigrants (30 percent arrived in 2000 or later) and are thus less likely to correctly respond to health-

[2] From hereafter, for simplicity, I will refer to non-Hispanic Blacks as 'Blacks', and non-Hispanic Whites as 'Whites'.

related questions in population surveys due to their lack of English proficiency; and (3) perhaps the most important, health selection processes may be linked to immigration, such that immigrants may be healthier than the population of their country of origin, and less healthy immigrants may be more likely to return to their home country, leaving the healthiest immigrant population in the United States. These issues are discussed in more detail in Chapter 2.

1.2 Research Goals

In this study, I contribute to the literature on health disparities—and Hispanic health in particular—by assessing the evidence supporting the existence of a Hispanic health advantage over non-Hispanics, and exploring the patterns of immigrant health over the life-course. My first contribution is the construction of an allostatic load index, an objective measure of health status not subject to biases due to group differences in culture or health literacy. Although allostatic load has been used before to explore the Hispanic Paradox (Crimmins et al. 2007), the measure I propose weighs its components accordingly to their independent associations with mortality, and accounts for non-linearities in these associations. In order to conduct a more thorough assessment of the existence of the Hispanic Paradox, I also conduct analyses using the Framingham risk score, a well-known measure of risk of coronary heart disease.

My second contribution is the use of semiparametric methods for the study of health disparities. First, I use propensity score-based methods to produce estimates of health differences between Hispanics and Whites that are not subject to potential biases tue to estimations outside the region of common support. Second, I use another semiparametric method (differencing the partial linear model) to explore the age and SES associations with

health among immigrants, and whether these associations vary with the length of residence in the United States.

More specifically, I address the following research questions:

1. Is there evidence supporting the existence of the Hispanic Paradox when comparing the health of non-Hispanic Whites and individuals of Mexican ethnicity living in the United States?
2. Does the answer to the previous question change when the Mexican sample is divided by country of birth (i.e., Mexico and the United States)?
3. Are there differences between Mexican immigrants and non-Hispanic Whites in the associations between age and socioeconomic status with health?
4. What factors could explain the patterns found in question 3? In particular:
    a. Is there evidence of immigrant health selection?
    b. Do the health-age and health-SES patterns change with immigrants' length of residence in the United States?
    c. Are there health differences between immigrant cohorts?
    d. Are there health differences between immigrants with different degrees of acculturation?

1.3 Organization

This dissertation is organized as follows: In Chapter 2, I briefly summarize the literature on Hispanic Health, in particular that on the Hispanic Paradox, and I discuss the hypothesis that can potentially explain some of the results found by previous studies that are relevant for my work on this dissertation. In Chapter 3, I examine the existence of the Hispanic Paradox using propensity score methods and two summary indicators of health status: allostatic

load and Framingham risk scores. In this chapter, I first discuss the concept of *allostatic load* as a measure of biological risk and the use of Framingham scores as measures of risk of coronary heart disease. Next, I describe the data from the National Health and Nutrition Examination Survey and I explain the procedure I follow to create a measure of allostatic load that accounts for its components' relationships with mortality and for non-linearities in those relationships. Later in the same chapter, I discuss propensity score methods and their advantages over commonly used regression, focusing on the *doubly robust* method of propensity-score weighted regression, which I use to estimate allostatic load and Framingham risk differences between Hispanics and non-Hispanic Whites. I present the results of these estimations and conclude this chapter with a discussion of policy and future research implications. In Chapter 4, I explore the *health-age* and *health-SES* trajectories of Mexican immigrants using semiparametric methods. I assess the evidence supporting several hypotheses regarding the health selectivity of migration and the changes in health over immigrants' lifetime. Finally, in Chapter 5, I summarize the results and discuss the implications for future research and policy.

Chapter 2.

Hispanic Health in the United States: A Review

In this chapter, I describe the socioeconomic and demographic characteristics of the Hispanic population in the United States, and discuss the findings of the literature on Hispanic health. Although Hispanics are not a homogenous population—there are significant health and socioeconomic differences between Hispanic subgroups—, I focus on Hispanics in general and Mexicans in particular. The main reason for this is that my empirical analyses in the following chapters use data only on Mexican Americans and Mexican immigrants. The review of the literature is in no way complete. Nonetheless, it presents a representative set of the studies most relevant to the issues addressed in this dissertation.

2.1 Socioeconomic Profile of the Hispanic Population in the United States[3]

The Hispanic population in the United States has grown at a notably fast rate over the last three decades and they are now the largest minority group in the country. Figures from the 2000 census placed Hispanics at around 35 million people, a 58 percent increase since 1990. Estimates from the American Community Survey indicate that this growth has hardly slowed down: Hispanics increased their numbers by 29 percent between 2000 and 2007, for an estimated Hispanic population of 45 million in 2007, the last year for which estimates are available. Notably, over the 21st century Hispanics have accounted for 50 percent of total population growth in the United States (Fry 2008).

[3] Unless otherwise noted, numbers cited in Section 2.1 were obtained from the Pew Hispanic Center's *Statistical Portrait of Hispanics in the United States, 2007,* online at http://pewhispanic.org/factsheets/factsheet.php?FactsheetID=46.

A distinctive characteristic of the Hispanic population in the United States is its large number of immigrants. In 2007, about 40 percent of Hispanics were foreign-born, compared to only 8 percent for non-Hispanics (U.S. Census Bureau 2009). By far, Mexico is the country that contributes the most immigrants, as over 11 million Mexican-born immigrants currently live in the United States. The number of Mexican immigrants, which increased dramatically in the 1990s, has continued to grow in recent years. As shown in Table 1, between 2000 and 2007 Mexico accounted for 37 percent of the total change in the size of the foreign-born population and, just over that period, the number of Mexican immigrants in the United States increased by 28 percent, from 9.2 million to 11.7 million.

Table 1. US Foreign-Born Population by Region of Birth, 2000-2007

Region	2007 Total Population (1000s)	Change 2000-2007 (1000s)	Percent Change	Share of Total Change (%)
Mexico	11,740	2,576	28.1	37.3
South and East Asia	8,973	1,777	24.7	25.7
Central America	2,706	677	33.4	9.8
South America	2,577	656	34.2	9.5
Caribbean	3,374	419	14.2	6.1
Middle East	1,315	177	15.6	2.6
All others	7,364	631	9.4	9.1
Total	38,048	6,914	22.2	100.0

Source: Pew Hispanic Center (2007)

Hispanics living in the United States are younger than non-Hispanics. For example, the median age in years is 27 for Hispanics and 40 for Whites. The contrast is even more dramatic when country of birth is taken into account. Hispanic immigrants have a median age of 36, while the median age of US-born Hispanics is 17, a result of the large number of immigrants who entered the United States over the last 20 years, and who have given birth to children in this country.

The socioeconomic status of Hispanics is well below that of Whites and similar to that of Blacks. About 20 percent of Hispanics live in poverty, compared to 23 percent of Blacks and

9 percent of Whites. Hispanic median household income in 2007 was $40,500, compared to $54,700 for Whites, and $33,800 for Blacks. In terms of educational attainment, 24 percent of Hispanics of age 25 and older have an education of less than 9th grade, compared to 3 percent of Whites and 6 percent of Blacks. Accordingly, only 13 percent of Hispanics have a college degree, compared with 31 percent of Whites and 17 percent of Blacks. In education, there are important differences between US-born and foreign-born Hispanics, as only 9 percent of native Hispanics have an education less than 9th grade, but the figure is 34 percent for Hispanic immigrants.

2.2 Hispanic Health

The health of Hispanics began to receive attention in the public health literature only in the last two decades. Before, the common assumption was that Hispanic health profiles were similar to those of other minorities with similar socioeconomic conditions, such as African Americans (Vega and Amaro 1994). However, an increasing number of studies indicate that the issues surrounding Hispanic health are significantly different from those of other minority groups. A topic that has recently dominated the literature on Hispanic health is the so-called "Hispanic Paradox", the apparent health advantage of Hispanics over other ethnic groups, despite their lower socioeconomic profile. More recently, a handful of studies have identified a "second paradox": the association between socioeconomic status and health appears to be weaker among Hispanics than among other groups. Below, I discuss the main findings and yet-to-be-answered questions related to Hispanic health.

Markides and Coreil (1986) were the first to refer to the health status of Hispanics in the United States as an "epidemiological paradox". Conducting a review of previous studies, they concluded that the health status of Hispanics in Southwestern United States was closer to the health status of Whites than to that of African Americans. Among the health indica-

tors where the authors found this health similarity between Hispanics and Whites were infant mortality, life expectancy, mortality from cardiovascular diseases, cancer, and measures of functional health. Markides and Coreil called this phenomenon a paradox because Hispanics have a risk profile more similar to that of Blacks than that of Whites in terms of their socioeconomic status.

Since Markides and Coreil's article, a number of studies have explored the existence of the Hispanic Paradox. Franzini, Ribble, and Keddie (2001) conducted a comprehensive review of the literature on Hispanic health between the years 1963 and 1999. Their search identified nearly 200 relevant articles, of which they chose 89 to summarize in their review. One interesting finding of their review is that the Hispanic Paradox appears to be a recent phenomenon, since mortality studies conducted in the 1950s and through the 1970s generally found higher mortality rates for Hispanics—identified at the time by their Spanish surnames—than for Whites. It was not until data from the 1980 US census became available that mortality rates were found to be lower for Hispanics than for Whites at certain ages.

Several articles reviewed by Franzini, Ribble, and Keddie studied the mortality of Hispanics. Among them, Liao et al. (1998) used 1986-1990 data from the National Health Interview Survey, linked to the National Death Index (NDI), to assess the mortality patterns of the adult Hispanic population in the US and compare it to mortality patterns of Whites and Blacks. They found that Hispanic *males* have higher mortality rates than Whites at ages 18 to 44 (rate ratio equal to 1.33), similar mortality rates at ages 45 to 64 (RR=0.92), and lower mortality rates at ages equal to or over 65 (RR=0.76). For females, the mortality ratios at the same age categories were 1.22, 0.75, and 0.70, respectively, where only the latter two were statistically significant. Blacks had consistently higher mortality rates than both Whites and Hispanics. Similar results are found by Sorlie et al. (1993), who used data from the Current Population Survey matched to the NDI, and found that Hispanics had lower all-

cause mortality rates than non-Hispanics, as well as lower mortality from cancer and cardiovascular disease; on the other hand, Hispanics had higher mortality from diabetes and homicide.

Other studies have used self-reports of health status or health conditions. Drawing on 1989-2004 data from the National Health Interview Survey, Cho et al. (2004) examined Hispanic subgroup differences in three measures of health status: self-reported overall health, daily activity limitations, and number of days spent in bed due to illness. They found that Puerto Ricans exhibit the worst health profiles among Blacks, Whites, and all Hispanic subgroups. Individuals of Central/South American and Mexican origin are found to have lower risk of activity limitations and number of bed days than Whites. Finally, all Hispanic subgroups were more likely to report fair or poor health status than Whites.

A Hispanic health advantage has been found not only for adults, but also in infant mortality and birthweight. Kleinman (1990) used 1983 and 1984 data on linked birth and infant-death records. He finds that "despite a high rate of poverty and low use of prenatal care, Mexicans have approximately the same [infant mortality rate] as non-Hispanic whites." Regarding the heterogeneity of infant health outcomes among Hispanics, Becerra et al. (1991) and Albrecht et al. (1996) obtain similar findings: Puerto Ricans are the least advantaged group among Hispanics, while Cubans are the most advantaged. The first of these two studies also found that Mexicans had an infant mortality risk similar to that of Whites. In a more recent study, Luke et al. (2005) found that Hispanic women had lower proportions of low birth weight and preterm births—and higher average birth weight and gestation periods—than White and Black women.

On the other hand, some studies have found no Hispanic health advantage, and even disadvantages in some health outcomes. For example, the results of several studies indicate a Hispanic disadvantage in the incidence of diabetes (Hamman et al. 1989; Marshall et al.

1993), metabolic syndrome (Park et al. 2003), and obesity (Abraido-Lanza, Chao, and Florez 2005). Mitchell et al. (1992) try to determine whether Mexican Americans are more resistant than Whites to the cardiovascular effects of diabetes. They formulate this hypothesis based on the fact that Mexican Americans have a high prevalence of diabetes when compared to Whites, but experience lower cardiovascular mortality. They find that the associations of diabetes with myocardial infarction and coronary heart disease (CHD) risk factors are at least as strong, if not stronger, in Mexican Americans as in Whites. Interestingly, they still conclude that Mexican ethnicity confers protective effects against CHD, but this protection may be obscured by their high prevalence of diabetes.

Markides and Eschbach (2005) review recent research on the existence of the Hispanic Paradox. They conclude that most evidence indicates the existence of the Paradox. Studies that used datasets with better data quality—such as Medicare data linked to records from the Social Security Administration—find a significantly lower mortality advantage of Hispanics over Whites than studies that used vital statistics or linkages to the National Death Index, which indicates that poor data quality may indeed bias the results towards a larger Hispanic mortality advantage. Nevertheless, they conclude that the evidence supports the existence of the Paradox, at least for individuals of Mexican origin.

An additional puzzling result regarding Hispanic health has been identified by a few recent studies: the almost universally accepted positive association between SES and health may be weak or non-existing among Hispanics in the US. This weak association between SES and health has been found for mortality and several variables related to health and health behaviors (Goldman et al. 2006; Turra and Goldman 2007; Kimbro et al. 2008; Acevedo-Garcia, Soobader, and Berkman 2007). In fact, at least one study found that worse health is associated with higher SES levels among Mexicans (Zsembik and Fennell 2005). That such a result has escaped the attention of most of the literature is puzzling by itself.

Turra and Goldman (2007) suggest that this may be because some scholars have focused on racial/ethnic health differences while others have been mainly interested in the association between SES and health, with both groups assuming a constant overall SES-health relationship, thus paying little attention to possible variations in the SES-health gradient across racial/ethnic groups. In fact, it is likely that the two phenomena discussed in this section are interconnected. Several of the processes that have been proposed to explain the Hispanic Paradox—described in more detail below—might also result in weak *Health-SES* gradients.

2.3 Theories of Hispanic Health and the Hispanic Paradox

Three major hypotheses have been proposed to explain the Hispanic Paradox, two of them associated with immigration. First, several authors have argued that the Paradox is a result of Hispanic culture, which promotes better health behaviors and stronger social support among Hispanics than among non-Hispanics (Markides and Coreil 1986; Mitchell et al. 1990; Vega et al. 1991). Hispanics are less likely than non-Hispanics to smoke tobacco and drink alcohol (National Center for Health Statistics 2007), two important risk factors for poor health outcomes. Moreover, it has been posited that social principles in Hispanic countries result in stronger social support that positively affects health (Kana'Iaupuni et al. 2005). If these behaviors and social principles are passed across generations, Hispanics that maintain cultural ties to their countries of origin—or of their ancestors—will enjoy better health and lower risk of mortality.

The second hypothesis to explain the Hispanic Paradox is usually called the "*healthy migrant*" theory (Abraido-Lanza et al. 1999; Jasso et al. 2004). Under this hypothesis, immigrants are assumed to be a non-representative sample from the population of their countries of origin. Because of the difficulties and risks associated with the process of emi-

gration, individuals who attempt and succeed in migrating to more developed countries are likely to be positively selected in a number of traits, including health status. Although this hypothesis directly explains only better-than-expected health among immigrants, a possible explanation for a health advantage of Hispanics in general over other ethnic groups is that the US-born offspring of recent migrants inherit this good health from their parents' genes. The latter supposition is not often mentioned in the literature, but it is an implicit assumption in studies that conclude that a health advantage exists for all Hispanics, and that it is a result of the healthy migrant effect. The *healthy migrant* hypothesis is, perhaps, the most commonly accepted assumption in the Hispanic health literature, even though the evidence for it is not very clear (Rubalcava et al. 2008).

Finally, a third potential explanation for the Hispanic Paradox is the *"salmon bias"* hypothesis, which states that certain immigrants may be more likely to return to their countries of origin, such as the unemployed, retired, or those who are ill (Abraido-Lanza et al. 1999). The latter group gives the hypothesis its name, as some of these migrants would be returning to their home country only to die. If returning Hispanic migrants are more likely to be in worse health than those who remain in the United States, measures of Hispanic morbidity and mortality collected in the United States will be biased downwards, indicating a spurious health advantage of Hispanics over other ethnic groups. Although some authors have argued that the salmon bias is an important explanation of the Hispanic Paradox (Palloni and Arias 2004), recent evidence indicates that its effect is of too small a magnitude to fully explain it (Turra and Elo 2008).

In addition to the hypotheses that have been suggested to explain the Hispanic Paradox, a common premise in the Hispanic health literature is that the health of Hispanics deteriorates as they assimilate into US culture and their health behaviors worsen (Antecol and Bedard 2006). The acculturation hypothesis originates from the apparent reduction in the

immigrant health advantage over time, which would result in a convergence of immigrant health to that of the native non-Hispanic population. Although acculturation is the most commonly proposed explanation for this phenomenon, other factors may produce similar results, such as recent immigrant cohorts that are healthier than earlier cohorts, a reversion of immigrant health to the average health levels in their countries, or the accumulation of adverse life events unrelated to acculturation (Stephen et al. 1994; Jasso et al. 2004; Hertzman 2004).

2.4 Limitations to Existing Data and Previous Analyses

An important issue in the study of Hispanic health is the lack of quality and availability of data that allows for an adequate assessment of the health of Hispanics relative to other ethnic groups, and of its changes over time. One and a half decades ago, Vega and Amaro (1994) described the limitations of the data systems available at the time:

> "(a) [T]hey do not collect appropriate and accurate data on Hispanic ethnicity; (b) they do not sample sufficiently large numbers of Hispanics; and (c) they fail to tabulate and report data separately for Hispanics. Moreover, the Council of Scientific Affairs of the American Medical Association concluded, 'Accurate estimates of Hispanic death rates are impossible to determine because, until 1988, the national model death certificates did not contain Hispanic identifiers'."

Ten years later, Palloni and Arias (2004) still found problems with the quality of data-sets of Hispanic mortality and morbidity, which included the underreporting of Hispanic ethnicity, the misreporting of ages, and the mismatching of death records. Each of these data artifacts identified by Palloni and Arias may lead to spurious estimates of a Hispanic health advantage. In fact, Smith and Bradshaw (2006) argue that the under-identification of Hispanic ethnicity in death statistics accounts for the differences in life expectancy between

Hispanics and non-Hispanic Whites, and thus they conclude that "there is no Hispanic Paradox." However, studies that have used better-quality data, have found mortality rates for Hispanics that, although higher than those estimated from vital statistics, are still lower than those of non-Hispanics (Elo et al. 2004; Hummer, Benjamins, and Rogers 2004).

Chapter 3.

Health Differences Between US-Born Mexicans, Mexican Immigrants and Non-Hispanic Whites: An Analysis of the Hispanic Paradox Using Propensity-Score Methods

3.1 Introduction

The evidence discussed in Chapter 2 indicates the existence of a Hispanic health advantage over other ethnic groups. However, in addition to the data problems related to assessing the mortality of Hispanics, discussed in section 2.4, there are other potential biases that may arise as a result of the use of self-assessments of health status and self-reports of health conditions. This is a particularly important issue for the study of Hispanic health because of the large number of Hispanics who are either immigrants or offspring of recent immigrants, which may result in low degrees of assimilation into US culture and its institutional setting for an important number of Hispanics. Commonly used self-reported health indicators may produce biased results when they are used to compare ethnic groups with different levels of acculturation, such as Hispanic immigrants and US-born Whites (Finch et al. 2002).

An illustration is probably useful to explain the last point. Table 2 compares self-reported health by racial/ethnic groups in the 1988-1994 and 1999-2004 National Health and Nutrition Examination Survey (NHANES). Whites and Blacks seem to enjoy better health than Mexicans in general, as they have higher proportions of individuals reporting "*excellent*" or "*very good"* health (57 percent for Whites, 41 percent for Blacks, 33 percent for Mexicans) and lower proportions reporting "*fair*" or *"poor"* health (14, 22, and 29 percent, respectively). Moreover, when Mexicans are divided by country of birth, immigrants

appear to be the least healthy ethnic group among those displayed in the table, while US-born Mexicans display self-rated health patterns similar to those reported by Blacks.

Table 2. Self-Rated Health, by Ethnic Group

	Excellent	Very Good	Good	Fair	Poor
Whites	23%	34%	30%	11%	3%
Blacks	18%	23%	36%	18%	4%
Mexicans (all)	14%	19%	38%	25%	4%
Mexicans:					
Immigrants	12%	14%	40%	29%	4%
US-Born	17%	26%	35%	18%	4%

Source: NHANES-III and NHANES 1999-2004

On the other hand, the picture becomes significantly less clear when the prevalence of chronic diseases (shown in Table 3) is examined by racial/ethnic group. In this case, Mexicans report the lowest incidence of five of six chronic conditions, diabetes being the only one where they report slightly higher prevalence than Whites. Furthermore, when Mexicans are examined by country of birth, immigrants are the ethnic group with the lowest disease prevalence, by far and across the board. Since immigrants are on average younger than the native US population, the differences observed in Table 3 could be explained simply because younger people are less likely to suffer from chronic conditions. However, Jasso et al (2004) examined similar tabulations, stratified by age groups, using data from the National Health Interview Survey and found similar patterns of self-rated health and self-reported chronic conditions. As they discuss, these figures could be interpreted as indicating that immigrants (or Mexicans in general) may subjectively self-report themselves as having worse health than they actually have. An alternative explanation is that Mexicans indeed have worse health than Whites and Blacks but under-report their suffering of specific chronic diseases, either because of cultural differences or lack of access to medical diagnoses. Determining which of these conjectures, if either, is correct cannot be done

without additional information, which makes clear the need for a more objective measure of health, not subject to these types of biases.

Table 3. Self-Reported Chronic Disease Prevalence, by Ethnic Group

	High Blood Pressure	Asthma	Arthritis	Chronic Bronchitis	Cancer	Diabetes
Whites	25%	10%	22%	7%	10%	5%
Blacks	30%	11%	19%	5%	3%	8%
Mexicans (all)	16%	6%	10%	3%	2%	6%
Mexicans:						
Immigrants	12%	3%	7%	2%	1%	5%
US-Born	20%	10%	15%	4%	3%	7%

Source: NHANES-III and NHANES 1999-2004

In terms of methodology, another important issue in the study of health disparities are the large and systematic differences in the distribution of individual characteristics—such as income, education, or employment—between ethnic groups. Regression techniques are commonly used to adjust estimates of health differences between groups, by controlling for their differences in covariate values. However, an issue that is mostly ignored in the literature on health disparities is that regression estimates may be very sensitive to model specification assumptions and, if the differences in individual characteristics are large, the calculations may be produced by extrapolating on the available data, which may lead to estimation biases (Heckman et al. 1998; Cameron and Trivedi 2005).

In this chapter, I assess the evidence supporting the existence of a health advantage for individuals of Mexican ethnicity over non-Hispanic Whites in the United States. I contribute to the literature on the Hispanic Paradox by creating an objective measure of health status based on the concept of allostatic load—the long-term wear and tear on the body due to cumulative physiological stress. In addition to allostatic load, I use the Framingham risk score, a well-known measure of coronary heart disease risk, to test the realiability of my results. In addition, I propose the use of propensity score methods as a valuable tool to

study health disparities because they allow the researcher to assess the lack of overlap in the characteristics of the groups being compared, an issue often overlooked in these studies.

Below, I discuss the concept of allostatic load and the Framingham risk score. In section 3.2, I describe the data from the National Health Nutrition and Examination Survey, introduce the measure of allostatic load, and describe the construction of the Framingham risk score. Section 3.3 discusses propensity score methods, and the doubly-robust estimator: a consistent estimator of differences in outcomes between groups, even in the presence of misspecifications in one of the two steps that compose it. In section 3.4, I discuss the results of the allostatic load measure construction, the assessment of overlap in the distribution of covariates between non-Hispanic Whites and Mexicans, and the estimations of health differences between these two groups using allostatic load and the Framingham score. Section 3.5 concludes the chapter with a discussion of the results and their implications.

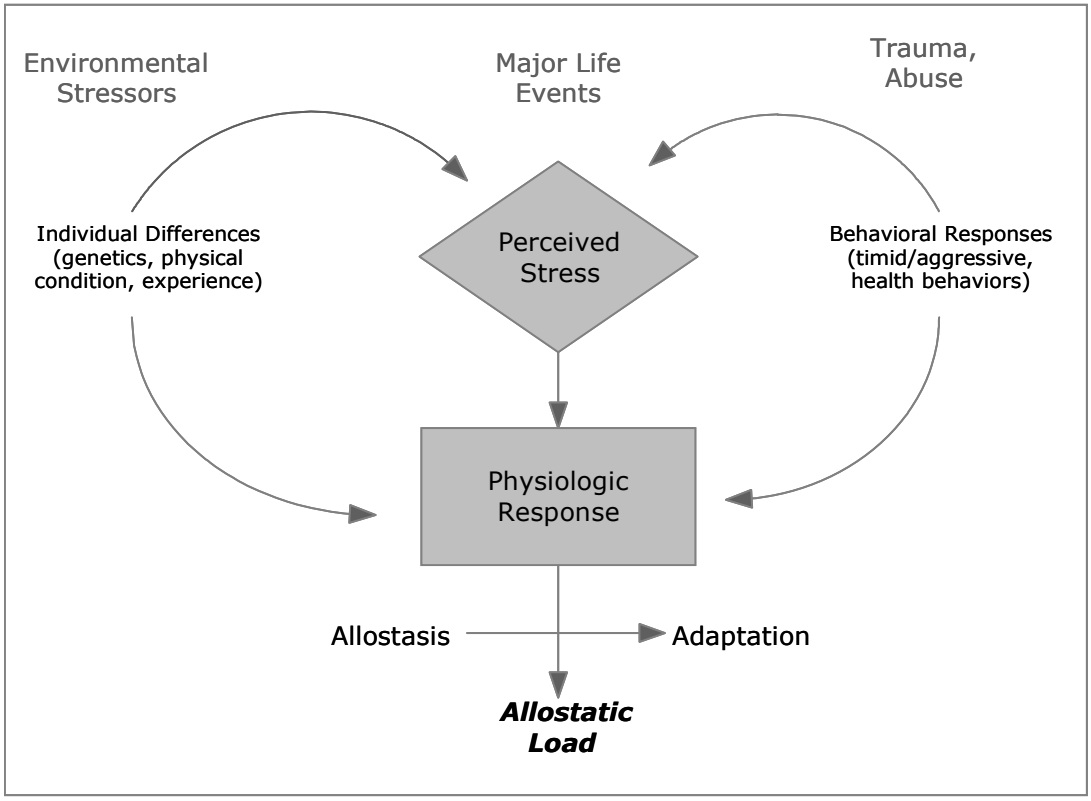
3.1.1 Allostatic Load

Stressful experiences—major life events, noise, hunger, isolation, temperature extremes, trauma, abuse, or infections—trigger physiological responses in an attempt to protect the body. Among others, the nervous, cardiovascular, metabolic, and immune systems activate biological mechanisms that seek to achieve stability through physiological adjustments. This ability of the human body to achieve stability through change is known as *allostasis* (Sterling and Eyer 1988).

Figure 1, adapted from McEwen (1998), depicts a conceptual model of the process of adaptation to stressful stimuli. An individual's ability to adapt to continuous or repeated stress depends on several factors, which include genetics, physical condition, and idiosyncrasy. Under normal circumstances, the physiological response to stress is sustained for an interval long enough to appropriately respond to the stressor and is then turned off. How-

ever, different situations may arise in which either frequent stress or insufficient adaptation result in physiological damage (McEwen and Wingfield 2003). This long-term wear and tear, experienced by the body as it struggles to achieve stability in stressful situations, has been called allostatic load (McEwen and Stellar 1993; McEwen and Seeman 1999).

Figure 1. Conceptual Model of Stress and Allostatic Load



The concept of allostatic load has been operationalized in recent studies through summary indices of several biomarkers. For example, Seeman et al (1997) developed a measure of allostatic load using 10 parameters: systolic and diastolic blood pressure, waist-hip ratio, serum high-density lipoprotein (HDL) and total cholesterol levels, plasma levels of glycosylated hemoglobin, serum dehydroepiandrosterone sulfate (DHEA-S), 12-hour urinary cortisol excretion, and 12-hour urinary norepinephrine and epinephrine excretion levels. For each of these parameters, individuals were classified into quartiles based on the sample's

distribution of their values, and the allostatic load score for each individual is created by adding the number of parameters for which the subject fell into the highest-risk quartile.

Allostatic load has been shown to be a consistent predictor of mortality, cardiovascular disease, decline in physical functioning, and decline in cognitive functioning (Seeman et al. 1997; Karlamangla et al. 2002; Seeman et al. 2001). Since allostatic load is a result of individual exposure to stress, and exposure is a function of several factors, including social and economic conditions, it has also been proposed as a likely mediator between socioeconomic status and health outcomes. Consistently with this idea, several studies have found significant associations between allostatic load and several measures of socioeconomic and psychosocial status such as education, hostility, disadvantaged environments, and poverty (Kubzansky, Kawachi, and Sparrow 1999; Evans 2003; Johnston-Brooks et al. 1998).

3.1.2 Framingham Risk Score

The Framingham risk score (FRS) is a widely used measure to predict the risk of coronary heart disease (CHD) in the general population. This score was derived from the results of the Framingham Heart Study (Anderson et al. 1991; Wilson et al. 1998), a series of studies of population-based samples with follow-up intervals of several years that allowed researchers to derive scoring algorithms to estimate the risk of CHD. The Framingham score includes risk factors such as age, gender, smoking, blood pressure, and cholesterol levels. The application of a scoring formula that accounts for different combinations of these factors results in a total score that can be used to estimate the 10-year risk of CHD for a given individual. The Framingham scoring system has been validated for individuals of ages 20-79 (NCEP 2001). Other studies have shown that the risk factors in the Framingham score do less well in predicting the risk of CHD for individuals of age 85 and older (de Ruijter et al. 2009).

3.2 Data and Measures

3.2.1 Data

I use data from the 1988-1994 and 1999-2004 releases of the National Health and Nutrition Examination Survey (NHANES-III and continuous NHANES), a series of cross-sections representative of the non-institutionalized US population (NCHS 2005). NHANES includes individual data on demographic and socioeconomic characteristics, diet, medical examinations, physical measurements, and laboratory tests. Mexican Americans and African Americans were over-sampled, so each of these groups represents around one fourth of each cross-section; the Mexican-born account for 50-60 percent of the Mexican-ethnicity samples. In addition, I use the NHANES-III National Death Index (NDI) linkage to construct the allostatic load measure; the NDI linkage provides mortality follow-up information from the date of survey participation (1988-1994) through December 31, 2000 for all NHANES-III adult participants (ages 17 and older).[4]

I restrict the data to individuals 20 years of age and older who attended the mobile examination centers where the physical exams and laboratory samples were collected. In addition, I remove women who reported to be pregnant or were found pregnant by the laboratory analyses, as is common practice when biomarkers are the outcome being analyzed. Finally, I exclude African Americans, individuals from other races, and non-Mexican Hispanics; I remove the first two groups to simplify the analyses and because the Hispanic Paradox is usually framed in terms of Hispanic mortality/health status relative to that of Whites, and the third group because there is evidence that health varies across Hispanic

[4] More detailed information on NHANES-III and the 1999-2004 NHANES surveys can be found online at http://www.cdc.gov/nchs/nhanes.htm.

subgroups (Markides and Eschbach 2005), but the sample of non-Mexican Hispanics in NHANES is too small to conduct separate analyses on them.

The study sample thus consists of 20,680 individuals—13,368 Whites and 7,312 Mexican Americans. Of this total, 11,064 come from NHANES-III and 9,616 from the 1999-2004 NHANES. However, the size of the estimation sample is 19,363 due to missing information in some of the covariates used in the analyses.[5]

3.2.2 Allostatic Load Measure

As described above, a common approach used in previous studies to operationalize the concept of allostatic load is to create an index that counts the number of biomarkers above or below a certain threshold of *biological risk*; the threshold values are chosen either by the distribution of the biomarkers in the sample, or using commonly accepted values of clinical risk (e.g., Seeman et al. 1997; Seeman et al. 2001; Crimmins et al. 2007). Although this method has some advantages such as being easy to implement and interpret, it also has the shortcoming of implicitly giving equal weight to all components of the index (Seeman et al. 2001). In addition, this approach does not account for the possibility of a non-linear relationship between each biomarker and health outcomes or interest, such as mortality.

In this study, I address these issues by following an approach similar to those of Karlamangla and colleagues (2006; 2002), who estimate the association between each biomarker and a health outcome of interest—mortality and functional decline, respectively—and construct their allostatic load measure by creating a linear combination of the biomarkers, weighting each of them with the estimated coefficients. For example, Karlamangla et al (2006) estimate a logistic regression of mortality on ten biomarkers and create their al-

[5] Unless otherwise specified, the NHANES sampling weights and sampling design variables were used in all analyses in this chapter.

lostatic load measure as a linear combination of the ten biomarkers, using as weights the coefficients estimated in the logistic regression.

To construct the allostatic load measure in this study, I use the NHANES-III NDI linkage, which includes information on mortality status, follow-up months from the exam date, and underlying cause of death. Using this information, I estimate semiparametric Cox proportional hazards models of time-until-death using eight biomarkers as the independent variables.[6] Since I am interested in exploring non-linear relationships between each biomarker and mortality, I create categorical variables for each biomarker's quintiles and include four of them in the survival regressions, using the third quintiles of each biomarker as references.[7] An alternative to using quintiles would have been to assume specific functional forms (such as quadratic or cubic) for the relationship between the biomarkers and mortality. I ran regressions with quadratic and cubic terms, but the estimated coefficients were not always consistent with findings from the medical literature about the health implications of higher or lower values of these biomarkers, which indicates that forcing those functional forms in the regression models was possibly not appropriate.

Thus, I estimate eight semiparametric survival models with the functional form

$$h\left(t_j\right) = h_0\left(t\right) \exp\left(\mathbf{x}'_j \beta\right), \tag{1}$$

where $h\left(t_j\right)$ is the hazard function, the probability of individual j dying at time t; $h_0\left(t\right)$ is the baseline hazard function, which is left unspecified in the Cox proportional hazards

[6] In the survival models, I exclude individuals who do not have follow-up information available (n=11), and individuals whose cause of death is listed as an accident (n=127).

[7] I use the third quintile as the reference group because the signs of the other four quintiles' coefficients allow me to easily determine the estimated shape of the relationship between the biomarker and the hazard function. For example, if the signs of the first two quintiles are negative and the signs of the 4th and 5th quintiles are positive, the relationship is increasing. This can obviously be done using any quintile as reference and I choose the third quintile just as a matter of personal preference.

model; $\mathbf{x}$ is a vector of dummy variables for the biomarker's 1st, 2nd, 4th, and 5th quintiles; and β is a vector of regression coefficients. The biomarkers in the $\mathbf{x}$ vectors are: C-reactive protein (CRP), serum albumin (ALB), glycosylated hemoglobin (GLY), total cholesterol (TCH), HDL cholesterol (HDL), resting heart rate (PUL), systolic blood pressure (SYS), and diastolic blood pressure (DIA).

The final step to construct the AL measure is to define it as a linear combination of the eight biomarkers' quintiles, using as weights the coefficients estimated in the survival regressions. More specifically, the allostatic load score for individual i is created using the formula:

$$\begin{aligned} AL_i = & \sum_{j\in\{1,2,4,5\}} \alpha_{j1}\cdot crp_{ij} + \sum_{j\in\{1,2,4,5\}} \alpha_{j2}\cdot alb_{ij} \\ & + \sum_{j\in\{1,2,4,5\}} \alpha_{j3}\cdot gly_{ij} + \sum_{j\in\{1,2,4,5\}} \alpha_{j4}\cdot tch_{ij} + \sum_{j\in\{1,2,4,5\}} \alpha_{j5}\cdot hdl_{ij} \\ & + \sum_{j\in\{1,2,4,5\}} \alpha_{j6}\cdot pul_{ij} + \sum_{j\in\{1,2,4,5\}} \alpha_{j7}\cdot sys_{ij} + \sum_{j\in\{1,2,4,5\}} \alpha_{j8}\cdot dia_{ij}. \end{aligned} \quad (2)$$

Where α_{jk} is the coefficient in equation (1) of the k^{th} biomarker's j^{th} quintile; for example, crp_{i1}, is a dummy variable equal to one if the individual is in the first quintile of the CRP distribution and zero otherwise. Thus, individuals with all biomarker values in the third quintile will have an allostatic load score of zero, and individuals in higher/lower quintiles of each biomarker will have allostatic load scores that will be higher or lower according to the relationship of those biomarkers with mortality, as estimated by the survival model. Using the coefficients in equation (2), I also create summary scores for three allostatic load sub-systems: *INFLAMMATORY* (consisting of CRP and ALB; that is, the first row in (2)), *METABOLIC* (GLY, HDL, and TCH), and *CARDIOVASCULAR* (PUL, SYS, and DIA).

To assess the robustness of these weights, I estimate similar logistic regressions of mortality on dummy variables of the four quintiles of each biomarker. In these regressions,

mortality is defined using a dummy variable equal to one if the survey participant died before December 31, 2000, and zero otherwise. The logistic regression estimates the association between the covariates and the probability of dying. Karlamangla et al (2006) used the coefficients estimated in a similar regression as weights in their measure of allostatic load.

I should remark that, since NDI linkage is not yet available for the 1999-2004 NHANES survey, the biomarker weights are estimated using only NHANES-III data, but the weights are used to create the allostatic load score for the entire study sample of NHANES-III and 1999-2004 NHANES participants. Thus, I make an implicit, but plausible, assumption that the relationship between the biomarkers and the probability of death remains similar across both datasets.

3.2.3 Framingham Risk Score (FRS)

I compute the 10-year risk of coronary heart disease (CHD) by using the 2001 Framingham point system guidelines from the National Cholesterol Education Program (NCEP 2001). The risk factors included in FRS are age, total cholesterol, HDL cholesterol, systolic blood pressure, tobacco smoking, and treatment for hypertension. Table 4 shows the FRS scoring system for men and women. For example, a 77-year-old man who is a smoker, has total cholesterol equal to 170, HDL cholesterol equal to 30, and *non-treated* systolic BP of 125, will have an FRS score of 16, which translates into a 10-year risk of CHD equal to 25%. Since this scoring system has only been validated for individuals of age 20-79, I conduct these analyses only with NHANES respondents within that age range. A potential source of confusion in the terminology when using the Framingham scoring system is the fact that the algorithm produces a point total which then is mapped into an estimate of 10-year risk of coronary heart disease. *Unless otherwise noted*, whenever I allude to the Framingham score or Framingham risk score, the reference is to the 10-year risk of CHD.

Table 4. Framingham Scoring for 10-year Risk of CHD (Men and Women)

Risk Factor		Age Interval 20-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79
		Scoring Points ***(Men / Women)***									
Age		-9 / -7	-4 / -3	0 / 0	3 / 3	6 / 6	8 / 8	10 / 10	11 / 12	12 / 14	13 / 16
Total Chol.	<160			0 / 0		0 / 0		0 / 0		0 / 0	
	160-199			4 / 4		3 / 3		2 / 2		1 / 1	0 / 1
	200-239			7 / 8		5 / 6		3 / 4		1 / 2	0 / 1
	240-279			9 / 11		6 / 8		4 / 5		2 / 3	1 /2
	≥280			11 / 13		8 / 10		5 / 7		3 / 4	1 / 2
Smoking	Smoker			8 / 9		5 / 7		3 / 4		1 / 2	1 / 1
HDL Chol.	<40						2 / 2				
	40-49						1 / 1				
	50-59						0 / 0				
	≥60						-1 / -1				
Hypertension Treatment				***Non-Treated***						***Treated***	
Systolic BP	<120			0 / 0						0 / 0	
	120-129			0 / 1						1 / 3	
	130-139			1 / 2						2 / 4	
	140-159			1 / 3						2 / 5	
	≥160			2 / 4						3 / 6	

RISK SCORING

Men Total Pts.	<0	0-4	5-6	7	8	9	10	11	12	13	14	15	16	≥17
10-year risk	<1%	1%	2%	3%	4%	5%	6%	8%	10%	12%	16%	20%	25%	≥30%
Women Total Pts.	<9	9-12	13-14	15	16	17	18	19	20	21	22	23	24	≥25
10-year risk	<1%	1%	2%	3%	4%	5%	6%	8%	11%	14%	17%	22%	27%	≥30%

3.2.4 Imputation of Income Categories

Most control variables used in this study are missing values for relatively few observations and thus the removal of those observations from the sample is not a source of concern in terms of potential bias in the results. One exception is the ratio of family income to poverty level (PIR), which is missing for 1,827 of the 20,608 observations, almost 9 percent of the study sample.

In order to prevent the exclusion of so many observations, I use a single imputation method (Donders et al. 2006). The imputation procedure is the following: First, using observed values, I create six PIR categories (PIR<1.0, 1.0≤PIR<2.0, 2.0≤PIR<3.0, 3.0≤PIR<4.0, 4.0≤PIR<5.0, and PIR≥5.0). I then estimate an ordinal logistic model of the PIR categories on a set of socio-demographic variables (age, gender, ethnicity, education, health insurance, employment status, type of work done the longest, and marital status), and for each observation in the sample missing a value of PIR I predict the probabilities of it belonging to each of the six PIR categories. Finally, to introduce an element of uncertainty—and thus realism, since not all individuals with, for example, a college education are at the top of the income scale—I generate a random variable with a uniform distribution between zero and one, and impute the PIR category of those observations missing PIR according to the cumulative probabilities of belonging in each category and the random number corresponding to that observation.[8]

[8] For example, if an observation has probabilities 0.10, 0.20, 0.40, 0.15, 0.10, and 0.05 of belonging in PIR categories 1-6, respectively, and the random number generated for that observation is 0.6, the imputed PIR category for that observation will be PIR3, but if the random number is 0.97 then the imputed category will be PIR6. Thus, this observation has a relatively large probability of being placed in category PIR3, but this result is not deterministic.

3.3 Methodology of Analyses

Propensity score methods, first introduced in Rosenbaum and Rubin (1983), are most commonly used in the program evaluation literature, where the main objective is to estimate the causal effect of a specific treatment on an outcome of interest. The vocabulary and mathematical notation used in this literature are often based on the counterfactual model, first formalized by Donald Rubin (1974, 1977), with important contributions from James Heckman (1978, 1989) and Charles Manski (1995, 2003, 2007), among others.

In short, the counterfactual model assumes that each individual in the population can be exposed to one of two (or more) states, usually called *treatment* and *control*. Although only one state is observed for each individual, the model assumes that a potential *what-if* outcome in the unobserved state exists for each individual (i.e., the counterfactual). The objective thus becomes to generate *defensible* counterfactual estimations so that treatment effects, the difference between the outcome under the observed state and the outcome under the counterfactual, can be estimated (Morgan and Winship 2007).

Heckman, Ichimura, Smith, and Todd (1998) provide a breakdown of the bias that may arise in the estimation of treatment effects using the counterfactual model:

(a) Bias due to counterfactual estimates in regions where there are no individuals belonging to one of the two comparison groups (i.e., estimates outside the region of common support)

(b) Bias due to improperly weighting the estimates due to differences in the distributions of observed characteristics between the treatment and control groups, even within the region of common support, and

(c) Bias due to unobserved characteristics that affect both the treatment assignment and the outcome (e.g., selection bias).

Matching is a commonly used approach that eliminates the *first two* sources of bias (Blundell, Dearden, and Sianesi 2005). Its basic idea is to generate a sample of individuals in the *control* state with a distribution of characteristics that closely resembles that of individuals in the *treatment* state. If the assumption of *selection on observables* holds, the third source of bias listed above is ignorable and the differences in outcomes between both groups can be attributed to the treatment.[9]

If the set of variables used to do the matching is large, finding exact matches can be difficult. Matching on the propensity score is an alternative to reduce the high dimensionality of this problem. The propensity score is simply the probability that an individual is in the treatment group, given the set of observed characteristics that are related to both treatment status and outcome; Rosenbaum and Rubin (1983) showed that matching on the propensity score achieves the same results as matching on the complete set of observed variables. More recently, methods that use the propensity scores as weights, rather than matching on them, have been developed. In Section 3.3.1, I discuss combining propensity score weighting and regression, the method I use in this study; a fairly comprehensive review of these and other "treatment effects" methodologies can be found in Imbens (2004).

As I mentioned above, propensity score methods are mostly used in the context of program evaluation, where the counterfactual is clearly defined—that is, some individuals receive the treatment but might not have received it, just as those who did not get the treatment could have received it; thus, in most propensity score applications, individuals in the treatment group could have been in the control group and vice versa. However, Frölich

[9] The selection on observables condition, often also called *unconfoundedness,* requires that, conditionally on the observed variables, the distribution of the counterfactual outcome in the treated group is the same as the observed distribution of the outcome in the control group. This condition implies that unobserved variables that affect the outcome should not also affect treatment assignment.

(2007) shows that propensity score methods can be used outside the sphere of treatment evaluation because the consistency of these methods does not depend on the concept of the counterfactual. Frölich, for example, uses propensity score matching to analyze the gender wage gap in the United Kingdom, where the *treatment* group are women, and the *control* group are men.

Although propensity score methods are often used as an alternative to regression analysis, both methods depend on the principle of unconfoundedness (i.e., ignorability of the third source of bias described above). There are, however, two key differences. First, propensity score methods can be nonparametric or semiparametric, depending on how the propensity score is estimated, while linear regression makes the assumption that conditioning on a linear combination of the covariates is enough to eliminate selection bias; if the covariate distributions are similar enough between both groups, the linearity assumption may not be important, but if the distributions differ substantially, parameter estimations may be based mostly on extrapolations, with the potential for substantial bias. Second, propensity score methods allow for a straightforward identification of the region of common support, highlighting the importance of removing individuals from the control (or treatment) group who do not have similar counterparts in the other group; this issue is usually ignored in studies that use regression methods, letting the linear functional form produce estimates outside the region of common support (Caliendo 2006). If overlap is a problem, however, neither regression methods nor propensity score models are appropriate, but common regression diagnostics do not focus on assessing this situation.

Nevertheless, it is important to note that if the assumptions of regression methods are met—particularly, if the specification of the functional form is correct and the region of common support covers the entire range of the data—the use of propensity score methods will not provide any benefits over regression in terms of unbiased estimation of the pa-

rameters. Still, under uncertainty, using propensity score methods has distinct advantages due to the key differences described in the previous paragraph (Rubin 1997; Harding 2003).

3.3.1 Doubly-Robust Estimator: Propensity Score-Weighted Regression

Although both regression and propensity score methods are used for the same purpose—to reduce estimation bias related to differences in observed covariates—there are potential advantages to combining them (Rubin and Thomas 2000; Ho et al. 2007). James Robins refers to the combination of propensity score and regression methods as a "doubly robust" estimator in the sense that, as long as the parametric model for either the propensity score or the outcome regression is correctly specified, the resulting estimator will be consistent. Thus, combining propensity score weighting with regression provides the researcher with "two chances" to get nearly correct inference about a causal effect (Robins and Rotnitzky 2001; Bang and Robins 2005).

The approach I use in this study roughly mirrors that of Hirano and Imbens (2001), who propose an estimator that combines propensity score (PS) weighting with regression adjustment. Specifically, I first estimate the propensity score model using a logistic regression of the form:[10]

$$\Pr\left(M_i = 1 \middle| Z_i = z\right) = \frac{\exp\left(z'\gamma\right)}{1+\exp\left(z'\gamma\right)}. \tag{3}$$

[10] In practice, the choice of a logistic or probit model is not critical, since both models produce similar results (Caliendo and Kopeinig 2008). However, since I use bootstrapping techniques to estimate the standard errors of the estimators (more details below in this section), I choose the logistic model because its computation is slightly faster.

In this equation, M_i is an indicator equal to one when the individual is of Mexican ethnicity and zero otherwise;[11] Z_i is a vector of covariates that vary across Mexicans and non-Mexicans; and γ is a vector of regression coefficients. Once this regression is estimated, I compute a propensity score, $\hat{p}(z_i)$, for every observation in the sample by predicting each individual's likelihood of *being Mexican* given her observed covariate values. To guarantee that estimates are computed in the region of common support, I drop observations in the Mexican sample with higher propensity scores than the maximum propensity score value in the comparison sample and, similarly, I drop observations in the comparison sample with propensity score values below the minimum propensity score in the Mexican sample.

After computing the propensity scores and limiting the sample to the region of common support, I estimate the average difference in allostatic load and Framingham risk scores between Mexicans and the comparison groups using two different methods: First, I use the traditional semiparametric approach—where the propensity scores are estimated using a parametric model, but no specific functional form is assumed for the allostatic load differences between groups—by taking a simple difference in means between both groups, but weighting each observation with the following weights:

$$w_i(m,w) = v_i\left[m_i + (1-m_i)\cdot\frac{\hat{p}(z_i)}{1-\hat{p}(z_i)}\right], \tag{4}$$

where v_i are the NHANES sampling weights, and m_i and $\hat{p}(z_i)$ are defined the same as above. Thus, the PS weights are equal to one (multiplied by the sampling weights) for Mexi-

[11] My analyses involve four different comparisons: (a) Whites with all Mexicans, (b) Whites with US-born Mexicans, (c) Whites with Mexican immigrants, and (d) US-born Mexicans with Mexican immigrants; thus, M_i is defined accordingly to the relevant comparison, with Mexican immigrants being the *treatment* group in the last of the four comparisons.

cans and $\hat{p}(z_i)/(1-\hat{p}(z_i))$ for the individuals in the comparison group. The semiparametric estimator of allostatic load differences between groups is then equal to:

$$\hat{\tau}_{a,s} = \frac{\sum_{i=1}^{n_t} w_i \cdot AL_i}{\sum_{i=1}^{n_t} w_i} - \frac{\sum_{j=1}^{n_c} w_j \cdot AL_j}{\sum_{j=1}^{n_c} w_j} \tag{5}$$

Similarly, the semiparametric estimator of Framingham risk score (FRS) differences between groups is:[12]

$$\hat{\tau}_{f,s} = \frac{\sum_{i=1}^{n_t} w_i \cdot FRS_i}{\sum_{i=1}^{n_t} w_i} - \frac{\sum_{j=1}^{n_c} w_j \cdot FRS_j}{\sum_{j=1}^{n_c} w_j} \tag{6}$$

Second, I compute the doubly-robust estimator of differences between ethnic groups by estimating least squares regressions with the form:

$$AL_i = \alpha_0 + \hat{\tau}_{a,p} \cdot M_i + \alpha_1' X_i + \alpha_2' \cdot M_i \cdot \left(X_i - \overline{X}_1\right) + \varepsilon_i \tag{7}$$

where AL_i is the allostatic load score of individual *i*; $\hat{\tau}_{a,p}$ is the doubly-robust parametric estimator of the difference in allostatic load between Mexicans and Whites (or the relevant comparison group); X_i is a vector of socioeconomic and demographic covariates; and $\overline{X}_1$ is the average of X for the relevant *treatment* group (i.e., all Mexicans, US-born Mexicans, or Mexican immigrants). The same PS weights in equation (4) are used to weigh the observations in these regressions.

I should observe that I do not produce a doubly-robust estimator for the FRS measure because the Framingham 10-year risk of CVD is top-coded at 30%—that is, for individuals

[12] As explained in section 3.2.3, the outcome variable in these analyses is the 10-year risk of CHD computed using the scoring system in Table 4. For simplicity, I refer to this risk as the Framingham risk score (FRS).

with 10-year risk of CHD above 30% I do not have an exact risk estimate, I only know that it is at least 30%. A common method to conduct analyses when the outcome measure is top-coded (i.e., right-censored) is the Tobit regression model (Tobin 1958; Schnedler 2005). Indeed, I could substitute this method for the ordinary least squares model in order to compute the doubly-robust estimator. However, Tobit models require stronger assumptions than ordinary least squares and are particularly vulnerable to biases in the presence of heteroskedasticity (Johnston and DiNardo 1997). In order to avoid these problems, I limit the analyses using the Framingham score to the semiparametric estimator in equation (6).

The specification in equations (4) – (7) produces an estimation of the *treatment effect for the treated* (ATT); that is, the estimators $\hat{\tau}_{a,s}$, $\hat{\tau}_{f,s}$, and $\hat{\tau}_{a,p}$ represent the average difference in allostatic load—or Framingham risk scores—between Mexicans and a sample of Whites with a covariate distribution similar to the Mexican sample. I consider this estimator to be more appropriate for this study than the more commonly used *average treatment effect* (ATE), which estimates the average difference between Mexicans and a sample with a covariate distribution similar to that of the entire sample.

A final important issue when propensity score methods are used is that the standard errors estimated by statistical software in the final step—equations (6) and (7)—are incorrect because the propensity score estimation in the first step and the use of PS weights in the second step introduce additional sources of variability that are not taken into account by the software that estimates either the differences in means or the WLS equation. Correctly estimating the standard errors is important in order to test the significance of the parameter estimates. Following a common approach in the literature (Caliendo and Kopeinig 2008), I use bootstrapping to estimate the standard errors of the main parameter estimates. For each of the four comparisons, 250 bootstrap draws are conducted, each of them reproducing the entire procedure described in this section; that is, in each repetition: (1) a new

sample with replacement is drawn, (2) the propensity scores are computed, (3) observations outside the region of common support are dropped, (4) propensity score weights are generated, (5) the average difference between Mexicans and the comparison group is estimated using a PS-weighted difference in means, and (6) the adjusted difference between Mexicans and the comparison group is estimated using a PS-weighted least squares regression.

3.3.2 Model Building

Choosing the sets of covariates to be included in the propensity score and outcome models (equations (3) and (7)) is not a straightforward decision. Some authors suggest including a comprehensive set of covariates and advise against removing variables when doubt exists about whether they should be included or not (e.g., Rubin and Thomas 1996). Conversely, others argue that the choice of variables must be made carefully and taking into account theory, previous research, and any additional knowledge relevant to the particular model being estimated (Caliendo and Kopeinig 2008).

Regardless of the approach used to build the models, since the main objective of using a propensity score method is to balance the distribution of characteristics in the treatment and control groups, one should always assess the quality of the matching method used. There are several alternatives to do this, but I choose the simplest one, which is to conduct two-sample t-tests to check if there are any significant differences in the means of the covariates in the treatment and control groups. If the t-test shows significant differences, then the propensity score model is misspecified and higher-order terms and interactions of the original covariates must be added to the model until the differences in covariate means are no longer significant.

The strategy I choose to build the propensity score and outcome models is similar to that described in Hirano and Imbens (2001), who estimate *k* logistic regressions of the treatment dummy on each individual covariate (where *k* is the number of covariates available in their data) and similarly estimate *k* ordinary least squares regressions of the outcome variable on the treatment dummy and each covariate. They then include in the propensity score model all covariates with a t-statistic above a threshold value t_p, and they include in the outcome model all covariates with a t-statistic above a threshold value t_{reg}. Although this method is easy to implement, it ignores issues such as the inclusion of potentially endogenous covariates in one of the models, or correlation between covariates that may lead to multicollinearity. I thus estimate *k* logistic and OLS regressions, but use the results only as guides to identify covariates that are clearly not significant in either the propensity score or outcome models and to make decisions regarding the collapsing of categorical variables into fewer groups. Following the suggestions of other authors (Caliendo and Kopeinig 2008), I include in the outcome model variables that previous research has shown to be significantly associated with health outcomes.

An additional complication I face is the fact that in this study I estimate a large number of outcome models: since I have four sets of comparisons (see footnote 11) and five sets of outcomes (allostatic load, inflammatory, metabolic, cardiovascular, and Framingham risk scores), my main analyses require the estimation of 20 models; in addition, my sensitivity analyses (see Section 3.3.3) involve similar estimations using a different definition of allostatic load, previously used by other authors; finally, I conduct additional estimations where I remove from the models variables that are potentially endogenous. In all, I estimate a total of 100 models. In order to simplify model selection, I include in each model all covariates with significant t-tests in any of the five outcomes, regardless of whether the

covariate is significant in each particular model or not. Finally, I include in the propensity score models all covariates present in the outcome models to guarantee that their distributions are balanced across the treatment and control groups.

3.3.3 Sensitivity Analyses

3.3.3.1 Traditional Definition of Allostatic Load

Many recent studies have defined the AL measure as a count of the number of biomarkers with values beyond a certain threshold of high risk (see Section 3.1.1 above). As I also commented earlier, these measures implicitly assign each biomarker the same importance and do not account for potential non-linearities in the relationship between the biomarkers and, say, mortality. Nevertheless, in order to assess the sensitivity of my results to the definition of allostatic load (and its three subcomponents) used in this study, I estimate weighted-least squares models—with the propensity score weights—in which the allostatic load variables are defined as in these studies.

In this definition, subjects are classified for each biomarker into quartiles based on the distribution of scores in the sample. The traditional allostatic load score is created by summing the number of biomarkers in the highest risk quartile, which is the top quartile for most of the eight biomarkers, except for HDL cholesterol and albumin, for which the bottom quartile is the highest risk group. A similar procedure is used to create summary scores for the inflammatory, metabolic, and cardiovascular systems.

3.3.3.2 Potential Endogeneity of Covariates

Although the main variable of interest—Mexican ethnicity—is clearly not endogenous in the allostatic load models because health status does not determine whether a person is Mexican or not,[13] some of the control variables used in both the propensity score and allostatic load models (e.g., income, education) are likely to be endogenous either in the propensity score or in the outcome models. For example, Mexicans are more likely to come from families with low income and are less likely to have health insurance. Since both income and insurance status are used in the allostatic load models as control variables, it is possible that the coefficient estimates of the main variable of interest are biased. In order to assess the importance of this problem, I estimate the models in Section 3.3.1 with and without the income and insurance variables.

3.4 Results

3.4.1 Construction of Allostatic Load Measure

The results of the survival and logistic regressions are shown in Table 5. Only the fourth and fifth quintiles are used for C-reactive protein because 60 percent of the observations had the same value due to the lowest possible measurement in NHANES-III, and thus the first three quintiles are the reference group in that case.[14] The association of the eight biomarkers with mortality varies significantly in terms of strength and direction. The coeffi-

[13] Endogeneity would be a concern if certain individuals were less likely to report being of Mexican ethnicity and their health status were different from those who do report being Mexican. Although a similar problem has been mentioned in studies that use death records to explore the Hispanic Paradox, I am not aware of any study that has found that ethnicity self-reporting varies across individuals of different health statuses.

[14] The lowest measured CRP value in the 1999-2004 NHANES was significantly lower than in NHANES-III. To avoid spurious results that lower CRP values in the latter NHANES imply better health than in NHANES-III, I set the minimum CRP value in 1999-2004 equal to that of NHANES-III, which resulted in 60% of the observations having this value.

cients in Table 5 show mostly the expected results: allostatic load increases with higher values of C-reactive protein, glycosylated hemoglobin, total cholesterol, pulse, and systolic blood pressure; allostatic load decreases with higher values of serum albumin; and, finally, the association of allostatic load with diastolic blood pressure and HDL cholesterol is U-shaped, with low (high) values of diastolic BP (HDL cholesterol) being associated with higher AL than values in the middle. Despite the U-shaped relationship, HDL values in the fifth quintile are still *better* than HDL values in the first quintile, as would be expected. The relative magnitudes of the coefficients are similar in both the survival and logistic regressions and most signs are identical (the only one that is different, fourth quintile of HDL cholesterol, is not significant in either regression. Given the overall consistency of the results, I conclude the coefficients are robust and I use those estimated in the survival regression as weights in the allostatic load score, since the survival models use the most information available from the data.

Allostatic load is thus defined as a linear combination of the quintiles of the eight biomarkers, where the linear combination's weights are the coefficients estimated in the survival regressions. Using equation (2), an individual's allostatic load score is:

$$\begin{aligned} AL_i = \; & 0.35crp_4 + 0.92crp_5 + 0.93alb_1 + 0.32alb_2 - 0.22alb_4 - 0.81alb_5 \\ & -0.58gly_1 - 0.29gly_2 + 0.59gly_4 + 1.40gly_5 - 0.47tch_1 - 0.14tch_2 + 0.23tch_4 + 0.64tch_5 \\ & +0.40hdl_1 + 0.12hdl_2 - 0.09hdl_4 + 0.26hdl_5 - 0.18pul_1 - 0.23pul_2 - 0.13pul_4 + 0.45pul_5 \\ & -0.68sys_1 - 0.40sys_2 + 0.54sys_4 + 1.58sys_5 + 0.24dia_1 + 0.01dia_2 - 0.03dia_4 + 0.25dia_5 \end{aligned} \tag{8}$$

Recall that, for example, crp_4 is a dummy variable equal to one if the respondent is in CRP's fourth quintile and zero otherwise, so that only one coefficient from each biomarker's set of dummy variables will be used in the AL score. Thus, an individual with values of all

eight biomarkers in the middle quintile would have an allostatic load score of zero. Higher allostatic load scores represent worse health. For ease of interpretation of the results, I transform the *AL*, inflammatory, metabolic, and cardiovascular scores so that each of them has a mean of zero and standard deviation equal to one. I use these standardized variables in all remaining analyses.

3.4.2 Descriptive Statistics

The descriptive statistics of the original sample of NHANES participants, weighted using the original NHANES sampling weights, are shown in Table 6. Clearly, there are significant differences in the distributions of covariates between Whites and Mexicans. The latter are, on average, almost ten years younger, have lower income, are less educated, and are more likely to have had blue collar jobs. Moreover, there are also significant differences between US-born Mexicans and Mexican immigrants. The characteristics of US-born Mexicans are generally at a midpoint between those of Whites and Mexican immigrants. An important characteristic of Mexican immigrants is that only 42 percent of them are female, compared to 51 percent for both Whites and US-born Mexicans. Differences in education are also striking: over 70 percent of Mexican immigrants do not have a high school diploma, compared to 17 percent of Whites and 33 percent of US-born Mexicans.

Table 5. Estimated Coefficients from Survival Models for Allostatic Load Weights

Biomarker	Quintile	Survival Models		Logistic Models		Shape
CRP	1st	*See note*		*See note*		*Increasing*
	2nd	*See note*		*See note*		
	3rd	*Reference*		*Reference*		
	4th	**0.350**	(0.107)	0.209	(0.119)	
	5th	**0.920**	(0.068)	**0.904**	(0.705)	
Albumin	1st	**0.933**	(0.098)	**0.844**	(0.107)	*Decreasing*
	2nd	**0.321**	(0.099)	**0.315**	(0.100)	
	3rd	*Reference*		*Reference*		
	4th	-0.221	(0.133)	-0.174	(0.133)	
	5th	**-0.811**	(0.160)	**-0.709**	(0.168)	
Glycosylated Hemoglobin	1st	**-0.579**	(0.150)	**-0.552**	(0.142)	*Increasing*
	2nd	**-0.289**	(0.127)	-0.253	(0.133)	
	3rd	*Reference*		*Reference*		
	4th	**0.589**	(0.115)	**0.609**	(0.121)	
	5th	**1.398**	(0.097)	**1.451**	(0.103)	
Total Cholesterol	1st	**-0.469**	(0.107)	**-0.485**	(0.106)	*Increasing*
	2nd	-0.140	(0.101)	-0.161	(0.109)	
	3rd	*Reference*		*Reference*		
	4th	**0.225**	(0.103)	**0.229**	(0.111)	
	5th	**0.643**	(0.093)	**0.679**	(0.093)	
HDL Cholesterol	1st	**0.398**	(0.115)	**0.414**	(0.116)	*U-Shaped*
	2nd	0.119	(0.105)	0.137	(0.107)	
	3rd	*Reference*		*Reference*		
	4th	-0.090	(0.114)	0.088	(0.117)	
	5th	**0.258**	(0.105)	**0.264**	(0.114)	
Pulse	1st	-0.182	(0.111)	-0.140	(0.118)	*Flat, then Increasing*
	2nd	-0.232	(0.125)	-0.216	(0.134)	
	3rd	*Reference*		*Reference*		
	4th	-0.132	(0.144)	-0.153	(0.149)	
	5th	**0.448**	(0.119)	**0.421**	(0.130)	
Systolic Blood Pressure	1st	**-0.683**	(0.137)	**-0.710**	(0.139)	*Increasing*
	2nd	**-0.404**	(0.135)	**-0.438**	(0.142)	
	3rd	*Reference*		*Reference*		
	4th	**0.536**	(0.101)	**0.567**	(0.105)	
	5th	**1.577**	(0.103)	**1.694**	(0.109)	
Diastolic Blood Pressure	1st	**0.238**	(0.102)	0.195	(0.103)	*U-Shaped*
	2nd	0.007	(0.112)	0.018	(0.119)	
	3rd	*Reference*		*Reference*		
	4th	-0.030	(0.104)	-0.024	(0.109)	
	5th	**0.250**	(0.086)	**0.276**	(0.086)	

NOTES: Standard errors are in parentheses. Coefficients in **bold typeface** are significant at the 5 percent confidence level. The survival regressions are semi-parametric Cox proportional hazards models. Due to the minimum level measured in NHANES-III, about 60% of the observations have the same C-reactive protein (CRP) value; thus, the first three quintiles are used as reference.

Table 6. Descriptive Statistics of Original Sample (Averages/Proportions)

Variable	Whites	All Mexicans	US-born Mexicans	Mexican Immigrants
N (unweighted)	12,778	6,583	3,095	3,488
Age	47	38	40	37
Female	51%	46%	51%	42%
Income/Pov. Ratio				
PIR < 1.0	9%	29%	21%	36%
1.0 ≤ PIR < 2.0	18%	34%	26%	39%
2.0 ≤ PIR < 3.0	19%	16%	18%	15%
3.0 ≤ PIR < 4.0	17%	9%	14%	6%
4.0 ≤ PIR < 5.0	13%	5%	8%	2%
PIR > 5.0	24%	7%	13%	2%
Education				
Less than HS	17%	55%	33%	71%
HS degree	35%	24%	34%	17%
College +	48%	21%	33%	12%
Insurance				
Private	78%	45%	61%	33%
Public	11%	12%	15%	10%
Uninsured	11%	43%	24%	57%
Type of work done the longest				
Never worked	2%	5%	3%	7%
White collar	26%	8%	13%	5%
Service	42%	36%	47%	28%
Blue collar	29%	50%	35%	60%
Military	1%	1%	2%	0%
Marital Status				
Single	14%	19%	20%	19%
Widowed/Divorced	18%	12%	16%	9%
Married	68%	69%	64%	72%

NOTES: All observations are weighted using the original NHANES sampling weights for the sample that received medical examinations. The sample size of US-born Mexicans and Mexican immigrants does not add up to the total of all Mexicans in the sample because a few respondents reported Mexican ethnicity but not their country of birth.

Average values of the biomarkers included in the allostatic load index for each ethnic group are shown in Table 7. Mexican immigrants have the 'best' average values in six of the biomarkers used in this study—C-reactive protein, serum albumin, total cholesterol, pulse, systolic blood pressure, and diastolic blood pressure—, while Whites had the best average values of serum albumin and HDL cholesterol. Table 7 also shows the average values of the

summary indices of health status. Mexican immigrants have the lowest allostatic load scores, while US-born Mexicans and Whites have the highest (recall that lower AL values are better, and the scores are centered around the mean, such that the average in the full sample equals zero). Not only do immigrants have the lowest overall allostatic load score, their inflammatory and cardiovascular scores are also the lowest among the three groups, although their metabolic score is slightly higher (but not statistically significant) than that of Whites. As for the Framingham risk score, Whites have the highest 10-year risk of coronary heart disease, (5.1 percent), followed by US-born Mexicans (3.6 percent), and Mexican immigrants (2.7 percent).

Table 7. Average Values of Eight Biomarkers, The Allostatic Load Index and its Subcomponents, and Framingham Risk Score; by Ethnic Group

	Non-Hispanic Whites		US-Born Mexicans		Mexican Immigrants	
C-reactive Protein	**0.419**	(0.007)	0.516	(0.027)	**0.414**	(0.014)
Serum Albumin*	4.285	(0.013)	4.308	(0.023)	**4.351**	(0.013)
Glycosylated Hemoglobin	**5.338**	(0.016)	5.464	(0.024)	5.469	(0.024)
Total Cholesterol	203.1	(0.599)	**196.3**	(1.153)	**194.8**	(1.062)
HDL Cholesterol*	**51.30**	(0.272)	50.56	(0.302)	48.15	(0.279)
Pulse	73.34	(0.249)	73.86	(0.392)	**71.09**	(0.278)
Systolic Blood Pressure	121.6	(0.302)	119.9	(0.573)	**117.2**	(0.484)
Diastolic Blood Pressure	72.32	(0.196)	71.93	(0.312)	**69.57**	(0.354)
Allostatic Load	0.065	(0.045)	0.065	(0.028)	**-0.137**	(0.030)
Inflammatory Score	0.049	(0.031)	0.109	(0.055)	**-0.147**	(0.031)
Metabolic Score	0.055	(0.025)	0.078	(0.035)	0.061	(0.030)
Cardiovascular Score	0.040	(0.018)	-0.016	(0.035)	**-0.226**	(0.030)
Framingham Risk Score	5.08%	(0.108)	3.59%	(0.138)	**2.72%**	(0.148)

NOTES: Standard errors are in parentheses. * Indicates biomarkers for which higher values represent better health status. **Bold** typeface indicates the group with the 'best' average value for each biomarker. If the differences between any group and the 'best' group are not statistically significant, both are in **bold** typeface.

3.4.3 Common Support

A first assessment of the region of common support can be made visually, by inspecting the distribution of propensity scores in the treatment and control groups (Lechner 2000). The main goal of this initial assessment is to spot any potential support problems, and then one can proceed to do a more precise assessment of the common support region and eliminate observations outside of it.

The distributions of the propensity scores in each of the four comparisons are shown in Figure 2.[15] Although the propensity score distributions of the White sample in the first three graphs have "thin" right tails, there is sufficient overlap between *treatment* and *control* groups in all four comparisons—the last graph is for the comparison of US-born Mexicans and Mexican immigrants. In order to guarantee that the common support condition holds, I apply the method of comparing minimum and maximum values (Caliendo and Kopeinig 2008). That is, I delete all observations with lower propensity scores than the minimum—and higher propensity scores than the maximum—in the opposite group.

The minima and maxima of the propensity scores, as well as the number of observations dropped due to being outside the common support region, are reported in Table 8. Not surprisingly, the comparison with the most dropped observations was that for Mexican immigrants and Whites, and even in that comparison less than 3 percent of the sample had to be dropped. Despite the considerable differences in the distribution of covariates reported in Table 6, the number of observations outside the region of common support is relatively small. Thus, common support does not appear to be a problem and valid estimates of health differences between the *treatment* and *control* groups can be estimated *in the common support region.*

[15] Non-parametric kernel methods were used to plot the propensity score distributions.

Table 8. Propensity Score Minima/Maxima and Observations Removed from Sample

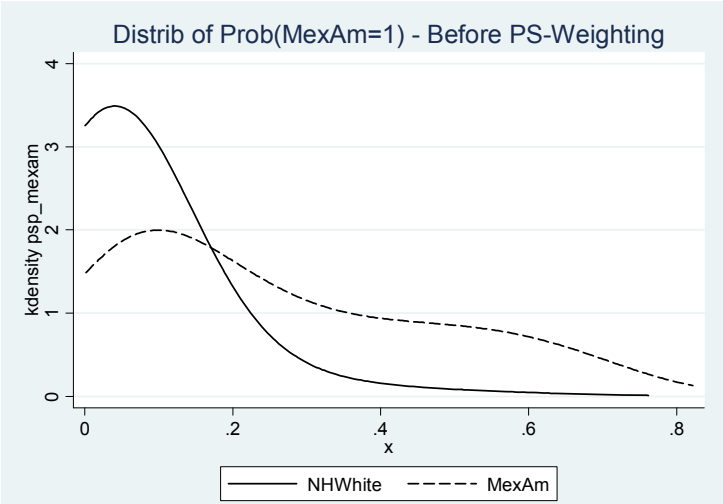
Comparison	Min/Max Control	Min/Max Treatment	Deleted Observations Control/Treatment		Total Obs. Remaining
All Mexicans vs. Whites	0.001/0.76	0.002/0.82	83	27	19,253
US-born Mexicans vs. Whites	0.001/0.27	0.002/0.30	37	2	15,834
Mexican immigrants vs. Whites	0.000/0.81	0.003/0.88	343	31	15,892
US-born Mex. vs. Immigrants	0.001/0.96	0.024/0.97	37	17	6,529

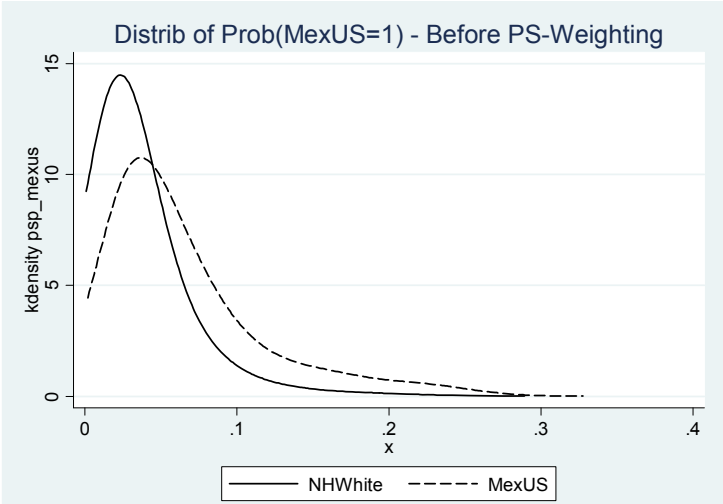
3.4.4 Matching Quality

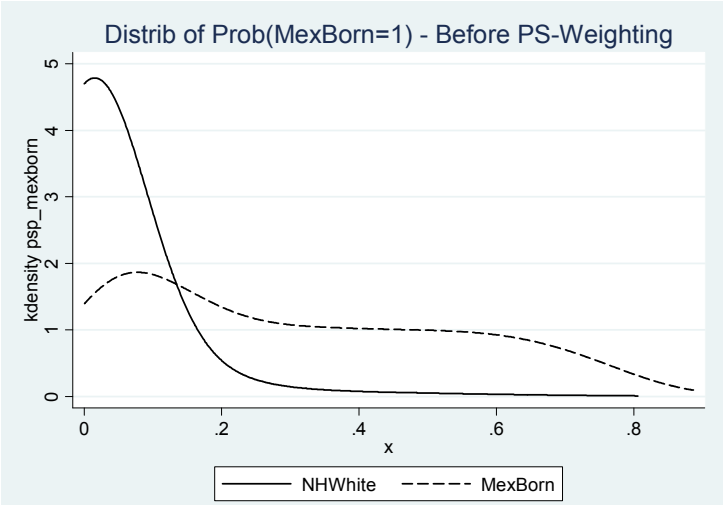
Table 9 reports the characteristics of the four groups after being balanced with the propensity score weights in equation (4). Clearly, weighting with the propensity scores produced samples with similar average values in all the variables for the treatment and comparison groups. Statistical tests found no significant differences in means between the treatment and control groups in any of the four comparisons.

A clearer representation of the balancing of the samples achieved by using the propensity score weights is depicted in Figure 3, which shows the t-statistics of the differences between the comparison groups in all covariates, both before and after weighting the samples with the propensity scores. The *Xs* represent t-statistic values of the mean difference between the comparison and treatment groups *before* PS-weighting, while black dots show the t-statistic values *after* PS-weighting. None of the t-statistic values after weighting is greater than 1.5, less than 3% are greater than 1.0, and 84% are lower than 0.5. Figure 4 provides yet another visualization of overall covariate balance. It shows the distribution of the propensity scores before and after weighting (the off-support observations are still shown in the left-hand side graphs, but omitted from those in the right-hand side). Overall, both figures show that the propensity score weights succeeded in balancing the samples of the comparison and treatment groups such that all covariates have similar distributions, except for the outcome variables (allostatic load and Framingham risk).

Figure 2. Common Support Assessment: Distributions of Propensity Scores Before Adjustments







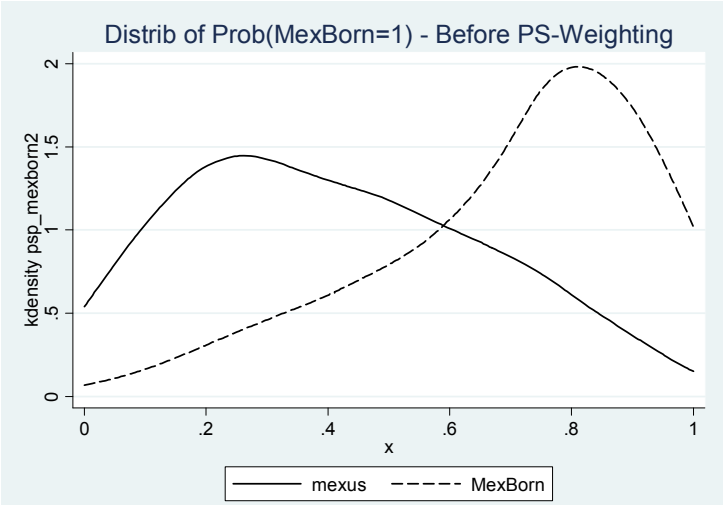


Table 9. Descriptive Statistics of Propensity Score-Weighted Samples—Estimated in the Common Support

	First Weighted Sample		Second Weighted Sample		Third Weighted Sample		Fourth Weighted Sample	
	Whites	**Mexicans**	**Whites**	**US Mexicans**	**Whites**	**Immigrants**	**US Mexicans**	**Immigrants**
N (unweighted)	12,695	6,558	12,741	3,093	12,435	3,457	3,058	3,471
Age	38	38	40	40	37	37	37	37
Female	46%	46%	50%	50%	43%	42%	43%	42%
Income/Pov. Ratio								
PIR < 1.0	30%	29%	20%	20%	37%	36%	38%	36%
1.0 ≤ PIR < 2.0	33%	34%	26%	26%	39%	40%	39%	39%
2.0 ≤ PIR < 3.0	16%	16%	18%	18%	15%	15%	13%	15%
3.0 ≤ PIR < 4.0	10%	10%	15%	15%	6%	6%	6%	6%
4.0 ≤ PIR < 5.0	5%	5%	8%	8%	2%	2%	2%	2%
PIR > 5.0	7%	7%	13%	13%	2%	2%	2%	2%
Education								
Less than HS	55%	55%	33%	33%	72%	71%	70%	71%
HS degree	24%	24%	34%	34%	16%	17%	17%	17%
College +	21%	21%	33%	33%	12%	12%	13%	12%
Insurance								
Private	44%	45%	60%	61%	32%	34%	33%	34%
Public	12%	12%	14%	15%	10%	10%	10%	10%
Uninsured	43%	43%	25%	24%	57%	56%	56%	56%
Type of work done the longest								
Never worked	6%	5%	3%	3%	9%	7%	7%	7%
White collar	8%	8%	13%	13%	5%	5%	5%	5%
Service	37%	36%	47%	47%	28%	28%	29%	28%
Blue collar	48%	50%	35%	35%	58%	60%	59%	60%
Marital Status								
Single	19%	19%	20%	20%	18%	19%	20%	19%
Widow/Divorced	12%	12%	15%	15%	9%	9%	11%	9%
Married	69%	69%	65%	65%	72%	72%	69%	72%

Figure 3. Covariate Balance Before and After Weighting with Propensity Scores

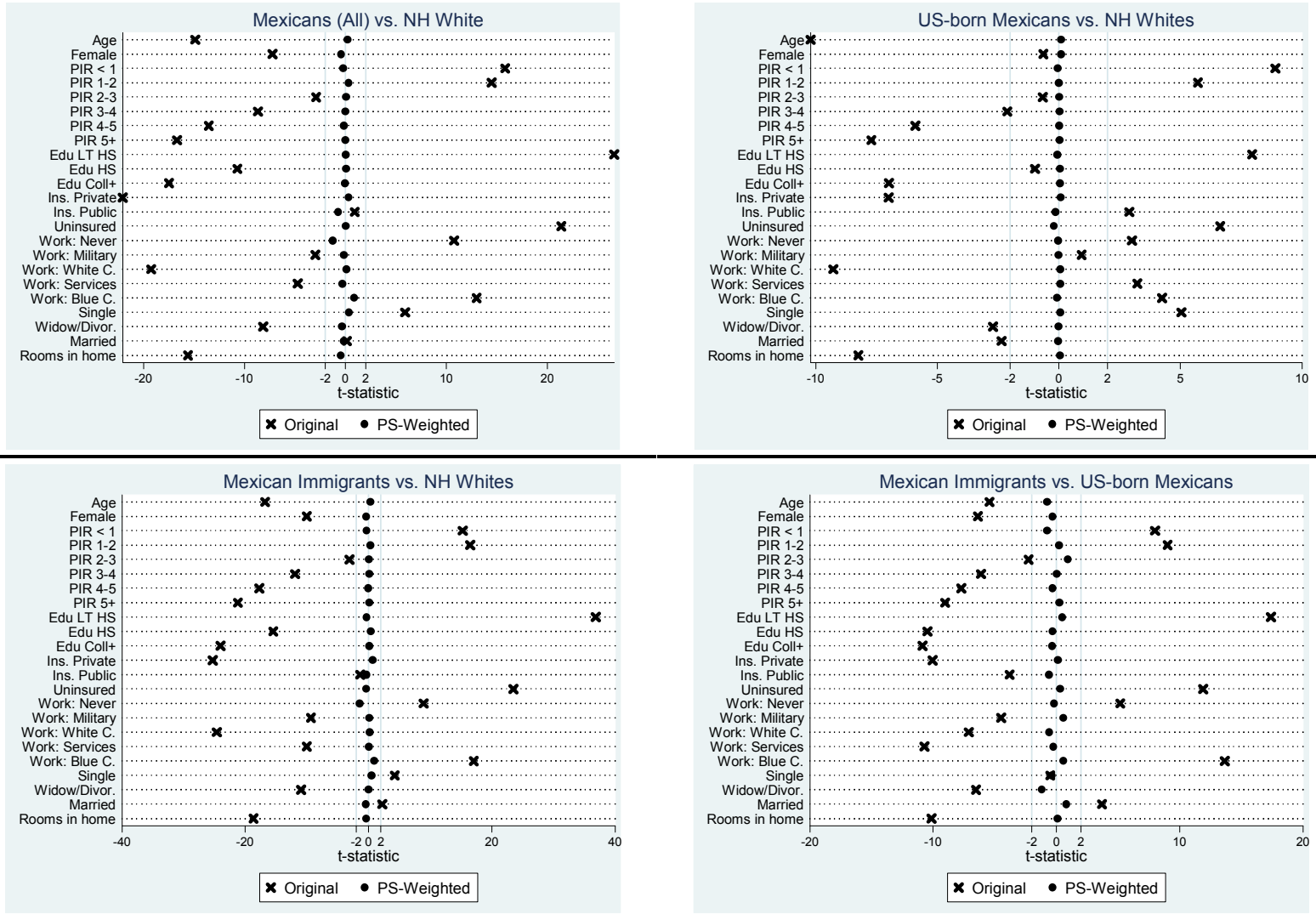
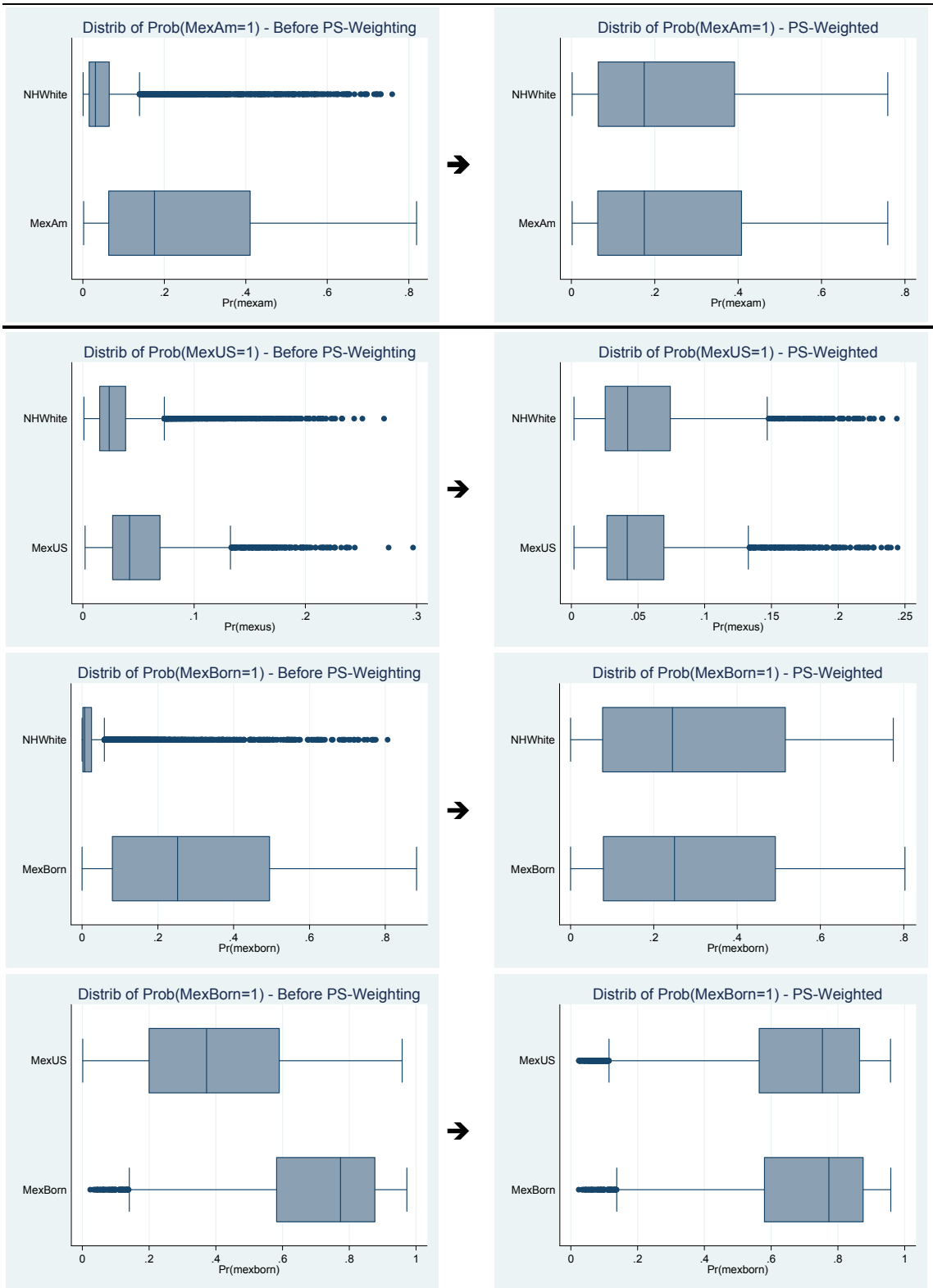


Figure 4. Boxplots of the Propensity Scores – Before and After PS-Weighting



3.4.5 Allostatic Load Model Results

The estimated differences in allostatic load between Whites, US-born Mexicans, and Mexican immigrants are presented in Table 10. Panels in the table represent separate comparisons (e.g., Mexicans vs. Whites or Mexican immigrants vs. US-born Mexicans) and rows within each panel represent models for different outcome variables: allostatic load, inflammatory, metabolic, or cardiovascular scores.

The first column in Table 10 shows the simple difference in average outcome values between ethnic groups with no adjustments. The second column shows the estimated coefficients of dummy variables for the ethnic group of interest (Mexicans, US-born Mexicans, or Mexican immigrants) in an ordinary least squares regression of allostatic load scores on this dummy variable and the covariates in Table 9. The third column gives the semi-parametric estimate of the difference in average outcomes between ethnic groups, estimated using the sample balanced with the PS weights and calculating the difference in average allostatic load scores between both groups. Finally, the last column shows the coefficients estimated with the *doubly robust estimator,* combining PS weighting with a parametric regression model.

Table 10. Ethnic Differences in Allostatic Load Scores

	Diff. in Means		OLS Model		PS-Weighted Diff. in Means		PS-Weighted OLS Model	
	COEFF	*SE*	*COEFF*	*SE*	*COEFF*	*BSE*	*COEFF*	*BSE*
All Mexicans vs. Non-Hisp. Whites								
Allostatic Load	**-0.115**	(0.044)	0.065	(0.033)	**0.054**	(0.027)	**0.050**	(0.026)
Inflammatory	-0.086	(0.046)	-0.015	(0.050)	-0.036	(0.031)	-0.036	(0.031)
Metabolic	0.013	(0.037)	**0.164**	(0.026)	**0.175**	(0.027)	**0.170**	(0.027)
Cardiovascular	**-0.177**	(0.033)	-0.019	(0.030)	-0.037	(0.029)	-0.039	(0.028)
US-Born Mexicans vs. Non-Hisp. Whites								
Allostatic Load	0.009	(0.055)	**0.171**	(0.040)	**0.162**	(0.024)	**0.165**	(0.024)
Inflammatory	0.061	(0.060)	**0.123**	(0.055)	**0.110**	(0.026)	**0.112**	(0.026)
Metabolic	0.022	(0.043)	**0.167**	(0.028)	**0.169**	(0.025)	**0.169**	(0.025)
Cardiovascular	-0.056	(0.042)	**0.080**	(0.035)	**0.076**	(0.027)	**0.076**	(0.027)
Mexican Immigrants vs. Non-Hisp. Whites								
Allostatic Load	**-0.202**	(0.043)	-0.014	(0.036)	-0.024	(0.040)	-0.035	(0.037)
Inflammatory	**-0.196**	(0.042)	**-0.117**	(0.056)	**-0.145**	(0.046)	**-0.148**	(0.046)
Metabolic	0.006	(0.042)	**0.162**	(0.034)	**0.189**	(0.042)	**0.174**	(0.041)
Cardiovascular	**-0.267**	(0.035)	**-0.092**	(0.036)	**-0.122**	(0.044)	**-0.124**	(0.041)
Mexican Immigrants vs. US-Born Mexicans								
Allostatic Load	**-0.211**	(0.050)	**-0.139**	(0.058)	**-0.181**	(0.042)	**-0.158**	(0.041)
Inflammatory	**-0.256**	(0.049)	**-0.187**	(0.084)	**-0.200**	(0.055)	**-0.192**	(0.052)
Metabolic	-0.016	(0.042)	0.078	(0.040)	0.026	(0.046)	0.041	(0.049)
Cardiovascular	**-0.211**	(0.046)	**-0.202**	(0.051)	**-0.216**	(0.047)	**-0.196**	(0.045)

NOTES: Each row represents a different dependent variable. The coefficients shown are for the dummy variable of the 'treatment' group (the first one mentioned in the heading of each panel). All models were adjusted for the variables shown in Table 6. Coefficients in **bold typeface** are significant at the 5 percent confidence level. Standard errors are in parenthesis; standard errors in the PS-weighted models were estimated using 200 bootstrap draws.

3.4.5.1 All Mexicans vs. Whites

A casual look at the difference in average allostatic load, inflammatory, and cardiovascular scores between Whites and Mexicans in the first column of Table 10 may lead one to conclude that there is evidence supporting the Hispanic Paradox for the entire population of Mexicans in the United States: Mexicans have lower values of three of the four scores (overall allostatic load, inflammatory, and cardiovascular), two of them statistically significant

and the third borderline significant, whereas for metabolic, although positive, the difference is not significant. Reaching this conclusion may be even more justifiable if one considers the differences in socioeconomic status evidenced in Table 6, which clearly show that Mexicans have a much lower socioeconomic status and lower access to health insurance than Whites. We might even guess that, after accounting for Mexicans' lower SES, their health advantage will become yet more evident.

However, another important difference between Mexicans and Whites is that the former are significantly younger, and younger individuals tend to have lower allostatic load scores. Thus, it is not clear in advance whether the differences in age or socioeconomic status will dominate the estimate of the disparities in allostatic load between Mexicans and Whites. The results of the OLS model (second column) are useful to answer this question. After controlling for the socioeconomic and demographic variables described above, two out of four coefficients are positive, one of them significant and the other borderline, while the other two negative coefficients are clearly not statistically different from zero. Thus, the OLS regressions indicate not only that Mexicans are not healthier than Whites, but also that they have worse health overall and in at least one allostatic load subsystem, since their total allostatic load score is 0.06 standard deviations—and their metabolic score 0.16 standard deviation—higher than that of Whites.

As explained above, OLS estimations are heavily dependent on the model's assumed functional form. If the model specification is incorrect, the results could be biased; this situation is of particular concern when the groups being compared differ substantially in their characteristics, which is the case in this study, because the parameters could be estimated by extrapolation outside the region of common support. Although Figure 4 suggests that support is probably not a cause for concern when comparing Whites and the entire

sample of Mexicans, there are still a few observations outside the region of common support, so I remove them before running the PS-weighted estimations.

The estimates of the semi-parametric and the doubly robust propensity score estimators are very similar, and they lead to conclusions similar to those of the OLS model: (1) Mexicans have allostatic load and metabolic scores higher than Whites (the former only slightly higher, but statistically significant), and (2) there are no significant differences in the inflammatory and cardiovascular scores of both groups. Regarding the differences between OLS and propensity score estimates, although the OLS estimate is 30 percent higher than the estimate of the doubly-robust model, this difference is not statistically significant.[16]

3.4.5.2 US-Born Mexicans vs. Whites

The picture becomes significantly clearer when Mexicans are divided according to their country of birth. For US-born Mexicans, all three approaches (OLS and the PS-weighted methods) provide coefficient estimates with the same sign and similar magnitude. In this comparison, the largest relative difference between the OLS and the doubly-robust model is in the inflammatory coefficient, which is 10% lower in the latter model. However, like in the models for all Mexicans, the differences between the OLS and propensity score models are not statistically significant.

The PS-weighted models indicate that US-born Mexicans have allostatic load scores that are higher than those of Whites by about 0.16 standard deviations. In addition, Mexicans have higher scores than Whites for all three subcomponents, with the difference ranging

[16] The coefficients estimated in the OLS model follow a t-student distribution, while the bootstrapped coefficients in the propensity score-weighted models follow a normal distribution. Since a t-student distribution is nearly identical to a normal distribution at sample sizes over 40 observations, I can compare the coefficients from both models and test for significance of the differences in their means assuming they are distributed normally.

from 0.08 standard deviations for cardiovascular to 0.17 standard deviations for metabolic. Therefore, not only there is no evidence of a Hispanic Paradox for US-born Mexicans, but we can safely conclude that they have worse health than Whites, at least when it is assessed using the biomarkers in the allostatic load index.

3.4.5.3 Mexican Immigrants vs. Whites

The allostatic load scores of Mexican immigrants and Whites are not significantly different from each other. However, this does not mean that both groups have similar biological risk profiles. There are significant differences in each of the three allostatic load subcomponents that cancel out when they are added together: Mexican immigrants have inflammatory and cardiovascular scores that are 0.15 and 0.13 standard deviations lower than those of Whites, providing evidence that supports the existence of the Hispanic Paradox for these two sets of biomarkers. However, the metabolic score is 0.18 standard deviations higher for Mexican immigrants than for Whites, which is consistent with the higher rates of obesity and diabetes observed in the former group.

Regarding the differences between OLS and propensity score methods, the estimates differ by a somewhat larger magnitude than in the previous two comparisons. For example, the coefficient estimates for the inflammatory and cardiovascular scores are larger (i.e., more negative) in the doubly robust model by about 0.03 standard deviations, representing relative differences between the OLS and doubly-robust estimator of 26 and 35 percent, respectively. These larger differences are probably due to the fact that Whites and Mexican immigrants are the two most dissimilar ethnic groups in the NHANES sample in terms of their socioeconomic and demographic characteristics, and required the largest adjustment to achieve common support. I should note that the coefficient differences between the OLS

and PS models, although larger than in other comparisons, are still not statistically significant.

3.4.5.4 Mexican Immigrants vs. US-Born Mexicans

All three estimators provide identical qualitative results in terms of the signs of the coefficient estimates. However, some magnitudes differ considerably, particularly between the OLS model and the semiparametric PS-weighted difference in means, which differ in their estimates of the allostatic load and metabolic coefficients by about 0.04 standard deviations. In addition, the OLS estimator in the metabolic model is positive and borderline significant, while the estimators using propensity scores, although positive, are clearly not statistically significant.

What can be safely concluded from this set of models is that Mexican immigrants are generally healthier than US-born Mexicans, since their overall allostatic load scores are lower (by 0.14-0.18 standard deviations) as well as their inflammatory (0.19-0.20 SDs) and cardiovascular (0.20-0.22 SDs) scores. There are no significant differences in metabolic score between these two groups.

3.4.6 Framingham Risk Score Results

Estimates of differences in 10-year risk of CHD—the Framingham risk score—between Whites, US-born Mexicans, and Mexican immigrants are presented in Table 11. Each row in this table represents a different comparison between two ethnic groups (e.g., Mexicans vs. Whites or Mexican immigrants vs. US-born Mexicans). The first column shows the simple difference in average outcome values between ethnic groups with no adjustments; the average 10-year risk of CHD is 5.1 percent for Whites, 3.6 percent for US-born Mexicans, and 2.7 percent for Mexican immigrants. Thus, before adjustments for covariate differences, Whites

have a risk of CHD that is 46% higher than that of US-born Mexicans, and 89% higher than that of Mexican immigrants.

The second column in Table 11 reports the semi-parametric estimator of the difference in average Framingham risk between ethnic groups, calculated using the PS weights . A more detailed discussion of each comparison is given below.

Table 11. Ethnic Differences in 10-year Risk of CHD in Framingham Risk Score Models, Propensity-Score Weighted Differences in Means

	Diff. in Means	PS-Weighted Difference in Means
All Mexicans vs. NH Whites	**-1.985** (0.155)	**-0.642** (0.114)
US-Born Mexicans vs. NH Whites	**-1.489** (0.180)	**-0.341** (0.099)
Mexican Immigrants vs. NH Whites	**-2.356** (0.184)	**-0.808** (0.179)
Mexican Immigrants vs. US-Born Mexicans	**-0.868** (0.214)	**-0.639** (0.226)

NOTES: The numbers shown represent the average difference in 10-year risk of coronary heart disease between the two groups in each row. Coefficients in **bold typeface** are significant at the 5 percent confidence level. Standard errors are in parentheses; standard errors in the PS-weighted models were estimated using 250 bootstrap draws.a

3.4.6.1 All Mexicans vs. Whites

Before any adjustments, Whites have a 10-year risk of CHD 65% higher than the pooled Mexican sample (5.1 vs. 3.1 risk percentage, for a difference of 1.99 percentage points shown in Table 11). Since the Framingham risk score includes age as a risk factor and Mexicans are on average younger than Whites, we expect this difference to be lower after adjusting the White sample so that it resembles the characteristics of the Mexican sample. Indeed, the average 10-year risk of CHD for Whites decreases significantly, from 5.1 to 3.7 percent, after adjusting the sample with the propensity score weights. Nevertheless, although smaller, the difference between Whites and Mexicans, 0.64 percentage points remains statistically significant.

3.4.6.2 US-Born Mexicans vs. Whites

In contrast to the allostatic load models, which found US-born Mexicans to have worse health in general than Whites, the propensity score-weighted comparison of Framingham risk between US-born Mexicans and Whites finds that the former have a 10-year risk of CHD lower than the latter: 3.59 percent vs. 3.93 percent, respectively, for an estimated difference of 0.34 percentage points. A number of factors may account for the differences between the AL and FRS comparison; they are discussed in Section 3.4.6.5 below.

3.4.6.3 Mexican Immigrants vs. Whites

Like in the allostatic load models, Mexican immigrants are found to be healthier than Whites when the Framingham risk score is used to assess health status. The semiparametric propensity score estimator indicates that Mexican immigrants have a 10-year risk of CHD 0.81 percentage points lower than Whites (2.74 percent vs. 3.55 percent).

3.4.6.4 Mexican Immigrants vs. US-Born Mexicans

The semiparametric estimator indicates that Mexican immigrants have an average 10-year risk of CHD lower than US-born Mexicans by 0.64 percentage points (2.73 percent vs. 3.37 percent). In order to avoid confusion, I should clarify that the PS-weighted difference in FRS means between Mexican immigrants and US-born Mexicans must not necessarily equal to the 'difference of the differences' between US-born Mexicans and Mexican immigrants with Whites for two reasons:[17] (1) in each case the White sample was weighted to resemble the characteristics of the respective 'treatment' group (i.e., when comparing US-

[17] For example, the PS-weighted difference between US-born Mexicans and Whites is 0.341, and the difference between Mexican immigrants is 0.808, so we would normally expect (if we were using a simple difference in means) the difference between Mexican immigrants and US-born Mexicans to be 0.808-0.341=0.467, but the difference reported in Table 11 is 0.639.

born Mexicans with Whites, the White sample was weighted to resemble the distribution of characteristics of the US-born Mexican sample, and similarly for the comparison of Mexican immigrants with Whites); and (2) in each comparison, the observations outside the region of common support were removed, and thus the same samples were not necessarily used across the different analyses in Table 11.

3.4.6.5 Analysis of Differences Between Framingham and Allostatic Load Estimations

The results of the allostatic load and Framingham score models (shown in Table 10 and Table 11, respectively) are not always consistent with each other. In particular, while the comparison of allostatic load scores between US-born Mexicans Whites indicates that the former have worse health than the latter, the comparison of Framingham risk scores between these groups results in a lower estimate of 10-year risk of CHD for US-born Mexicans than for Whites. Two explanations might account for these inconsistencies. First, unlike allostatic load, the Framingham scoring system (summarized in Table 4) takes into account not only biological markers of health status, but also includes three other risk factors: age, smoking, and hypertension treatment status. The statistical analyses I conduct in this study control for age, but not for smoking or hypertension treatment. Therefore, differences between Whites and Mexicans in either or both of these variables may result in inconsistent findings between analyses that use allostatic load as the outcome and similar analyses that use Framingham risk. Second, the allostatic load score is composed by eight biomarkers that are indicators of inflammatory, metabolic, and cardiovascular functioning, while the Framingham risk score includes only three of these indicators. Therefore, if the between-group differences in the five indicators included only in the allostatic load score have different patterns than the between-group differences in the three indicators present in both measures, we may find differences in the results obtained using each of these indices. Be-

low, I explore whether each of these two explanations accounts for the inconsistencies between the AL and FRS models.

As can be seen in Table 12, there are significant differences in smoking and hypertension treatment rates between Mexicans and Whites. Whites are significantly more likely to smoke (27 percent) than US-born Mexicans (23 percent) and Mexican immigrants (22 percent), and they are also more likely to receive treatment for hypertension (20, 14, and 8 percent, respectively). Thus the differences between the AL and FRS analyses may be due to the fact that, on average, US-born Mexicans smoke less and are less likely to receive treatment for hypertension than Whites.

Table 12. Rates of Smoking and Hypertension Treatment, Mexicans and Whites

	Current Smokers (%)	Receiving Treatment for Hypertension (%)
Non-Hispanic Whites	27%	20%
US-born Mexicans	23%	14%
Mexican immigrants	22%	8%

Source: NHANES 1988-1994 and 1999-2004.

One potential solution to address this incompatibility between the AL and FRS analyses would be to control for differences in smoking and hypertension treatment in the statistical analyses where allostatic load is the outcome measure. However, my main interest in this study is to explore ethnic differences in health after controlling for demographic and socioeconomic characteristics, not for health behaviors or use of healthcare services. A second alternative, which I choose, is to calculate '*reduced*' Framingham scores that exclude the points attributable to smoking and hypertension treatment, and conduct the statistical analyses with this 'reduced' score. If there are no differences between the results of the allostatic load models and those of the *reduced* Framingham risk score models, it is reasonable to conclude that the differences between the original analyses can be attributed to the inclusion of these two risk factors in the Framingham scoring system.

The results of analyses using the *reduced* Framingham score are shown in Table 13.[18] Clearly, these results differ significantly from those reported in Table 11: once the points attributable to smoking and hypertension treatment are removed from the Framingham score, the PS-weighted differences in FRS means between Whites, US-born Mexicans, and Mexican immigrants are no longer significant, in any of the four comparisons. The results shown below not only are different from those obtained in the original FRS models: they are also entirely different from the results obtained in the AL models as well. The allostatic load models indicate worse overall health for *U.S.-born* Mexicans when compared to Whites, and better overall health for Mexican immigrants when compared to Whites. Conversely, the *reduced* FRS models indicate that all three ethnic groups have similar values of the Framingham score once smoking and hypertension are removed from the scoring system.

Table 13. Ethnic Differences in *Reduced* Framingham Score, Propensity Score-Weighted Differences in Means

	PS-Weighted Difference in Means	
US-Born Mexicans vs. NH Whites	0.125	(0.091)
Mexican Immigrants vs. NH Whites	0.147	(0.189)
Mexican Immigrants vs. US-Born Mexicans	-0.190	(0.271)

NOTES: The numbers shown represent the average difference in *reduced* Framingham score between the two groups in each row. The *reduced* Framingham score excludes the points due to smoking and hypertension treatment from the original Framingham score. None of the coefficients are significant at the 5 percent confidence level. Standard errors are in parentheses and were estimated using 250 bootstrap draws.

As explained above, another potential explanation for inconsistencies between AL and FRS models is the inclusion of five biomarkers in the allostatic load score that are not considered in the Framingham risk scoring system. Tentative evidence for this explanation can be found in Table 14, which reports the *PS-weighted* average values of each biomarker in

[18] Since the mapping of total points to 10-year risk of CHD in the Framingham risk scoring system was designed only with the inclusion of both smoking and hypertension treatment status as risk factors, I cannot calculate the 10-year risk of CHD. Thus, in the analyses of this sub-section the outcome variable is the total point score.

the allostatic load index for the three ethnic groups. There are no significant differences between Whites and Mexican immigrants in the average values of the three biomarkers included in the *reduced* Framingham score (total cholesterol, HDL cholesterol, and systolic blood pressure). In addition, although there are differences between Whites and US-born Mexicans in the average values of HDL cholesterol and systolic blood pressure, these differences are in the opposite direction. That is, US-born Mexicans have *better* (higher) levels of HDL cholesterol, but *worse* (higher) systolic blood pressure. Furthermore, Table 14 indicates the existence of significant differences between the three groups in average values of CRP, serum albumin, glycosylated hemoglobin, resting heart rate (pulse), and diastolic blood pressure.

Table 14. Average Biomarker Values in NHANES Sample After Propensity Score-Weighting, Non-Hispanic Whites and Mexicans

	Non-Hispanic Whites		US-Born Mexicans		Mexican Immigrants	
C-reactive Protein	0.422	(0.017)	0.512	(0.038)	**0.398**	(0.011)
Serum Albumin*	4.308	(0.015)	4.319	(0.018)	**4.364**	(0.008)
Glycosylated Hemoglobin	**5.348**	(0.035)	5.505	(0.031)	5.511	(0.022)
Total Cholesterol	196.9	(1.516)	195.6	(1.902)	196.1	(0.927)
HDL Cholesterol*	47.45	(0.551)	**49.09**	(0.587)	47.64	(0.273)
Resting heart rate	75.45	(0.494)	74.20	(0.543)	**70.56**	(0.248)
Systolic Blood Pressure	**118.3**	(0.578)	121.7	(0.897)	**118.0**	(0.316)
Diastolic Blood Pressure	73.01	(0.422)	73.23	(0.664)	**70.41**	(0.249)

NOTES: Shaded rows represent biomarkers that are also included in the Framingham risk score. Propensity score weights are applied such that the distributions of the White and US-born Mexican samples resemble the Mexican immigrant sample. Bootstrapped standard errors are in parentheses. * Indicates biomarkers for which higher values represent better health status. **Bold** typeface indicates the group with the 'best' average value for each biomarker. If the differences between any group and the 'best' group are not statistically significant, both are in **bold** typeface. If there are no significant differences between any of the three groups, *no* coefficients are shown in **bold** typeface.

The means shown in Table 14 suggest that analyses that use a health measure that includes all eight biomarkers (e.g., allostatic load) could find significant health differences between the three groups, while analyses that use a health measure that includes only the

three shaded biomarkers (e.g., the *reduced* FRS) might find no differences between them, which is consistent with the results of the models I have estimated so far. Nevertheless, average biomarker values do not provide enough evidence to conclude that the *reduced* FRS models are consistent with the AL models, because the latter gives different weights to each biomarker and to each quintile within a biomarker. In order to test this explanation, I computed a *reduced* AL score; that is, an allostatic load score that includes only the three biomarkers present in the Framingham scoring system.

The results of analyses using the *reduced* AL score—after removing the biomarkers not present in the *reduced* Framingham score—are shown in Table 15. Like in the analyses with the full AL score, these estimates indicate a health disadvantage of US-born Mexicans when compared to Whites. In fact, the results in this table are no different from those using the full AL score: there are no significant health differences between Whites and Mexican immigrants, and Mexican immigrants have a health advantage over US-born Mexicans.

Table 15. Ethnic Differences in *Reduced* Allostatic Load Score, Propensity Score-Weighted Differences in Means

	PS-Weighted Difference in Means	
US-Born Mexicans vs. NH Whites	**0.187**	(0.045)
Mexican Immigrants vs. NH Whites	-0.023	(0.041)
Mexican Immigrants vs. US-Born Mexicans	**-0.210**	(0.030)

NOTES: The parameters shown represent the average difference in the *reduced* allostatic load score between the two groups in each row. The *reduced* AL score includes only the biomarkers present in the Framingham score: total cholesterol, HDL cholesterol, and systolic blood pressure. Parameters in **bold typeface** are significant at the 5 percent confidence level. Standard errors are in parentheses and were estimated using 250 bootstrap draws.

A summary of the analyses conducted in this Chapter is shown in Table 16. Although the analyses using *reduced* FRS and AL measures did not fully reconcile the differences when using the original scores, the picture regarding the health of Mexican immigrants and US-born Mexicans relative to Whites is not entirely unclear. The least definite result is the one regarding the comparison of US-born Mexicans with Whites: the analyses with the two

AL scores indicate a health advantage of the latter group when compared to the former, but the analysis that used the full Framingham score indicated the opposite, while the comparison that used the reduced Framingham score found no differences between these two groups. For the comparison of Mexican immigrants with Whites, three of the four analyses found no significant differences between them, while the analysis that used the full Framingham score found lower 10-year risk of CHD among Mexican immigrants than among Whites. Finally, three of the four analyses found a health advantage of Mexican immigrants over US-born Mexicans, only the comparison that used the reduced Framingham score found no significant differences between them.

Table 16. Summary of AL and FRS Propensity Score-Weighted Estimates of Differences in Health Status Between Mexicans and Whites

	AL PS-Weighted Difference in Means	**FRS PS-Weighted Difference in Means**	***Reduced* FRS PS-Weighted Difference in Means**	***Reduced* AL PS-Weighted Difference in Means**
US-Born Mexicans vs. NH Whites	Positive	Negative	N.S.	Positive
Mexican Immigrants vs. NH Whites	N.S.	Negative	N.S.	N.S.
Mexican Immigrants vs. US-Born Mexicans	Negative	Negative	N.S.	Negative

NOTES: N.S. indicates "non-significant" differences between groups. The AL columns refer to the models where the outcome is the total AL score.

3.4.7 Sensitivity Analyses

3.4.7.1 Alternative Allostatic Load Measure

The first column of coefficients in Table 17 shows the results of the sensitivity analyses described in Section 3.3.3.1, corresponding to the PS-weighted regressions using an alternative allostatic load definition (a count of the number of biomarkers with values beyond cut points of "high risk"). It is important to note that only coefficient signs, but not magnitudes, are comparable with other results in this table or in Table 10 because these results are ex-

pressed in different units (*number of biomarkers at high risk levels*) from the units in previous analyses (*standard deviations*).

Clearly, the most significant difference between these results and those reported previously is the sign of the Mexican dummy coefficient in the first model (which compares allostatic load scores of *all* Mexicans with those of Whites). This coefficient is positive and *significant* in the PS-weighted regression in Table 10, but negative and *significant* in Table 17. A closer examination of the remaining coefficients in the same column of Table 17 reveals the reason for this discrepancy: in the models that use the traditional AL definition no significant differences are found in *metabolic* scores between Mexican Americans and Whites, or between Mexican immigrants and Whites, while the results using my allostatic load definition found higher metabolic scores for Mexicans than for Whites.

Table 17. Sensitivity Analyses: Coefficient Estimates under Alternative Models

	PS-Weighted (alternative AL definition)		PS-Weighted (no income/poverty)		PS-Weighted (no insurance)	
	COEFF	*BSE*	*COEFF*	*BSE*	*COEFF*	*BSE*
All Mexicans vs. Non-Hisp. Whites						
Allostatic Load	**-0.109**	(0.042)	**0.079**	(0.026)	0.046	(0.024)
Inflammatory	-0.007	(0.020)	-0.004	(0.031)	-0.039	(0.028)
Metabolic	0.015	(0.023)	**0.200**	(0.027)	**0.161**	(0.026)
Cardiovascular	**-0.112**	(0.024)	-0.034	(0.029)	-0.033	(0.027)
US-Born Mexicans vs. Non-Hisp. Whites						
Allostatic Load	**0.138**	(0.041)	**0.174**	(0.024)	**0.165**	(0.024)
Inflammatory	**0.085**	(0.019)	**0.121**	(0.027)	**0.110**	(0.026)
Metabolic	0.024	(0.022)	**0.178**	(0.025)	**0.167**	(0.024)
Cardiovascular	0.030	(0.024)	**0.078**	(0.027)	**0.078**	(0.027)
Mexican Immigrants vs. Non-Hisp. Whites						
Allostatic Load	**-0.282**	(0.060)	0.015	(0.038)	-0.042	(0.035)
Inflammatory	**-0.078**	(0.028)	**-0.092**	(0.045)	**-0.152**	(0.042)
Metabolic	0.020	(0.032)	**0.236**	(0.041)	**0.164**	(0.038)
Cardiovascular	**-0.215**	(0.034)	**-0.125**	(0.044)	**-0.117**	(0.038)
Mexican Immigrants vs. US-Born Mexicans						
Allostatic Load	**-0.349**	(0.072)	**-0.157**	(0.041)	**-0.147**	(0.037)
Inflammatory	**-0.152**	(0.031)	**-0.192**	(0.052)	**-0.191**	(0.046)
Metabolic	-0.009	(0.048)	0.034	(0.048)	0.043	(0.042)
Cardiovascular	**-0.202**	(0.038)	**-0.195**	(0.044)	**-0.177**	(0.038)

NOTES: The coefficients in the second column are not comparable to those in the rest of this table, or to those in Table 10 because the units in this AL definition are different. Each row represents a different dependent variable. All models were adjusted for the variables shown in Table 6, except for those indicated in the first rows for columns 3 and 4. The coefficients shown are for the dummy variable of the 'treatment' group (the first one mentioned in the heading of each panel). Coefficients in **bold typeface** are significant at the 5 percent confidence level. Standard errors are in parenthesis and were estimated using 250 bootstrap draws.

Although these analyses do not allow me to directly examine the reason for this discrepancy, a first potential explanation is that Mexicans have higher (adjusted) values of the biomarkers that compose the metabolic score (hemoglobin, total and HDL cholesterol), but these differences are not captured by the traditional allostatic load measure, which only considers biomarker values that are in the highest-risk quartiles. These differences are, on the other hand, measured by my allostatic load definition, which captures not only high-risk

values, but also any deviations—positive or negative—from the third quintile. If this were the case, I argue that my allostatic load measure produces better estimations of the differences in metabolic scores between Mexicans and Whites. For example, using my allostatic load definition, a person with a hemoglobin value in the first quintile of the sample's distribution receives a lower metabolic score than a subject with a value in the third quintile, but both of them would receive the same score using the traditional allostatic load definition. Since the coefficient for the first hemoglobin quintile is significant in Table 5, I consider it more appropriate to assign a lower metabolic score to the first individual, as my allostatic load measure does.

Consider now a second alternative explanation. Say, for example, that Mexicans have higher average (adjusted) values of hemoglobin than Whites, lower average values of total cholesterol, and not significantly different values of HDL cholesterol. In the traditional metabolic score these differences are likely to cancel each other out and there would be no significant metabolic differences between Mexicans and Whites. However, these differences do not necessarily cancel out in the metabolic score used in my previous analyses because hemoglobin values receive higher weights than total cholesterol due to its stronger association with mortality (see Table 5). Again, I think this makes the case for trusting the estimations from my allostatic load definition over the traditional definition, because the latter does not account for the relative importance of biomarkers in explaining mortality.

3.4.7.2 Potentially Endogenous Covariates

The last two coefficient columns in Table 17 show the results of PS-weighted regressions where either income/poverty ratio or insurance status were excluded from the mod-

els.[19] Although coefficient magnitudes differ between Table 17 and the last coefficients column in Table 10, the differences are generally small. Most importantly, the statistical significance patterns are identical in all three sets of regressions, with the only exception being the *treatment* coefficient in the allostatic load model for *all* Mexicans vs. Whites; this coefficient is positive and statistically significant in the model that includes all variables and in the model that excludes PIR, but is not statistically significant in the model that excludes insurance status.

Nevertheless, I consider this a minor issue because (a) the coefficient is in fact borderline significant (p-value=0.06); (b) its magnitude is in fact almost identical to that of the original model (0.048 vs. 0.045); and (c) my main focus are the regressions where I divide Mexicans in US-born and immigrants, and in those models the significance patterns are identical and the coefficient magnitudes are very similar. Therefore, I conclude that the relevant estimations from my original models are robust to the exclusion of potentially endogenous variables.

3.5 Conclusion

In this study, I sought to corroborate the existence of a health advantage of individuals of Mexican ethnicity over non-Hispanic Whites. The results reported above indicate that there is *not* a general health advantage of Mexicans over Whites. Rather, the health advantage that has been reported in numerous previous studies is apparently enjoyed only by Mexican immigrants, and not in all the health indicators used in this study. Mexican Americans (i.e., US-born) were found to be disadvantaged not only in the overall allostatic load score, but also in each of its three subcomponents, and in a reduced version that included

[19] These variables were excluded both from the propensity score and allostatic load models.

only three biomarkers, which calls into question the existence of an overall Hispanic Paradox. These results are not only robust to the use of alternative measures of allostatic load and to the exclusion of potentially endogenous covariates, but are also consistent with those of Crimmins et al (2007), who found similar patterns of biological risk differences between Mexican Americans, Mexican immigrants, and Whites, using (a) a different methodology, (b) a different operationalization of allostatic load, and (c) a sample of individuals of age 40 and older.

Nonetheless , the allostatic load results are not fully supported by the analyses using the Framingham risk score. In those analyses, not only Mexican immigrants, but also US-born Mexicans were found to have a lower 10-year risk of CHD when compared to Whites. When the points attributable to smoking and hypertension treatment are excluded from the FRS measure, the propensity score-weighted analyses indicate that there are no significant health differences between Whites, U.S.-born Mexicans, and Mexican immigrants. When I reduce the allostatic load score by keeping only the biomarkers present in the Framingham scoring system, the results of the analyses using both measures are still not reconciled. In fact, the results using the *reduced* AL measure are qualitatively identical to the results using the *full* AL measure.

Overall, I cannot categorically conclude that US-born Mexicans are less healthy than Whites, but I believe there is significant evidence to suggest this conclusion. The average values of each biomarker, shown in Table 14, suggest that US-born Mexicans have worse levels than Whites of C-reactive protein, glycosylated hemoglobin, and systolic blood pressure. According to the coefficients in Table 5, systolic blood pressure and glycosylated hemoglobin are the two biomarkers most strongly associated with mortality, and thus it is difficult to argue in favor of the Hispanic Paradox for US-born Mexicans.

Focusing at the moment on the results using allostatic load as the indicator of health status, it is clear that studies of Hispanic health need to focus on why health advantages are enjoyed by immigrants but not by their descendents. Moreover, future research should also try to determine why Mexicans (both US-born and immigrants) are at a disadvantage in biomarkers associated with the metabolic system, a result that has been found by other authors (e.g., Park et al. 2003). As Markides and Eschbach (2005) discuss, a perception that Hispanics enjoy health advantages over other populations may result in public policies that ignore disadvantages that in fact exist; therefore it is important to acknowledge existing disadvantages and promote their awareness among policymakers and other stakeholders.

I conclude this chapter with a remark on the use of propensity score methodologies in analyses of health disparities. Although the qualitative results of this study do not change when traditional regression analysis is used, I found that the quantitative differences between the regression and propensity score methodologies were largest for the comparisons of health status between Mexican immigrants and non-Hispanic Whites. This is not surprising since the largest socioeconomic differences are between these groups, and it underscores the importance of assessing the potential lack of common support to avoid the first source of bias discussed in Heckman et al. (1998). This is particularly relevant in the study of health disparities, where comparisons are usually made between groups with substantial differences in their distributions of observed characteristics. Propensity score methods have rarely been used in this literature, probably at least in part because of lack of familiarity with them, but also due to misunderstandings about their applicability outside the realm of program evaluation. Even in those cases where researchers prefer to use regression analyses, propensity scores can be useful to assess the lack of overlap, and eliminate observations outside the common support area.

Chapter 4.

Changes in Immigrant Health with Length of Residence in the United States: A Semiparametric Analysis

4.1 Introduction

In Chapter 2, I described two important results found in recent studies of immigrant health: (a) the association between socioeconomic status and health appears to be weaker among Hispanic immigrants than among non-Hispanic Whites; and (b) the health status of immigrants deteriorates faster than that of US natives as their length of residence in the US increases. In this chapter, I discuss how these two phenomena might be related to each other, and I assess the evidence supporting four hypotheses that could explain them.

4.1.1 Hypotheses of Immigrant Health-SES Gradient

Although the finding of a weak Health-SES gradient among immigrants is relatively recent, researchers have already offered some theories to explain this result. Goldman et al. (2006) and Kimbro et al. (2008) describe three potential explanations, two of them migration-related. The first is that Health-SES gradients in migrant-sending countries are weak or inverted relative to those in the US. These authors have noted that certain health behaviors like alcohol drinking and smoking are more prevalent among Mexicans of higher SES levels, which may result in a weaker or even positive gradient. If that were the case, Mexican immigrants in the United States could also exhibit these gradients and transmit them to subsequent generations.

A second potential explanation is the *healthy migrant effect* (see Section 2.3). In particular, if this effect exists and low-SES emigrants are more likely to be positively selected on health status than high-SES emigrants, a weak Health-SES gradient would be observed

among immigrants in the US, even if the Health-SES gradients in Mexico and the US are similar. Finally, a third hypothesis offered by Goldman and colleagues is related to the *acculturation effects* discussed in Section 4.1.2 below: Hispanics with high SES levels are likely to be more assimilated into US culture, either because they have lived longer in the US or because they belong to the second or higher generation. Since the public health literature suggests that higher acculturation is associated with worse health behaviors, low-SES Hispanics would exhibit better-than-expected health status when compared to high-SES Hispanics. It is worth noting that these authors consider the negative health consequences associated with cumulative stress as part of this *acculturation hypothesis*; below, I classify this effect as part of a separate hypothesis related to health trajectories over the life course.

Zsembik and Fennell (2005) offer a single explanation that is essentially a combination of those described in the previous paragraph: the health of Mexicans is first determined by the positive health selectivity of migration, but acculturation moderates this health advantage, such that worse health is observed among individuals with higher levels of SES and acculturation. Similarly, Acevedo-Garcia et al. (2007) discuss three potential explanations. Two of them match the first two hypothesis discussed by Goldman et al (2006). The third is related to social and cultural factors, but does not rely on *acculturation*; rather, the authors hypothesize that protective social and cultural factors are present across all SES levels among immigrants, thus attenuating the Health-SES gradient.

An additional hypothesis consistent with a weak Health-SES gradient is related to imperfections in the labor market for immigrants. According to the theory of health capital (Grossman 1972), health is an important component of an individual's human capital, such that characteristics commonly associated with higher socioeconomic status—e.g., energy and ambition—are also found among healthy people, which then leads to a positive correlation between health and SES. However, if these attributes among immigrants are imper-

fectly rewarded in the U.S. labor market (perhaps due to illegal immigration, language proficiency, or lack of familiarity with the U.S. institutional setting), it may happen that healthy and skilled individuals, who would have attained a relatively high SES in their country of origin, attain lower-than-expected SES levels in the U.S. labor market.

4.1.2 Hypotheses of Immigrant Health Deterioration

As discussed in Chapter 2, several studies have found a faster decline in the health of immigrants with increases in their length of residence in the United States (e.g., Stephen et al. 1994; Cho et al. 2004; Zsembik and Fennell 2005; Uretsky and Mathiesen 2007). Perhaps the most commonly offered explanation for this result is that immigrant health deteriorates faster with acculturation into US culture, possibly as a result of an adoption of negative health habits such as alcohol drinking, tobacco smoking, or drug use (Vega and Amaro 1994; Escarce, Morales, and Rumbaut 2006; Diez Roux et al. 2005). This hypothesis is consistent with several studies that have found worse health behaviors among immigrants as their time of residence in the US increased (Abraido-Lanza, Chao, and Florez 2005; Dubowitz et al. 2007).

A potentially important limitation in many of these studies is the assumption that length of residence in the US is a reliable proxy for acculturation. Although acculturation is more likely to increase with duration of residence in the US, the latter does not necessarily imply higher acculturation. Acculturation is a complex and multidimensional construct, and it is not difficult to imagine situations in which some Hispanics with, say, 20 years of residence in the US would be less acculturated than others who immigrated more recently. Lara et al. (2005) describe different measures and scales commonly used as proxies for acculturation. Although these scales often contain several components, language is the most commonly included construct, and psychometric studies have shown that it explains most of the varia-

tion in multidimensional acculturation scales. When language has been used as a proxy for acculturation in studies of acculturation and health, results have been mixed: some have supported the acculturation hypothesis (Finch, Frank, and Vega 2004), while others did not find a significant association between language use and health outcomes (Zsembik and Fennell 2005).

Furthermore, several other hypothesis are consistent with accelerated health deterioration with increases in length of US residence, independently of acculturation. First, the *cohort effects hypothesis* states that recent immigrant cohorts might be healthier than earlier immigrant cohorts, at the time of their arrival to the United States (Stephen et al. 1994). In this case, if recent cohorts continue being healthier than earlier cohorts as their length of residence increases, we would observe better average health outcomes among recent immigrants than among immigrants who arrived a longer time ago, independently of the acculturation levels of either group.

Second, the *life-course hypothesis* departs from the fact that migration can be a difficult and stressful process, since it often involves separation from family and friends, adaptation to a different culture, and going through hardships during the migration trip and upon arrival (Jasso et al. 2005). These difficulties may in fact be related to the health selectivity of migration, since the process may require physical fitness and other individual traits associated with better health (CONAPO 2008). This hypothesis states that the cumulative effect of the adversities associated with migration might cause faster declines in health status among immigrants than among the native US population, independently of acculturation. In fact, under this *life-course hypothesis* (Hertzman 2004), higher acculturation could be beneficial because it might reduce the negative effects of adaptation and diminish the cultural barriers of access to healthcare.

Finally, a hypothesis that has been rarely mentioned in the literature is the *regression to the mean hypothesis:* the decline in immigrant health over time may simply be a result of a process of "regression to the mean," in which emigrants self-select based on their advantageous health status at the time of migration. Since migrant self-selection is based only on their observable health status but not on their underlying biological profiles, the health status of immigrants will tend over time to return to the average levels in their country of origin (Chiswick, Lee, and Miller 2008; Jasso et al. 2004). Like under the previous two hypotheses, this process would be independent of acculturation.

In this chapter, I seek to provide evidence to better understand the health selectivity and health trajectories of Mexican immigrants. This work contributes to the literature on immigrant health by exploring the evidence supporting several of the hypotheses described above. Unlike in the previous chapter, where the *treatment* of interest was a binary variable indicating membership in a specific ethnic group, in this chapter I focus on the nature of the *Health-Age* and *Health-SES* relationships. In order to avoid biases that may result from incorrect parametric specifications, I use semiparametric methods to explore these relationships.

First, I compare the *Health-Age* and *Health-SES* trajectories of immigrants to those of Whites. In particular, I am interested in evidence regarding a weaker *Health-SES* gradient among immigrants than among Whites, and I test a number of parametric specifications for these trajectories. Second, I *indirectly* test the health selectivity of migrants by comparing the health of *recent* Mexican immigrants with that of Whites. Third, I test the effect of length of US residence on immigrant health by comparing the health trajectories of *recent* immigrants with those of *earlier* immigrants. Fourth, I assess the existence of health differences between immigrant cohorts by comparing the health of a cohort of recent immigrants in NHANES-III with that of a cohort of recent immigrants in the 2001-2004 NHANES. Finally, I

seek evidence of an effect of acculturation on immigrant health, independent of length of US residence, by comparing the health of immigrants who have been in the US for at least ten years and who speak Spanish at home with the health of immigrants with a similar length of US residence but who speak English at home. Overall, these analyses will allow me to assess the health selectivity of migration, and the evidence supporting the hypothesis that can potentially explain the patterns previously found in the literature on immigrant health.

4.2 Data and Methodology

4.2.1 Data

I use the 1988-1994 and 1999-2004 NHANES data and allostatic load measure described in Chapter 3. I limit the sample to Mexican immigrants and US-born non-Hispanic Whites. Since one of the focal points of this chapter is the relationship between allostatic load and income/poverty ratio (PIR) and the semiparametric analyses require continuous measures, I remove from the original data the observations that are missing the value of the unimputed PIR variable. The *unweighted* sample sizes are shown in Table 18. The age and income variables are categorized in the table to show their distribution and sample sizes, but they are continuous in all the analyses in this chapter. The White sample has a relatively large number of observations at all income and age levels, but the Mexican immigrant sample has relatively few observations at PIR levels above three, and for the subsamples of English speakers. Therefore, we can expect the standard errors of the semiparametric analyses to be high at the right tails when the variable in the *x-axis* is age, and in the analyses that are stratified by language spoken at home.

Table 18. Sample Sizes of Mexican Immigrant and White Samples in Chapter 4

Variable	Mexican Immigrants	Non-Hispanic Whites
N	3,112	11,372
Income/Poverty Ratio		
0 - 1	1,215	1,105
1 - 2	1,211	2,541
2 - 3	411	2,216
3 - 4	151	1,819
4 - 5	62	1,283
5 or more	62	2,408
Age		
20-29	863	1,449
30-39	707	1,743
40-49	605	1,649
50-59	238	1,604
60-69	388	1,658
70 or older	311	3,219
Years in the US		
0- 4	550	-
5-14	947	-
15 or more	1,399	-
Language spoken at home		
Spanish only	2,580	-
English only	167	-
English and Spanish	361	-

4.2.2 Methods

All three types of analyses used in the previous chapter—ordinary least squares, semi-parametric propensity score weighting, and doubly-robust propensity score weighting—have at least one parametric component: the OLS model is a fully parametric estimation, where a specific functional form is assumed for the relationship between the outcome variable and the regressors; the two-step PS-weighting method has a parametric component in the first step, where the propensity scores are computed, and a nonparametric component in the second, where the mean outcomes are compared; and the doubly-robust estimator consists of two parametric steps, first to estimate the propensity scores and then to estimate the differences between the groups. However, most of the variables in these models are *inherently* categorical (e.g., *sex* has two categories, *education* has three categories, etc.)

and thus no parametric assumptions are needed regarding the association between them and allostatic load.

On the other hand, two variables in these analyses are not *inherently* categorical: age and income/poverty ratio (PIR). In Chapter 3, in order to use these variables in the allostatic load models, I entered age as a polynomial with linear, quadratic, and cubic terms (age, age^2, and age^3), which is common in health studies that use regression and include age in the right-hand side of the models. In addition, in those analyses I divided PIR into six categories in order to be able to impute income groups for observations missing a PIR value, almost nine percent of the sample. Since these two variables—*age* and *PIR*—are the focus of this chapter, I pay considerably more attention to the nature of their relationship with allostatic load, and I do not use observations for which a PIR category was imputed in the analyses of Chapter 3.

In order to explore the relationships between allostatic load and age, and allostatic load and PIR, I use the semiparametric method of differencing the partial linear model. This method can be used for the consistent estimation of a nonparametric effect and to conduct specification tests of parametric functional forms for the same effect (Yatchew 1988). The partial linear model is a regression specification that is useful when the researcher is confident about the functional form of some parts of a regression model, but the form of other parts is uncertain (Lokshin 2006). This model consists of a parametric and a nonparametric component:

$$y = z\beta + f\left(x\right) + \varepsilon \tag{9}$$

No assumptions are needed of the function $f\left(\bullet\right)$, except that it is smooth. Rice (1984) and Yatchew (1988) showed that the nonparametric effect can be removed from this equation by implementing a *differencing* algorithm that subtracts the values of one observation

from those of other nearby observations. In this procedure, the sample is sorted according to the values of the variable x such that $x_1 \leq x_2 \leq \ldots \leq x_n$, and each observation is subtracted from the next one, which yields:

$$y_i - y_{i-1} = (z_i - z_{i-1})\beta + \left[f(x_i) - f(x_{i-1}) \right] + \varepsilon_i - \varepsilon_{i-1}.$$

It can be shown that this differencing procedure removes not only the direct nonparametric effect of *x* on y, but also any indirect effect of *x* on *y* thru *z*. Furthermore, the estimation of an ordinary least squares regression on the differenced data

$$\tilde{y}_j = \tilde{z}_j \beta_{diff} + \tilde{\varepsilon}_i, \tag{10}$$

where $\tilde{y}_j = y_i - y_{i-1}$, $\tilde{z}_j = z_i - z_{i-1}$, and $\tilde{\varepsilon}_j = \varepsilon_i - \varepsilon_{i-1}$ produces $\hat{\beta}_{diff}$, a consistent estimator of β. As discussed in detail in Yatchew (2003, Ch. 4), a more complex procedure with higher-order differences improves the efficiency of this estimator. Furthermore, the residuals from equation (10) provide information on $f(x)$ that can be combined with nonparametric smoothing techniques—such as *lowess* or locally weighted polynomials—to approximate f's shape. Because the $\hat{\beta}_{diff}$ coefficients estimated in (10) converge to the true coefficients at a fast rate, these nonparametric estimates of f are consistent and the construction of confidence intervals for f is valid (Lokshin 2006).

Michael Lokshin's *plreg* command can be used in Stata to estimate the partial linear model and implement the differencing algorithm. There are two advantages to using this method over other nonparametric techniques: (1) it allows the researcher to control for the effect of other variables (e.g., gender or education) on allostatic load while the nonparametric effects are estimated; and (2) specification tests can be conducted to compare fully parametric models with the model estimated by *plreg*, which allows one to choose a paramet-

ric specification suggested by the graphical approximation of f, and conduct significance tests on it (Yatchew 1999). A disadvantage of *plreg* is that it does not allow for the use of weights, and thus the samples cannot be balanced with the propensity score weights.[20]

4.2.3 Estimation of AL-Age and AL-SES Relationships by Differencing

In order to explore how allostatic load changes with age and socioeconomic status—using PIR as a proxy for the latter—and whether there are any differences in these relationships between Whites and Mexican immigrants, I follow the steps described below.

First, I estimate the partial linear models shown below, separately for Whites and immigrants:[21]

$$\begin{aligned} AL &= z\beta + f\left(age\right) + \varepsilon_a \\ AL &= z\gamma + g\left(pir\right) + \mu_p \,, \end{aligned} \tag{11}$$

where *z* is a matrix of the socioeconomic characteristics; β and γ are vectors of coefficients; and f and g are smooth functions of *age* and *PIR*, respectively. I perform differencing on these models in order to remove the nonparametric effects, and estimate the remaining linear models using ordinary least squares:

$$\begin{aligned} \left(AL_i - AL_{i-1}\right) &= \left(z_i - z_{i-1}\right)\beta + \left(\varepsilon_1 - \varepsilon_{i-1}\right) \\ \left(AL_i - AL_{i-1}\right) &= \left(z_i - z_{i-1}\right)\gamma + \left(\mu_1 - \mu_{i-1}\right), \end{aligned}$$

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[20] An alternative to differencing the partial linear model is to conduct nonparametric smoothing of the age and PIR variables using Stata's *lpoly* command, which accepts weights and thus would allow me to balance the samples with the PS weights. The results reported below are similar when using this method.

[21] Ideally, one would prefer to estimate a single model with a joint function for *age* and *PIR*, $h\left(age, pir\right)$. Unfortunately, *plreg* does not allow for this joint analysis, so separate functions must be estimated.

The residuals of these regressions provide information on f and g while controlling for the linear effects of z, so I use nonparametric techniques to obtain graphical approximations of their shapes. Next, I estimate various fully parametric models where *age* and *PIR* have different functional forms (e.g., linear, quadratic), including those suggested by the graphs produced in the previous step. I conduct the specification tests described in Yatchew (1999) to assess whether these parametric specifications adequately capture the *AL-age* and *AL-SES* relationships for both Whites and Mexican immigrants. These specification tests are important because they will indicate whether parametric models that use one of these functional forms (e.g., quadratic or logarithmic) may be biased.

4.2.4 Examining Hypotheses of Causes of Immigrant Health Patterns Over Duration of Residence in the United States

To test the hypothesis that health varies with immigrants' length of residence in the United States, an obvious first alternative is to estimate a regression model with time since immigration—usually measured in years—as one of the covariates. A major problem with this approach, however, is that *age* and *years since immigration* are likely to be highly correlated, which may cause multicollinearity in a regression model, especially since Mexican immigrants tend to arrive to the US at similar ages.[22] Although a viable alternative is to categorize the variable "years in the US" and enter the categories as dummy variables in a parametric model, I am less interested in estimating a parametric effect for "years in the US" and more interested in assessing whether the *AL-age* and *AL-SES* relationships vary across different levels of that variable. A good option in this situation is to stratify the sample, di-

[22] Nearly 70 percent of *recent* Mexican immigrants in NHANES are of age 30 or younger. This accentuates the collinearity problem because immigrants arrive at similar ages and thus the years of immigration variable moves nearly parallel to the age variable.

viding it into groups according to their length of residence in the US. An important benefit of analyses that are stratified by length of US residence is that it allows to at least partially control for the health-age selectivity of migration since, for example, a 60-year-old recent immigrant is likely to be different from a 60-year-old person who immigrated to the United States 20 years ago (Jasso et al. 2004).

The exact number of years since immigration is available for NHANES-III observations, but the 1999-2004 NHANES data contains only a categorical variable with nine different ranges of length of US residence. The categories of this variable are shown in Table 19 (the table shows the frequencies for observations that are not missing a value of allostatic load; the distribution is very similar for the entire sample of Mexican immigrants.) Based on the sample sizes, and seeking to create categories that are meaningful in terms of length of stay, I stratify the sample into the following groups: (a) less than 5 years in the US, (b) between 5 and 15 years in the US, and (c) 15 years or more in the US.

Table 19. Frequencies of Length of Time in the United States—Mexican Immigrants

Category	Number of Observations	Weighted Percentage	Cumulative Percentage
Less than 1 year	99	4.5%	
1 year – less than 5 years	451	18.5%	23.0%
5 years – less than 10 years	484	19.8%	42.8%
10 years – less than 15 years	463	17.2%	60.0%
15 years – less than 20 years	341	12.6%	72.6%
20 years – less than 30 years	505	17.5%	90.1%
30 years – less than 40 years	272	5.9%	96.0%
40 years – less than 50 years	175	2.7%	98.7%
50 years or more	106	1.3%	100.0%

I conduct stratified analyses by "years since immigration" subgroups. In these analyses, I use the same methodology of differencing the partial linear model, described in Section 4.2.3. That is, within each immigration group, I difference out the nonparametric effects of *age* and *PIR* on allostatic load, estimate an ordinary least squares regression on the differ-

enced data, and then I conduct a nonparametric regression smoothing on the residuals of this regression to obtain a graphical estimation of the shape of the nonparametric functions $AL = f(age)$ and $AL = g(pir)$. In these analyses, described in more detail below, I seek to assess the existence of migrant health selection, and I explore several hypotheses that have been mentioned in the literature to explain observed health patterns among immigrants.

4.2.4.1 Immigrant Health Selection

To test for immigrant health selection, I compare health patterns of *recent immigrants* (i.e., those who have resided in the US for less than 5 years) with those of *non-Hispanic Whites*. As Jasso et al (2004) argue, this comparison provides indirect but convincing evidence of migrant health selection because, since most measures of population health indicate that the US population is healthier than the population of Mexico, a finding that recent Mexican immigrants are healthier than US-born Whites would clearly indicate that those immigrants are a selected healthier sample than the population of Mexico. I should remark that it is important to conduct this comparison using only recent immigrants, because several factors may influence the health of immigrants over time, changing their trajectories and thus invalidating any comparisons between Whites and immigrants that seek to explore selectivity. Since I compare not only average allostatic load levels, but the entire age and SES trajectories, I am also able to assess whether the health selection of immigrants changes with age, and whether the *AL-SES* relationships are similarly strong for recent immigrants and Whites.

4.2.4.2 Changes in Immigrant Allostatic Load Trajectories With Length of US Residence

Since the NHANES data is cross-sectional, I cannot make a longitudinal assessment of the changes in allostatic load for immigrants after they arrive to the United States. However, a comparison of the allostatic load trajectories of *recent immigrants* (those who arrived less than five years ago) and two groups of *earlier immigrants* (those who arrived between five and fifteen years ago, and those who have lived in the US for more than fifteen years) will provide evidence of any health differences between immigrants with difference lengths of US residence. If existing, these differences would indicate the possibility of health faster health deterioration after migration. Combined with the analyses described below, I will be able to make more concrete inferences about the origin of these health patterns.

4.2.4.3 Exploring Cohort Effects

As discussed above, a potential explanation for the result found in previous studies that immigrant health deteriorates faster following immigration is that recent immigrant cohorts are healther than earlier immigrant cohorts. The possibility of having healthier recent immigrant cohorts certainly exists, because Mexico experienced a significant improvement in population health in the second half of the 20th century, which led to an increase in life expectancy from 61 years in 1970 to 70 years in 1990 (UNICEF 2009), and this progress has not slowed down much since then, as life expectancy increased to 75 years in 2005 (OECD 2007). However, an increase in the health of the general population does not necessarily translate into better health for new migrant cohorts. For example, since previous migrant cohorts probably were already selected from the healthiest segments of the Mexican population, their health status might be similar to that of new migrant cohorts because increases

in life expectancy are likely to be higher among populations with poor health profiles, and migrants are unlikely to come from these populations.[23]

Comparing the health of *recent* immigrants between NHANES-III (1988-1994) and the more recent NHANES releases (1999-2004) may provide some evidence to assess the validity of this hypothesis. This comparison is obviously limited by the fact that not many years passed between these data collections. In order to somewhat reduce this problem, I remove the observations from the 1999-2000 NHANES such that there are at least seven years between observations—and as many as 16 years between the first and latest NHANES data collections.[24] If cohort effects are responsible for the results described above, we would expect *recent* immigrants in the 2001-2004 sample to be healthier than *recent* immigrants in the 1988-1994 sample.

4.2.4.4 Testing for Assimilation Effects

Finally, I examine whether it is likely that acculturation leads to poorer health. Although there are many indicators of acculturation, I am limited to the language spoken at home. Since recent immigrants are evidently more likely to speak Spanish and to be healthier (assuming I find evidence of selection), a full comparison of Spanish-speaking vs. English-speaking immigrants will be biased. Instead, I restrict the sample to immigrants who have lived in the US for *at least* 10 years, which I consider long enough to observe differences in acculturation. In addition, limiting the comparison to those who have lived in the

[23] An interesting implication of this argument is that increases in population health in Mexico may result in larger migrant cohorts due to increases in the health levels among populations with the lowest health profiles. This is, however, an issue unrelated to the main focus of this study.

[24] Neither NHANES-III nor the 1999-2004 NHANES allow for the identification of the specific year when an observation was collected. The recent NHANES samples can be divided in two-year periods, which allowed me to remove the 1999-2000 sample. NHANES-III can be divided in two subsamples: 1988-1991 and 1991-1994; however, removing the 1991-1994 data resulted in a too-small sample of Mexican immigrants, and thus in this analysis I must include the entire NHANES-III data.

US for at least 15 or 20 years would reduce the sample by nearly 17 and 30 percent, respectively (see Table 19).

NHANES provides information on three categories according to language spoken *at home*: only Spanish, only English, and both English and Spanish. Since the samples of "English only" and "both English and Spanish" are too small, I compare those who speak only Spanish with the other two groups (i.e., those who speak at least some English at home). There are non-minor limitations to this analysis, the most important being that acculturation is a multi-dimensional construct, certainly not fully characterized by the language spoken at home. However, there are not many indicators of acculturation other than language spoken at home available in both NHANES-III and the 1999-2004 NHANES. Nevertheless, psychometric analyses have shown that language items explain most of the variance observed in more complex measures of acculturation (Lara et al. 2005). In addition, it is not clear how language would affect health other than through acculturation in a way that would confound these analyses.

Overall, the analyses described above will provide a comprehensive assessment of the health trajectory of Mexican immigrants, and allow for an evaluation of hypotheses found in the literature on immigrant health, such as that of decreasing health with acculturation, and healthier recent cohorts.

4.3 Results

4.3.1 Changes in Allostatic Load With Age and Income: Whites and Immigrants

4.3.1.1 Nonparametric Estimates

The results of estimating the models in equation (11), after differencing out the non-parametric effects of $f(age)$, are shown in Table 20. The last row in the table reports the significance test of the nonlinear effect of *age*. The estimator *V* is distributed standard normal under the null hypothesis that a linear model without an *age* effect is preferred to the partial linear model. The significance test indicates that the effect of age on allostatic load is highly significant, for both Whites and Mexican immigrants.

Table 20. Results of Partial Linear Model Estimation—After Differencing out *f(age)*[25]

	Non-Hispanic Whites		Mexican Immigrants	
Variable	**Coefficient**	**SE**	**Coefficient**	**SE**
Female	**0.082**	(0.018)	**0.096**	(0.034)
Education				
Less than High school	**0.156**	(0.024)	0.097	(0.055)
High school degree	**0.135**	(0.018)	0.096	(0.063)
College or higher	*Reference*			
Insurance				
Private	**-0.060**	(0.028)	-0.018	(0.036)
Public	-0.031	(0.034)	**0.127**	(0.051)
Uninsured	*Reference*		*Reference*	
Type of work				
Never worked	0.086	(0.056)	**0.172**	(0.059)
White collar	**-0.106**	(0.023)	-0.084	(0.074)
Service sector	-0.024	(0.021)	-0.009	(0.037)
Blue collar	*Reference*		*Reference*	
Marital status				
Single	0.051	(0.028)	-0.052	(0.047)
Widowed/Divorced	0.031	(0.020)	-0.015	(0.047)
Married	*Reference*		*Reference*	
Significance test on f(age)	*V* = 159.33, p-value=0.000		*V* = 75.70, p-value=0.000	

[25] The models in this table include a linear effect for *PIR*, and the models in Table 21 include a linear effect for *age*.

The results of a similar analysis, but where the effect of $g(pir)$ is being differenced out, are reported in Table 21. The significance test on $g(pir)$ indicates that this variable is significant for both Whites and Mexican immigrants, although its significance is not as strong as that of age; in fact, the effect for immigrants is not significant at a 1 percent confidence level. This suggests that *age* is a significantly more important determinant of allostatic load than socioeconomic status. The rest of the coefficients in this table are very similar to those in Table 20, with the exception of public insurance, which was significant for immigrants in the previous table.

Table 21. Results of Partial Linear Model Estimation--After Differencing out *g(pir)*

	Non-Hispanic Whites		**Mexican Immigrants**	
Variable	**Coefficient**	**SE**	**Coefficient**	**SE**
Female	**0.077**	(0.018)	**0.096**	(0.034)
Education				
Less than High school	**0.151**	(0.024)	**0.126**	(0.055)
High school degree	**0.138**	(0.019)	**0.117**	(0.063)
College or higher	*Reference*			
Insurance				
Private	**-0.054**	(0.029)	-0.026	(0.036)
Public	-0.033	(0.034)	0.026	(0.048)
Uninsured	*Reference*		*Reference*	
Type of work				
Never worked	0.051	(0.057)	**0.171**	(0.059)
White collar	**-0.106**	(0.023)	-0.067	(0.075)
Service sector	-0.031	(0.021)	0.011	(0.037)
Blue collar	*Reference*		*Reference*	
Marital status				
Single	0.016	(0.027)	-0.065	(0.045)
Widowed/Divorced	0.007	(0.021)	-0.032	(0.048)
Married	*Reference*		*Reference*	
Significance test on g(pir)	V = 5.57, p-value=0.000		V = 1.86, p-value=0.032	

Next, I use the residuals of the partial linear model regressions to obtain graphical estimations of the shapes of f and g. I estimate nonparametric smoothing regressions on the

residuals. The results of the nonparametric smoothing are shown in Figure 5.[26] The graph on the upper left suggests that the shape of the allostatic load relationship with age is very similar for Whites and Mexican immigrants, although immigrants appear to have a slight health advantage at younger ages that gradually disappears, such that between ages 45 and 55 the allostatic load scores of both groups are nearly identical. At older ages, Whites have a growing health advantage over Mexican immigrants. However, when we examine the 90 percent confidence intervals of these lines, shown in the upper right graph, it appears that these differences are not large enough to be significant, with the possible exception of ages younger than 40, where there are some segments in which the confidence intervals do not overlap. In terms of candidate functional forms to test for *age*, the shape of these lines suggest either a quadratic, cubic, or logarithmic form.

Figure 5's bottom row shows the estimated nonparametric regression lines for *PIR*. A striking feature of this graph are the entirely different *Health-SES* trajectories of Whites and Mexican immigrants. At low PIR values, the line for Whites starts at a relatively high allostatic load level and then decreases monotonically as PIR increases until a PIR value of about three, after which it increases slightly. On the other hand, the line for Mexicans at a PIR value of zero is about 0.25 *AL* units below the line for Whites, remains nearly flat as PIR increases until PIR levels close to two, and then increases slightly after that. An examination of the confidence intervals shows that, although the intervals overlap at high PIR levels, there are significant differences at PIR levels below 1.5. At first sight, it is not clear what functional forms can be tested for PIR, but it is possible that a cubic form can approximate these shapes.

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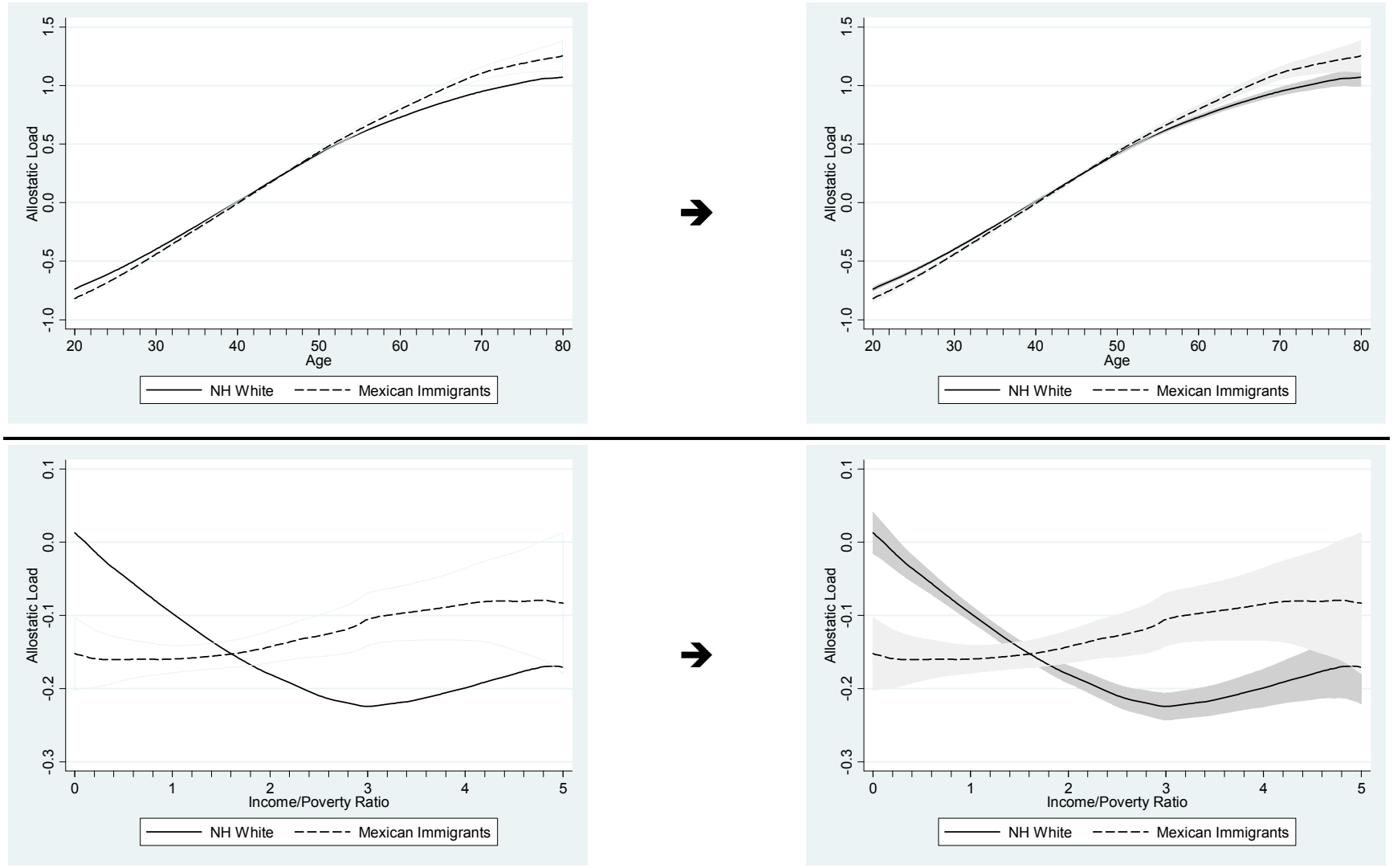
[26] I use a version of *plreg*—modified with permission from its author, Michael Lokshin—that allows for the estimation of confidence intervals.

Regardless of the appropriate functional form, the *AL-PIR* graph suggests two significant results. First, income is associated more strongly with health status for Whites than it is for Mexican immigrants. It is not difficult to notice that the *PIR* line for immigrants is nearly flat, while the line for Whites has a clear negative slope from low income up until *PIR* values near three. Second, not only is the relationship between *AL* and *SES* weak, but it could even be positive, such that higher income levels are associated with somewhat worse health status. Given the widely acknowledged strong and negative relationship between health and socioeconomic status, if confirmed, this is an important finding, and it is consistent with previous findings of weak *Health-SES* gradients among Hispanics in the United States (Goldman et al. 2006; Turra and Goldman 2007) and of a positive *Health-SES* gradient among Mexican Americans (Zsembik and Fennell 2005).

It is important to note, however, that other confounding factors not taken into account in the previous analyses may produce the observed patterns, even if the *Health-SES* gradient is strong and negative for Mexican immigrants. Below, I examine whether immigrants' length of residence in the United States could be a factor explaining these *Health-SES patterns*, and I evaluate whether other evidence supports some of the potential hypotheses to explain the apparent faster decline in health among Mexican immigrants.

Figure 5. Nonparametric Estimation of *f(age)* and *g(pir)*—Non-Hispanic Whites and Mexican Immigrants



4.3.1.2 Parametric Estimates

I estimate the following regression models, some of them suggested by the nonparametric regressions in Figure 5, and others to avoid the potential of missing a better specification than those suggested by the graphs:

$$\begin{aligned} &1.\ AL = z\beta + x\cdot\gamma_1 + \varepsilon \\ &2.\ AL = z\beta + x\cdot\gamma_1 + x^2\cdot\gamma_2 + \varepsilon \\ &3.\ AL = z\beta + x\cdot\gamma_1 + x^2\cdot\gamma_2 + x^3\cdot\gamma_3 + \varepsilon \\ &4.\ AL = z\beta + \log(x)\cdot\gamma_1 + \varepsilon. \end{aligned} \tag{12}$$

Where $x = \{age,\ pir\}$, and the other terms are the same as defined above. The significance test for each of these functional forms is conducted using the mean square residuals of the partial linear models in (11) and the parametric models in (12). The test statistic is:

$$V = \sqrt{m\cdot n}\,\frac{\left(s_{res}^2 - s_{diff}^2\right)}{s_{diff}^2} \xrightarrow{d} N(0,1). \tag{13}$$

Where *m* is the order of differencing used to estimate the partial linear model (higher orders are more efficient, *plreg* can estimate up to 10th-order differencing); *n* is the sample size before differencing; s_{res}^2 is the mean square residual of the parametric model; and s_{diff}^2 is the mean square residual of the partial linear model. Under the null hypothesis that the parametric model is correct, *V* is distributed standard normal.

The results of the significance tests for all models in (12) are reported in Table 22. The estimated p-values indicate that, *at a significance level of 5 percent*, none of the parametric models adequately capture the relationships allostatic load-age and allostatic load-PIR. Nevertheless, the cubic form of age (both Whites and Mexican immigrants), and the quadratic form of PIR (Mexican immigrants) are satisfactory at a significance level of 1 percent.

On the other hand, none of the four parametric specifications are adequate for $g(PIR)$ for Whites, since their specification tests are highly significant. This function probably requires a more complex specification than those I explore in this chapter. I conclude that none of these parametric forms adequately approximate the functional form of $g(PIR)$ for Whites, and that $f(age)$ for both groups and $g(pir)$ for Mexican immigrants can be somewhat reasonably approximated by cubic and quadratic forms, respectively.

Table 22. Specification Tests of Parametric Models—Non-Hispanic Whites

Parametric Model	Parametric Mean Square Residual	PLM Mean Square Residual	V	p-value	Best
Whites					
Age linear	0.5925	0.5782	8.09	0.000	
Age quadratic	0.5838		3.17	0.001	
Age cubic	0.5818		2.04	0.021	✓
Age logarithmic	0.5869		4.93	0.000	
PIR linear	0.5925	0.5829	5.39	0.000	
PIR quadratic	0.5924		5.33	0.000	
PIR cubic	0.5924		5.33	0.000	
PIR logarithmic	0.5919		5.05	0.000	✓
Mexican Immigrants					
Age linear	0.5798	0.5662	4.00	0.000	
Age quadratic	0.5737		2.21	0.014	
Age cubic	0.5720		1.71	0.044	✓
Age logarithmic	0.5770		3.18	0.001	
PIR linear	0.5798	0.5730	2.41	0.008	✓
PIR quadratic	0.5799		2.01	0.022	
PIR cubic	0.5801		2.06	0.020	
PIR logarithmic	0.5820		2.62	0.004	

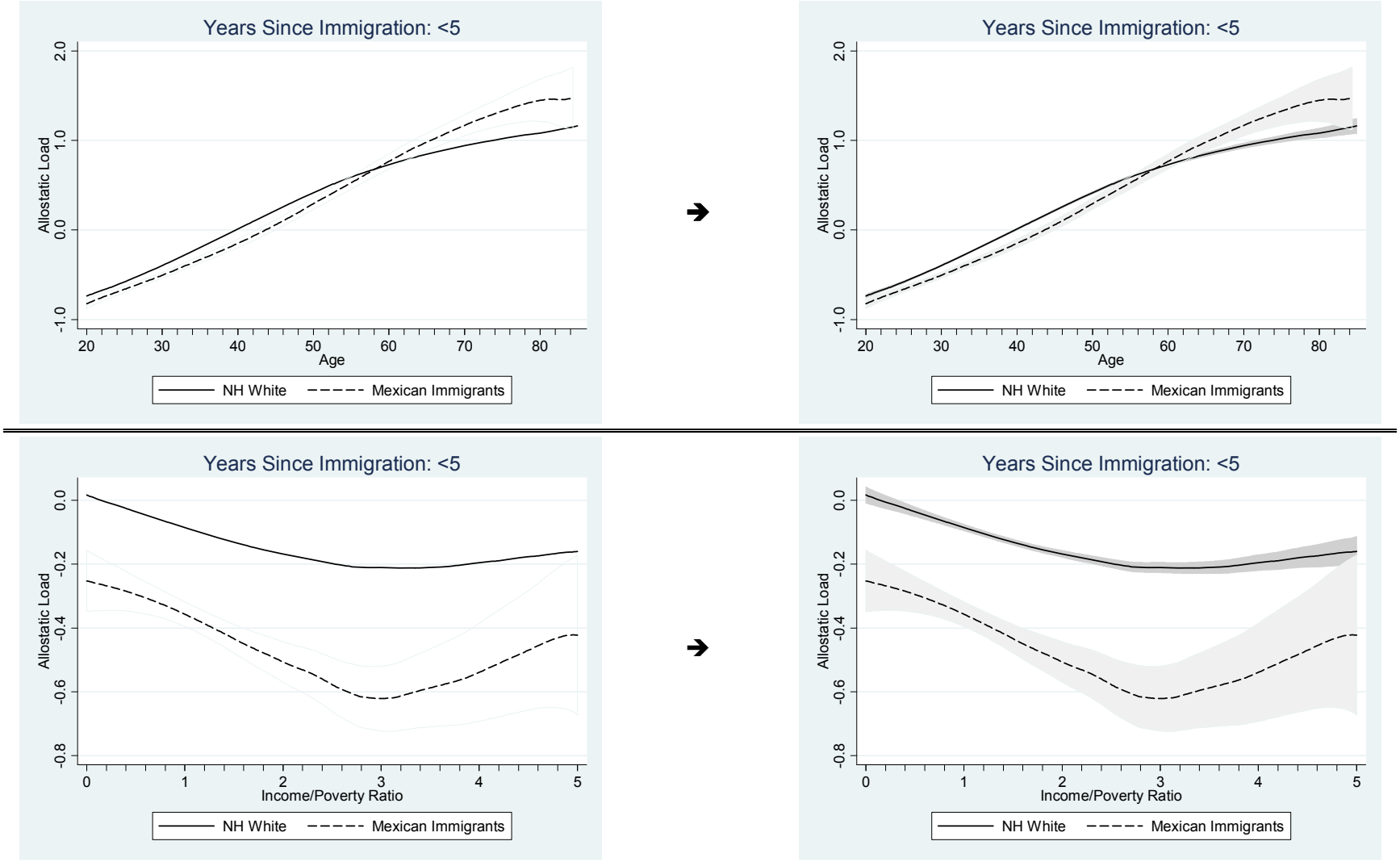
4.3.2 Evidence For and Against Hypotheses of Changes in Immigrant Health Over Length of US Residence

4.3.2.1 Immigrant Health Selection

The semiparametric estimation of the allostatic load-age and -SES trajectories for *recent immigrants* is shown in Figure 6. Three important conclusions can be derived from this figure: (1) there is clear evidence of health selection: most recent immigrants have lower levels of allostatic load than Whites of similar age and socioeconomic status; (2) this selection appears to be limited to young and middle-aged immigrants, since those of age 60 and older are increasingly less healthy than Whites; and (3) the striking result found in section 4.3.1, that the *AL-SES* relationship is weaker for immigrants than for Whites, disappears once we limit the comparison to recent immigrants; not only is the shape of this relationship similar, with the exception of an inverse "hump" at mid-income levels, but also the strength of the association is similar for immigrants and Whites, and perhaps even stronger for immigrants. As expected, the standard errors of these plots are larger than before, but they are sufficiently small at young ages and low income levels to be confident about these conclusions.

The results in this figure highlight the importance of taking into account factors that may have a significant role in mediating relationships such as that of allostatic load and age, or allostatic load and SES. While from Figure 5 we could have concluded that the AL-SES relationship is weak and perhaps positive for immigrants, the graphs in Figure 6 indicate that that is not the case, at least for recent immigrants.

Figure 6. Allostatic Load Age and SES Trajectories—Recent Immigrants vs. Non-Hispanic Whites

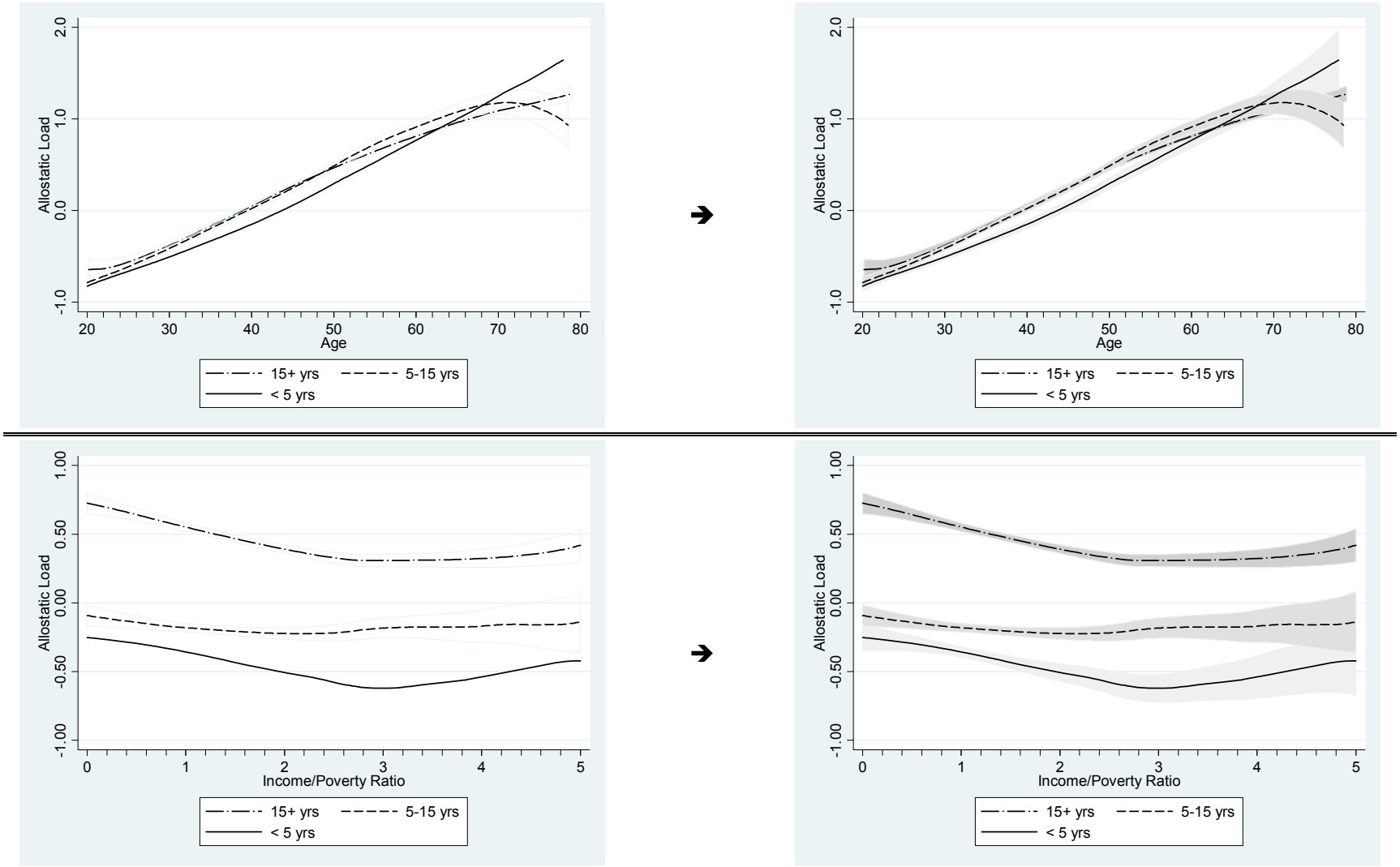


4.3.2.2 Immigrant Health-Age and Health-SES Trajectories by Length of US Residence

Comparisons of the estimated f and g functions for different immigrant groups—categorized by their length of US residence—are shown in Figure 7. Because there were too few observations at older ages, which resulted in extremely large confidence intervals that limited the usefulness of the graph because of the scale of the y-axis, I restricted these graphs to individuals of age 78 or younger. The fitted lines in the top panel indicate that young and middle-aged *recent* immigrants are healthier than immigrants of similar ages who arrived to the US more *five or more years earlier*. In particular, for immigrants between roughly 32 and 54 years of age, the estimated differences are large enough to be outside the area of overlap of the 90 percent confidence intervals. At ages over 70, *recent* immigrants appear to be less healthy than the other two immigrant groups, although there is considerable overlap of the confidence interval at all ages over 60. Worse health status of elderly *recent* immigrants is consistent with the negative selection at older ages found above.

With respect to the *AL-SES* trajectories, the picture is even clearer: recent immigrants are healthier than earlier immigrants of similar income levels across the entire range of income/poverty ratios reported in NHANES. Moreover, we now see that not only *recent* immigrants, but also immigrants with over 15 years of residence in the US, have a negative and similarly strong relationship with *PIR*. The only group with a weaker *AL-SES* relationship, and that resembles the plot shown in Figure 5, are immigrants with a US residence length between 5 and 15 years. Therefore, the weak *Health-SES* patterns found above appear not to be an intrinsic trait of Mexican immigrants, but rather a result of the initial health selectivity of migration and an apparent decline in health over the length of immigrants' residence in the United States.

Figure 7. Allostatic Load-Age and -SES Trajectories--By Length of US Residence



4.3.2.3 Comparing Immigrant Cohorts: NHANES-III vs. 2001-2004 NHANES

One potential explanation for the apparent decline in immigrant health as the length of residence in the United States increases does not involve a health decline over time, but rather the possibility that recent immigrant arrival cohorts are healthier than earlier arrival cohorts. This would explain both the *Health-SES* patterns found in Figure 5, because earlier arrival cohorts would have higher SES levels due to having lived in the US longer and would be less healthy due to the hypothesized cohort differences, as well as the differences between immigrants according to the length of their US residence found in Figure 7.

The available information on time since arrival to the US in the 1999-2004 NHANES severely limits my ability to construct accurate arrival cohorts. Thus, I can only define two arrival cohorts: those who were *recent* arrivals during in NHANES-III, and those who were *recent* arrivals in the 1999-2004 NHANES. The estimated f and g functions for these two immigrant arrival cohorts are shown in Figure 8. In this case, *recent immigrants* are defined as those having arrived within 10 years of the survey. I change the definition of recent immigrant in order to maintain a reasonable sample size and yet, the standard errors are high at older ages and high income levels. Nevertheless, the plots at young and middle ages, and at low and middle income levels, suggest that there are no significant differences in allostatic load scores between these two arrival cohorts. Furthermore, if there are any differences, the more recent immigrant cohort appears to be less healthy than the 1988-1994 immigrant cohort, exactly the opposite of what we would expect if cohort effects were to explain the patterns found in Figure 7. These results are consistent with those of Antecol and Bedard (2006), who did not find significant health differences between several immigrant arrival cohorts (1980 and before, 1981-1985, 1986-1990, and 1991-1996)

4.3.2.4 Acculturation Effects—Spanish- vs. English-Speaking Immigrants

Finally, to assess whether there is any indication that health declines with acculturation, I restrict the sample to immigrants who have lived in the United States for *10 years or longer* and I stratify the semiparametric analyses by the language spoken at home. The results are shown in Figure 9. There is no indication that Spanish-speaking immigrants are healthier than English-speaking immigrants. In fact, Spanish speakers have higher allostatic load values than English speakers over nearly the entire age range, although these differences are generally not large enough to be outside the confidence intervals. One limitation of these analyses is that they do not account for the possibility that English-speaking immigrants were healthier than Spanish-speaking immigrants at the time of arrival to the US. Unfortunately, I cannot test for this possibility because the sample size of recent immigrants who speak English is too small (less than 5 percent of the sample of recent Mexican immigrants). In fact, this can be considered as an argument for the validity of these analyses: 10 years after arrival, nearly 32 percent of the sample speak at least some English, which indicates that over 25 percent of immigrants have acculturated to some extent, and the acculturated are healthier than the unacculturated.

Nevertheless, as discussed above, acculturation is multi-dimensional and language is only one of its many components. Therefore, it may still be the case that acculturation in other dimensions may have negative health consequences. Results using other measures of acculturation such as diet, cultural beliefs, or civic participation could be different from those found here. Nevertheless, language spoken at home remains a valid indicator of acculturation, and the results reported in Figure 9 do suggest that more acculturated immigrants are healthier than the less acculturated.

Figure 8. Allostatic Load Age and SES Trajectories—NHANES-III and 2001-2004 NHANES

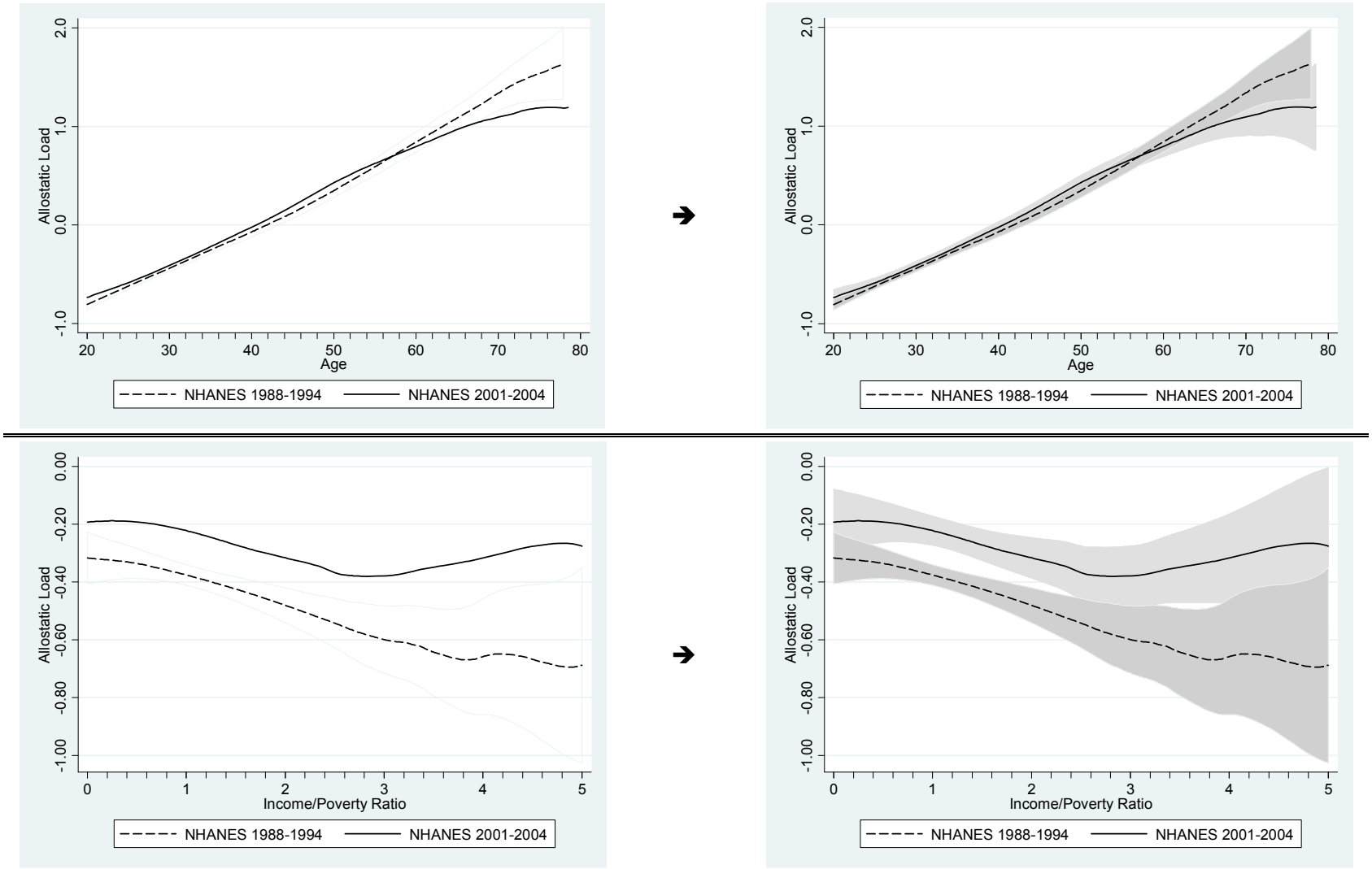
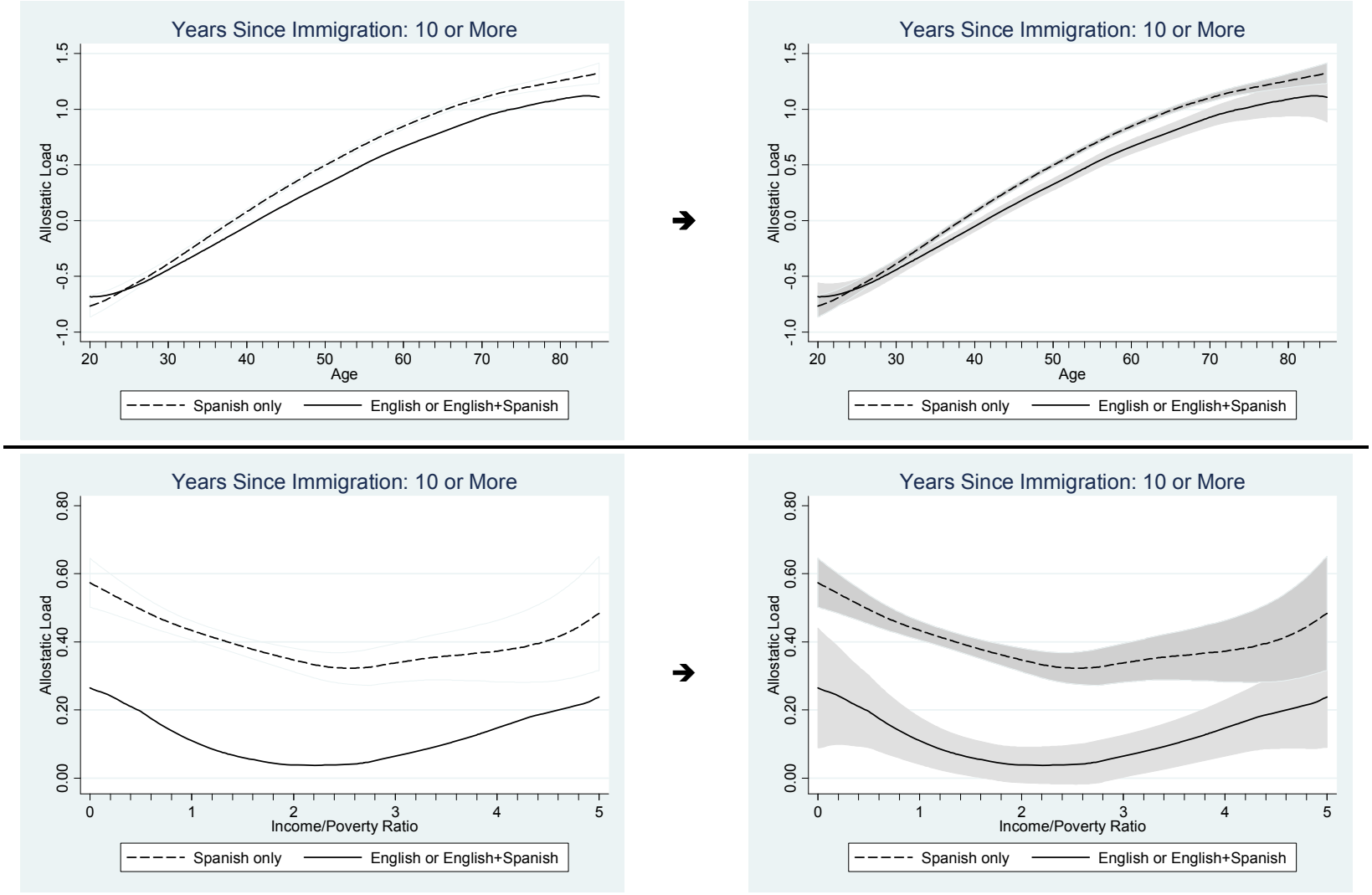


Figure 9. Allostatic Load Age and SES Trajectories—Spanish- vs. English-Speaking Immigrants



4.4 Conclusion

The analyses in this chapter support some of the hypotheses that are common in the literature on immigrant health, but raise some doubts about others. First, the semiparametric analyses I conduct on the full sample of Mexican immigrants indicate that (1) immigrant health declines relative to that of US-born Whites as the length of US residence increases; and (2) the *Health-SES* gradient of immigrants is flatter than that of Whites, and it might be slightly positive at higher income levels.

However, when I divide the sample of Mexican immigrants according to length of residence in the United States, the health patterns are somewhat different from those described above. More specifically, a comparison of *recent* Mexican immigrants with Whites suggest that immigrants are indeed positively selected on health status, because their allostatic load levels are lower than those of Whites at ages below 60. On the other hand, the health selectivity of older Mexican immigrants appears to be negative, which is consistent with younger Mexicans being likely to migrate for economic reasons, but older Mexicans being more likely to migrate to reunite with their family. These comparisons also reveal a *Health-SES* gradient for recent immigrants similar to that of Whites, suggesting that the weak gradient found in other studies is an artifact of health changes over immigrants' life course.

Indeed, when the allostatic load patterns of recent immigrants are compared to the patterns of immigrants with 5-15 years of residence in the US, and to those of immigrants with over 15 years of US residence, the results show a better health status for the former group compared to the latter two. This is consistent with the result, often found in the literature, that immigrant health decreases with length of residence in the United States. However, it is important to note that this does not necessarily imply that immigrant health decreases

with acculturation since, as discussed above, increased length of residence is not necessarily a good indicator of acculturation.

One potential hypothesis to explain the worsening of immigrant health with length of residence in the United States is the existence of health differences between different immigrant cohorts. However, when I test this hypothesis by comparing recent immigrants in NHANES-III with recent immigrants in the 2001-2004 NHANES, I do not find significant differences between both cohorts. If anything, the results suggest that the NHANES-III cohort of recent immigrants might have been healthier than its counterpart in the 2001-2004 NHANES, contrary to the hypothesis that would explain the results described above. The confidence intervals are too wide in this analysis, though; thus, larger sample sizes are required to draw any definitive conclusions.

Finally, I try to identify acculturation effects on immigrant health by comparing the allostatic load patterns of Mexican immigrants who have lived in the US for at least ten years and who speak *only* Spanish at home, with those of immigrants with at least ten years of US residence, but who speak at least some English at home. The results indicate opposite results to those expected under the *acculturation hypothesis*: more acculturated (English speaking) immigrants are healthier than less acculturated immigrants. Although the differences are not always outside the 90 percent confidence intervals, they are large enough to be statistically significant between ages 40 and 70, and at most PIR levels below 3.5.

Overall, the analyses in this chapter do not support the hypothesis of *acculturation* or *healthier recent cohorts*, suggesting that either the *life-course* hypothesis or the *regression to the mean* hypothesis are more likely to be correct. That is, the observed decline in immigrant health with increases in length of US residence may be due to either (a) the cumulative effect of the hardships migrants experience over their lifetime, or (b) a convergence of immigrant health status to the average health levels in their home countries.

An important contribution of this analysis is the insight it provides into what Goldman and colleagues (2006) have called a "second Hispanic Paradox": the apparent weak or even flat *Health-SES* gradient among Hispanics living in the United States. My results indicate that the *Health-SES* gradient among Mexican immigrants is indeed negative and about as steep as that of non-Hispanic Whites. The weak gradient that is observed when all immigrants are examined together is an artifact of the decline in immigrant health with length of US residence: as immigrants' duration of residence increases, so does their income, but given the decline in health associated with longer stay in the US, we observe better health among low-income immigrants (who are more likely to be recent immigrants) than among high-income immigrants (who are likely to have lived for more years in the US). As Figure 7 suggests, when immigrants are divided according to their length of US residence, their *Health-SES* gradients resemble those of US-born Whites (the exception appears to be immigrants with a residence between 5 and 15 years.) I should note, however, that several of the studies that found a weak gradient among Hispanics used education as the measure of SES instead of income. In this study I focused on income because it is a continuous variable that allows me to conduct semiparametric analyses that I consider provide a greater insight into the nature of the Health-SES relationship. Nevertheless, future studies can explore the gradient while stratifying the sample by length of US residence, in order to verify if results similar to mine are obtained.

Chapter 5.
Discussion

This work sought to provide a better understanding of the issues surrounding the health of Hispanics in general and Hispanic immigrants in particular. A first contribution of this study is the creation of an objective measure of biological risk that weighs each of its components according to their association with mortality, and takes into account potential nonlinearities in those associations. A second contribution is my emphasis on the potential usefulness of semiparametric methods in the study of health disparities. I apply two such methods in my analysis: First, I use propensity score estimators to estimate the health gap between Whites, US-born Mexicans, and Mexican immigrants. Second, I use a semi-parametric differencing estimator combined with nonparametric smoothing in order to analyze the health patterns of Mexican immigrants, and how they compare to the health patterns of Whites. Below, I summarize my main findings and discuss the implications of this research.

5.1 Main Findings

In Chapter 3, I examined the existence of a health advantage of individuals of Mexican ethnicity over non-Hispanic Whites in the United States, a phenomenon commonly known as the "Hispanic Paradox." My main results indicate that this paradox is a phenomenon related to immigration. When I use allostatic load to compare health status between ethnic groups, US-born Mexicans are found to be less healthy than both Whites and Mexican immigrants. This health disadvantage exists not only for the summary index of allostatic load (AL), but also for each of its three subcomponents (inflammatory, metabolic, and cardiovascular). Mexican immigrants have lower (i.e., better) allostatic load scores than US-born Mexicans, and lower inflammatory and cardiovascular scores than Whites. However, the

metabolic scores of Mexican immigrants are higher than those of Whites, and so the overall allostatic load score is not significantly different between Whites and immigrants.

On the other hand, similar analyses that use the 10-year risk of coronary heart disease as the outcome, estimated using the Framingham scoring system (FRS), produce somewhat different results. In this case, both US-born Mexicans and Mexican immigrants are found to have lower risk of coronary heard disease (CHD) than Whites, which is consistent with a general Hispanic Paradox. I attempt to reconcile the FRS and AL results by first removing from the Framingham score the risk factors not included in the AL measure, and then removing from allostatic load the biomarkers not included in the Framingham score. Although some differences persist when I use these 'reduced' measures, the FRS models are generally consistent with the AL models regarding the health of Mexican immigrants (i.e., Mexican immigrants enjoy health similar to Whites, although they have advantages in some areas and disadvantages in others). Regarding US-born Mexicans, I find the evidence as convincingly suggesting that they are less healthy than Whites.

In Chapter 4, I explore the *Health-age* and *Health-SES* trajectories of Mexican immigrants. I use semiparametric methods to estimate these trajectories over the full age and SES range available in NHANES. Moreover, I assess the evidence supporting several hypotheses that have been presented in the literature on immigrant health. Consistently with results in other research, I find *indirect* evidence supporting the positive health selection of Mexican migrants and a decline in immigrant health with increases in duration of residence in the United States. However, unlike other studies, I find that the *Health-SES* gradients are similar for Whites, recent immigrants, and immigrants who have lived in the US more than 15 years. Only immigrants with 5-15 years of residence appear to have a weaker gradient when compared to US-born Whites. In addition, I do not find support for the hypotheses that immigrant health worsens with acculturation, or that recent immigrant cohorts are

healthier than earlier immigrant cohorts. Overall, the results in this chapter suggest that the observed decline in immigrant health with increases in duration of US residence are more likely to be due to either (a) the cumulative negative effect of the adversities associated with the process of migration, or (b) a regression of immigrant health to the average health levels in their countries of origin.

5.2 Future Research and Policy Implications

An important first implication of the analyses in Chapter 3 is that researchers need to be cautious when using self-assessments of health status or self-reports of health conditions to compare the health of different ethnic groups. As shown in Tables 2 and 3, the results of those analyses are ambiguous and largely dependent on the measure chosen. Objective health measures that use biomarkers or other unbiased indicators of health should be used in these studies whenever possible.

Further research is needed to verify my result that US-born Mexicans are less healthy than non-Hispanic Whites and Mexican immigrants. If confirmed, this is an important result with at least three significant implications: (1) future analyses of ethnic disparities should divide the samples by country of birth, or should add country of birth interaction terms if the analyses are pooled across ethnic groups; (2) other studies need to assess whether this disadvantage exists not only for US-born Mexicans, but also for other US-born Hispanics; and (3) a focus of future research should be identifying the causes of this health disadvantage.

With respect to Mexican immigrants, it is necessary to establish why positive health selection of migrants does not translate in a health advantage in biomarkers of metabolic functioning. Evidently, the processes that lead to better health status of migrants in inflammatory and cardiovascular functioning do not produce the same result for metabolic

indicators. This is important not only from an academic perspective, but also because diabetes and obesity are increasingly important health problems for Hispanics in the United States and in countries like Mexico. If the root causes of this disadvantage are cultural, both US and Mexican governments need to engage in public health campaigns to neutralize them before the diabetes and obesity epidemics grow even more in importance.

Another important implication of this work is the lack of support for the *acculturation hypothesis*. This is a theory that now has deep roots in the health literature, so obviously further efforts are needed to verify these findings using better measures of acculturation. Nevertheless, if it is indeed the case that acculturation does not necessarily lead to worse health outcomes, researchers and policymakers need to become aware of it. Moreover, my results suggest that acculturation—at least language acculturation—may actually be beneficial for health. This could occur through better access to the healthcare system, increased health literacy, or simply through a feeling of belonging to US society. All of these alternatives need to be explored because they could be of great importance to future policy efforts aimed towards immigrants.

My results regarding the health trajectories of Mexican immigrants are consistent with two hypothesis: (1) the *life-course* hypothesis, which states that the adversities associated with migration are the cause of the decline of immigrants' health with increased length of US residence, and (2) the *regression to the mean* hypothesis, which states that this health decline is simply a result of a convergence of immigrant health to the health profiles in their country of origin. The available data does not allow me to specifically test either of these two hypotheses against each other. I am only aware of two studies that have attempted to test for *regression to the mean* in a health context. Chiswick et al. (2008) found that the health of four different types of immigrants declined at different rates over a three-year period. Since three of the four types had similar rates of good health status upon arrival,

under reversion to the mean we would expect their health status to decline at similar rates, thus their results cast some doubt on the regression to the mean hypothesis. The authors also conclude that the observed health declines may be caused by several factors associated to initial adjustment to a new country, including "the stress of immigration" (i.e., the *life-course hypothesis*), but they never formally test this premise. Biddle et al. (2007) also examine the extent to which the health of immigrants to Australia varies over time. They consider cohorts of immigrants from three different groups of countries of birth, and expect similar rates of health decline among the three groups under the *regression to the mean hypothesis*, but instead find differences in health decline between these groups, thus concluding that declines in health are more likely to be affected by "culture and environment." For example, a recent study found that Mexican immigrants in the US and Mexicans with a family member in the US are at a higher risk to have suicidal thoughts than Mexicans with no migration history and no migrant relatives (Borges et al. 2009). This suggests that the process of migration places migrants and their families under additional stress, above and beyond the stress experienced by non-migrant households, which is consistent with the life-course perspective.

Future research should explicitly test these two hypotheses, especially since the potential policy implications under each of them are considerably different. In particular, the *regression to the mean* hypothesis implies that the decline in immigrant health is expected as a result of natural biological processes, and thus policies should focus on identifying the health risks likely to result from the convergence of immigrant health to the health profiles in their countries of origin (e.g., could the high incidence of diabetes among Hispanics have been anticipated given the diabetes epidemic that has affected countries like Mexico?). On the other hand, if declines in immigrant health are produced by higher cumulative stress throughout the life course, policies should focus on identifying the sources of additional

stress in order to mitigate their effects and reduce, for example, the higher risk of suicidal thoughts among Mexicans found by Borges et al. (2009). A potential research approach to test these hypotheses is to compare the health trajectories of undocumented immigrants with those of legal immigrants. Since the former are more likely to undergo a difficult migration process, under the *life-course* hypothesis we would expect their health to decline faster than for legal immigrants. On the other hand, under *regression to the mean* there is no a priori reason to expect that both groups would have different rates of health decline over time.

Two final remarks are warranted regarding alternative theories of the causes of immigrant health deterioration. First, it is possible that differential use of—or lack of access to—the healthcare system is a contributing factor to explain a faster health decline among immigrants than among the native US population. For example, immigrants might be healthier upon arrival to the US, but lack of access to healthcare services may result in worse health outcomes over time. Research indicates that immigrants use less healthcare services than the native US population (Goldman, Smith, and Sood 2006). Although this may be due to positive health selection and younger age among immigrants, lower use of medical care may result in worse health outcomes over time. In addition, even if immigrants increase their use of healthcare services as they acculturate, prejudice among healthcare providers and imperfect communication between patients and providers may result in lower quality in the provision of healthcare (Escarce 2005; Balsa, McGuire, and Meredith 2005). Therefore, another item for future research is the role of differences in the use of healthcare services and in the quality of services received as contributors to the deterioration of health status among immigrants.

Second, contextual factors may mediate the relationship between immigrant health status and duration of residence in the receiving country. A theory related to this perspec-

tive is that of *segmented assimilation* (Portes and Zhou 1993). This theory maintains that immigrants with low SES levels may have no choice but to assimilate into low-SES areas in the United States, thus experiencing both the adversities and negative influences associated with these areas. On the other hand, other immigrants, most likely those with high SES levels upon arrival, may be able to replicate the traditional model of acculturation and integration into the White middle-class. In addition, under a third pattern of assimilation, immigrants may preserve their most important cultural traits while at the same time they assimilate into mainstream society, thus enjoying the benefits associated with assimilation.

Although this theory is usually posited in the context of socioeconomic outcomes, it has been mentioned recently as a potential framework to explain the trajectories of immigrant health (Goldman et al. 2006). Testing this theory, Finch et al. (2007) hypothesize that neighborhood disadvantage and acculturation moderate the relationship between place of birth and low birth weight, such that (a) immigrant women who live in disadvantaged neighborhoods are more likely to give birth to low-weight infants than similar women living in more advantaged neighborhoods, (b) immigrant women who live in *unacculturated* neighborhoods are less likely to give birth to low-weight infants than similar women living in more *acculturated* neighborhoods, and (c) the association between neighborhood disadvantage and low birth weight is similar for native women and immigrant women, but the relationship between neighborhood *acculturation* and low birth weight is opposite between both groups of women. Their findings generally support these hypotheses, thus suggesting that contextual factors indeed influence the health outcomes of immigrants. Whether these effects result in different health patterns than those observed in this study is another issue that needs to be explored in future research.

Bibliography

Abraido-Lanza, A. F., M. T. Chao, and K. R. Florez. 2005. Do healthy behaviors decline with greater acculturation? Implications for the Latino mortality paradox. *Social Science & Medicine* 61 (6):1243-1255.

Abraido-Lanza, A. F., B. P. Dohrenwend, D. S. Ng-Mak, and J. B. Turner. 1999. The Latino Mortality Paradox: A Test of the "Salmon Bias" and Healthy Migrant Hypotheses. *American Journal of Public Health* 89 (10):1543-1548.

Acevedo-Garcia, Doiores, Mah-J. Soobader, and Lisa F. Berkman. 2007. Low birthweight among US Hispanic/Latino subgroups: The effect of maternal foreign-born status and education. *Social Science & Medicine* 65:2503-2516.

Albrecht, S. L., L. L. Clarke, M. K. Miller, and F. L. Farmer. 1996. Predictors of differential birth outcomes among Hispanic subgroups in the United States: The role of maternal risk characteristics and medical care. *Social Science Quarterly* 77 (2):407-433.

Anderson, K. M., P. M. Odell, P. W. Wilson, and W. B. Kannel. 1991. Cardiovascular disease risk profiles. *Am Heart J* 121 (1 Pt 2):293-8.

Antecol, Heather, and Kelly Bedard. 2006. Unhealthy assimilation: Why do immigrants converge to American health status levels? *Demography* 43 (2):337-360.

Balsa, A. I., T. G. McGuire, and L. S. Meredith. 2005. Testing for statistical discrimination in health care. *Health Serv Res* 40 (1):227-52.

Bang, Heejunt, and James M. Robins. 2005. Doubly Robust Estimation in Missing Data and Causal Inference Models. *Biometrics* 61:962-972.

Becerra, J. E., C. J. R. Hogue, H. K. Atrash, and N. Perez. 1991. Infant-Mortality among Hispanics - a Portrait of Heterogeneity. *Jama-Journal of the American Medical Association* 265 (2):217-221.

Biddle, Nicholas, Steven Kennedy, and James T. McDonald. 2007. Health assimilation patterns amongst Australian immigrants. *The Economic Record* 83 (260):16-230.

Blundell, Richard, Lorraine Dearden, and Barbara Sianesi. 2005. Evaluating the Effect of Education on Earnings: Models, Methods and Results from the National Child Development Survey. *Journal of the Royal Statistical Society, Series A* 168 (3):473-512.

Borges, Guilherme, Joshua Breslau, Maxwell Su, Matthew Miller, Maria Elena Medina-Mora, and Sergio Aguilar-Gaxiola. 2009. Immigration and Suicidal Behavior Among Mexicans and Mexican Americans. *American Journal of Public Health* 99 (4):728-733.

Caliendo, Marco. 2006. *Microeconometric Evaluation of Labour Market Policies*. Vol. 568, *Lecture Notes in Economics and Mathematical Systems*. Berlin: Springer.

Caliendo, Marco, and Sabine Kopeinig. 2008. Some Practical Guidance for the Implementation of Propensity Score Matching. *Journal of Economic Surveys* 22 (1):31-72.

Cameron, A. C., and P. K. Trivedi. 2005. *Microeconometrics. Methods and Applications*. New York, NY: Cambridge University Press.

Chiswick, B. R., Y. L. Lee, and P. W. Miller. 2008. Immigrant Selection Systems and Immigrant Health. *Contemporary Economic Policy* 26 (4):555-578.

Cho, Youngtae, W. Parker Frisbie, Robert A. Hummer, and Richard G. Rogers. 2004. Nativity, Duration of Residence, and the Health of Hispanic Adults in the United States. *International Migration Review* 38 (1):184-211.

CONAPO. 2008. Migration and Health: Latinos in the United States. Mexico, D.F.: Consejo Nacional de Población.

Cooper, R., J. Cutler, P. Desvigne-Nickens, S. P. Fortmann, L. Friedman, R. Havlik, G. Hogelin, J. Marler, P. McGovern, G. Morosco, L. Mosca, T. Pearson, J. Stamler, D. Stryer, and T. Thom. 2000. Trends and disparities in coronary heart disease, stroke, and other cardiovascular diseases in the United States - Findings of the National Conference on Cardiovascular Disease Prevention. *Circulation* 102 (25):3137-3147.

Crimmins, Eileen M., Jun K. Kim, Dawn E. Alley, Arun Karlamangla, and Teresa Seeman. 2007. Hispanic Paradox in Biological Risk Profiles. *American Journal of Public Health* 97 (7):1305-1310.

de Ruijter, W., R. G. Westendorp, W. J. Assendelft, W. P. den Elzen, A. J. de Craen, S. le Cessie, and J. Gussekloo. 2009. Use of Framingham risk score and new biomarkers to predict cardiovascular mortality in older people: population based observational cohort study. *BMJ* 338:a3083.

Diez Roux, A. V., R. Detrano, S. Jackson, D. R. Jacobs, Jr., P. J. Schreiner, S. Shea, and M. Szklo. 2005. Acculturation and socioeconomic position as predictors of coronary calcification in a multiethnic sample. *Circulation* 112 (11):1557-65.

Donders, A. Rogier T. , Geert J.M.G. van der Heijdenn, Theo Stijnen, and Karel G.M. Moons. 2006. Review: A gentle introduction to imputation of missing values. *Journal of Clinical Epidemiology* 59:1087-1091.

Dubowitz, Tamara, Stephanie A. Smith-Warner, Doiores Acevedo-Garcia, S.V. Subramanian, and RD Karen E. Peterson, ScD. 2007. Nativity and Duration of Time in the United States: Differences in Fruit and Vegetable Intake Among Low-Income Postpartum Women. *American Journal of Public Health* 97 (10):1787-1790.

Elo, I. T., C. M. Turra, B. Kestenbaum, and B. R. Ferguson. 2004. Mortality among elderly Hispanics in the United States: Past evidence and new results. *Demography* 41 (1):109-128.

Escarce, J. J. 2005. How does race matter, anyway? *Health Services Research* 40 (1):1-7.

Escarce, Jose J., Leo S. Morales, and R. G. Rumbaut. 2006. The health status and health behaviors of Hispanics. In *Hispanics and the Future of America*, edited by M. Tienda and F. Mitchell. Washington, DC: National Academy Press.

Evans, G. 2003. A multimethodological analysis of cumulative risk and allostatic load among rural children. *Developmental Psychology* 39 (5):924-933.

Finch, B. K., R. Frank, and W. A. Vega. 2004. Acculturation and acculturation stress: A social-epidemiological approach to Mexican migrant farmworkers' health'. *International Migration Review* 38 (1):236-262.

Finch, Brian K., Robert A. Hummer, Maureen Reindl, and William A. Vega. 2002. Validity of Self-Rated Health Among Latino(a)s. *American Journal of Epidemiology* 155 (8).

Finch, Brian K., Nelson Lim, William Perez, and D. Phuong Do. 2007. Toward a population health model of segmented assimilation: the case of low birth weight in Los Angeles. *Sociological Perspectives* 50 (3):445-468.

Franzini, L., J.C. Ribble, and A.M. Keddie. 2001. Understanding the Hispanic Paradox. *Ethnicity and Disease* 11:496-518.

Frölich, Markus. 2007. Propensity Score Matching Without Conditional Independence Assumption--With an Application to the Gender Wage Gap in the United Kingdom. *Econometrics Journal* 10:359-407.

Fry, Richard. 2008. Latino Settlement in the New Century. Washington, DC: Pew Hispanic Center.

Goldman, Dana, James P. Smith, and Neeraj Sood. 2006. Immigrants And The Cost Of Medical Care. *Health Affairs* 25 (6):1700-1711.

Goldman, Noreen. 2001. Social inequalities in health: disentangling the underlying mechanisms. *Annals of the New York Academy of Sciences* 954:118-139.

Goldman, Noreen, Rachel T. Kimbro, Cassio M. Turra, and Anne R. Pebley. 2006. Socioeconomic Gradients in Health for White and Mexican-Origin Populations. *American Journal of Public Health* 96 (12):2186-2193.

Grossman, Michael. 1972. The Demand for Health: A Theoretical and Empirical Investigation. In *NBER Occasional Papers*. New York: National Bureau of Economic Research.

Hahn, R. A., and S. Eberhardt. 1995. Life Expectancy in 4 Us Racial Ethnic Populations - 1990. *Epidemiology* 6 (4):350-355.

Hamman, R. F., J. A. Marshall, J. Baxter, L. B. Kahn, E. J. Mayer, M. Orleans, J. R. Murphy, and D. C. Lezotte. 1989. Methods and prevalence of non-insulin-dependent diabetes mellitus in a biethnic Colorado population. The San Luis Valley Diabetes Study. *Am J Epidemiol* 129 (2):295-311.

Harding, David J. 2003. Counterfactual Models of Neighborhood Effects: The Effect of Neighborhood Poverty on Dropping Out and Teenage Pregnancy. *American Journal of Sociology* 109 (3):676-719.

Heckman, James. 1978. Dummy Endogenous Variables in a Simultaneous Equation. *Econometrica* 46:931-961.

———. 1989. Causal Inference and Nonrandom Samples. *Journal of Educational Statistics* 14:159-168.

Heckman, James, Hidehiko Ichimura, Jeffrey Smith, and Petra Todd. 1998. Characterizing Selection Bias Using Experimental Data. *Econometrica* 66 (5):1017-1098.

Hertzman, Clyde. 2004. The life-course contribution to ethnic disparities in health. In *Critical Perspectives on Racial and Ethnic Differences in Health in Late Life*, edited by N. B. Anderson, R. A. Bulatao and B. Cohen. Washington, DC: National Academies Press.

Hirano, Keisuke, and Guido Imbens. 2001. Estimation of Causal Effects using Propensity Score Weighting: An Application to Data on Right Heart Catheterization. *Health Services & Outcomes Research Methodology* 2:259-278.

Ho, Daniel E., Kosuke Imai, Gary King, and Elizabeth A. Stuart. 2007. Matching as Nonparametric Preprocessing for Reducing Model Dependence in Parametric Causal Inference. *Political Analysis* 15 (3):199-236.

Hummer, Robert A., Maureen R. Benjamins, and Richard G. Rogers. 2004. Racial and Ethnic Disparities in Health and Mortality Among the U.S. Elderly Population. In *Critical Perspectives on Racial and Ethnic Differences in Health in Late Life*, edited by N. B. Anderson, R. A. Bulatao and B. Cohen. Washington, D.C.: National Academy Press.

Imbens, Guido. 2004. Nonparametric Estimation of Average Treatment Effects Under Exogeneity: A Review. *Review of Economics and Statistics* 86 (1):4-29.

Jasso, Guillermina, D. S. Massey, Mark S. Rosenzweig, and James P. Smith. 2004. Immigrant Health: Selectivity and Acculturation. In *Critical Perspectives on Racial and Ethnic Differences in Health in Late Life*, edited by N. B. Anderson, R. A. Bulatao and B. Cohen. Washington, D.C.: The National Academies Press.

Jasso, Guillermina, Douglas S. Massey, Mark R. Rosenzweig, and James P. Smith. 2005. Immigration, Health, and New York City: Early Results Based on the U.S. New Immigrant Cohort of 2003. *Economic Policy Review, Federal Reserve Bank of New York* 11 (2):127-151.

Johnston-Brooks, C. H., M. A. Lewis, G. Evans, and Whalen C. K. 1998. Chronic stress and illness in children: the role of allostatic load. *Psychosomatic Medicine* 60 (5):597-603.

Johnston, Jack, and John DiNardo. 1997. *Econometric Methods*. Fourth ed. New York: McGraw-Hill.

Kana'Iaupuni, S. M., K. M. Donato, T. Thompson-Colon, and M. Stainback. 2005. Counting on kin: Social networks, social support, and child health status. *Social Forces* 83 (3):1137-1164.

Karlamangla, Arun S., Burton H. Singer, and Teresa Seeman. 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.

Karlamangla, Arun, Burton H. Singer, Bruce S. McEwen, John W. Rowe, and Teresa E. Seeman. 2002. Allostatic load as a predictor of functional decline MacArthur studies of successful aging. *Journal of Clinical Epidemiology* 55:696-710.

Keppel, K. G. 2007. Ten largest racial and ethnic health disparities in the United States based on healthy people 2010 objectives. *American Journal of Epidemiology* 166 (1):97-103.

Kimbro, Rachel T., Sharon Bzostek, Noreen Goldman, and Germán Rodríguez. 2008. Race, ethnicity, and the education gradient in health. *Health Affairs* 27 (2):361-372.

Kleinman, Joel C. 1990. Infant Mortality Among Racial/Ethnic Minority Groups, 1983-1984. *Morbidity and Mortality Weekly Report* 39:31-39.

Kubzansky, L. D., I. Kawachi, and D. Sparrow. 1999. Socioeconomic status, hostility, and risk factor clustering in the normative aging study: Any help from the concept of allostatic load? *Annals of Behavioral Medicine* 21 (4):330-338.

Lara, M., C. Gamboa, M. I. Kahramanian, L. S. Morales, and D. E. Bautista. 2005. Acculturation and Latino health in the United States: a review of the literature and its sociopolitical context. *Annu Rev Public Health* 26:367-97.

LaVeist, Thomas A. 2005. *Minority Populations and Health. An Introduction to Health Disparities in the United States*. San Francisco, CA: Jossey-Bass.

Lechner, Michael. 2000. A note on the common support problem in applied evaluation studies. University of St. Gallen, Discussion Paper No 2001-01.

Liao, Youlian, Richard S. Cooper, Guichan Cao, Ramon Durazo-Arvizu, Jay S. Kaufman, Amy Luke, and Daniel L. McGee. 1998. Mortality Patterns among Adult Hispanics: Findings from the NHIS, 1986 to 1990. *American Journal of Public Health* 88 (2):227-232.

Lokshin, Michael. 2006. Difference–based semiparametric estimation of partial linear regression models. *The Stata Journal* 6 (4):377-383.

Luke, B., M. B. Brown, R. B. Misiunas, V. H. Gonzalez-Quintero, C. Nugent, C. van de Ven, F. R. Witter, R. B. Newman, M. D'Alton, G. D. V. Hankins, D. A. Grainger, and G. A. Macones. 2005. The Hispanic Paradox in Twin Pregnancies. *Twin Research and Human Genetics* 8 (5):532-537.

Manski, Charles. 1995. *Identification Problems in the Social Sciences*. Cambridge, MA: Harvard University Press.

———. 2003. *Partial Identification of Probability Distributions*. New York, NY: Springer.

———. 2007. *Identification for Prediction and Decision*. Cambridge, MA: Harvard University Press.

Markides, K. S., and J. Coreil. 1986. The Health of Southwestern Hispanics: An Epidemiological Paradox. *Public Health Reports* 101:253-265.

Markides, K. S., and K. Eschbach. 2005. Aging, migration, and mortality: Current status of research on the Hispanic paradox. *Journals of Gerontology Series B-Psychological Sciences and Social Sciences* 60:68-75.

Marshall, J. A., R. F. Hamman, J. Baxter, E. J. Mayer, D. L. Fulton, M. Orleans, M. Rewers, and R. H. Jones. 1993. Ethnic differences in risk factors associated with the prevalence of non-insulin-dependent diabetes mellitus. The San Luis Valley Diabetes Study. *Am J Epidemiol* 137 (7):706-18.

McEwen, B.S. 1998. Seminars in Medicine of the Beth Israel Deaconess Medical Center: Protective and Damaging Effects of Stress Mediators. *New England Journal of Medicine* 338 (15):171-179.

McEwen, B.S., and E. Stellar. 1993. Stress and the individual. Mechanisms leading to disease. *Archives of Internal Medicine* 153 (18):2093-2101.

McEwen, Bruce S., and Teresa .E. Seeman. 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* 896:30-47.

McEwen, Bruce S., and John C. Wingfield. 2003. The concept of allostasis in biology and biomedicine. *Hormones and Behavior* 43 (1):2-15.

Mitchell, B. D., S. M. Haffner, H. P. Hazuda, J. K. Patterson, and M. P. Stern. 1992. Diabetes and coronary heart disease risk in Mexican Americans. *Annals of Epidemiology* 2 (1-2):101-106.

Mitchell, B. D., M. P. Stern, S. M. Haffner, H. P. Hazuda, and J. K. Patterson. 1990. Risk factors for cardiovascular mortality in Mexican Americans and non-Hispanic whites. San Antonio Heart Study. *American Journal of Epidemiology* 131 (3):423-33.

Morgan, Stephen L., and Christopher Winship. 2007. *Counterfactuals and Causal Inference. Methods and Principles for Social Research*. New York, NY: Cambridge University Press.

National Center for Health Statistics. 2007. Health, United States, 2007 with chartbook on trends in the health of Americans. Hyatsville, MD: U. S. Department of Health and Human Services.

NCEP. 2001. Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *Journal of the American Medical Association* 285 (19):2486-2497.

NCHS. 2005. National Health and Nutrition Examination Survey, 2005-2006. Overview. Hyattsville, Maryland: National Center for Health Statistics.

OECD. 2007. Health at a Glance 2007. OECD indicators. Paris: Organisation for Economic Co-operation and Development.

Palloni, A., and E. Arias. 2004. Paradox lost: Explaining the Hispanic adult mortality advantage. *Demography* 41 (3):385-415.

Park, YW, S Zhu, L Palaniappan, S Heshka, MR Carnethon, and SB Heymsfield. 2003. The metabolic syndrome: prevalence and associated risk factor findings in the US population from the Third National Health and Nutrition Examination Survey, 1988-1994. *Archives of Internal Medicine* 163:427-436.

Pew Hispanic Center. 2009. *Statistical Portrait of the Foreign-Born Population in the United States,* 2007 [cited March 11 2009]. Available from http://pewhispanic.org/factsheets/factsheet.php?FactsheetID=45.

Portes, Alejandro, and Min Zhou. 1993. The new second generation: segmented assimilation and its variants. *Annals of the American Academy of Political and Social Science* 530:74-96.

Rice, John. 1984. Bandwidth choice for nonparametric regression. *Annals of Statistics* 12:1215-1230.

Robins, James M., and Andrea Rotnitzky. 2001. Comment on 'Inference for Semiparametric Models: Some Questions and an Answer?' by Bickel and Kwon. *Statistica Sinica* 11:920-936.

Rosenbaum, Paul R., and Donald B. Rubin. 1983. The Central Role of the Propensity Score in Observational Studies for Causal Effects *Biometrika* 70 (1):41-55.

Rubalcava, L. N., G. M. Teruel, D. Thomas, and N. Goldman. 2008. The healthy migrant effect: new findings from the Mexican Family Life Survey. *Am J Public Health* 98 (1):78-84.

Rubin, Donald B. 1974. Estimating Causal Effects of Treatment in Randomized and Non-Randomized Studies. *Journal of Educational Psychology* 66:688-701.

———. 1977. Assignment to Treatment Group on the Basis of a Covariance. *Journal of Educational Statistics* 2 (1):1-26.

———. 1997. Estimating Causal Effects from Large Data Sets Using Propensity Scores. *Annals of Internal Medicine* 127 (8S):757-763.

Rubin, Donald B., and Neal Thomas. 1996. Matching Using Estimated Propensity Scores: Relating Theory to Practice. *Biometrics* 52 (1):249-264.

———. 2000. Combining Propensity Score Matching With Additional Adjustments for Prognostic Covariates. *Journal of the American Statistical Association* 95 (450):573-585.

Schnedler, Wendelin. 2005. Likelihood estimation for censored random vectors. *Econometric Reviews* 24 (2):195-217.

Seeman, T.E. , B.H. Singer, J.W. Rowe, Horwits R.I., and B.S. McEwen. 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., B.S. McEwen, J.W. Rowe, and B.H. Singer. 2001. Allostatic load as a marker of cumulative biological risk: MacArthur studies of successful aging. *Proceedings of the National Academy of Sciences of the United States of America* 98:4770-4775.

Smith, D. P., and B. S. Bradshaw. 2006. Rethinking the Hispanic paradox: Death rates and life expectancy for US non-Hispanic white and Hispanic populations. *American Journal of Public Health* 96 (9):1686-1692.

Sorlie, P. D., E. Backlund, N. J. Johnson, and E. Rogot. 1993. Mortality by Hispanic status in the United States. *Journal of the American Medical Association* 270 (20):2464-2468.

Stephen, Elizabeth H., Karen Foote, Gerry E. Hendershot, and Charlotte A. Schoenborn. 1994. Health of the foreign-born population: United States, 1989-90. *Advance Data From Vital and Health Statistics* (241):1-10.

Sterling, Peter, and Joseph Eyer. 1988. Allostasis: A New Paradigm to Explain Arousal Pathology. In *Handbook of Life Stress, Cognition and Health*, edited by S. Fisher and J. Reason. New York: John Wiley.

Tobin, James. 1958. Estimation for relationships with limited dependent variables. *Econometrica* 26 (1):24-36.

Turra, C. M., and I. T. Elo. 2008. The Impact of Salmon Bias on the Hispanic Mortality Advantage: New Evidence from Social Security Data. *Popul Res Policy Rev* 27 (5):515-530.

Turra, Cassio M., and Noreen Goldman. 2007. Socioeconomic Differences in Mortality Among U.S. Adults: Insights Into the Hispanic Paradox. *Journals of Gerontology Series B-Psychological Sciences and Social Sciences* 62B (3):S184-S192.

U.S. Census Bureau. 2009. *American FactFinder* 2009 [cited March 29 2009]. Available from http://factfinder.census.gov/.

UNICEF. *At a glance: Mexico*. United Nations Children's Fund 2009 [cited March 28, 2009. Available from http://www.unicef.org/infobycountry/mexico_statistics.html.

Uretsky, Matthew Cory, and Sally G. Mathiesen. 2007. The effect of years lived in the United States on the general health status of California's foreign-born populations. *Journal of Immigrant Health* 9:125-136.

Vega, W. A., B. Kolody, R. Valle, and J. Weir. 1991. Social Networks, Social Support, and Their Relationship to Depression among Immigrant Mexican Women. *Human Organization* 50 (2):154-162.

Vega, William A., and H. Amaro. 1994. Latino Outlook: Good Health, Uncertain Prognosis. *Annual Review of Public Health* 15:39-67.

Williams, David R., and Chiquita Collins. 1995. US Socioeconomic and Racial Differences in Health: Patterns and Explanations. *Annual Review of Sociology* 21:349-386.

Wilson, P. W., R. B. D'Agostino, D. Levy, A. M. Belanger, H. Silbershatz, and W. B. Kannel. 1998. Prediction of coronary heart disease using risk factor categories. *Circulation* 97 (18):1837-47.

Yatchew, Andrew. 1988. Some tests of nonparametric regression models. In *Dynamic Econometric Modeling, Proceedings of the Third International Symposium in Economic Theory and Econometrics*, edited by W. Barnett, E. Berndt and H. White. Cambridge: Cambridge University Press.

———. 1999. Differencing methods in nonparametric regression: simple techniques for the applied econometrician. University of Toronto.

———. 2003. *Semiparametric regression for the applied economist*. New York, NY: Cambridge University Press.

Zsembik, Barbara A., and Dana Fennell. 2005. Ethnic Variation in Health and the Determinants of Health Among Latinos. *Social Science & Medicine* 61:53-63.