Race/Ethnicity, Nativity and Morbidity Trajectories*

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Abstract

Cross-sectional studies have shown that morbidity levels vary by race/ethnicity and nativity, however, little is known about racial/ethnic/nativity disparities in the rate of accumulation of lifethreatening chronic conditions (e.g. cancer, diabetes, heart disease, hypertension, and stroke). This study integrates demographic and developmental perspectives and methods by utilizing a life course framework, seven waves of data from the Health and Retirement Study, and growth curve models to investigate race/ethnic/nativity inequalities in morbidity age-trajectories. We test whether health inequalities decrease, remain stable, or increase with age, consistent with agingas-leveler, persistent inequality, and cumulative disadvantage hypotheses, respectively. In addition, we examine the extent to which group differences in life course capital (e.g. social origins, SES, marriage, and health behaviors) account for health disparities. Results reveal that both chronic condition levels and rates of accumulation vary by race/ethnicity/nativity, net of death and dropout attrition. Native-born blacks and Hispanics have worse morbidity trajectories than their white counterparts, and there is clear evidence of an immigrant health advantage across all racial/ethnic groups. While aging-as-leveler, persistent health inequality and cumulative disadvantage hypotheses are typically framed as competing hypotheses, support for each of them is found, depending on the life stages and groups being analyzed.

Introduction

Overall, the health of older Americans is improving, yet large health disparities between different demographic groups exist, and these disparities do not appear to be narrowing over time (Manton and Gu 2001; Martin et al. 2007). Although previous research has found evidence that health levels vary by race/ethnicity and nativity, it remains unclear how life course health trajectories differ across racial/ethnic/nativity subgroups. Understanding how both race/ethnicity and nativity shape mid- and late-life health is particularly important, as people of color and immigrants will comprise an increasing proportion of older adults in the U.S., owing to several population processes including growth in racial/ethnic diversity, unprecedented high levels of immigration in recent decades and population aging. Between 2010 and 2030, the number of adults aged 65 and over is projected to jump from 40 million to 72 million, an increase from 13 percent to 20 percent of the population, with significant increases in the number of foreign born persons, especially immigrants from Latin America and Asia (He 2002; He et al. 2005). Moreover, whereas the proportion non-Hispanic white (hereafter "white") older adults is expected to decline by 13 percent between 2003 and 2030, the proportions of non-Hispanic black (hereafter "black") and Hispanic older adults are expected to increase by 25 and 83 percent, respectively (He et al. 2005).

An abundance of empirical research shows that health varies by race/ethnicity, with people of color often exhibiting worse health outcomes than whites. Compared to whites, blacks and Hispanics have worse physical and functional health (Blackwell, Collins, and Coles 2002; CDC 2004; Kelley- Moore and Ferraro 2004; Markides, Coreil, and Rogers 1989; Markides et al. 1997; Schoenbaum and Waidmann 1997; Stern and Haffner 1990; Taylor 2008). However, despite their structural and socioeconomic disadvantages compared to whites, Hispanics have

comparable or better adult health on several measures of health including cancer, heart disease and mortality, a phenomena known as the '*Hispanic paradox*' (Elo and Preston 1997; Hummer et al. 1999; 2000; Markides and Coreil 1986; Markides et al. 1997; Palloni and Arias 2004; Singh and Siahpush 2001). Also, within racial/ethnic groups, there is a robust relationship between nativity and health. On average, immigrants have lower rates of chronic conditions than their native-born counterparts (*Dey and Lucas 2006; Jasso et al. 2004; Read and Emerson2005; Sing and Siahpush 200)*. Racial/ethnic and nativity differences in health levels are welldocumented; however, to-date no study has examined how the rate of accumulation of lifethreatening chronic conditions (e.g. cancer, diabetes, heart disease, hypertension, and stroke) varies by race/ethnicity and nativity.

Prior research on race/ethnicity, nativity and health has tended to examine betweenperson differences, as is the case with most sociological and demographic research. In contrast, developmental life course research aims to explain long-term, intra-individual patterns of stability and change. Whereas a between-person design is useful for investigating group differences in health, a within-person approach focuses on patterns of intra-individual health decline or improvement with age (George, forthcoming). Importantly, this study integrates demographic and developmental perspectives (Alwin, Hofer and McCammon 2006) by using both between- and within-person approaches to investigate how morbidity trajectories vary across racial/ethnic/nativity groups among blacks, Hispanics and whites born in the U.S. and abroad.

At what age do health disparities emerge? Do health disparities shrink, remain stable or grow with age, and by how much? We empirically test three hypotheses on the dynamic nature of intracohort inequalities in health across the life course: the *aging-as-leveler* (Dowd and

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Bengtson 1978), *persistent inequality* (Ferraro and Farmer 1996; Henretta and Campbell 1976), and *cumulative disadvantage* hypotheses (Dannefer 1987; 2003; DiPrete and Eirich 2006; Ferraro, Shippee and Schafer 2009; O'Rand 1996; Willson, Shuey and Elder 2007) posit that, with age, racial/ethnic disparities in health decrease, remain stable and increase, respectively. This study examines racial/ethnic/nativity disparities in morbidity and whether group differences are attenuated, persistent or accentuated with age.

The lack of knowledge about whether and how age-trajectories of health vary by both race/ethnicity and nativity is due to the limitations of the methodological and conceptual choices of prior studies. First, many studies have used cross-sectional data to infer aging processes. However, longitudinal data measuring health at numerous points in the life course is necessary to understand diverse aging experiences. Second, even among longitudinal studies of health disparities with respondents of dissimilar ages, most have used survey waves instead of age as the time metric, which obscures group differences in age-related changes in health (Adkins et al. 2009). Third, many explore transitions over relatively short periods. Fourth, although Hispanics and immigrants comprise a large and growing proportion of the population, few studies have investigated their health trajectories. Fifth, many studies fail to account for nonrandom attrition (e.g., mortality selection), which may bias estimates of disparities in health dynamics given group difference in mortality rates (see Beckett 2000; Herd 2006; Hummer et al. 2000; Palloni and Arias 2004). Sixth, although, we know that health is shaped by the interaction of various forms of life course capital (e.g., human, economic, and social capital) over time (O'Rand 2006), no prior studies have examined how these forms of capital may mediate racial/ethnic/nativity disparities in morbidity trajectories.

The proposed study extends previous research on health disparities by drawing on life course theory, and by both conceptualizing and modeling chronic condition trajectories as dynamic life course processes. Specifically, this study 1) investigates racial/ethnic/nativity differences in intra-individual health changes on numerous occasions, 2) analyzes intra-individual change as a function of age, 3) examines morbidity trajectories over an extended period (ages 51-73), 4) includes native- and foreign-born blacks, Hispanics and whites, 5) accounts for death and dropout attrition, and 6) investigates the extent to which group differences in life course capital account for racial/ethnic/nativity disparities in morbidity intercepts (initial levels) and slopes (rates of change). In addition, this study examines the whether length of residence in the U.S. is associated with morbidity levels and rates of change among immigrants. Overall, results show that examining racial/ethnic/nativity disparities in health trajectories is useful for understanding intra-cohort inequality dynamics and diverse aging experiences.

Background

Racial/Ethnic Health Disparities

Race/ethnicity and social location shape inequality in access to resources, exposure to risks and consequently health. Racially stratified opportunity structures result in the accumulation of disadvantages for people of color and disparate aging experiences. Life course perspectives emphasize the notion that health is shaped by the interaction of various forms of life course capital over time (O'Rand 2006). A large body of research has documented the protective effects of social factors including childhood SES (Hamil-Luker and O'Rand 2007), educational attainment (Dupre 2007; Ross and Wu 1996), income (Mirowsky and Hu 1996; Rogers 1992), wealth (Bond et al. 2003; Smith 1999), and marriage (Waite 1995; Williams and Umberson 2004); because blacks and Hispanics are disadvantaged relative to whites on these key social

determinants of health, one would expect them to exhibit worse health profiles than whites (Brown and Warner 2008; Newman 2002; Shuey and Willson 2008).

Indeed, racial/ethnic disparities in health have been well-documented over the last several decades (Williams and Collins 1995). Blacks experience worse health than whites on an array of health outcomes. For example, black adults have a higher prevalence of diabetes, arthritis, hypertension, strokes, and heart disease than their white counterparts (Blackwell, Collins, and Coles 2002; Reed, Darity, and Robertson 1993; Schoenbaum and Waidmann 1997). Blacks also report having worse self-rated health (Farmer and Ferraro, 2005; Shuey and Willson 2008) and higher rates of disability than whites (Kelley- Moore and Ferraro 2004; Manton and Gu 2001; Taylor 2008). Moreover, compared to whites, blacks have a shorter life expectancy, have higher all-cause mortality rates, and mortality due to diseases of the heart, malignant neoplasms, and cerebrovascular diseases (Elo and Preston 1997; National Center for Health Statistics 2003). Overall, previous research suggests that racial/ethnic socioeconomic inequality accounts for much, but not all of racial/ethnic disparities in health (Crimmins, Hayward and Seeman 2004; Hayward, et al. 2000). Mounting empirical evidence shows that perceptions of racial discrimination negatively affect health (Brondolo et al. 2003; Krieger 2000; Paradies 2006; Williams and Neighbors 2001), and that minorities' higher levels of perceived discrimination result in elevated levels of stress and contribute to health disparities (Geronimous 1996; Hummer 1996; Krieger 2000; Williams et al. 2003).

The picture of Hispanic health and well-being is complex. On the one hand, Hispanics exhibit higher rates of hypertension, kidney disease and diabetes than whites (CDC 2004; Markides, Coreil, and Rogers 1989; Stern and Haffner 1990). Additionally, compared to whites, Hispanics consistently report worse self-assessed health (Angel and Angel 1996; Hummer et al.

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2004) and poorer functional health, indicated by their higher rates of disability (Markides et al. 1997). On the other hand, Hispanics have been shown to have a health advantage over whites in terms heart disease and cancer (Markides et al. 1997). Moreover, Hispanics have lower all-cause mortality rates than whites in mid- and later-life (Elo and Preston 1997; Hummer et al. 1999; 2000; Marides and Coreil 1986; Markides et al. 1997; Singh and Siahpush 2001; 2002). Because of the positive association between SES and health, and the fact that Hispanics have less human and economic capital than whites, one would expect Hispanics to have worse health than whites. The counterintuitive yet increasingly common finding that Hispanics have lower morbidity and mortality rates than whites despite their disadvantaged socioeconomic positions is known as the 'Hispanic paradox' (Marides and Coreil 1986).

Several competing theories have been proposed to explain Hispanic's surprisingly good health given their structural disadvantages relative to whites, which will be discussed below. At the core of each of these explanations is a composition argument. Specifically, the Hispanics population in the U.S. is thought to have better health than expected as a result of disproportionately high rates of immigration: nearly half of the Hispanics residing in the U.S. are foreign-born, compared to approximately 6% of blacks and 5% of whites (Hummer et al. 1999). Immigration rates have significant consequences for overall health profiles of racial/ethnic groups because health varies by nativity.

Nativity and Health

Overall, immigrants exhibit better health than their U.S.-born counterparts. For instance, immigrants have lower rates of chronic conditions such as cancer, diabetes, heart disease, hypertension, and stroke (Dey and Lucas 2006; Jasso et al. 2004; Read and Emerson 2005; Singh and Siahpush 2002), and fewer functional disabilities (Read and Emerson 2005; Stephen et al.

1994). Moreover, within racial/ethnic groups, immigrants have lower mortality rates than the native-born population (Hummer et al. 1999; 2000; Markides and Eschbach 2005; Palloni and Arias 2004; Sing and Siahpush 2001;2002; Turra and Goldman 2007). The immigrant health advantage appears to apply to all racial/ethnic groups, though the magnitude of the nativity effect may vary by race/ethnicity (Hummer et al. 1999; Palloni and Arias 2004; Read and Emerson 2005; Sing and Siahpush 2001).

Three central hypotheses have been proposed to explain immigrants' better health vis-àvis native-born populations. Although most of the theorizing on explanations for an immigrant health advantage has been based on Hispanics, these explanations may apply more generally, across racial/ethnic groups. The *healthy migrant hypothesis* posits that migration is positively selective on an array of characteristics and that those who migrate to the U.S. are healthier than their native-born counterparts (Jasso et al. 2004; Landale, Oropesa and Gorman 2000). Several mechanisms may lead to immigrants being selected for good health including health screenings of prospective immigrants, and the fact that individuals who migrate often do so for occupational reasons and to improve their lives—both of which are associated with good health (Markides 2001; Markides and Eschbach 2005). Moreover, gaining entry to the U.S. without documentation can be an arduous process, requiring good health. Akresh and Frank (2008) found strong evidence of positive health selection of immigrants of all racial/ethnic backgrounds.

The *salmon bias hypothesis* asserts that the relatively good health and low rates of adult mortality among Hispanics in the U.S. is a statistical artifact, owing to the return migration of migrants in poor health. Indeed, Paloni and Arias (2004) showed that accounting for emigration explained foreign-born Mexicans' health advantage over non-Hispanic whites (see also Markides and Eschbach 2005). In their review of empirical evidence over a 20-year period, Franzini and

colleagues (2001) conclude that selective immigration and emigration likely play roles in but do not completely account for the immigrant health advantage, suggesting that other factors such as health behaviors likely play a role.

The *health behavior hypothesis* posits that foreign-born persons have better health than their native-born counterparts because they have more favorable health behaviors and risk profiles (Abraido-Lanza et al. 1999; Abraido-Lanza, Chao, and Florez 2005; Hummer et al. 1999; Markides and Coreil 1986). Indeed, relative to their U.S.-born counterparts, black, Hispanic, and white immigrants are less likely to smoke, drink alcohol excessively, or be obese (Dey and Lucas 2006; Hummer et al. 1999; Singh and Siahpush 2002). According to the acculturation hypothesis, greater exposure to mainstream society leads to the erosion of immigrants' initial advantages in health behaviors and, over time, the health of immigrants should decline. Empirical evidence suggests that although acculturation (typically measured by nativity and length in the U.S.) results in the adoption unhealthy behaviors and poorer health (Clark and Hofsess 1998; Vega and Amaro 1994), it also is associated with higher levels of exercise (Abraido-Lanza et al. 2005; Crespo et al. 2001); thus, evidence for the health behavior and acculturation hypotheses is mixed. Overall, racial/ethnic and nativity differences in health at a point in time are well-documented, yet less is known about racial/ethnic/nativity disparities in age-trajectories of health.

Race/Ethnicity, Nativity and Health Trajectories

Health trajectories are in influenced by the accumulation risks and resources over time (Ferraro et al. 2009). As noted above, the lives of racial/ethnic minorities and immigrants evolve differently than their white and U.S.-born counterparts with respect to human, economic and social capital; consequently, morbidity trajectories are likely to vary across racial/ethnic/nativity

groups. Three competing hypotheses have emerged to explain intracohort inequality as the cohort ages. The aging-as-leveler hypothesis posits that aging involves negative health consequences for both advantaged and disadvantaged populations, and that those with health advantages earlier in life have the most to lose in terms of health decline (Dowd and Bengtson 1978). Therefore, group differences in health should attenuate later in life. The *persistent* inequality hypothesis asserts that intracohort stratification is constant as the cohort ages (Henretta and Campbell 1976). Demographic and socioeconomic factors have consistent effects on wellbeing over time. According to this hypothesis, racial/ethnic/nativity inequalities in health are expected to remain stable with age (e.g., Clark and Maddox 1992; Ferraro and Farmer 1996; Taylor 2008). Alternatively, the *cumulative advantage/disadvantage* hypothesis posits that intracohort inequality increases as the cohort ages (Dannefer 1987; 2003; DiPrete and Eirich 2006; O'Rand 1996; Willson et al. 2007). On the one hand, advantages are magnified with age through a "cumulative process of differentiation" (Dannefer 1988: 16), whereby individuals with an initial advantage have increasing access to resources and exposure to opportunities (Ferraro et al. 2009). On the other hand, disadvantages early in life shape social and developmental pathways and lead to subsequent disadvantages and exposure to risk (O'Rand and Hamil-Luker 2005). Thus, racial/ethnic/nativity health disparities are hypothesized to increase with age, consistent with several studies showing that black-white disparities in health are accentuated over time (Ferraro, Farmer, and Wybraniec 1997; Kelley-Moore and Ferraro 2004).

The relatively few studies on race differences in adult health trajectories have focused on disparities in self-rated health and disability between blacks and whites (Clark and Maddox 1992; Ferraro and Farmer 1996; Ferraro et al. 1997; Kelley-Moore and Ferraro 2004; Shuey and Willson 2008; Taylor 2008; Yao and Robert 2008), excluding Hispanics and neglecting other

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important health outcomes such as chronic conditions. Furthermore, no studies to-date have examined nativity differences in health trajectories across racial/ethnic groups. The present study aims to fill these gaps in the literature by investigating racial/ethnic/nativity differences in morbidity trajectories among black, Hispanic, and white U.S.- and native-born populations. Additionally, because we know that health is shaped by various forms of life course capital (O'Rand 2006), this study assesses the extent to which group differences in social origins, SES, marriage, and health behaviors account for racial/ethnic inequalities in health trajectories.

Importantly, previous research on racial/ethnic health disparities over time may have been biased due to inattention to nonrandom attrition in panel studies. Longitudinal data measuring health at three or more times is necessary to estimate trajectories of health. A drawback of longitudinal data, however, is nonrandom attrition due to refusal, loss to follow-up, institutionalization, and death (Beckett 2000; Lillard and Panis 1998). Racial/ethnic minorities, lower SES individuals and those in poorer health have higher rates of mortality; thus, the surviving sample is likely to be disproportionately healthy, wealthy and white (Hummer, Rogers and Eberstein 1998; Hummer et al. 2000). Studies that fail to account for attrition are likely to underestimate inequalities in health changes (Kelley-Moore and Ferraro 2004; Shuey and Willson 2008; Taylor 2008). To minimize selection bias, the present study accounts for both death and dropout attrition.

Data and Methods

Sample

Data from waves 1 through 7 of the Health and Retirement Study (HRS) are utilized. The target population for the HRS includes all English or Spanish-speaking adults in the contiguous United States, aged 51-61 in 1992 (spouses of respondents were interviewed regardless of age-

eligibility), who reside in households. Respondents were re-interviewed biennially between 1992 and 2004. Blacks and Hispanics were oversampled to allow independent analysis of racial groups. Only a minor proportion of individuals are institutionalized at the target ages of this study; respondents remain in the study in the event that they are institutionalized between 1992 and 2004. Nonetheless, levels of morbidity may be somewhat understated given the exclusion of institutionalized populations at baseline. Analyses are based on 9,011 native- and foreign-born black, Hispanic and white respondents aged 51 to 61 in 1992. Other racial/ethnic groups are excluded due to extremely small sample sizes.

Dependent Variable

Serious chronic conditions. Respondent answered the question, "Has a doctor ever told you that you have (had a) [condition]." Life-threatening conditions examined in this study include cancer, diabetes, heart disease, hypertension, and stroke. A summary measure of the total number of conditions ever diagnosed was constructed, ranging between 0 and 5, consistent with previous research (e.g. Farmer and Ferraro 2005). While one might be concerned about the quality of self-reported data on chronic conditions, previous research has shown there to be high levels of agreement between self-reported and physician-evaluated morbidity for serious conditions among older adults (Simpson et al. 2004; Skinner et al. 2005). Moreover, selfreported conditions appear to have better predictive validity than physician-evaluated morbidity on subsequent health declines (Ferraro and Su 2000).

Demographic Variables

Six dummy variables index *racial/ethnic/nativity groups:* 1) white, native-born (omitted), 2) white, foreign-born, 3) black, native-born, 4) black, foreign-born, 5) Mexican American, native-born, and 6) Mexican American, foreign-born. Individuals are classified as Mexican

origin if they report being Mexican on a question concerning one's ethnicity. Respondents are considered white if they do not report Hispanic ethnicity and report being white; similarly, individuals are classified as being black if they report being black and non-Hispanic. Supplemental analyses explored disaggregating other Hispanic populations into subgroups by ethnicity (e.g. Cuban, Puerto Rican, other Hispanic) and nativity; however, growth curves could not be estimated for these subgroups due to small sample sizes, therefore, these groups are not included (see Brown and Warner 2008). To be consistent with prior research racial/ethnic/nativity differences in health, native-born whites are the reference group (e.g. Hummer et al. 1999; Paloni and Arias 2004; Singh and Siahpush 2001; 2002). Gender is measured by a dummy variable (1=female; 0=male). Both age and age^2 are included in the analysis to capture health changes with age. A dummy indicator for birth cohort distinguishes between respondents born between 1931-36 from those born between 1937-41. Length of residence in the U.S. is measured in years for immigrants and ranges from 0 to 61 years. To account for differential rates of dropout and death attrition, a measure of the number of waves interviewed (1-7) and a dummy indicator of whether the respondent died during the observation are included in the models.

Social Origins

A substantial body of literature has documented a link between disadvantage in early-life and poor health in later-life (Ben-Shlomo and Kuh 2002; Elo and Preston 1992; O'Rand and Hamil-Luker 2005). Childhood SES measures include indicators of, *whether the family was poor*, and the respondents' *mother's and father's educational attainment* (less than high school=1; 0 otherwise).

Socioeconomic Variables

Adult SES indicators include respondent's *educational attainment* (in years), *logged household earnings* (includes monies from wages and salaries for both spouses in the case of marriage), *logged household social security income*, and *logged household net worth* (total assets – total liabilities), and *health insurance coverage*.

Health Behaviors

Indicators of respondents' health behaviors include measures of *obesity* (1= BMI >30; 0=otherwise), *smoking history* (1=ever smoked; 0=otherwise), whether they *currently smoke*, and whether they *drink heavily* (1= 3+ drinks/day; 0=otherwise).

Family and Regional Context

Marriage is known to be protective of health (Umberson 1987; Williams and Umberson 2004) and vary by race/ethnicity (Cherlin 1992), therefore, a dummy variable for *marital status* (unmarried=1; 0 otherwise). A series of dummy variables indicate the region in which respondents currently reside (e.g., Northeast (ref.), Mid West, South, or West).

Analytic Strategy

Developmental and life course theory posit age as the appropriate metric in the study of health changes. However, the HRS is organized by wave, not by age. Due to considerable age heterogeneity within each wave of the HRS (a range of 11 years), it was necessary to reorganize the data from wave to age in order to accurately test the hypotheses. This transformation is referred to as an accelerated longitudinal design, which is commonly used in developmental, survey research (see Herd 2007; Willson et al. 2007; Shuey and Willson 2008; Yang 2007).

Random coefficient growth curves were modeled within a mixed model (i.e., hierarchical linear model) framework to investigate racial/ethnic/nativity differences in health trajectories between the ages of 51 and 73. These models are well-suited for the assessment of individual change with age (Raudenbush and Byrk 2002). A hierarchical strategy is used, where repeated observations (Level 1) are nested within respondents (Level 2).

Growth curve models generate individual trajectories that are based on estimates of person-specific intercepts (initial value) and slopes (rate of change) that describe intra-individual patterns of change in health as a function of age. Comparisons of nested likelihood ratio tests (LRTs) of various shapes of health trajectories (e.g. linear, quadratic or cubic models), suggested that a quadratic growth curve with random intercepts and random linear and quadratic age slopes provided the best fit to the data. After developing an accurate model of the unconditional trajectory, independent variables are added to the model in order to examine the extent to which they explain racial/ethnic disparities in health. To estimate the effects of the covariates on the trajectory slope, covariates are interacted with age and age-squared. All variables are timevarying except measures of race/ethnicity/nativity, gender, cohort, social origins, and smoking history. Covariates are mean-centered to facilitate model interpretation. When independent variables are not mean-centered, the fixed effects of age and age² represent the trajectory shape for respondents with values of zero on all covariates; however, when independent variables are mean-centered, the fixed effects of age and age² represent the mean trajectory shape for individuals with average values on the continuous measures and zero values on the dummy variables, which is more substantively interesting (Singer and Willett 2003).

The following represents the growth curve models for the number of chronic conditions of respondent *i* at time *t*:

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Level 1 model:

Number of Chronic Conditions_{ti} = $\pi_{0i} + \pi_{1i}Age_{ti} + \pi_{2i}Age_{ti}^2 + e_{ti}$

where π_{0i} captures the number of chronic conditions at age 51 for individual *i*; π_{1i} is the individual- and time-specific growth rate, representing the change in number of chronic conditions with each additional year of age, and π_{2i} is the quadratic or rate of accleration for respondent *i* at time *t*.

Level 2 model:

- $\pi_{0i} = \beta_{00} + \beta_{01} \text{Foreign-Born Whites}_i + \beta_{02} \text{Native-Born Blacks}_i + \beta_{03} \text{Foreign-Born Blacks}_i + \beta_{04} \text{Native-Born Mexican Americans}_i + \beta_{05} \text{Foreign-Born Mexican Americans}_i + \beta_{06} \text{Female}_i + \beta_{07} \text{Older Cohort}_i + \beta_{08} \text{Number of Waves}_i + \beta_{09} \text{Died}_i + r_{0i}$
- $\pi_{1i} = \beta_{10} + \beta_{11} \text{Foreign-Born Whites}_i + \beta_{12} \text{Native-Born Blacks}_i + \beta_{13} \text{Foreign-Born Blacks}_i + \beta_{14} \text{Native-Born Mexican Americans}_i + \beta_{15} \text{Foreign-Born Mexican Americans}_i + \beta_{16} \text{Female}_i + \beta_{17} \text{Older Cohort}_i + \beta_{18} \text{Number of Waves}_i + \beta_{19} \text{Died}_i + r_{1i}$
- $\pi_{2i} = \beta_{20} + \beta_{21} \text{Foreign-Born Whites}_i + \beta_{22} \text{Native-Born Blacks}_i + \beta_{23} \text{Foreign-Born Blacks}_i + \beta_{24} \text{Native-Born Mexican Americans}_i + \beta_{25} \text{Foreign-Born Mexican Americans}_i + \beta_{26} \text{Female}_i + \beta_{27} \text{Older Cohort}_i + \beta_{28} \text{Number of Waves}_i + \beta_{29} \text{Died}_i + r_{2i}$

where the coefficients β_{pq} are the effects of individiual characteristics on the π_{0i} intercept and π_{1i} , π_{2i} slope parameters, and r_{pi} are error terms. Ordinary least squares models were utilized, though the Gaussian distribution assumption is violated due to the slightly skewed distribution of the outcome (skew of the chronic conditions measure =.98). Additional analyses (not shown) conducted using log- and square root-transformed outcomes as well as Poisson models produced comparable results, suggesting that the results are not sensitive to the normality assumption (see Haas 2007; Herd 2006).

Number of waves respondents were interviewed varied by race/ethnicity/nativity, with native-born blacks, foreign-born blacks, native-born Mexican Americans, foreign-born Mexican Americans, native-born whites and foreign-born whites averaging 5.5, 5.5, 5.7, 5.5, 5.9 and 5.8 interviews, respectively. Mortality rates also differed across sub-groups. Morality rates for native-born blacks, foreign-born blacks, native-born Mexican Americans, foreign-born Mexican Americans, native-born whites and foreign-born whites were 23%, 5%, 17%, 9%, 13% and 11%, respectively (see Table 1 for information on statistically significant differences in dropout and death attrition). Given racial/ethnic/native differences in number of waves interviewed and mortality rates, and the fact that attrition is related to health, conventional methods that incorporate only respondents with complete cases lead to biased estimates of disparities in health trajectories. To avoid such biases, this study utilizes hierarchical linear models in tandem with maximum likelihood estimation, which has the advantage of being able to incorporate all respondents who have been observed at least once including those who attrit during the observation period in the sample, and is consistent with the approaches of recent high-quality studies on disparities in health trajectories (see Herd 2007; Shuey and Willson 2008; Taylor 2008; Willson et al. 2007; Yang 2007). Under these circumstances, Raudenbush and Bryk (2002) note that 1) the data may be assumed to be missing at random (MAR), meaning that the probability of missing a time point is independent of missing data given the observed data, and 2) this is a reasonable assumption when the observed data include variables related to both missingness and the dependent variable. Assuming the data are MAR, because all of the data are used in the analysis and a fully efficient estimation procedure (maximum likelihood) is utilized, estimates from the growth curve models are asymptotically unbiased (Raudenbush and Bryk 2002). Furthermore, to account for racial/ethnic/nativity differences in death and dropout

attrition, indicators of death and number of waves the respondent was interviewed are included in the models

Although one could argue that the independent variables should be lagged such that the dependent variable at wave t is predicted by the covariates at wave t-1, the focus of this study is not on establishing a causal relationship between the time-varying covariates and health, but rather on determining the extent to which racial/ethnic differences in life course factors mediate racial/ethnic inequalities in health trajectories. For this reason and the fact that lagging the independent variables reduces the sample size by more than 15%, the independent and dependent variables are modeled concurrently. Importantly, ancillary analyses (not shown) reveal that results are findings from this study are robust after lagging the covariates.

[Table 1, about here]

Results

Descriptive Statistics

Table 1 presents descriptive statistics (percentage distribution or mean) for each of the covariates separately by race/ethnicity/nativity. Findings show racial/ethnic/nativity differences in an array of factors. As expected, people of color are generally disadvantaged, relative to whites, in terms of measures of life course capital. For example, compared to native-born whites, Blacks and Mexican Americans born in the U.S. are more likely to have been in poverty during childhood, are less likely to have parents who graduated from high school, have less education, earnings and wealth, are less likely to be employed, and are more likely to be uninsured and unmarried. Interestingly, whereas foreign-born blacks and whites have more favorable social origins and SES than their native-born counterparts, the reverse is true with regards to Mexican Americans. Consistent with the health behaviors hypothesis, within racial/ethnic groups, foreign-

born respondents are less likely than those who are native-born to exhibit risky health behaviors. Overall, descriptive results reveal advantages for whites and immigrants, with few exceptions.

[Table 2, about here]

Chronic Condition Trajectories

Table 2 provides estimates from growth curve models of serious chronic conditions. Overall, results in Table 2 show that morbidity intercepts and slopes vary considerably by both race/ethnicity and nativity. Controlling for gender, cohort, and death and dropout attrition, Model 1 indicates that, on average, native-born whites have .389 chronic conditions at age 51 and their number of conditions increases with age at an accelerating rate, indicated by the significant and positive coefficients for the linear and quadratic slopes (.026 and .002, respectively); supplemental calculations based on the coefficients in presented in Model 1 reveal that they have 1.964 conditions by the time they reach the age of 73.

[Figure 1, about here]

Figure 1 shows disparities in morbidity between race/ethnic/nativity groups and nativeborn whites across ages 51-73. Compared to native-born whites, native-born blacks have .229 more conditions at age 51, a steeper linear slope and a flatter quadratic slope, resulting in a health gap that initially increases with age, peaking at age .349 at age 62 before waning back to .229 by age 73 (see Table 2 and Figure 1). Foreign-born blacks are at parity with native-born whites in terms of baseline levels of chronic conditions, though a foreign-born black health disadvantage emerges during the 50s and swells to .428 at age 64 and subsequently declines to .189 at age 73. Importantly, the health patterns of native- and foreign-born blacks vis-à-vis native-born whites are in line with both cumulative disadvantage and aging-as-leveler hypotheses. Whereas native-born Mexican Americans have .126 more chronic conditions than their white counterparts at age 51, persons residing in the U.S who were born in Mexico have .149 fewer conditions than native-born whites (Model 1); these inequalities appear to be relatively stable between ages 51 and 73. Consistent with the immigrant health advantage and persistent inequality hypotheses, relative to native-born whites, foreign-born whites have .152 fewer chronic conditions at midlife, a gap that remains constant throughout the observation period (Figure 1). Supplemental analysis (not shown) revealed further evidence of an immigrant health advantage, with foreign-born blacks and Hispanics exhibiting significantly lower morbidity intercepts and similar slopes vis-à-vis their native-born counterparts, as predicted by the persistent health inequality hypothesis.

Other demographic factors such as gender, cohort and mortality are also associated with morbidity trajectories Compared to men, Women appear to have higher levels of chronic conditions at age 51, though this effect is only marginally significant (p<.10). Respondents born between 1931 and 1936 have flatter linear slopes but steeper quadratic slopes than those born between 1937 and 1941. As one might expect, respondents who died during the observation period had more chronic conditions at baseline and experienced more rapid condition growth with age than those who did not, which underscores the importance of accounting for differential mortality rates.

Model 2 adds controls for social origins, human capital, adult SES, marriage, health behaviors, and region. Overall, there is little evidence that racial/ethnic/nativity differences in these factors mediate group differences in morbidity trajectories given the minimal changes in the magnitude and statistical significance of the coefficients for race/ethnicity/nativity between models 1 and 2. A noteworthy exception is that native-born Mexican Americans no longer exhibit higher morbidity intercepts than native-born whites, net of controls. Also, the size of the foreign-born Mexican American advantage in chronic conditions at age 51 increased by 66 percent (from -.149 to -.248) between Models 1 and 2. In both instances, ancillary analyses revealed that the inclusion of SES measures primarily drove improvements in the health of foreign-born Mexican Americans relative to native-born whites. While immigrants have more favorable health behaviors than U.S.-born persons, controlling for health behaviors did not abate the immigrant health advantage. Furthermore, after accounting for the possibility of return migration by controlling for dropout attrition, immigrants continue to exhibit better morbidity trajectories. Thus, results do not support either the healthy behavior or salmon bias hypotheses, suggesting that other factors such as positive health selection of immigration may explain the immigrant health advantage.

Consistent with a large body of research documenting the protective effects of education on health (Dupre 2007; 2008; Ross and Wu 1995), greater levels of education are associated with lower chronic condition intercepts. A number of other covariates are predictive of health trajectories including social security income, being in the labor force, health insurance, obesity, smoking history and marriage.

Table 3 presents the effects of length of residence in the United States on morbidity trajectories among immigrants after adjusting for gender, cohort, and attrition rates. Surprisingly, length of residence in the U.S. is not predictive of morbidity levels or rates of change for any of the racial/ethnic groups. Thus, results do not support the acculturation hypothesis. Supplemental analyses (not shown) revealed similar findings after operationalizing length of residence as a categorical variable and controlling for social origins, SES, marital status, health behaviors, and region.

[Table 3, about here]

Discussion

Population aging is likely to affect many aspects of U.S. society including educational, financial, familial, and health care institutions. Understanding health patterns among diverse racial/ethnic/native groups is particularly important given the projected increasing proportion older people of color and immigrants over the next several decades (He 2002). Previous research has shown that, compared to whites, blacks have higher levels of morbidity, whereas Hispanics exhibit a higher prevalence of certain chronic conditions and a lower rate of other conditions (Blackwell et al. 2002; *CDC 2004;* Markides et al. 1997). An immigrant health advantage has also been established in the literature (Singh and Siahpush 2002). Though cross-sectional disparities in chronic conditions are well-documented, little is known about racial/ethnic/nativity differences in morbidity age-trajectories. Do disparities in morbidity decrease, remain stable, or increase with age? This is the first study to both conceptualize and model morbidity trajectories for various racial/ethnic/nativity groups. Findings show that demographic subgroups differ in terms of both morbidity levels and rates of change.

While aging-as-leveler, persistent health inequality and cumulative disadvantage hypotheses have been framed as competing hypotheses, support for each of them is found, depending on the life stages and the groups being analyzed. For example, there is support for both the cumulative disadvantage and aging-as-leveler hypotheses when comparing the health trajectories of native- and foreign-born blacks to those of native-born whites: both native- and foreign-born blacks experience more rapid health deterioration during their 50s and early 60s, and subsequently exhibit slower rates of accumulation of conditions, never reaching parity with native-born whites. Compared to native-born whites, native-born Mexican Americans have

higher levels of chronic conditions, and foreign-born whites and Mexicans have fewer chronic conditions, as expected; interestingly, these health gaps remain stable as they age, consistent with the persistent inequality hypothesis. That support for the three hypotheses varied across context, suggests that rather than conceptualizing these hypotheses as competing frameworks that are mutually exclusive, a more flexible, life course contingency perspective that takes context into account more accurately illustrates how racial/ethnic/native differences in health evolve with age.

Findings on nativity effects reveal clear evidence of an immigrant health advantage, with foreign-born individuals exhibiting more favorable morbidity trajectories than their native-born counterparts, a pattern that holds across all racial/ethnic groups. This pattern persists even after accounting for group differences in health behaviors and attrition, indicating that factors besides health behaviors and emigration, such as healthy migrant selection (see Akresh and Frank 2008), may play a role in their immigrants more favorable morbidity trajectories. Taken as a whole, the literature on immigration and health provides suggests that healthy immigrant selection, health behaviors, and selective return migration in later life all play roles in creating an immigrant health advantage (Abraido-Lanza et al. 1999; Hummer et al. 2000; Landale et al. 2000; Markides and Coreil 1986; Paloni and Arias 2004). In addition, one could argue that lower rates of reported doctor-diagnosed conditions among foreign-born persons are because they have less access to health care. However, this study controlled for insurance coverage and previous research has shown that the immigrant health advantage persists among a subsample of immigrants who have seen a doctor within the last year (see Jasso et al. 2004), thus suggesting that the immigrant health advantage is not an artifact of misreporting. Determining the relative

contributions of each of these mechanisms is not possible with HRS data and is beyond the scope of this study; however, future research should explore this topic.

Interestingly, results do not support an acculturation hypothesis. The lack of association between duration in the U.S. and morbidity may be due to the countervailing effects of acculturation on health risk and protective factors. For example, longer durations in the U.S. have been shown to lead to worse health behaviors (i.e. increasing rates of smoking and problem drinking), yet increasing levels of exercise (Abraido-Lanza et al. 2005; Clark and Hofsess 1998; Crespo et al. 2001). Moreover, Jasso and colleagues (2001; 2004) have show large economic gains to immigration that appear to increase over time and positively affect subsequent health, thus providing a prediction counter to the common assumption that health deteriorate with increasing length of residence in the U.S. (see also Angel, Buckley and Sakamoto 2001). Further research on the impact of acculturation on health trajectories in needed.

We know that health is shaped by the interaction of various forms of life course capital (e.g., human, economic, and social capital) over time (O'Rand 2006), yet few studies have examined how these forms of capital may mediate racial/ethnic disparities in health trajectories. The lives of whites, blacks, and Hispanics evolve in very different ways with respect to protective resources and risk factors for chronic conditions. People of color are disadvantaged in terms of social origins, SES, family histories, and health behaviors (Brown and Warner 2008; Newman 2002). Minorities face structural disadvantages that result in greater exposure to risks and less access to opportunities and resources. The present study draws on life course themes in the status attainment tradition, which highlight how one's social origins and structural position in social and economic institutions influence attainment processes and an array of subsequent life chances and outcomes (Blau and Duncan 1967; Teachman 1987). Results reveal that various

forms of life course capital are predictive of chronic conditions, and that racial/ethnic inequality in life course capital account for some, but not all disparities in morbidity trajectories. For example, after controlling for a wide array of factors, native-born Mexican Americans do not exhibit more rapid accumulation of chronic conditions than native-born whites; however, only a small portion of blacks' excess morbidity is explained by racial inequality in life course capital (see Hayward et al. 2000).

The fact that health disparities are not eliminated after adjusting for group differences in social origins, human capital, SES, marriage, health behaviors and attrition, suggests that other factors such as discrimination and unequal exposure to stressors may play a major role. Several studies find that although SES is protective for both blacks and whites, blacks are more likely than whites to experience diminishing returns of SES (Farmer and Ferraro 2005; Shuey and Willson 2008). Pearson (2008) posits that acquiring traditional socioeconomic resources is likely to entail negative health consequences for minorities, owing to unequal barriers and opportunities for different racial/ethnic groups. While this seems plausible, the present study found relatively few instances of racial/ethnic/nativity differences in health returns to socioeconomic resources, with no apparent patterns emerging from the data. More research is needed to determine whether there are subgroup differences in the health benefits of upward mobility.

Subjective experiences of racism increase levels of stress, elevate risks for stress-related diseases and contribute to health disparities (Geronimus 1996; Harrell, et al. 2003; Krieger 2000; Williams et al. 2003). Additionally, Williams and Jackson (2005) argue that racial residential segregation is a fundamental cause of health disparities because it leads to differential exposure to societal risks and resources. They note that minority neighborhoods are disadvantaged in

terms of neighborhood safety, accessibility of recreational facilities, green spaces and healthy products in grocery stores, and marketing of tobacco and alcohol—all of which influence health behaviors (see also MMWR 1999; Williams and Collins 2001; Yao and Roberts 2008. Accounting for racial/ethnic differences in neighborhood socioeconomic context has been shown to reduce the minority health disadvantage (Cagney, Browning and Yen 2005; Robert and Ruel 2006; Yao and Robert 2008). Unfortunately the HRS does not collect information on perceptions of racism or neighborhood characteristics. Further research is needed on the roles that racism and neighborhood context play in generating disparities in health trajectories.

This study uses an index of serious chronic conditions because previous research has shown that this measure provides a more parsimonious approach to understanding broad dimensions of well-being than analyzing single items (Farmer and Ferraro 2005). In addition, analyses of summary health measures are less likely than those of binary outcomes to encounter issues such as insufficient statistical power (Ferraro and Wilmoth 2000). That said, health conditions differ in terms of their etiology, and race/ethnicity/nativity and social factors are likely to influence the trajectories of specific diseases differently. Ancillary analyses (not shown) indicated that results of this study are robust to the exclusion each of the life threatening conditions (i.e. cancer, diabetes, heart disease, hypertension, and stroke) as well as the inclusion of other health issues such as arthritis, psychological problems and lung disease. The health conditions analyzed in this study are consistent with previous research (e.g. Farmer and Ferraro 2005) and have in common that they are all life-threatening medical conditions; however, future research should examine racial/ethnic differences in trajectories of specific diseases.

This study utilizes an aggregate approach to investigate racial/ethnic/nativity disparities in average health trajectory intercepts and/or slopes. In contrast, a disaggregated approach is

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well-suited to determine whether there are group differences in the risks of following distinct classes of health trajectories (George 2003; Nagin 2005). Previous studies using a disaggregated or group-based approach to trajectories have identified a number of qualitatively distinct trajectories of health (e.g., constant good health, constant poor health, linear decline, precipitous decline, and decline and recovery) within the population (e.g., Clipp et al. 1992; Hamil-Luker and O'Rand 2007; O'Rand and Hamil-Luker 2005). Racial/ethnic/nativity groups are likely to differ in terms of risks of following specific classes of health trajectories. Although the topic is beyond the scope of this study, research on the issue is need.

Despite oversampling blacks and Hispanics, the HRS has too few respondents to detect differences in the health trajectories of many subgroups in the black and Hispanic populations. For example, sample sizes are inadequate to yield reliable estimates for blacks of different countries of origin. Similarly, small sample sizes of Hispanics with origins in places other than Mexico (e.g. Cuba, Honduras, Puerto Rico, Spain, etc.) preclude an examination of their health trajectories (See Brown and Warner 2008). Future analyses and additional data collection efforts are needed to investigate heterogeneity among older foreign-born populations (Jasso et al. 2004).

Although the analyses control for prospective death and dropout attrition, left-censoring may be an issue. Results presented here may be biased given racial/ethnic/nativity differences in mortality rates, and the fact that inclusion in the HRS sample is conditional upon survival to midlife (Shuey and Willson 2008; Taylor 2008). Accordingly, findings should be interpreted as conditional on survival to mid-life.

Empirical research on racial/ethnic/nativity differences in morbidity trajectories continues to lag behind theories on the matter. This study is among the first to investigate how serious chronic condition trajectories vary by race/ethnicity/nativity. Findings from this study illustrate

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the utility of integrating demographic and developmental perspectives for understanding intracohort inequality dynamics in general, and racial/ethnic/nativity differences in disease trajectories in particular. Demographic groups differ in both their chronic condition levels and rates of change. It is important to note that health disparities have already emerged by midlife. Thus, future research should investigate racial/ethnic/nativity differences in health trajectories and what contributes to them earlier in the life course in order to better understand and eliminate health disparities.

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Figure 1. Disparities in Morbidity Trajectories between Racial/Ethnic/Nativity Groups and U.S.-born Whites*

*Simulations are based on significant coefficient estimates in Model 1 of Table 2

	Blacks		Mexican Americans		Whites	
	Native-born	Foreign-born	Native-born	Foreign-born	Native-born	Foreign-born
Covariates						
Age	55.77 †	54.83 *	55.44	55.18 *	55.77	55.90
Female	.58 *	.58	.50	.51	.51	.56
Older cohort (1931-36)	.53 †	.37 *	.43 *	.41 *	.52	.52
Number of waves	5.46 *	5.48	5.73	5.54 *	5.86	5.80
Died	.23 *†	.05 *	.17 †	.09	.13	.11
Poor during childhood	.32 *	.27	.37 *	.39 *	.24	.20
Mother had < H.S. Education	.04 *	.05	.01 *†	.03 *	.11	.12
Father had < H.S. Education	.03 *†	.07	.01 *	.02 *	.12 †	.22 *
Years of Education	11.23 *	11.13 *	9.21 *†	4.91 *	12.66	12.58
Earnings (Ln)	7.77 *†	9.66	7.98 *†	7.16 *	9.28	9.29
Social Security Income (Ln)	1.89 *†	.43 *	1.73 *	1.49	1.29	1.25
Employed	.62 *	.83 *	.59 *†	.49 *	.71 †	.65 *
Net Worth (Ln)	7.45 *	7.69 *	9.74 *†	7.49	11.24 †	11.82 *
Uninsured	.21 *	.20 *	.32 *†	.62 *	.13	.16
Unmarried	.48 *	.42 *	.28 *	.27 *	.21	.19
Obese	.35 *†	.23	.28 *	.33 *	.21 †	.16 *
Ever smoked	.64 †	.39 *	.65 †	.51 *	.65	.63
Currently smoke	.31 *†	.12 *	.26	.21 *	.27 †	.21 *
Heavy Drinker	.05	.01	.07	.04	.05	.06
Midwest	.20 *†	.01 *	.06 *	.04 *	.28 †	.22 *
West	.06 *†	.02 *	.49 *	.46 *	.15 †	.22 *
South	.56 *†	.23 *	.45	.50 *	.39 †	.21 *
Years in the U.S.		19.97		24.96		30.99
<u>N</u>	1558	83	290	226	6551	303

Table 1. Means and Percentages of Covariates, by Race/Ethnicity/Nativity

* Statistically significant (p<.05) difference in covariate mean between racial/ethnic/nativity group and native-born whites

† Statistically significant (p<.05) difference in covariate mean between native- and foreign-born groups within race/ethnicity

	Model 1			Model 2		
	Initial Status	Linear Slope	Quadratic Slope	Initial Status	Linear Slope	Quadratic Slope
	(π_{0i})	(π_{1i})	(π_{2i})	(π_{0i})	(π_{1i})	(π_{2i})
Fixed Effects						
Intercept	.3887 ***	.0256 ***	.0023 ***	.5044 ***	.0211 *	.0021 ***
White, foreign-born	1517 *	.0146	0010 +	1639 **	.0158	0011 +
Black, native-born	.2288 ***	.0204 ***	0010 ***	.1850 ***	.0208 ***	0012 ***
Black, foreign-born	0396	.0603 **	0027 *	0676	.0553 *	0024 *
Mexican American, native-born	.1259 *	0177	.0008	.0588	0142	.0005
Mexican American, foreign-born	1486 *	.0181	0003	2476 **	.0208	0009
Female	.0402 +	0033	0003	.0225	0028	0003
Older cohort (1931-36)	.0256	0309 ***	.0007 **	.0216	0291 ***	.0005 *
Number of waves	0113	0028	.0001	0024	0025	.0001
Died	.3748 ***	.0166 +	.0013 **	.3517 ***	.0193 *	.0009 *
Poor during childhood				0088	.0033	<.0001
Mother had < H.S. Education				0683	0047	.0001
Father had < H.S. Education				0077	0008	.0003
Years of Education				0099 *	.0001	0001
Earnings				0011	<.0001	<.0001
Social Security Income				.0117 ***	0016 **	.0001 *
Employed				1196 ***	.0129 ***	0006 **
Net Worth				0023	<.0001	<.0001
Uninsured				0415 +	.0085	0006 +
Obese				0304 +	.0040	<.0001
Ever smoked				.1092 ***	0075	.0006 *
Currently smoke				1168 ***	0040	.0001
Heavy Drinker				.0275	0128	.0004
Unmarried				.0514 *	0065	.0002
Midwest				.0001	0039	.0003
West				.0091	0106	.0004
South				0298	.0012	.0001
Random Effects						
Level 1 Residual	.2534 ***			.2528 ***		
Level 2 Age	.1290 ***			.1263 ***		
Level 2 Age2	.0065 ***			.0063 ***		
Level 2 Intercept	.7560 ***			.7378 ***		
Ν	9011			9011		
Log Likelihood	-30326			-29825		

 Table 2. Race/Ethnicity, Nativity and Morbidity Trajectories (ages 51-73); Random Coefficient Growth Models

 Model 1

+p <.1; *p<.05; **p<.01; ***p<.001

$\begin{array}{ c c c c c c c c c c c c c c c c c c c$			Blacks				
$\begin{array}{c c c c c c c c c c c c c c c c c c c $		Initial Status	Linear Slope	Quadratic Slope			
$\begin{array}{c c c c c c c c c c c c c c c c c c c $		(π_{0i})	(π_{1i})	(π_{2i})			
$\begin{array}{lll} Intercept &0591 & .1186 * &0014 \\ Years in the U.S. & .0111 &0018 & .0001 \\ Female & .4248 + &0429 & .0015 \\ Older cohort (1931-36) &3222 & .0652 &0036 \\ Number of waves &0712 & .0271 &0017 \\ Died & 1.2678 + & .1420 & .0054 \\ Random Effects & & & & & \\ Level 1 Residual & .2452 *** & & & \\ Level 2 Age & .1398 *** & & \\ Level 2 Age & .1398 *** & & \\ Level 2 Age & .0064 *** & & & \\ Level 2 Age & .0064 *** & & & \\ Level 2 Age & .0064 *** & & & \\ Level 2 Age & .0001 & -246 & & & & \\ \hline \hline Mittal Status & Linear Slope & Quadratic Slope \\ \hline (\pi_{60}) & (\pi_{11}) & (\pi_{22}) & & \\ \hline Fixed Effects & & & & \\ Intercept & .2560 & .0828 *** & .0001 & \\ Years in the U.S. & .0016 & .0017 & .0001 & \\ Years in the U.S. & .0016 & .0017 & .0001 & \\ Years in the U.S. & .0016 & .0017 & .0001 & \\ Years in the U.S. & .0016 & .0017 & .0001 & \\ Female & .0487 & .0013 & .0011 & \\ Older cohort (1931-36) & -1.724 & .0065 & < .0001 & \\ Died & .1341 & .0087 & .0027 & \\ Random Effects & & & \\ Level 1 Residual & .2857 *** & \\ Level 2 Age & .1331 *** & \\ Level 2 Age & .0004 *** & \\ Level 2 Age & .0003 & .0002 & \\ \hline \hline Fixed Effects & & & \\ Intercept & .1384 & .0593 & .0008 & \\ Years in the U.S. &0013 & .0003 & .0002 & \\ Fixed Effects & & & \\ Intercept & .1384 & .0593 & .0008 & \\ Years in the U.S. &0013 & .0003 & .0002 & \\ Female & .2811 ** & .0376 + & .0010 & \\ Older cohort (1931-36) & .0439 & .0051 & .0003 & \\ Number of waves &0331 & .0052 & .0004 & \\ Died & .2364 & .0106 & .0004 & \\ Random Effects & & \\ Level 2 Age &0331 & .0052 & .0004 & \\ Died & .2364 & .0106 & .0004 & \\ Random Effects & & \\ Level 2 Age &0331 & .0052 & .0004 & \\ Died & .2364 & .0106 & .0004 & \\ Random Effects & & \\ Level 2 Age &0331 & .0052 & .0004 & \\ Died & .2364 & .0106 & .0004 & \\ Random Effects & & \\ Level 2 Age &0331 & .0052 & .0004 & \\ Died & .2364 & .0106 & .0004 & \\ Random Effects & & \\ Level 2 Age &0331 & .0052 & .0004 & \\ Died & $	Fixed Effects						
Years in the U.S. 0111 -,0018 .0001 Female .4248 + .0429 .0015 Older cohort (1931-36) 3222 .0652 0036 Number of waves 0712 .0271 0017 Died 1.2678 1420 .0054 Random Effects	Intercept	0591	.1186 *	0014			
Female 4248 + 0429 .0015 Older cohort (1931-36) 3222 .0652 0036 Number of waves 0712 .0017 .0017 Died 1.2678 + 1420 .0054 Random Effects	Years in the U.S.	.0111	0018	.0001			
$\begin{array}{llllllllllllllllllllllllllllllllllll$	Female	.4248 +	0429	.0015			
Number of waves 0712 .0271 0017 Died 1.2678 + 1420 .0054 Random Effects	Older cohort (1931-36)	3222	.0652	0036			
Died $1.2678 +$ 1420 .0054 Random Effects	Number of waves	0712	.0271	0017			
Random Effects Level 1 Residual .2452 *** Level 2 Age .1398 *** Level 2 Age .0064 *** Level 2 Intercept .7782 *** N 83 Log Likelihood -246 Mexican Americans Mexican Americans Mexican Americans Kade Effects Intercept .2560 .0828 *** 0001 Years in the U.S. .0016 0017 .0001 Years in the U.S. .0016 0065 <0001	Died	1.2678 +	1420	.0054			
Level 1 Residual .2452 *** Level 2 Age .1398 *** Level 2 Age 2 .0064 *** Level 2 Intercept .7782 *** N 83 Log Likelihood -246 Mexican Americans Initial Status Linear Slope Quadratic Slope (π_{0i}) (π_{1i}) (π_{2i}) Fixed Effects .0016 0017 .0001 Female .0487 .0013 .0011 Older cohort (1931-36) 1724 0065 <.0001	Random Effects						
Level 2 Age	Level 1 Residual	.2452 ***					
Level 2 Age2 .0064 *** Level 2 Intercept .7782 *** N 83 Log Likelihood -246 Mexican Americans Initial Status Initial Status Linear Slope Quadratic Slope (π_{ti}) (π_{ti}) (π_{2i}) Fixed Effects Intercept .2560 .0828 *** 0001 Years in the U.S. .0016 0017 .0001 Years in the U.S. .0016 0017 .0001 Older cohort (1931-36) 1724 0065 <0001	Level 2 Age	.1398 ***					
Level 2 Intercept .7782 *** N 83 Log Likelihood -246 Mexican Americans Initial Status Linear Slope Quadratic Slope (π_{0i}) (π_{1i}) (π_{2i}) Fixed Effects Intercept .2560 .0828 *** 0001 Years in the U.S. .0016 0017 .0001 Pemale .0487 .0013 .0011 Older cohort (1931-36) 1724 0065 <0001	Level 2 Age2	.0064 ***					
N 83 Log Likelihood -246 Mexican Americans Initial Status Linear Slope (π_{00}) (π_{11}) (π_{20}) Fixed Effects Intercept .2560 .0828 *** 0001 Years in the U.S. .0016 0017 .0001 Pemale .0487 .0013 .0011 Older cohort (1931-36) 1724 0065 <0001	Level 2 Intercept	.7782 ***					
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Random Effects Level 1 Residual .2857 *** Level 2 Age .1331 *** Level 2 Age2 .0064 *** Level 2 Age2 .0064 *** Level 2 Intercept .7228 *** N 226 Log Likelihood -809 Whites Initial Status Linear Slope Quadratic Slope (π_{0i}) (π_{1i}) (π_{2i}) Fixed Effects Intercept .1384 .0593 .0008 Years in the U.S. 0013 0003 0002 Female .2811 ** 0376 + .0010 Older cohort (1931-36) .0439 0051 .0003 Number of waves 0331 .0052 0004 Died .2364 .0106 .0004 Random Effects Itevel 1 Residual .2240 *** Itevel 2 Age .1299 *** Level 2 Age2 .0060 *** Itevel 2 Age2 .0060 *** Itevel 2 Age2 .0060 *** Level 2 Intercept .6444 *** N .303 Itevel 2 Age2 <td>Died Pandom Effects</td> <td>.1341</td> <td>.0087</td> <td>.0027</td>	Died Pandom Effects	.1341	.0087	.0027			
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(π_{0i}) (π_{1i}) (π_{2i}) Fixed EffectsIntercept.1384.0593.0008Years in the U.S001300030002Female.2811 **0376 +.0010Older cohort (1931-36).04390051.0003Number of waves0331.00520004Died.2364.0106.0004Random EffectsLevel 1 Residual.2240 ***Level 2 Age.1299 ***Level 2 Intercept.6444 ***N.303Log Likelihood818		Initial Status	Linear Slope	Quadratic Slope			
Fixed Effects Intercept .1384 .0593 .0008 Years in the U.S. 0013 0003 0002 Female .2811 ** 0376 + .0010 Older cohort (1931-36) .0439 0051 .0003 Number of waves 0331 .0052 0004 Died .2364 .0106 .0004 Random Effects . . . Level 1 Residual .2240 *** . . Level 2 Age .1299 *** . . Level 2 Age2 .0060 *** . . Level 2 Intercept .6444 *** . . N		(π_{0i})	(π_{1i})	(π_{2i})			
Intercept.1384.0593.0008Years in the U.S. 0013 0003 0002 Female.2811 ** 0376 +.0010Older cohort (1931-36).0439 0051 .0003Number of waves 0331 .0052 0004 Died.2364.0106.0004Random EffectsLevel 1 Residual.2240 ***Level 2 Age.1299 ***Level 2 Age2.0060 ***Level 2 Intercept.6444 ***N.303Log Likelihood818+p <.1; *p<.05; **p<.01; ***p<.001	Fixed Effects						
Years in the U.S. 0013 0003 0002 Female $.2811 **$ $0376 +$ $.0010$ Older cohort (1931-36) $.0439$ 0051 $.0003$ Number of waves 0331 $.0052$ 0004 Died $.2364$ $.0106$ $.0004$ Random EffectsLevel 1 Residual $.2240 ***$ Level 2 Age $.1299 ***$ Level 2 Age2 $.0060 ***$ Level 2 Intercept $.6444 ***$ N $.303$ Log Likelihood 818	Intercept	.1384	.0593	.0008			
Female $.2811 **$ $0376 +$ $.0010$ Older cohort (1931-36) $.0439$ 0051 $.0003$ Number of waves 0331 $.0052$ 0004 Died $.2364$ $.0106$ $.0004$ Random EffectsLevel 1 Residual $.2240 ***$ Level 2 Age $.1299 ***$ Level 2 Age2 $.0060 ***$ Level 2 Intercept $.6444 ***$ N $.303$ Log Likelihood 818	Years in the U.S.	0013	0003	0002			
Older cohort (1931-36) .0439 0051 .0003 Number of waves 0331 .0052 0004 Died .2364 .0106 .0004 Random Effects .2240 ***	Female	.2811 **	0376 +	.0010			
Number of waves 0331 .0052 0004 Died .2364 .0106 .0004 Random Effects	Older cohort (1931-36)	.0439	0051	.0003			
Died .2364 .0106 .0004 Random Effects .0004 .0004 Level 1 Residual .2240 *** .0004 Level 2 Age .1299 *** .00060 *** Level 2 Age2 .0060 *** .0004 Level 2 Intercept .6444 *** .00060 *** Log Likelihood -818 .0004	Number of waves	0331	.0052	0004			
Random Effects Level 1 Residual .2240 *** Level 2 Age .1299 *** Level 2 Age2 .0060 *** Level 2 Intercept .6444 *** N 303 Log Likelihood -818 +p <.1; *p<.05; **p<.01; ***p<.001	Died	.2364	.0106	.0004			
Level 1 Residual .2240 *** Level 2 Age .1299 *** Level 2 Age2 .0060 *** Level 2 Intercept .6444 *** N 303 Log Likelihood -818 +p <.1; *p<.05; **p<.01; ***p<.001	Random Effects						
Level 2 Age .1299 *** Level 2 Age2 .0060 *** Level 2 Intercept .6444 *** N .303 Log Likelihood -818 +p <.1; *p<.05; **p<.01; ***p<.001	Level 1 Residual	.2240 ***					
Level 2 Age2 .0060 *** Level 2 Intercept .6444 *** N 303 Log Likelihood -818 +p <.1; *p<.05; **p<.01; ***p<.001	Level 2 Age	.1299 ***					
Level 2 Intercept .6444 *** N 303 Log Likelihood -818 +p <.1; *p<.05; **p<.01; ***p<.001	Level 2 Age2	.0060 ***					
N 303 Log Likelihood -818 +p <.1; *p<.05; **p<.01; ***p<.001	Level 2 Intercept	.6444 ***					
Log Likelihood -818 +p <.1; *p<.05; **p<.01; ***p<.001	Ν	303					
+p <.1; *p<.05; **p<.01; ***p<.001	Log Likelihood	-818					
	+p <.1; *p<.05; **p<.01; ***	^c p<.001					

Table 3. Length of Residence in U.S. and Morbidity