Essays on Race/Ethnic Variations in the Dynamics of Chronic Diseases Among Middle and Old Aged Americans

by

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To the memory of my father, Ricardo C. Quiñones,
who always encouraged me to keep learning.

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ABSTRACT

This dissertation is composed of three empirical papers on ethnic disparities in chronic disease morbidity. The first paper analyzes intra- and interpersonal differences in comorbidity burden reported by white, black and Mexican Americans. Data come from Health and Retirement Study (HRS) participants aged 51 and over from 1995-2006. Hierarchical linear models are employed to analyze ethnic variations in temporal changes of reported comorbidities. On average, participants have nearly two chronic diseases at the baseline, which increased to almost three conditions over 11 years. Mexican Americans demonstrate lower initial levels and slower accumulation of comorbidities relative to whites. In contrast, blacks show an elevated level of comorbidity, although their rate of change decelerated over time relative to whites.

The second paper examines ethnic variations in the onset of hypertension diagnosis for white, black and Mexican Americans age 51 and over. Data came from HRS respondents who report being hypertension-free at the baseline for up to five time intervals (1995-2006). Discrete-time survival models are used to analyze ethnic variations in the probability of developing hypertension. We find the risk of newly diagnosed hypertension increased for all participants. Relative to white and Mexican Americans, black Americans had an elevated risk of incident hypertension throughout the 11-year period of observation. These variations persisted even when differences in health

behaviors, socioeconomic status, demographic, and time-varying health characteristics were adjusted.

The third paper examines the onset of diabetes mellitus diagnosis for HRS participants 51 and over who report being free of diabetes at the baseline. Discrete-time survival models are used to analyze ethnic variations in the probability of developing diabetes in up to five time intervals (1995-2006). We find the risk of newly diagnosed diabetes increased for all study participants. Relative to white and black Americans, Mexican Americans have a significantly elevated risk of diabetes. Increases in diabetes incidence for Mexican Americans persist through adjustment of health behaviors, socioeconomic status, demographic characteristics and changing health status. In contrast, increases in incident risk for black Americans relative to white Americans operate largely through changes in time-varying health status.

Our findings of continued racial and ethnic disparities in chronic disease burden as well as hypertension and diabetes mellitus incidence suggest there are still improvements to be made in prevention efforts aimed at middle and older aged minorities. These empirical papers highlight the importance of social and structural factors as critical policy levers for mitigating chronic disease burden as well as reducing the elevated risk of two pervasive chronic diseases for minorities in the U.S.

CHAPTER 1:

Introduction

The demographic shift of the U.S. population over the next few decades has many implications for how best to care for and treat disability and disease among the aged. However, the specific relationship of disease accumulation and onset trends on sub-sections of the population is unclear. Despite some cross-sectional evidence of ethnic differences in disease onset, it is not clear to what extent observed differentials are due to socioeconomic and ethnic patterning of aging processes. Although the existence and persistence of ethnic disparities in health outcomes has been extensively documented in the literature, disparities among older Hispanics have been less well explored. This dissertation focuses specifically on minority aging by examining dynamics of health from a more inclusive, nationally-representative perspective, as well as through the lens of ethnic correlates that may mediate these health dynamics.

A primary objective of this dissertation research is the focus on charting differences in chronic disease dynamics for older minorities that comprise large demographic segments of the U.S. population. In particular, this research compares chronic disease dynamics for older non-Hispanic white, non-Hispanic black and Mexican Americans. Another objective of this research is to clarify our understanding of age-related changes for one specific group of older Hispanics in the U.S. Lumping together

individuals from very different social, historical and political backgrounds confounds a critical aspect of ethnic identity. Much more needs to be understood about the morbidity burden of the various heterogeneous Hispanic sub-populations as they progress into advanced ages. This dissertation takes one such step in examining disease dynamics for Mexican Americans as differentiated from the nonspecific Hispanic moniker.

This dissertation is composed of three empirical papers which examine race/ethnic differences in disease dynamics for middle and old aged individuals. In Chapter 2, I present the first paper which is co-authored with Jersey Liang, and compares how individuals from three race/ethnic groups in the U.S. differ in their trajectories of chronic disease comorbidity. It focuses on how changes in comorbidity vary across white, black and Mexican Americans. Specifically, this paper examines intra- and interpersonal differences in co-occurring chronic disease reported by Americans aged 51 and over for a period up to 11 years. In order to do this, the Health and Retirement Study data from 1995-2006 is employed. These years of data enable us to examine data from a nationally-representative sample of older individuals with up to seven repeated observations. In this paper, we employ hierarchical linear models for longitudinal data (also known as growth curve models) to analyze ethnic variations in temporal changes of reported comorbidities. Using these model estimates, we are able to determine the level of reported co-occurring chronic disease for older white, black and Mexican Americans at the baseline, as well as the rate of accumulation of comorbidities for the 11 years of observation.

After examining the gross level of chronic disease comorbidity burden for the three race/ethnic groups, we then turn to examining individual disease dynamics. In

Chapter 3, the second paper estimates the period-by-period risk of self-reported hypertension, and is co-authored with Jersey Liang and Wen Ye. Hypertension is not only the most prevalent of the chronic diseases examined in the Health and Retirement Study, but is also an important contributor to rising health care costs in the U.S. This paper research examines the risk of developing hypertension for Americans 51 and older who report being hypertension-free at baseline. It focuses on how incidence in self-reported hypertension varies across white, black and Mexican Americans. We rely again on the Health and Retirement Study data of repeat observations for individuals for up to five time intervals (1995-2006). Because we are examining biennial survey data, we employ discrete-time survival models in order to analyze ethnic variations in the probability of developing hypertension in follow-up interviews. These models allow us to determine whether there are ethnic differences in the probability of reporting new hypertension for previously non-hypertensive individuals.

Chapter 4 presents the final paper on the risk of being diagnosed with diabetes mellitus, which also involves a chronic condition with high health care costs for the U.S. population. This paper is also co-authored with Jersey Liang and Wen Ye. In this paper, we analyze the probability of developing self-reported diabetes for older Americans who previously report not having diabetes. We again take advantage of the longitudinal design of the Health and Retirement Study to examine diabetes onset for a period of up to 11 years and five repeated time intervals (1995-2006). Differences in period-by-period self-reported diabetes incidence is examined for white, black and Mexican Americans age 51 and older. We again use discrete-time survival models to analyze ethnic variations in the probability of developing diabetes for middle aged and older adults. Again, we are

able to plot out the differential risk accruing to the three race/ethnic groups for the time periods in our observation window.

Chapter 5 offers some concluding remarks regarding the overall findings of the dissertation. These comments are couched in the context of the findings from the three individual papers. These three papers are sequential investigations and natural extensions of a broader policy issue: anticipating the changing needs of an ethnically diverse aging U.S. population as dictated by their respective chronic disease burden. Each of these papers examines the extent to which differences among race/ethnic groups in the dynamics of chronic disease morbidity persist and change into old age, if at all.

This dissertation provides empirical estimation of dynamic changes in chronic diseases for black, white and Mexican Americans. Although there has been much research documenting changes in individual chronic disease incidence and prevalence, fewer studies chart comparative changes for three large race/ethnic groups in the U.S. In addition, recent gerontological research has not focused on total chronic disease burden Americans face as they age. New experiences of chronic disease and the burden chronic disease comorbidities have on older individuals is largely unexplored. As a result, this dissertation provides insight into the dynamics of chronic disease changes for older Americans. This research also informs our understanding of the current state of chronic disease disparities, suggests that improvements in prevention efforts into old age should be made. In addition, this research implies that upstream structural factors play an important role in shaping differential chronic disease risk.

CHAPTER 2:

How Does the Trajectory of Comorbidity Vary Across Black, White, and Mexican Americans in Middle and Old Age?

2.1 Introduction

Over the last 40 years, the prevalence of chronic disease has increased substantially in the United States (Crimmins, 2004; Freedman, Martin & Schoeni, 2002). This increase is documented not only for the oldest of the old, but also for the middle-aged and the earlier-old. Moreover, the greatest growth in prevalence has been in the concurrent presence of multiple chronic diseases (Paez, Zhao & Hwang, 2009), which is commonly referred to as comorbidity (Fried, Ferrucci, Darer, et al., 2004; Verbrugge, Lepkowski & Imanaka, 1989). In 2005, 45.3% of the community-residing Americans aged 65-79 and 54.2% of those aged 80 and older reported multiple chronic diseases (Paez et al., 2009). In addition, Medicare claims data have documented that two thirds of all beneficiaries aged older than 65 years have two or more chronic conditions, and one third have four or more (Fried et al., 2004). Comorbidity is associated with high health care utilization and expenditures, and more importantly, it increases the likelihood of disability and mortality, over and above the risk from individual diseases (Fried et al., 2004).

There is an extensive literature documenting a disproportionate share of chronic disease morbidity and mortality for ethnic minorities (Hayward, Miles, Crimmins et al., 2000; Lantz, Lepkowski, Williams et al., 1998; Wong, Shapiro, Boscardin, et al., 2002; Cooper, Cutler, Desvigne-Nickens, et al., 2000). Much of the research is based on cross-sectional data and tends to focus on individual disease prevalence among blacks and Hispanics relative to whites in the U.S. (Lynch & Smith, 2005; Freedman et al., 2002). Various putative mechanisms such as double-jeopardy (Ferraro & Farmer, 1996) and lifestyle choices and discrimination (Williams & Collins, 1995) have been proposed in order to account for these observed ethnic differences. While these studies have contributed significantly to our knowledge, we do not know very much about how co-occurring chronic diseases are distributed across ethnic groups.

Even when longitudinal studies are undertaken, investigators tend to focus either on multiple diseases in single minority group or a single disease across different ethnic groups. For instance, Otiniano and colleagues (2003) examine longitudinal rates of heart attack mortality for Mexican elders and find patients are more likely to be male, older, and have co-occurring diabetes mellitus, hypertension and stroke. Wray and colleagues (2006) find that blacks and Latinos have higher prevalence of diabetes and increased odds of incidence net of social factors such as educational attainment, economic resources and parental social status. More importantly, most investigators have examined transitions in morbidity between two points in time which often do not reflect accurately the dynamic nature of health, as they provide no basis for distinguishing among alternative growth curves or trajectories (Rogosa, 1988). Connected by health transitions across successive years, a health trajectory imparts a form and meaning distinct from

those of health transitions (Clipp, Pavalko, & Elder, 1992). Accordingly, a more complete understanding of ethnic differences in health requires an analysis of health trajectory in terms of both the level as well as rate of change across these groups.

This research aims to contribute to current knowledge on aging and health in three respects. We first offer quantitative estimates of the trajectory of comorbidity by using longitudinal data derived from a national sample of Americans aged 51 and over for a period of up to 11 years (1995-2006). Second, we examine how the level and rate of change associated with comorbidity differ among black, Mexican, and white middle age and older adults. Finally, we explore how ethnic differences in the trajectory of comorbidity interface with socioeconomic status and time-varying health status.

2.2 Hypotheses

To address our research questions, we pose the following hypotheses.

H_1: Comorbidity increases with time (H_{1a}) as well as age (H_{1b}).

Given extensive evidence of increases in chronic condition prevalence in the U.S. (Wray et al., 2006; Geronimus et al., 2007), we hypothesize the level of comorbidity to be increasing over time. In addition, the presence of comorbidity increases markedly with age, largely because of the rise in the risk of individual chronic conditions (Fried et al., 2004).

H_2: Black middle-aged and older adults have higher initial levels of comorbidity and greater rates of change over time compared to whites.

Epidemiological and demographic research suggests that blacks have higher prevalence of disease and thus live in suboptimal health longer than their white counterparts (Freedman, et al., 2002). According to this research, blacks exhibit illness earlier and die at younger ages than whites. High levels of socioeconomic inequality account for much of the observed disparities in health at younger ages and early adulthood, with these differences narrowing into old age (Beckett, 2000). These seemingly inconsistent findings may be attributed to racial crossovers in morbidity and mortality (where age-specific rates of mortality and chronic disease among minorities converge and cross-over with rates of more advantaged social groups), and selective mortality due to the accumulation of health disadvantages over the lifecourse.

With considerable social stratification throughout the life course, social structure influences individual trajectories of health status by ethnicity, gender, and socioeconomic status dimensions (House, Lepkowski, Kinney, et al., 1994; Ross & Wu, 1996; Hagestad & Dannefer, 2001; Williams, 1997; Williams, 2005; Lynch & Smith, 2005). Ethnic differences in education, income levels, and segregated living conditions define what Link and Phelan (1995) term the fundamental causes of disease and disability. Disadvantages afforded by social inequalities lead to differences in health through divergent employment and occupational experiences, income and wealth streams, life styles, and health behaviors (Dannefer, 2003; Hayward, Miles, Crimmins, et al., 2000; Bulatao & Anderson, 2004; Hertzman, 2004). These mechanisms combine to affect racial differences in health in complex ways. Differences in health status trajectories likely reflect differences in the intersection of these factors over the lifecourse (Hayward et.al, 2000). We next hypothesize that social processes of cumulative disadvantage over

the lifecourse represent systematic assaults to health throughout the lifespan. Consistent with the concept of cumulative disadvantage, blacks are expected to demonstrate disease earlier in the lifespan, and are hypothesized to experience greater levels of co-occurring disease relative to whites.

H_3: Mexican-origin adults have lower initial levels of comorbidity and slower rates of change relative to white adults, or initial levels and rates of change that are very similar to whites.

The evidence on health trends for older Hispanics is mixed. Recent work examines the current state of health research on Hispanic populations, particularly the Hispanic Health Paradox (Markides & Eschbach, 2005). This epidemiological paradox refers to the finding that for some health outcomes, most notably mortality, Hispanics are comparable to whites despite being socioeconomically similar to black Americans. There are several explanations for this, among them, poor data quality with respect to the reporting of age and the ascertainment of mortality statistics; cultural advantages of Hispanics that yield protective effects on health; healthy migrant effects, where more robust individuals self-select in migrating; and, salmon bias, where frail individuals self-select with respect to out-migration back to their home country, and are no longer captured by U.S. morbidity or mortality statistics (Markides & Eschbach, 2005).

However, evidence of a Hispanic Health Paradox is not universally supported (Palloni & Morenoff, 2001). While studies utilizing several nationally-representative data sources find evidence for a Hispanic mortality advantage (Markides & Coreil, 1986; Franzini, Ribble & Keddie, 2001), other studies find no such advantage (Carrasquillo,

Lantigua, Shea, 2000). Thus, the state of research pertaining to heterogeneous Hispanic subpopulations remains mixed, prompting us to examine Mexican-Americans as a standalone ethnic group. It is unclear that the Hispanic Paradox advantage will materialize for older adults in specific Hispanic ethnic subgroups. Considering Mexican-origin individuals comprise the largest group among Hispanics in the U.S., we tentatively hypothesize that Mexican-origin individuals will exhibit similar outcomes or health advantages relative to white Americans.

2.3 Methods

2.3.1 Data

This study uses data from the Health and Retirement Study (HRS) at the University of Michigan's Institute for Social Research. The HRS respondents are a nationally-representative sample of community-based adults aged 51 and over and identified through screening of an area probability sample of households. The study includes individuals from several age cohorts: the Asset and Health Dynamics of the Oldest Old (AHEAD; born prior 1924), the Children of the Depression Age (CODA; born 1924-1930), the Health and Retirement Study cohort (HRS, born 1931-1941) and War Babies (WB, born 1942-1947).

Due to wave incompatibility of key independent variables, these analyses use seven waves of data from the HRS (1995-2006). Analyses are conducted with Stata 10.0 (Stata Corp., College Station, Texas) and HLM 6.05 (Scientific Software Int., Lincolnwood, Illinois).

2.3.2 Measures

2.3.2.1 Self-reported disease

The Health and Retirement Study asks respondents about a variety of diseases each interview year. In subsequent interviews, individuals were given the option to dispute their preloaded responses from the previous interview. In order to deal with responses that offer conflicting information, we examine additional information reported by respondents. Consultations with geriatric physicians provided the clinical criteria for satisfying the burden of proof for each of the seven reported diseases. For each disease, a dispute was corroborated by examining the evidence variables from the previous interview. For example, if an individual has conflicting reports of having had cancer, we utilize information on year cancer was diagnosed or receipt of cancer therapies (radiation, surgery, chemotherapy) to verify the diagnosis of cancer.

Self-reported disease indicators are used to measure comorbidity in the analyses. Measures for self-reported health status and disease have been well established and validated in earlier studies (Johnson & Wolinsky, 1993; Ferraro & Wilmoth, 2000; Mensah, Mokdad, Ford et al., 2005), and are widely used in aging and epidemiological research. In addition, nationally representative data collection instruments provide consistent estimates with incidence when compared to clinical studies of specific diseases (Glymour & Avendano, 2009).

These analyses involve growth curve models of a total count of comorbidity. Total comorbidity consists of physician-diagnosed hypertension, heart disease, diabetes, cancer, lung disease, arthritis and stroke, as reported by respondents. The distribution of

the comorbidity variable is sufficiently normal to treat it as a continuous variable (mean= 2.079, s.d.=1.355). The skewness (skewness=0.444) indicates the data are slightly right or positive-skewed. The kurtosis (kurtosis= 2.833), closely approximates the kurtosis of a standard normal distribution.

Additional analyses were undertaken to determine the sensitivity of our simple comorbidity count with one akin to a Charlson Comorbidities Index (CCI) type of adjustment. We derived separate indices weighting each disease with coefficient values derived from logistic regressions that capture each disease's predictive contribution to mortality (not shown), similar to other studies (Bravo, Dubois, Hébert, et al., 2002). In this way, a disease that is more predictive of death has a greater weight in the total comorbidity score. We then examined the correlation between this alternate comorbidity index with our simple sum (ρ=0.98). Given the high correlation between the indices, we are satisfied that using the sum of conditions is a sufficient measure of the comorbidity burden assumed by study participants.

2.3.2.2 Ethnicity

The principal covariates of interest in the analyses are indicators for self-reported black and Mexican ethnicity. Ethnicity is constructed as mutually-exclusive indicator variables for non-Hispanic white, non-Hispanic black and Mexican-ethnicity individuals. Other race and other Hispanic types are excluded from the analyses. Dummy variables for black and Mexican are included in the analytic models and are each interpreted relative to white study participants. Inability to identify other Hispanic subgroups in the HRS data (i.e., Cuban and Puerto Rican) prevented us from including them in the

analyses as additional and separate ethnic groups. Consequently, we chose to focus solely on Mexican ethnicity among Hispanics in these analyses.

2.3.2.3 Social stratification and social support

Various controls for demographic and socioeconomic factors are included as independent, time-constant and time-varying covariates in the analysis. Age is measured as age in 1995 for all individuals in the study, regardless of entry cohort. Education is measured as a continuous variable denoting years of schooling (range 0-17). Income is included as a time-varying covariate and introduced in the analyses. Lagged (reported values at time t-1) and change in income is inflation-adjusted to 2006 levels, and was also re-scaled (reported per 1000s of dollars) to facilitate its estimation in the multilevel models. Marital status is conceptualized as an indicator of social support for individuals, and is constructed as a time-varying covariate. The change in marital status (range -1 to 1) reflects dissolution/widowhood, no change, and acquisition of partners at each point in time over the study period.

2.3.2.4 Health status

Several covariates are used to mark the physical and mental health status of respondents in accounting for changes in comorbidity, and are included in the analyses as time-varying covariates. Self-rated ill health (SRH) is measured with a 5-item scale (1=excellent, 2=very good, 3=good, 4=fair and 5=poor). Functional status (0-11) incorporates both, activities of daily living (ADL, 0-6) and instrumental activities of daily living (IADL, 0-5), with higher scores reflecting increasing number of difficulties with any of the ADL or IADL activities. Depressive symptoms are measured with the Center for Epidemiological Studies Depression Scale (CES-D, 0-9) with a higher score reflecting

higher depressive symptoms. Lagged covariates and covariates denoting the change (current minus previous wave) for all of these health status variables are included in the analyses.

Time-varying health covariates are intended to provide some control of population heterogeneity in health status. We conceptualize health status as multidimensional, where physical and mental health limitations work in concert to influence chronic disease emergence. Specifically, health status in previous time periods has a bearing on current period chronic disease development. That is, global self assessment of health, functional limitations and depressive symptoms are conceptualized as confounding variables in an individual's future experience with chronic disease.

2.3.3 Data analysis

One of the limitations of using longitudinal data is the possibility of missing data at follow-up due to item non-response, survey non-response, and mortality (Little & Rubin, 1987). Selection bias may occur if any of these situations results in a nonrandom subset of the study population, affecting both internal and external validity (Berk, 1983). Data cleaning procedures include employing multiple imputation (Schafer & Graham, 2002) of incomplete multivariate data under a normal model software (NORM) to deal with missing data inherent in longitudinal data collection efforts. This represents a significant improvement to the use of older procedures, such as case deletion and single imputation, or assuming data are missing at random (MAR). Specifically, three complete data sets were imputed, and analyses are replicated on each of these data sets, following the standard algorithms to compute point estimates and standard errors. Estimates are then averaged across multiple imputations to generate a single point-estimate. In recent

analyses of health trajectories, multiple imputation was employed (Liang, Shaw, Krause et al., 2003; 2005).

Models are estimated by growth curves—multilevel models of longitudinal data. Conventional multiple regression models ignore the multilevel structure of the data, or at best, correct standard errors for the nested structure of the data, but do not model variation at higher levels (Raudenbush & Bryk, 2002). Independent variables are measures of elapsed time. In this way, modeling the two equation system yields estimates of within-individual changes across time (level 1) and estimates of between-individual differences defined by covariates in the model (level 2).

Hypotheses concerning the heterogeneity of health status trajectories are tested by applying multilevel models to repeated observations of study participants. An important aspect of Equation 1 is the assumption that the parameters vary across individuals. Thus, individual growth curve parameters (i.e., intercept and slopes of time-related changes) are allowed to vary randomly and are estimated as dependent variables in the level 2 (or person-level) models. In Equation 2, X_{qi} represents included baseline covariates (e.g., age, gender, marital status, health conditions/comorbidities, ethnic group identification) associated with individual i and, β_{pq} represents the effect of X_q on the pth growth parameter (b_{pi}). U_{pi} is a random effect with mean of zero.

Level 1: $$Y_{it} = \pi_{0i} + \pi_{1i}\text{TIME} + \Pi_{2I}X_{\textit{Time-Varying Covariates}} + \varepsilon_i \qquad (1)$$

Level 2: $$\pi_{0i} = \beta_{00} + \beta_{01}X_{\textit{Mexican}} + \beta_{02}X_{\textit{Black}} + \Sigma\beta_{pq}X_{qi} + U_{00} \qquad (2)$$

$$\pi_{1i} = \beta_{10} + \beta_{11}X_{\textit{Mexican}} + \beta_{12}X_{\textit{Black}} + \Sigma\beta_{pq}X_{qi} + U_{11} \qquad (3)$$

In Equation 1, Y_{iA} is health status for individual i at time t; b_{0i} is the intercept of comorbidity for individual i; b_{1i} is the rate of change (slope) in comorbidity for individual i across different time periods; and ε_{iA} represents random error in health status for individual i at time t.

In the proposed analysis, both linear and non-linear changes in disease were considered. Disease is modeled as a linear, quadratic, and cubic function of time. Using model diagnostics for the significance levels of the linear, quadratic and cubic terms, the linear functional form was selected as the most appropriate for the time norm. In addition, time-varying covariates are included in the level-1 equations of this model. The level 2 equations (2) and (3), allow for the random modeling of the intercept, π_{0i} and slope parameters, π_{1i}.

2.3.3.1 Death, attrition and proxy interviews

Measures for mortality, attrition, and proxy status are used in the models for the sole purpose of controlling selection bias associated with these factors. Indicator variables detailing whether or not a respondent died or had a proxy give an interview anytime in the interval between their baseline year and 2006 were used as controls for selection bias in the analyses. Seven percent of interviews were given by proxy respondents, although some measures such as CES-D, were not obtained by proxy interviews. In order to handle potential biases associated with excluding proxy interviews or attrition, we apply multiple imputation procedures (Schafer & Olsen, 1998) to minimize the loss of subjects due to item missing. Deleting proxy interviews was not considered a viable option given that it could lead to serious selection bias. Additionally, Beckett et al. (2000) note that including proxy interviews in analyses examining self-

reported disease of older respondents is imperative when proxy caregivers are in a position of providing a more accurate reporting of conditions that cause cognitive or physical impairment. Consequently, proxy interviews with imputed data are included in the analyses.

2.4 Findings

Table 2.1 details descriptive statistics for the total sample as well as by white, black and Mexican-origin individuals in the study. Table 2.2 offers descriptive statistics for the time-varying covariates of the analyses by study year. The mean time since the baseline across repeated observations is 5.64 years, while the mean level of total disease comorbidity is 2.08. In addition, the range of comorbidity experienced is a minimum of 0 and a maximum of 7. Approximately 12% of respondents do not experience any chronic conditions. At the upper range of chronic disease in this sample, only 1% of respondents experience 6-7 conditions. Although the full range of comorbidities is realized, very few respondents experience all measured comorbidities.

At the person-level, 26% of respondents died during the observation period and 9% have a missing wave(s) of information at some point between baseline year and 2006. Additionally, 14% of respondents are black and 4% are of Mexican origin.

Table 2.3 offers the hierarchical linear model results for comorbidity burden. Progressively complex models are explored for each of the dependent variables, starting with time-constant models until the time-varying model is presented. By order of presentation in Table 2.3, the unconditional model (M_0) is followed by a model where controls for proxy response, death and attrition are included (M_1), followed by models

with demographic covariates except education (M_2), as well as a model with demographic factors including education (M_3). These two demographic models are included so as to compare the net effect of education in addition to ethnicity. Finally, models are analyzed which include time-varying marital, proxy and health covariates (M_4). Figure 2.1 offers graphical results of M_4 by ethnic group for the trajectories of total comorbidity burden.

Hypothesis H_1 proposes that comorbidity burden increases with both time (H_{1a}) and age (H_{1b}). From the unconditional model, M_0, we see that the unadjusted comorbidity trajectory increases linearly with mean time (in M_0, b=0.114, p<.001), offering support to H_{1a}. This linear increase with time is also seen net of the fully time-constant and time-varying adjusted model M_4, offering additional support to H_{1a} (in M_4, b= 0.111, p<.001). As for testing the increase of comorbidity burden with age per hypothesis H_{1b}, M_2 first introduces the age covariate in the models. According to the analytic models, age demonstrates a higher intercept in M_2 (b=0.020, p<.001) as well as in M_4 (b=0.028, p<.001). However, although age has a nominally significant slope in M_2 (b=0.000, p<.01), the slope is no longer significant in M_4 (b=0.000). This translates to older individuals exhibiting higher initial levels of comorbidity burden at the beginning of the observation period, but no significant increases in the change of comorbidity when we include time-varying health covariates in the model.

The analytic models also offer some insight into racial and ethnic differences in comorbidity burden. These results offer partial support for hypothesis H_2, where black Americans were hypothesized to have higher comorbidity burden trajectories relative to whites. There are significant differences between blacks and whites in both the intercept

(in M_4, b=0.102, p<.001) and slope over time (in M_4, b=-0.008, p<.05). According to these results, blacks exhibit significantly increased initial levels of comorbidity relative to whites and a slower rate of disease accumulation over time. However, in hypothesis H_2, we proposed that both the level and rate of change would be greater for blacks relative to whites. From our model results, there is support only for increased initial comorbidity levels for blacks. The negative slope of the trajectory for black Americans indicates it is decreasing over time, possibly approaching a ceiling of comorbid conditions.

There are significant differences between the comorbidity trajectories of Mexican-origin individuals and whites. Results also support H_3, which proposes that Mexican-origin individuals demonstrate either a health advantage over whites with lower intercepts and slopes for comorbidity, or similar trajectories to whites. Both the intercept (in M_4, b=-0.334, p<.001) and rate of disease accumulation (in M_4, b=-0.014, p<.05) for Mexican-origin adults are lower than that for white Americans, demonstrating a comorbidity burden advantage for Mexican Americans. Figure 2.1 shows the graphical model results for comorbidity by the three racial and ethnic groups. From the plot, we see that the trajectory of comorbidity for black individuals is higher than the trajectories for Mexican and white individuals, with Mexican individuals enjoying the lowest initial level and trajectory of comorbidity burden. These analyses offer a more complete picture amount and rate of comorbidity burden these three ethnic groups are espoused with as they age.

Focusing on the results from the time-varying model (M_4), various included covariates play an important role in the shaping of comorbidity trajectories. For example, higher education is associated with fewer reported disease conditions at the outset (in M_4,

b=-0.024, p<.001) as well as a lower slope of the trajectory (in M_4, b=-0.001, p<.01). Moreover, the addition of education to the model moderately attenuates the already significant relationship between comorbidity and black and the addition of education to the model significantly alters the direction of the relationship between comorbidity and Mexican ethnicity. In comparing M_2 and M_3, we verify that the black effect persists despite accounting for varying education levels. However, including education in the model results in significant findings for Mexican-origin adults, who have lower levels and slower rates of accumulation of comorbidity burden in relation to whites.

From model M_4 we also determine that the lagged and change in health covariates all play significant roles in the tracing of comorbidity trajectories. Higher functional impairment, greater reporting of ill self-rated health, greater numbers of depressive symptoms, and higher BMI in the previous period all contribute to higher trajectories of comorbidity. In addition, greater changes in these health covariates in adjacent time periods also contribute significantly to greater comorbidity. It is interesting to note that greater time-varying health limitations consistently contribute toward higher comorbidity trajectories.

2.5 Discussion

This research provides new information concerning ethnic variations in health changes by quantitatively depicting the trajectory of comorbidity in black, white, and Mexican Americans. Middle-aged and older Americans have on average two chronic diseases at the baseline, with an increase of 0.11 per year to nearly three conditions in 2006. White Americans differ from black and Mexican Americans in terms of level and rate of change of comorbidity. Mexican Americans demonstrate lower initial levels and

slower accumulation of comorbidities relative to whites. In contrast, blacks showed an elevated level of comorbidity throughout the 11-year period of observation, although their rate of change decelerated over time relative to whites.

Complementing prior observations of ethnic disparities in mortality and single diseases (Angel & Angel, 2006; Hummer, Benjamins & Rogers, 2004; Mensah et al., 2005), our research extends our understanding of differences in comorbidity across black, white and Mexican Americans. The difference between black and white Americans can be largely characterized as persistent inequality (Ferraro & Farmer, 1996). However, because of the smaller rate of change in comorbidity among blacks, this differentiation is diminishing over time. Specifically, comorbidity among blacks was 9% higher than that of whites (1.87/1.72) in 1995, which narrowed to 2% (3.01/2.96) in 2006 (Figure 2.1). If this rate of change persists for another decade, comorbidity between blacks and whites may fully converge. This would be consistent with the prediction of the age-as-leveler hypothesis. On the other hand, increasing disparity in comorbidity as implied by the hypothesis of cumulative disadvantage does not appear to apply in this context.

According to our findings, the comorbidity trajectory for Mexican Americans is lower than that of white Americans, hence aligning broadly with the concept of the Hispanic Paradox or perhaps more specifically, the Mexican Paradox. Still, these results should be regarded as a preliminary step in understanding disease trajectories for Mexican Americans. The Paradox moniker describes Hispanics that display advantageous outcomes relative to their low socio-economic status. However, further examination of immigration dynamics for this group is warranted. Specifically, parsing out trajectories for Mexicans while considering immigration dynamics (nativity status,

generational status, time since migration, and education level at the time of migration) would offer insight into the development of the comorbidity trajectory for Mexican-origin adults that are foreign-born relative to U.S.-born.

Comorbidity was measured in this research by a composite of seven chronic conditions. These seven diseases would probably differ substantially in their etiologies and health consequences. Therefore, parallel analyses focusing on single diseases, one at a time, are needed to further understand the trajectories of chronic conditions. Furthermore, for a given level of comorbidity, the disease mix may differ. How these trajectories interface with one another and how they jointly affect health outcomes (e.g., disability, depressive symptoms) remain important topics for future research. More importantly, research concerning how these processes differ across blacks, whites, and various Hispanic subgroups is critical for a more complete understanding of ethnic variations in health dynamics. Disentangling these would allow for improved management and treatment of multiple conditions.

In addition to ethnicity, this research also shed some light on the influences of other dimensions of social stratification (e.g., age, gender, and SES) on comorbidity. For instance, individuals in an older age group experienced a higher level of chronic diseases. Nonetheless, age difference in the rate of change was largely a function of socioeconomic status and prior health. Although women did not differ from men in their initial level of comorbidity they did differ from men in the rate of change. Those with more education experienced less comorbidity as well as slower rates of comorbidity change over time. It is therefore important to take these variables into account when examining ethnic differences in comorbidity. Furthermore, how various dimensions of social stratification

interact in affecting comorbidity remains to be analyzed.

The present study can be improved in several aspects of which future research is required. First, HRS tracks individuals 51 years of age and over. Health disadvantages and differential mortality that may have occurred before middle age are not traced here, and are an important consideration when interpreting the findings. To gain a more complete understanding of ethnic variations in health changes over the life span, longitudinal data base including individuals under the age of 51 would be extremely useful.

Second, nativity has consistently been an important predictor when examining Hispanic disease profiles in the U.S. (Crimmins et al., 2007; Angel, Buckley, & Sakamoto, 2001). Covariates for foreign-born Mexican and age at immigration were included in exploratory analyses (not shown) to address some of these concerns although neither of these factors were significant. It is possible that there is insufficient sample size within subgroups (e.g., foreign born Mexican-origin individuals, n=313) to detect differences between the groups. In addition, we have concerns of potential bias introduced by socioeconomic differences in return migrants. Wong, Palloni and Soldo (2007) find a positive long-term effect of U.S. migration for Mexican return migrants. That is, older Mexicans who return from a migration spell in the U.S. are more likely to be in the wealthiest strata of the income distribution in Mexico. It is possible that we are not observing total reported comorbidity for Mexican-Americans if HRS Mexican-ethnicity participants with more means and access to health care—and thus, diagnoses—are migrating back to Mexico and no longer captured by our analyses. Further research is

necessary to disentangle the complex relationship between immigration and health over time for Mexican-origin individuals.

Third, there is a concern related to the use of self-reported disease indicators, particularly with reference to the extent of inconsistencies over time (Beckett, Weinstein, Goldman, et al., 2000). In addition, self-report and clinical records of diagnosed diseases depend greatly on the health care seeking behavior of individuals. Differences between ethnic groups in access to healthcare have important implications to this work. It is possible that under-diagnosis may obscure even greater differences across ethnic groups. Hence, limited access to diagnosis by Mexican-origin immigrants, particularly recent immigrants with fewer resources and less familiarity with available health care options may lead to an underestimation of differences in the burden of comorbidity. Still, this concern is likely to be small for older adults. Previous reports from the Hispanic Established Populations for the Epidemiologic Studies of the Elderly indicate that 87% of Mexican Americans in the Southwest are covered by Medicare (Markides, Rudkin, Angel, et al., 1997, p. 295).

Fourth, there might be significant heterogeneity in changes in comorbidity, which is not explored in this research. For instance, recent analysis of data from the Health and Retirement Study has shown that underlying the average trajectory of disability, there are five distinct courses of change including (a) excellent functional health, (b) good functional health with small increasing disability, (c) accelerated increase in disability, (d) high but stable disability, and (e) persistent severe disability (Liang, Xu, Bennett, Ye, & Quiñones, 2009). Similar heterogeneity may exist for the trajectory of comorbidity. Future work is needed to model groups that may experience substantially different levels

of comorbidity.

Although age-based analysis would be appealing in the interpretation of model results, age-based analysis is appropriate only in cases where there is an adequate number of repeated observations for each age so as to capture the proper form of the within-individual growth (Mehta & West, 2000). Due to the wide age differences among HRS respondents at the baseline and an inadequately small age distribution at the older age spectrum, age and cohort effects are highly confounded. Because there is insufficient data to estimate cohort effects, we instead focus our analyses on time-related changes. Still, further disentangling these age-period-cohort effects in our analyses would provide great interpretive gains of comorbidity dynamics attributable to secular time and age-related changes, and should be explored in future analyses (Jacobs, Hannan & Wallace, et al., 1999).

Many middle-aged and older Americans face multiple chronic conditions simultaneously which are increasing with time and age. Black Americans showed an elevated level of comorbidity throughout the 11-year period of observation, while Mexican Americans show a favorable trajectory of co-existing chronic conditions relative to white Americans. Further research is needed concerning the impact of left truncation, nativity, nature of self-reported diseases, and heterogeneity underlying the average trajectory of comorbidity.

Table 2. 1 Descriptive Statistics for Hierarchical Linear Models

	Total		White		Black		Mexican	
Measures	**mean**	**(s.d.)**	**mean**	**(s.d.)**	**mean**	**(s.d.)**	**mean**	**(s.d.)**
Level 1 (*intrapersonal changes*)	n=67,358		n=55,051		n=9,220		n=3,087	
Disease burden (index 0-7)	2.08	(1.35)	2.04	(1.35)	2.34	(1.35)	2.00	(1.36)
Time since baseline year	5.64	(2.74)	5.65	(2.74)	5.58	(2.74)	5.67	(2.75)
Proxy, time (t-1)	0.06	(0.25)	0.06	(0.23)	0.08	(0.28)	0.13	(0.33)
Married, time (t-1)	0.69	(0.46)	0.72	(0.45)	0.51	(0.50)	0.72	(0.45)
Household income, time (t-1)	63.74	(97.66)	69.39	(95.5)	40.90	(53.9)	34.87	(182.93)
Body mass index, time (t-1)	27.71	(5.34)	27.32	(5.09)	29.55	(6.19)	28.85	(5.42)
Functional status, time (t-1)	0.49	(1.43)	0.41	(1.27)	0.87	(1.93)	0.82	(1.92)
Self-rated health, time (t-1)	2.78	(1.11)	2.67	(1.09)	3.17	(1.10)	3.32	(1.09)
Depressive symptoms, time (t-1)	1.86	(2.06)	1.73	(1.98)	2.34	(2.24)	2.58	(2.43)
Δ Proxy	0.01	(0.21)	0.01	(0.19)	0.01	(0.24)	0.01	(0.29)

Δ Marital status	-0.03	(0.21)	-0.03	(0.21)	-0.03	(0.22)	-0.03	(0.20)
Δ Household income	0.04	(102.03)	-0.48	(100.6)	2.27	(64.3)	2.19	(185.18)
Δ BMI	-0.03	(2.06)	-0.02	(1.91)	-0.07	(2.65)	-0.02	(2.47)
Δ Functional status	0.19	(1.31)	0.18	(1.21)	0.22	(1.66)	0.21	(1.68)
Δ Self-rated health	0.10	(0.91)	0.11	(0.89)	0.08	(0.99)	0.10	(0.97)
Δ Depressive symptoms	0.12	(2.00)	0.13	(1.94)	0.09	(2.18)	0.08	(2.48)
Level 2 (*interpersonal differences*)	n=17,517		n=14,279		n=2,461		n=777	
Died (between baseline and 2006)	0.26	(0.44)	0.26	(0.44)	0.28	(0.45)	0.24	(0.43)
Ever attritted (between baseline and 2006)	0.09	(0.29)	0.09	(0.28)	0.11	(0.32)	0.09	(0.29)
Age in 1995	64.33	(10.26)	64.65	(10.26)	63.23	(10.26)	62.41	(9.80)
Female	0.57	(0.50)	0.56	(0.50)	0.62	(0.48)	0.54	(0.50)
Education (in years)	12.00	(3.32)	12.53	(2.82)	10.61	(3.65)	6.98	(4.57)

Note: Level 1 is associated with repeated observations for survey participants. Level 2 is associated with individuals at the baseline (i.e., 1995 for AHEAD, 1996 for HRS, and 1998 for WB and CODA).

Table 2. 2 Time-Varying Covariates and Year of Survey

	1998		2000		2002		2004		2006	
Measures	**mean**	**(s.d.)**	**mean**	**(s.d.)**	**mean**	**(s.d.)**	**mean**	**(s.d.)**	**mean**	**(s.d.)**
Proxy, time (t-1)	0.07	(0.25)	0.07	(0.25)	0.07	(0.25)	0.07	(0.26)	0.06	(0.23)
Married, time (t-1)	0.72	(0.45)	0.71	(0.45)	0.68	(0.47)	0.67	(0.47)	0.66	(0.47)
Household income, time (t-1)	58.45	(82.76)	62.08	(119.35)	62.40	(103.86)	60.81	(89.89)	72.6	(73.92)
Body mass index, time (t-1)	27.51	(5.27)	27.58	(5.22)	27.76	(5.30)	27.79	(5.37)	27.75	(5.49)
Functional status, time (t-1)	0.44	(1.39)	0.44	(1.42)	0.45	(1.42)	0.60	(1.51)	0.62	(1.52)
Self-rated health, time (t-1)	2.71	(1.14)	2.85	(1.14)	2.75	(1.11)	2.79	(1.09)	2.85	(1.09)
Depressive symptoms, time (t-1)	1.76	(1.96)	1.97	(2.11)	1.94	(2.09)	1.89	(2.12)	1.83	(2.12)
Δ Proxy	0.02	(0.22)	0.02	(0.20)	0.01	(0.21)	-0.00	(0.20)	-0.01	(0.20)
Δ Marital status	-0.02	(0.14)	-0.05	(0.26)	-0.03	(0.21)	-0.02	(0.20)	-0.03	(0.20)
Δ Household income	-0.30	(91.0)	-1.96	(118.02)	-3.68	(88.63)	10.42	(106.92)	-3.36	(93.80)

Δ Body mass index	0.08	(1.84)	0.12	(1.96)	-0.04	(2.09)	2.09	(2.04)	-0.10	(2.20)
Δ Functional status	0.18	(1.37)	0.12	(1.28)	0.32	(1.37)	0.19	(1.27)	0.127	(1.31)
Δ Self-rated health	0.26	(0.94)	-0.03	(0.92)	0.11	(0.90)	0.11	(0.88)	0.076	(0.88)
Δ Depressive symptoms	0.38	(2.05)	0.06	(2.03)	0.03	(2.02)	0.03	(1.99)	0.106	(1.95)

Table 2. 3 Hierarchical Linear Model Results for Total Comorbidities

	Model 0		**Model 1**		**Model 2**		**Model 3**		**Model 4**	
Covariates	**Coefficient**	**p**	**Coefficient**	**p**	**Coefficient**	**p**	**Coefficient**	**p**	**Coefficient**	**p**
Fixed Effect										
Time-Varying Variables										
Proxy, time (t-1)									0.030	
Married, time (t-1)									-0.014	
Household income, time (t-1)									0.000	*

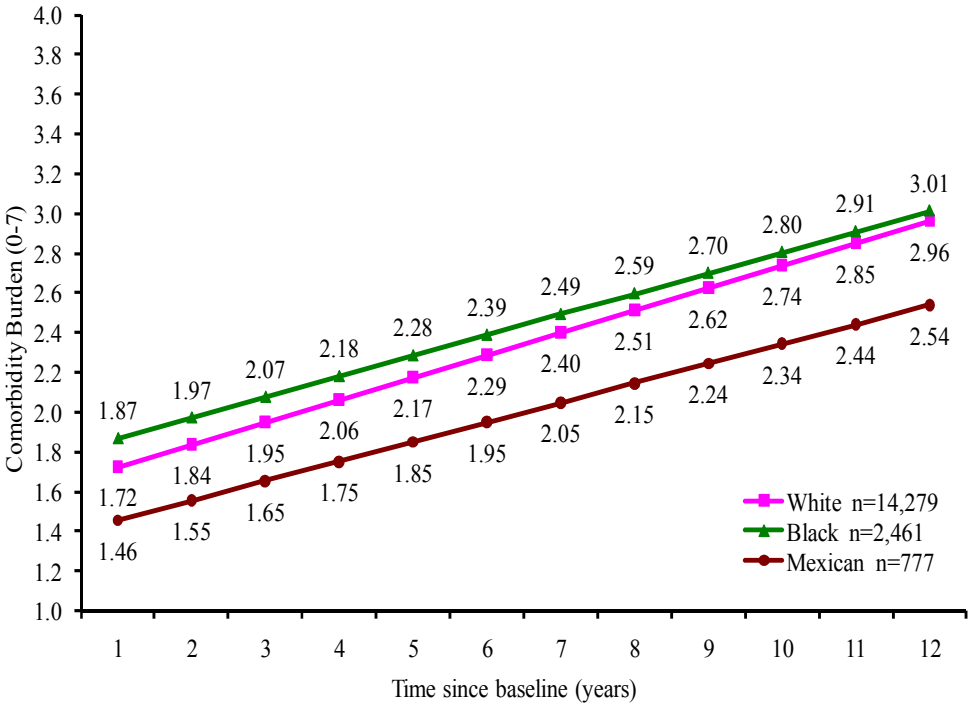
Body mass index, time (t-1)					0.028***
Functional status, time (t-1)					0.043***
SRH, time (t-1)					0.233***
Depressive symptoms, time (t-1)					0.023***
Δ Proxy					0.026
Δ Marital status					0.007
Δ Household income					0.000
Δ Body mass index					0.009***
Δ Functional status					0.025***
Δ SRH					0.137***
Δ Depressive symptoms					0.013***
For Intercept, B0					
Intercept	2.168***	2.185***	2.188***	2.188***	2.244***
Death		0.842***	0.630***	0.600***	0.626***

Ever attrit		-0.122***	-0.111***	-0.121***	-0.111***
Proxy		0.021	0.020	-0.056	
Age (in 1995)			0.020***	0.017***	0.028***
Female			0.008	-0.008	-0.009
Non-Hispanic black			0.301***	0.199***	0.102***
Mexican			0.003	-0.293***	-0.334***
Education				-0.053***	-0.024***
For Time slope, B1					
Intercept	0.114***	0.120***	0.120***	0.120***	0.111***
Death		0.035***	0.030***	0.030***	0.036***
Ever attrit		-0.004	-0.002	-0.002	0.002
Proxy		0.000	-0.003	-0.004	
Age (in 1995)			0.000*	0.000*	0.000
Female			-0.012***	-0.012***	-0.011***

Non-Hispanic black			-0.009**	-0.010**	-0.008*
Mexican			-0.004	-0.008	-0.014*
Education				-0.001	-0.001**
Random Effect					
Intercept 1	1.673***	1.551***	1.506***	1.483***	1.139***
Time slope	0.012***	0.012***	0.012***	0.012***	0.013***
Level-1, R	0.109	0.109	0.109	0.109	0.100

Note: Reliability estimates are based on 15,677 of 17,517 units that had sufficient data for computation. Fixed effects and variance components are based on all the data. Household income is inflation-adjusted and re-scaled; it is reported per 1000s of dollars.
*p<.05, **p<.01, ***p<.001

Figure 2. 1 Trajectories of Comorbidity Burden



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CHAPTER 3:

Risk of Incident Hypertension Among Black, White and Mexican Americans After Age 50

3.1 Introduction

Middle aged and older Americans are increasingly burdened by more than one chronic condition at a time, with cardiovascular disease foremost among these comorbidities. Hypertension continues to be the most prevalent form of cardiovascular disease, and an important contributor to morbidity and mortality in the U.S. (Mensah & Brown, 2007; Cooper, Cutler, Desvigne-Nickens, et al., 2000). Projected demographic changes for the coming decades add to concerns of a continued upward trend in cardiovascular burden. Aging baby-boomers and rising obesity prevalence among younger Americans have the potential of greatly exacerbating the state of cardiovascular illness in the U.S. Associated with these increases in morbidity are rising health care costs, making efforts to ramp up prevention and better elucidate trends in hypertension risk a policy imperative (Mensah & Brown, 2007; Vasan, Beiser, & Seshadri, et al., 2002).

Despite national goals to reduce and eliminate ethnic disparities in health outcomes (Smedley, Stith & Nelson, 2002), disparities in cardiovascular conditions continue to be persistent. Surveillance data describe high prevalence of hypertension among black adults, and cite Hispanics and persons of low socioeconomic status to be

most at risk for developing cardiovascular disease (Mensah, Mokdad & Ford, et al., 2005; Mensah & Brown, 2007). Although control of well-established clinical risk factors has improved for adults that age into Medicare, ethnic and socioeconomic disparities in cardiovascular disease burden have not diminished (McWilliams, Meara & Zaslavsky, et al, 2009). To the contrary, recent findings from Geronimus and colleagues (2007) cite greater increases in hypertension prevalence for black Americans, with widening disparities over working ages.

Less is known about the incidence of hypertension among older adults. An increasing amount of work focuses on the social patterning of cardiovascular risk factors and incident hypertension for young adults. For example, Wang et al. (2006) observe significant differences in the trajectories of ambulatory blood pressure between black and white young adults that persist even after controlling for socioeconomic factors. Similarly, Matthews et al. (2002) assess the effect of changes in socioeconomic status on incident hypertension over a 10-year period. According to their analyses, young black adults have much higher odds of developing hypertension relative to white young adults. In addition, low socioeconomic status is strongly and independently associated with incident hypertension. However, less is known about hypertension incidence for the near-elderly and elderly.

Few studies focus on hypertension incidence for older minority adults over time. Vasan and colleagues (2002) examine factors associated with hypertension risk for older Americans. They determine that residual lifetime risk of developing hypertension is high for both, participants at 55 years of age as well as participants 65 years of age. High residual lifetime risk of hypertension represents a large public health burden and dictates

efforts toward primary prevention not be limited to middle-aged and younger individuals. Still, these studies do not compare differences in the risk of developing hypertension for various ethnic groups in the U.S.

Current work yields valuable insight into hypertension risk for Americans at middle and old age for three different ethnic groups. This work aims to add to current understanding in health disparities and aging by examining incident hypertension over a period of 11 years for aging white, black and Mexican Americans in the U.S.

3.2 Hypotheses

Hypothesis 1 (H_1): The risk of hypertension increases with time (H_{1a}). In addition, the risk of hypertension increases for older age groups relative to middle aged individuals (H_{1b}).

Hypertension incidence is expected to increase over time. This secular time trend is ascribed to documented increases in the literature of incidence as well as the rise in risk factor prevalence for hypertension. In addition, the risk factors for hypertension onset are well established and validated on young adults as well as the elderly (Mosley & Lloyd-Jones, 2009). Abdominal obesity, high levels of blood lipids (Mensah & Mokdad, 2005) and smoking (Cooper et al., 2000) have been identified as critical risk factors for the onset of hypertension. Among the elderly with no history of hypertension, the residual lifetime risk of developing the disease is upwards of 90% (Vasan et al., 2002).

Biological changes in individuals with age are strong correlates for disease onset. Hemodynamic trends of linear increases of systolic blood pressure (SBP) coupled with vascular changes (i.e., arterial hardening that occurs with age) are associated with higher

hypertension incidence (Mosley & Lloyd-Jones, 2009). In addition, examination of blood pressure changes for the U.S. population in the late 1980s and early 1990s indicates that systolic blood pressure increases with age for non-Hispanic white, non-Hispanic black and Mexican Americans (Burt, Whelton, Roccella, et al., 1995). Although these studies are not able to follow-up previously non-hypertensive individuals over long periods of time, they do provide a rationale for expecting increased hypertension incidence with age.

Hypothesis 2 (H_2): Incident risk of hypertension for our sample of black adults will be higher relative to white adults (H_{2a}). The inclusion of time-varying health covariates will attenuate but not entirely account for this gap (H_{2b}).

Various social factors influence the development of chronic disease and disability for middle aged and older adults in the U.S. A considerable amount of the literature describes observed differences in health status among blacks and Hispanics relative to whites in the U.S. (Lynch & Smith, 2005; Freedman, Martin & Schoeni, 2002). Several studies examine whether minorities are placed in double jeopardy—where poor and declining health is not only due to the aging process, but also due to occupying a low-status segment in society. However, this hypothesis does not garner much empirical support (Ferraro & Farmer, 1996). Other hypothesized explanations include lifestyle choices and discrimination (Williams & Collins, 1995), which have been proposed to account for observed differences in minority health status.

Evidence from the epidemiological and demographic literature suggests that blacks have a higher prevalence of disability and disease and thus live in suboptimal health longer than their white counterparts (Freedman et al., 2002). According to this research, blacks exhibit illness earlier and die at younger ages than whites. High levels of socioeconomic inequality account for much of the observed disparities in health at younger ages and early adulthood, with socioeconomic differences narrowing into old age (Beckett, 2000). These may be attributed to racial crossovers in morbidity and mortality and selective mortality due to the accumulation of health disadvantages over the lifecourse.

In addition, given considerable social stratification throughout the lifecourse, social structure influences health status through ethnicity, gender, and socioeconomic status dimensions (House et al., 1994; Ross & Wu, 1996; Hagestad & Dannefer, 2001; Williams, 1997; Williams, 2005; Lynch & Smith, 2005). Race and ethnic differences in education, income levels, and segregated living conditions are described by Link and Phelan (1995) as more distal *fundamental causes* of disease. In this framework, disadvantages stemming from social inequalities accumulate over the lifespan and result in later-in-life differences in health. These may operate through divergent employment and occupational experiences, income and wealth streams, life styles, and health behaviors (Dannefer, 2003; Hayward, Miles, Crimmins, et al., 2000; Bulatao & Anderson, 2004; Hertzman, 2004). Differences in risk profiles for disease reflect differences in the combination of these factors over time (Hayward et.al, 2000).

Social processes over the lifecourse work to systematically and negatively affect health throughout the lifespan. Blacks are expected to demonstrate increased risk of

hypertension incidence relative to whites. In addition, epidemiological evidence for hypertension disease rates suggest that rates for black adults are higher than white Americans net of demographic socioeconomic and health covariates (Mensah et al., 2005; Crimmins et al., 2007; Geronimus et al., 2007). Despite the inclusion of controls for baseline health status in prior studies, black Americans still demonstrate higher a propensity to have hypertension relative to white Americans.

Hypothesis 3 (H_3): Incident risk of hypertension for Mexican American adults in our study will be elevated relative to white Americans (H_{3a}). The inclusion of time-varying health covariates will attenuate but not entirely account for this gap (H_{3b}).

Recent work examines the state of health research on Hispanic populations, and specifically addresses the Hispanic Health Paradox (Markides & Eschbach, 2005; Crimmins, Kim, Alley, et al., 2007). This epidemiological paradox refers to the finding that for some health indicators, Hispanics demonstrate outcomes comparable to white Americans despite being socioeconomically similar to black Americans. There are several proposed explanations for this phenomenon. Among them are poor reporting of mortality statistics, health-protective cultural advantages, self-selection of healthy immigrants, and return-migration by frail individuals so that they are no longer captured by U.S. morbidity or mortality statistics (Markides & Eschbach, 2005).

However, the ubiquitousness of the Hispanic Health Paradox is often challenged (Palloni & Morenoff, 2001). While studies utilizing several nationally-representative data sources find evidence for a Hispanic mortality advantage (Markides & Coreil, 1986;

Franzini, Ribble & Keddie, 2001), other studies find no such advantage (Carrasquillo, Lantigua & Shea, 2000). The balance of the evidence suggests that the Hispanic Paradox is most strongly supported by a mortality advantage for Mexican-born persons, males, and older individuals (Crimmins et al., 2007). Despite our analyses focusing on middle and older aged Mexican Americans, prior studies find evidence of higher risk of cardiovascular disease for Hispanics (Mensah & Brown, 2007). Given these epidemiological findings, we expect incident hypertension to be higher for our sample of Mexican Americans, net of time varying health status. Although we expect that including changing health profiles will diminish hypertension risk, we do not anticipate the risk for Mexican Americans and whites to converge.

3.3 Methods

3.3.1 Data

This study uses data from the Health and Retirement Study (HRS), collected at the University of Michigan's Institute for Social Research. The HRS respondents are a nationally-representative sample of community-based adults aged 51 and over and identified through screening of an area probability sample of households. The study involves individuals from several birth cohorts, including: the Asset and Health Dynamics of the Oldest Old (AHEAD; born prior 1924), the Children of the Depression Age (CODA; born 1924-1930), the Health and Retirement Study cohort (HRS, born 1931-1941) and War Babies (WB, born 1942-1947). In 1998, the Health and Retirement Study added the CODA and WB cohorts to the study and consolidated data collection efforts for all birth cohorts. Prior to 1998, data collection on the original HRS cohort occurred on even years and in odd years for AHEAD data collection.

Due to wave incompatibility of key independent variables, these analyses use seven waves of data from the HRS (1995-2006). Data management and analysis is conducted with Stata 10.0 (Stata Corp., College Station, Texas).

3.3.2 Measures

3.3.2.1 Hypertension

An indicator variable for self-reported hypertension is used to measure disease status in the analyses and used to estimate hypertension incidence. Self-reported health and disease status have been well established and validated in earlier studies (Johnson & Wolinsky, 1993; Ferraro & Wilmoth, 2000; Mensah et al., 2005), and is widely used in aging and epidemiological research. In addition, nationally representative data collection instruments provide consistent estimates of incidence when compared to clinical studies (Glymour & Avendano, 2009).

The Health and Retirement Study asks respondents if they have been diagnosed with hypertension each interview year. In subsequent interviews, individuals were given the option to dispute their preloaded responses from the previous interview. In order to deal with responses that offer conflicting information, we examine additional information reported by respondents. Consultations with geriatric physicians provided the clinical criteria for satisfying the burden of proof for each of the seven reported diseases. For each disease, a dispute was corroborated by examining the evidence variables from the previous interview. For example, if an individual gives conflicting reports of hypertension diagnosis, we utilize information on use of hypertensive medication to verify the diagnosis.

3.3.2.2 Ethnicity

The principal covariates of interest in the analyses are indicators for self-reported black and Mexican ethnicity. Ethnicity is constructed as mutually-exclusive indicator variables for non-Hispanic white, non-Hispanic black and Mexican-ethnicity individuals. Consequently, dummy variables for black and Mexican are included in the analytic models and are each interpreted relative to white study participants. Only Hispanic respondents self-reporting as having Mexican ancestry are included in these analyses. Ideally, we would analyze other Hispanic subgroups, however the HRS does not track each specific Hispanic subgroup for the time period examined. Other race and other Hispanic types are excluded from these analyses.

3.3.2.3 Health status

Several covariates are used to mark the physical and mental health status of respondents in accounting for onset of hypertension. Health status is operationalized by covariates for self-rated health, functional limitations and depressive symptoms. These are included in the analyses as time-varying lagged values as well as change scores. Both, previous period health information and net health change are conceptualized as contributing unique information in models of incident hypertension.

Lagged covariates are constructed as health status at time (t-1) and predict hypertension risk at time t. Lagged self-rated ill health (SRH) is measured with a 5-item scale (1=excellent, 2=very good, 3=good, 4=fair and 5=poor). Lagged functional status (0-11) incorporates both, activities of daily living (ADL, 0-6) and instrumental activities of daily living (IADL, 0-5), with higher scores reflecting increasing number of difficulties with any of the ADL or IADL activities. Lagged depressive symptoms are

measured with the Center for Epidemiological Studies Depression Scale (CES-D, 0-9) with a higher score reflecting higher depressive symptoms.

Change scores are constructed as the difference in health status between time t and time (t-1). Change in self-rated ill health (SRH) reflects current wave SRH minus previous wave SRH. Analogously, changes in functional status and depressive symptoms also represent current minus previous wave values. Positive change scores reflect an increase in the burden of health grievances, while negative scores denote improvement via divestment of health problems. A change score of zero denotes no change in health conditions across adjacent waves.

Time-varying health covariates are intended to provide some control of population heterogeneity in health status. We conceptualize health status as multidimensional, where physical and mental health limitations work in concert to influence chronic disease emergence. Specifically, health status in previous time periods has a bearing on current period chronic disease development. That is, global self assessment of health, functional limitations and depressive symptoms are conceptualized as confounding variables in an individual's future experience with chronic disease.

3.3.2.4 Health behaviors

In addition to physical and mental health measures, we also incorporate several behavioral risk factors for hypertension into our model. We include body mass index (BMI) as lagged and change score covariates. BMI is calculated using respondents' self-reported weight for each interview year and height reported at baseline. We also include previous wave smoking status as well as a change score covariate.

3.3.2.5 Social stratification and social support

Various controls for demographic and socioeconomic factors are included as independent, time-constant and time-varying covariates in the analysis. Age is measured as age in 1995 for all individuals in the study, regardless of entry cohort. Education is measured as a continuous variable denoting years of schooling (range 0-17). Lagged and change in income is inflation-adjusted to 2006 levels, and was also re-scaled (reported per 1000s of dollars) to facilitate its estimation. Marital status is conceptualized as an indicator of social support for individuals, and is constructed as a lagged time-varying covariate. The change in marital status (range -1 to 1) reflects dissolution/widowhood, no change, and acquisition of partners at each point in time over the study period.

3.3.3 Data analysis

Discrete-time survival analysis is used to model the onset of hypertension. The discrete-time hazard represents the risk of event occurrence in each discrete time period among people in the risk set. An important feature of the analysis is that the probability is conditional—the risk of hypertension is estimated for those respondents who have not been previously diagnosed (Singer and Willett, 1993). Consequently, persons already diagnosed with hypertension were excluded from the analyses. 44% (n=7719) of the baseline sample are excluded from the analyses due to prevalent hypertension diagnoses. Of these, black hypertensives comprise 18% (n=1525) and Mexican hypertensives 4% (n=334) of the excluded cases.

We use Stata macros developed by Dinno (2002) to estimate the model using the logit link and adjust for clustering within households. In estimating discrete time hazard

models, the logistic transformation yields the conditional log-odds of the risk of hypertension onset:

$$\log \frac{h(t_{ij})}{1-h(t_{ij})} = [\alpha_1 D_{1ij} + \alpha_2 D_{2ij} + \ldots + \alpha_J D_{Jij}] + [\beta_1 X_{1ij} + \beta_2 X_{2ij} + \ldots + \beta_P X_{Pij}] \quad (1)$$

Equation (1) describes the conditional probability that individual i will experience hypertension diagnosis in time period j, given that individual i is free of hypertension in time period j-1. D_{1ij}- D_{Jij} represent indicators for each time period. The associated parameters $\alpha_1 - \alpha_J$ represent multiple intercepts for each time period. Taken together, these form the log-odds of the baseline hazard of hypertension occurrence. The slope term parameters β_1- β_P assess the unit-difference effects of predictors X_{1ij}-X_{Pij} on the risk of developing hypertension.

A consideration in using longitudinal data is the possibility of missing data at follow-up due to item non-response, survey non-response, and mortality (Little & Rubin, 1987). Selection bias may occur if any of these situations results in a nonrandom subset of the study population, affecting both internal and external validity (Berk, 1983). We employ multiple imputation (Schafer & Graham, 2002) of incomplete multivariate data under a normal model software (NORM) to deal with missing data inherent in longitudinal data collection efforts. This represents a significant improvement to the use of older procedures, such as case deletion and single imputation, or assuming data are missing at random (MAR). Specifically, three complete data sets were imputed, and analyses are replicated on each of these data sets, following the standard algorithms to compute point estimates and standard errors. Estimates are then averaged across multiple

imputations to generate a single point-estimate. In recent analyses of longitudinal health data, multiple imputation was employed (Liang, Quiñones & Bennett, et al., 2010).

3.3.3.1 Mortality

In order to account for competing risks to disease incidence, separate discrete-time survival models for mortality are also examined. A full model of the hazard of mortality is estimated for the same population of respondents free of hypertension at the outset, and includes controls for demographic, socioeconomic and health status changes over time. The risk of dying is examined to determine whether selective mortality is occurring for black and Mexican Americans relative to white Americans in our sample.

3.4 Findings

Table 3.1 details sample descriptive statistics collected at the baseline interview. Similarly, Table 3.2 presents descriptive statistics for the sample at each time interval. The mean age for our total sample is 63 years, and 56% are female. The ethnic composition of our sample shows 10% of respondents are black and 5% are of Mexican origin.

Table 3.3 presents the discrete-time hazard model results for the risk of developing hypertension. We first present the unconditional model ($M_{3.0}$) to ascertain the shape of the baseline hypertension hazard. This is followed by progressively more complex models. Table 3.3 reports the model results as fitted odds ratios. Model coefficients are transformed into predicted probabilities of the hazard using the formula, $\frac{1}{e^{-(\alpha jDj+\beta pXp)}}$ (Dinno, 2002). Figure 3.1 presents the plot of the predicted probability of developing hypertension for the model which includes only ethnic group identification.

Figures 3.2 and 3.3 present plots detailing the predicted probability of developing hypertension for the time-varying health adjusted model ($M_{3.3}$) by race/ethnic group and by race/ethnic age groups for each time period, respectively.

Hypothesis H_{1a} proposes that the incidence of hypertension increases with time. This hypothesis is supported by model results. The baseline hazard for hypertension in $M_{3.0}$ (unadjusted by any predictors) demonstrates a steady increase in the risk of incident hypertension over time. The probability of being diagnosed with hypertension in the first period is 0.089 and rises to 0.134 by the final time period. We also hypothesize in H_{1b} that the risk of hypertension is higher for older versus younger ages. This hypothesis is supported by model results. Older individuals have higher odds of developing hypertension (in $M_{3.3}$, OR=1.007, $p<.01$).

Figure 3.3 shows $M_{3.3}$ results but with an added dimension of examining race/ethnic by age groups. This figure plots the probability of developing hypertension by both, ethnic and age group. We examine ethnic differences in onset of hypertension by 55 year olds (25^{th} percentile of the age distribution) and 70 year olds (75^{th} percentile of the age distribution). It is interesting to note that both, the younger and older aged black Americans exhibit significantly higher probabilities of hypertension than all other age/ethnic groups examined. Although older black Americans demonstrate the highest probability of developing hypertension, which supports H_{1b}, middle-aged blacks have higher odds of incident hypertension than either older white or older Mexican Americans.

Hypothesis H_{2a} states that the odds of hypertension are higher for black relative to white Americans. In examining models $M_{3.1}$ through $M_{3.3}$, we are able to determine

pronounced differences in the probability of newly diagnosed hypertension for black relative to white Americans. $M_{3.1}$-$M_{3.3}$ detail significantly higher odds of hypertension for blacks (in $M_{3.1}$, OR=1.483, p<.001; in $M_{3.2}$, OR=1.392, p<.001; in $M_{3.3}$, OR=1.278, p<.001). All model results provide support for H_{2a}. Hypothesis H_{2b} states that the inclusion of time-varying health covariates will attenuate but not entirely account for the difference in odds between black and white Americans. Across the three models, the odds are somewhat attenuated but remain significantly higher relative to whites after accounting for differences in demographic and socioeconomic covariates and health status, respectively. The slightly reduced yet significant odds throughout $M_{3.1}$-$M_{3.3}$ support H_{2b}.

Figures 3.1 and 3.2 show sequential model results by ethnic group. Figure 3.1 plots $M_{3.1}$ results and figure 3.2 shows $M_{3.3}$ results by ethnic group. The plots offer visual confirmation of increased probability of developing hypertension for black relative to white and Mexican Americans. Figure 3.2 shows $M_{3.3}$ model results and demonstrates that the conditional probabilities of developing hypertension are highest for black Americans in the time-varying health adjusted model. The figure also presents interesting dynamics over time. Overall, the risk of newly diagnosed hypertension increased between 1995 and 2006 for all Americans over age 50. The probability of incident hypertension among black Americans was 0.10 during the period of 1996-1998, which increased steadily to 0.16 during 2004-2006. In contrast, among white Americans the risk was 0.08 during 1996-1998 and 0.13 during 2004-2006. For Mexican Americans, the probability increased from 0.08 during 1996-1998 to 0.13 during 2004-2006.

Hypothesis H_{3a} suggests that the odds of hypertension are higher for Mexican relative to white Americans. This hypothesis is not supported by model results. Although $M_{3.1}$ details higher odds of hypertension for Mexican-ethnicity individuals (OR=1.303, p<.001), once we account for differences in demographic and socioeconomic covariates, and later for changing health status, Mexican Americans no longer have significantly different odds of incident hypertension relative to whites (in $M_{3.2}$, OR=1.085, p>.05; in $M_{3.3}$, OR=0.966 , p>.05). Given these results, we find no support for H_{3a}. In addition, H_{3b} proposes that the inclusion of time-varying health covariates will attenuate the gap in odds between Mexican and white Americans. This hypothesis is also not supported by the model results, given that the difference in odds observed in $M_{3.1}$ converges after accounting for differences in demographic and socioeconomic status in $M_{3.2}$.

Figures 3.1 and 3.2 offer these results visually. For Mexican Americans, the probability of reporting new hypertension cases is markedly different between the models. Mexican Americans have significantly higher odds of incident hypertension relative to whites in figure 3.1. However, comparing figures 3.1 and figure 3.2, once socioeconomic, health behavior and health status are accounted for, we no longer observe these differences between Mexican and white Americans.

We also examine the competing risk of mortality through separate discrete-time survival models of non-hypertensive persons at the baseline. These results are presented in Table 3.4. In estimating these discrete-time hazard models of mortality, we include a measure of comorbidities. We note that hypertension diagnosis is counted among the

time-varying comorbidities measure in $M_{4.3}$, along with diagnosis of cancer, diabetes mellitus, stroke, heart disease, arthritis and lung disease.

There are interesting dynamics in the probability of dying for the three race/ethnic groups that are non-hypertensive at baseline. During the time period examined, there were significantly lower odds of dying for Mexican Americans relative to whites (in $M_{4.3}$, OR=0.608, $p<.01$) but no differences in the risk of dying for black relative to white Americans (in $M_{4.3}$, OR=0.881, $p=.202$). Mexican Americans 51 and older who are not burdened with hypertension in their entry into the study have lower risk of mortality compared to whites.

3.5 Discussion

This study moves beyond the cross-sectional design of disease prevalence as well as longitudinal studies that only consider transitions between two points in time, and examines hypertension incidence for middle-aged and older individuals in the U.S. In addition, it involves the comparison of both black and Mexican to white Americans. This research is able to determine how interval-by-interval changes in hypertension risk differ for black, Mexican, and white Americans over and 11-year period. The cumulative incidence of developing hypertension over the entire period for the aggregate sample is 44%, with cumulative incidence of 43% for white Americans, 51% for black Americans, and 42% for Mexican Americans. In addition, in examining competing mortality risks, black participants in the HRS had similar mortality risk with respect to whites. However, Mexican Americans demonstrate a mortality advantage with respect to white Americans in their risk of dying over the 11-year period.

The risk of incident hypertension increases over time for all individuals in the HRS. To a significant extent, increases in incident hypertension were a result of changing demographic and health attributes. Relative to white and Mexican Americans, black Americans were 30% more likely to develop hypertension throughout the 11-year period of observation. These variations persisted even when differences in health behaviors, socioeconomic status, demographic, and time-varying health characteristics were adjusted. These results largely support previous studies of increasing hypertension incidence with age (Mosley & Lloyd-Jones, 2009), and extends beyond these studies by tracing persistent ethnic disparities in disease incidence into old age.

Examination of model findings by age groups also revealed interesting results. Although we plotted findings for the 25th and 75th age percentiles expecting a clear pattern of higher odds accruing to older age groups, this was not the case. Both, younger and older blacks in our sample had the highest odds of developing hypertension relative to Mexican and white Americans. Previously undiagnosed blacks of any age in the HRS are most at risk for developing hypertension.

This study was able to examine hypertension risk for one ethnic group among several Hispanic groups in the U.S. The results indicate that hypertension onset for Mexican ethnicity individuals broadly aligns with the concept of the Hispanic Paradox. Mexican individuals in our study did not exhibit significantly different risk of developing hypertension compared to whites. In addition, in examining competing mortality risks, Mexican individuals in the HRS had favorable mortality risk with respect to whites. These results have interesting implications for Mexican Americans. According to these findings, Mexican Americans experience lower mortality risk and similar hypertension

risk compared to whites with comparable health profiles. It is possible that we are not observing true mortality for this ethnic group if the hypothesized salmon bias process is occurring for sick Mexican Americans. However, we cannot confirm this hypothesis with this data.

Addtionally, there could be bias introduced by socioeconomic differences in return migrants. Wong, Palloni and Soldo (2007) suggest that older Mexicans who return from a migration spell in the U.S. are more likely to be in the wealthiest strata of the income distribution. It is possible that we are not observing higher rates of newly diagnosed hypertension if HRS Mexican-ethnicity participants with more means and access to health care are migrating back to Mexico and are no longer in our study sample. Further study of immigration dynamics for Mexican Americans is needed to investigate these results and better sort out heterogeneity by nativity and return-migration behavior. These should be addressed in future studies.

There are several important limitations that need to be acknowledged. First, an important concern to using self-reported health measures, and specifically, self-reported hypertension indicators, is the reliability of responses and consistency in subsequent respondent re-interviews (Beckett et al., 2000). Specific procedures to explore the extent of inconsistencies across an individual's longitudinal record and provide more time-consistent disease indicators is reflected in these analyses. In addition, individuals may be unaware or underreport diagnoses of hypertension due to age and race/ethnicity biases and differential access to good quality clinical practitioners. Ostchega and colleagues (2008) find some substantiation for this concern. Using NHANES surveillance data, they report that blacks with hypertension are more likely to be aware of having hypertension

compared to Mexican Americans with hypertension. However, the authors do not find evidence of differential awareness of diagnosed hypertension between white and black or white and Mexican Americans with hypertension. It is possible to use ongoing and recent HRS collection of measured blood pressure to corroborate reported hypertension. Initial comparison of self-reported and measured blood pressure data in the HRS indicate that the two are reasonably correlated (Weir, 2006). Given these considerations, self-reported receipt of hypertension diagnosis from a physician is a relevant measure of the presence of hypertension.

Second, these findings are largely reliant on individuals in the HRS having access to a hypertension diagnosis, presumably through physician visits and health care system use. Measures of diagnosed disease from self-report and clinical records depend greatly on the health-seeking behavior of individuals. Differences in health-seeking behavior could drive differences between ethnic groups. Although the majority of the study population is Medicare age-eligible, additional analyses (not shown) included time-varying health insurance coverage. Even after controlling for middle-aged and older adults potentially moving in and out of health insurance coverage, incident hypertension is elevated for black relative to white Americans, and statistically similar for whites and Mexican Americans. In recent studies, differences in the control of cardiovascular risk factors between predominantly Spanish-speaking and English-speaking Hispanics are discernible (Eamranond, Legedza & Diez-Roux, 2009). Although we also attempt to control for Spanish-language interview (analyses not shown), as well as sub-analyses on only Mexicans who give interviews in Spanish versus English (analyses not shown), we find no differences in hypertension risk. Language of interview is admittedly a rough

proxy for predominant language use. Further exploration of cultural factors for Mexican Americans with suitable data is warranted.

Finally, it is difficult to ascertain a clear picture of minority health with the HRS sample since the study tracks individuals from middle age into old age and is not able to capture earlier-life selective mortality and hypertension onset. Health disadvantages and differential mortality that may have occurred before middle age are not traced here, and are an important consideration when interpreting the findings. The analyzed sample of black, Mexican and white Americans that survive to middle and old age without diagnosed hypertension is very different than their respective representative population due to differential morbidity and mortality that occurs earlier in the lifecourse. In addition, further efforts to disentangle age-period-cohort effects in our analyses should be explored in future analyses (Jacobs, Hannan & Wallace et al., 1999). Still, these results indicate that hypertension prevention efforts for black populations in the U.S. lag behind those for whites.

Our results indicate aggressive prevention of hypertension even into old age is essential, particularly for black minorities in the U.S. Similar to the recommendations from Mosley and Lloyd-Jones (2009), the dissemination of information and aggressive treatment of hypertension and risk factors for hypertension need not be targeted only to young adults. More targeted efforts are needed to reduce hypertension burden for older aged Americans, and more specifically, to older aged black Americans.

Our research focuses on incidence of hypertension for older adults in the U.S. A second complementary piece to this research would be to examine health trajectories

among individuals who have been diagnosed with prevalent hypertension for older white and minority Americans. Although we do not explicitly model changes in prevalence, we are able to determine that the cumulative incidence of hypertension for the observation period of 1995-2006, which is 44% for the entire sample. It is remarkable that in our analyses of hypertension incidence for the 11-year period, nearly half of the sample is newly diagnosed with hypertension. In addition, we are able to determine that 44% of the baseline sample was diagnosed with hypertension, which represents prevalence in the HRS study population for the period up to 1995. Still, future studies are needed to examine changes in prevalence of hypertension for older Americans given its binary absorbing state specification.

In addition, many adults in the U.S. increasingly face multiple chronic conditions as they age (Paez, Zhao & Hwang, 2009). Potential complications arising from a new hypertension diagnosis has vast implications on the management of multiple diseases for elderly Americans. Further detailing disease case mix for older adults and any resulting ethnic variations in disease profiles would lead to better tailored chronic disease care efforts. Understanding disease case mix and acuity, the extent to which left truncation influences ethnic differences in hypertension incidence are important areas for further study.

Table 3. 1 Sample Descriptive Statistics at the Baseline by Ethnic Group

	Total		**White**		**Black**		**Mexican**	
	N= 9259		**N= 7880**		**N= 936**		**N= 443**	
	Mean	s.d	Mean	s.d	Mean	s.d	Mean	s.d
Proxy, time (t-1)	0.063	0.244	0.058	0.234	0.073	0.261	0.130	0.336
Married, time (t-1)	0.755	0.430	0.773	0.419	0.589	0.492	0.790	0.408
Household income, time (t-1)	66.000	94.460	70.776	99.213	44.239	61.616	28.113	27.801
Functional status, time (t-1)	0.305	1.144	0.270	1.070	0.497	1.428	0.519	1.592
Self-rated health, time (t-1)	2.422	1.090	2.357	1.075	2.701	1.075	2.971	1.143
Depressive symptoms, time (t-1)	1.489	1.779	1.417	1.731	1.788	1.872	2.124	2.189
Comorbidities, time (t-1)	0.758	0.853	0.730	0.795	0.891	1.084	0.667	0.707
Δ Proxy	0.017	0.207	0.016	0.194	0.039	0.255	0.000	0.304
Δ Marital status	-0.019	0.135	-0.018	0.133	-0.023	0.150	-0.017	0.131
Δ Household income	0.753	114.775	0.921	123.110	-1.929	50.316	3.466	29.074

Δ Functional status	0.127	1.146	0.117	1.088	0.199	1.444	0.159	1.398
Δ Self-rated health	0.271	0.938	0.275	0.912	0.268	1.042	0.207	1.134
Δ Depressive symptoms	0.357	1.952	0.339	1.872	0.450	2.187	0.476	2.663
Δ Comorbidities	0.132	0.361	0.116	0.335	0.217	0.465	0.000	0.325
Age in 1995	63.251	10.264	63.490	10.272	62.202	10.396	61.233	9.479
Female	0.559	0.497	0.558	0.497	0.581	0.494	0.521	0.500
Education (in years)	12.303	3.202	12.707	2.785	11.170	3.542	7.499	4.594

Notes:
(1) Household income is inflation-adjusted and reported per 1000s of dollars
(2) Baseline interview varies by study entry cohort: 1995 for AHEAD, 1996 for HRS, and 1998 for WB and CODA

Table 3. 2 Sample Descriptive Statistics by Time Interval

	1995/1996-1998		**1998-2000**		**2000-2002**		**2002-2004**		**2004-2006**	
	N= 7183		**N= 8427**		**N= 7514**		**N= 6955**		**N= 6401**	
	Mean	(s.d)	Mean	(s.d)	Mean	(s.d)	Mean	(s.d)	Mean	(s.d)
Proxy, time (t-1)	0.063	0.244	0.063	0.244	0.065	0.247	0.068	0.251	0.052	0.222
Married, time (t-1)	0.755	0.430	0.743	0.437	0.710	0.454	0.697	0.460	0.687	0.464
Household income, time (t-1)	66.000	94.460	71.151	149.756	70.923	115.601	68.771	100.486	78.369	76.539
Functional status, time (t-1)	0.305	1.144	0.297	1.144	0.302	1.147	0.398	1.200	0.443	1.289
Self-rated health, time (t-1)	2.422	1.090	2.577	1.105	2.496	1.074	2.560	1.066	2.621	1.067
Depressive symptoms, time (t-1)	1.489	1.779	1.708	1.973	1.684	1.946	1.669	2.014	1.574	1.958
Comorbidities, time (t-1)	0.758	0.853	0.965	0.944	1.078	0.971	1.206	1.010	1.334	1.042
Δ Proxy	0.017	0.207	0.013	0.196	0.014	0.204	-0.006	0.192	-0.009	0.195
Δ Marital status	-0.019	0.135	-0.045	0.255	-0.029	0.212	-0.021	0.203	-0.026	0.203
Δ Household income	0.753	114.775	-2.574	151.103	-4.292	95.823	8.595	117.412	-3.876	97.966

Δ Functional status	0.127	1.146	0.105	1.105	0.240	1.190	0.158	1.084	0.108	1.149
Δ Self-rated health	0.271	0.938	-0.012	0.919	0.127	0.900	0.120	0.881	0.093	0.879
Δ Depressive symptoms	0.357	1.952	0.060	1.985	0.049	1.945	-0.032	1.904	0.111	1.856
Δ Comorbidities	0.132	0.361	0.156	0.396	0.176	0.427	0.168	0.417	0.166	0.423

Note: Household income is 2006 inflation-adjusted and reported per 1000s of dollars

Table 3. 3 Discrete-Time Hazard Model Results for Hypertension Diagnosis

	Model 3.0 ($M_{3.0}$)		**Model 3.1 ($M_{3.1}$)**		**Model 3.2 ($M_{3.2}$)**		**Model 3.3 ($M_{3.3}$)**	
Covariates	**OR**	**p-value**	**OR**	**p-value**	**OR**	**p-value**	**OR**	**p-value**
Time period D_1	0.098	***	0.092	***	0.081	***	0.014	***
Time period D_2	0.123	***	0.117	***	0.101	***	0.019	***
Time period D_3	0.128	***	0.121	***	0.108	***	0.019	***
Time period D_4	0.138	***	0.132	***	0.121	***	0.021	***

Time period D_5	0.155***	0.147***	0.136***	0.024***
Non-Hispanic black		1.483***	1.392***	1.278***
Mexican		1.303***	1.085	0.966
Proxy, time (t-1)			1.050	1.249
Δ Proxy			1.437***	1.656***
Age in 1995			1.007**	1.007**
Education			0.971***	0.987
Female			1.181***	1.209***
Married, time (t-1)			0.999	0.963
Δ Married			1.050	1.113
Income, time (t-1)			0.999*	1.000
Δ Income			0.999*	0.999
BMI, time (t-1)				1.038***
Δ BMI				1.030**

SRH, time (t-1)			1.153***
Δ SRH			1.276***
CES-D, time (t-1)			1.011
Δ CES-D			1.013
Functional, time (t-1)			0.966
Δ Functional			0.962
Comorbidities, time (t-1)			1.043
Δ Comorbidities			1.458***
Smoking, time (t-1)			0.846*
Δ Smoking			0.824
Goodness-of-fit LL	-10560.04	-9739.32	-6887.80
Wald Chi-squared	-10560.04	-9739.32	-6887.80
Probability>chi-squared	0.000	0.000	0.000

*p<.05, **p<.01, ***p<.001

Table 3. 4 Discrete-Time Hazard Model Results for All-Cause Mortality

	Model 4.0 ($M_{4.0}$)		**Model 4.1 ($M_{4.1}$)**		**Model 4.2 ($M_{4.2}$)**		**Model 4.3 ($M_{4.3}$)**	
Covariates	**OR**	**p-value**	**OR**	**p-value**	**OR**	**p-value**	**OR**	**p-value**
Time period D_1	0.081	***	0.081	***	0.000	***	0.006	***
Time period D_2	0.085	***	0.086	***	0.000	***	0.005	***
Time period D_3	0.068	***	0.068	***	0.000	***	0.004	***
Time period D_4	0.059	***	0.059	***	0.000	***	0.003	***
Time period D_5	0.064	***	0.064	***	0.000	***	0.006	***
Non-Hispanic black			1.069		0.890		0.881	
Mexican			0.834		0.713	**	0.608	**
Proxy, time (t-1)					2.682	***	1.499	**
Δ Proxy					2.975	***	1.583	**
Age in 1995					1.094	***	1.028	***
Education					0.979	**	1.008	

Female	0.616***	0.521***
Married, time (t-1)	0.717***	0.731***
Δ Married	0.824	0.836
Income, time (t-1)	0.996***	0.998*
Δ Income	0.998***	0.999
BMI, time (t-1)		0.967***
Δ BMI		0.908***
SRH, time (t-1)		1.567***
Δ SRH		1.497***
CES-D, time (t-1)		0.995
Δ CES-D		1.001
Functional, time (t-1)		1.194***
Δ Functional		1.164***
Comorbidities, time (t-1)		1.199***

Δ Comorbidities				1.342***
Smoking, time (t-1)				1.624***
Δ Smoking				0.987
Goodness-of-fit LL	-9059.638	-9057.431	-7193.613	-3487.550
Wald Chi-squared	15770.350	15761.780	11178.160	7585.980
Probability>chi-squared	0.000	0.000	0.000	0.000

*p<.05, **p<.01, ***p<.001

Figure 3. 1 Probability of Developing Hypertension by Ethnic Group ($M_{3.1}$)

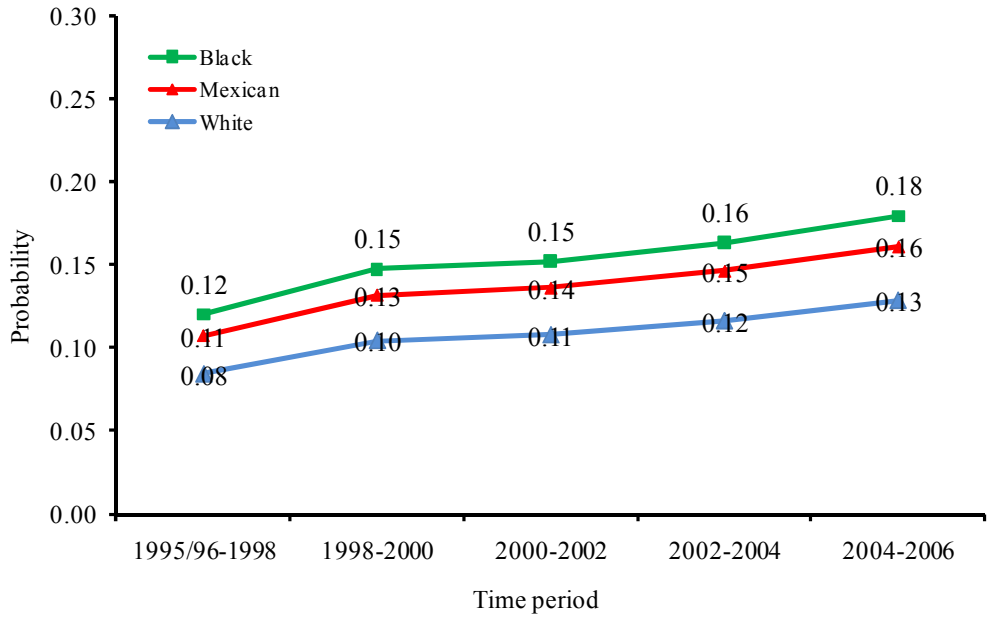
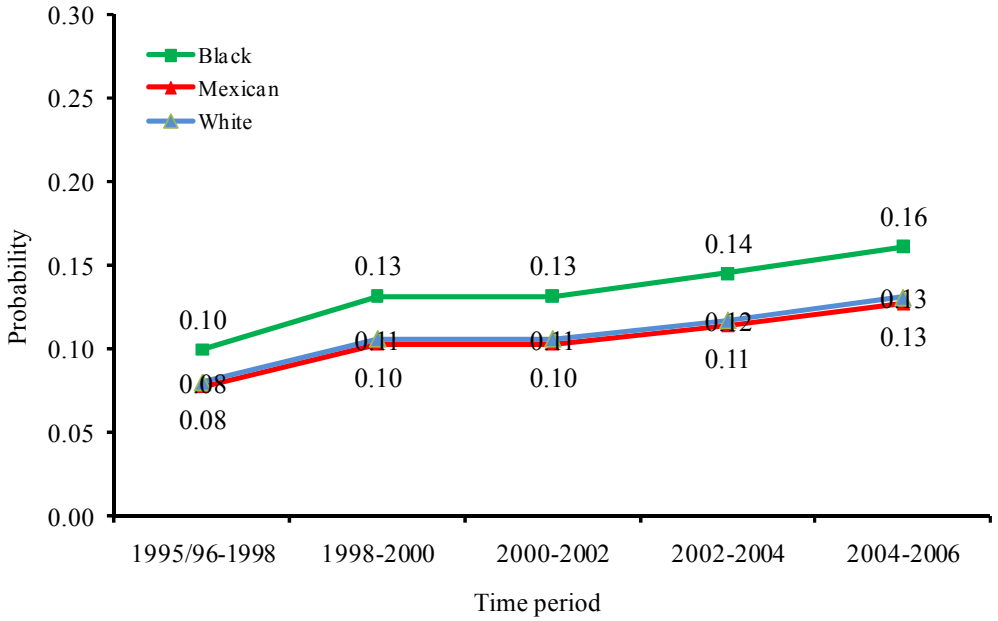
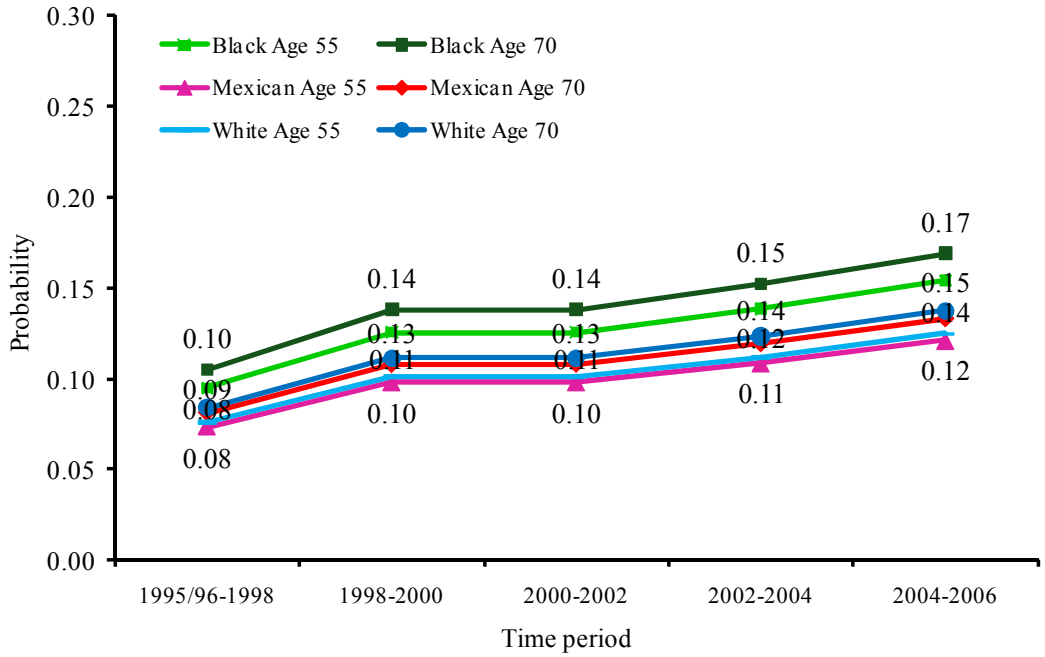


Figure 3. 2 Probability of Developing Hypertension by Ethnic Group ($M_{3.3}$) †



†Conditional probabilities evaluated at the means of other $M_{3.3}$ model covariates

Figure 3. 3 Probability of Developing Hypertension by Ethnic and Age Group ($M_{3.3}$)†



†Conditional probabilities evaluated at the means of all $M_{3.3}$ model covariates

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CHAPTER 4:

Disparities in Diabetes Mellitus Risk for Middle and Old Aged Black, White and Mexican Americans

4.1 Introduction

The public health burden and high social cost of diabetes is extensively documented and increasingly recognized as a domestic and international epidemic (Boyle, Honeycutt & Narayan, et al., 2001; Black, 2002; Brancati, Kao & Folsom et al., 2000; Ujcic-Voortman, Schram & Jacobs-van der Bruggen, et al., 2009). In the U.S., diabetes alone accounts for over 100 billion dollars in treatment costs, over 3 million hospital stays and 300,000 deaths annually (Brancati et al., 2000). By 2025, an estimated 22 million Americans will be diagnosed with type II, or adult-onset diabetes (Black, 2002). Specifically, surveillance estimates from the National Health and Nutrition Examination Survey (NHANES) find 7.7% of adults aged 20 and older self-report a diagnosis of diabetes in the period of 2003-2006 (Centers for Disease Control and Prevention, 2008). In addition, diabetics are at increased risk of diabetic complications and comorbid conditions, including disability, depression, cognitive impairment and low quality of life (Black, 2002).

Ethnic differences in diabetes prevalence and incidence in the U.S. is also well documented (Wray, Alwin & McCammon, et al., 2006; McBean, Li & Gilbertson, et al, 2004). Although much of the research documenting disparities in diabetes rates remains

cross-sectional, there is an increasing amount of work focusing on longitudinal diabetes trends. For example, Wray et al. (2006) examine diabetes in a multi-ethnic, comparative framework to find that older adults, men, blacks and Latinos have higher odds of prevalent diabetes. In addition, they find the odds of incident diabetes increase for blacks and Latinos net of social factors such as educational attainment, economic resources and parental social status. In examining the population of Medicare beneficiaries, McBean and colleagues (2004) also find higher prevalence and incidence of diabetes for black, Asian and Hispanic adults relative to white adults. These studies offer insight into the presence and persistence of disparities in diabetes morbidity in the U.S. However, these studies do not explicitly compare differences in the risk of developing diabetes for middle-aged and older black, Mexican and white adults in the U.S.

In addition, several studies focus on modifiable health behaviors as important risk factors associated with diabetes. Family history, adiposity and sedentary lifestyles are all established factors related to diabetes for older individuals in the U.S. (Suchindran, Vana & Shaffer, et al., 2009; Brancati, et al., 2000). However, less is known about how individual changes in health profiles may bear on diabetes incidence for older aged adults. Although there is acknowledgement that diabetes often co-occurs with other chronic diseases and physical limitations among the elderly (Black, 2002), there is little effort to account for changing health status in studies of diabetes incidence. Few studies consider changing levels of reported functional limitations, health ratings, depressive symptoms, and chronic disease comorbidities when examining incident diabetes.

Existing research provides valuable insight into determining diabetes risk for Americans from various ethnic groups. However, there is less research on how diabetes

risk might vary for older minorities with age-related changes in health profiles. This work aims to add to current understanding in health disparities and aging by examining incident diabetes over a period of 11 years for aging white, black and Mexican Americans in the U.S. using discrete-time survival analysis.

4.2 Hypotheses

Hypothesis 1 (H_1): The risk of diabetes increases with time (H_{1a}). That is, throughout the 11-year observation window, the risk of diabetes will increase in subsequent discrete time periods.

In stark contrast to Type I and gestational diabetes, Type II diabetes is diagnosed in middle and old age. The convergence of various factors later in life influences the development of chronic disease and disability for middle aged and older adults in the U.S. Low socioeconomic status, family history and obesity are all identified as critical risk factors for the onset of diabetes in middle age and late life (Black, 2002; Suchindran, et al., 2009). Several studies document increasing prevalence and incidence of adult-onset diabetes in the U.S. In particular, McBean and colleagues (2004) estimated a 37% increase in diabetes incidence between 1994 and 2001. In addition, after changing demographic factors and prevalence rates are taken into consideration, the projected burden of diabetes in the United States is expected to increase by 165% by the year 2050 (Boyle, et al., 2001). Given these documented increases and projections, we expect the risk of diabetes for our study population to also display an increasing secular time trend.

Hypothesis 2 (H_2): The odds of adult-onset diabetes for our sample of older black adults will be higher relative to white older

Americans (H_{2a}). Despite accounting for changing health profiles for black Americans, incident diabetes will remain significantly higher relative to whites (H_{2b}).

Several mechanisms have been proposed to account for observed disparities in health status between minorities and whites in the U.S. (Lynch & Smith, 2005; Freedman, Martin & Schoeni, 2002). One premise centers on the concept of double jeopardy, where poor health is not only ascribed to declines associated with the aging process but also to low socioeconomic status. However, there has been little empirical support for this hypothesis (Ferraro & Farmer, 1996). Other explanations include lifestyle choices and discrimination (Williams & Collins, 1995), as well as limited access to high-quality medical care and preventive health behaviors due to low socioeconomic status. This lack of access disproportionately exposes minorities to risk factors for the development of diabetes such as low medication adherence, poor diet and inactivity (Winston, Barr & Carrasquillo, et al., 2009).

The demographic literature suggests that blacks have a higher prevalence of disability and disease and live in poor health longer than their white counterparts (Freedman, et al., 2002). This research describes blacks exhibiting illness earlier and dying at younger ages than whites due to the accumulation of health disadvantages over the lifecourse. High levels of socioeconomic inequality account for much of the observed disparities in health at younger ages and early adulthood, as these differences narrow in old age (Beckett, 2000).

In addition, social structure influences health status through a host of factors, most notably, through ethnicity, gender, and socioeconomic status (House, Lepkowski & Kinney, et al., 1994; Ross & Wu, 1996; Hagestad & Dannefer, 2001; Williams, 1997; Williams, 2005; Lynch & Smith, 2005). Race and ethnic differences in education, income levels, and segregated housing are conceptualized as "fundamental causes" of disease (Link & Phelan, 1995). According to this framework, health insults accumulate over the lifespan and account for large disparities in later-life health. These may operate through differences in employment and occupational opportunities, income and wealth streams, life styles, and health behaviors (Dannefer, 2003; Hayward, Miles, Crimmins, et al., 2000; Bulatao & Anderson, 2004; Hertzman, 2004). Differences in risk profiles for disease reflect differences in the combination of these factors over time (Hayward, et al., 2000).

Social processes of cumulative disadvantage over the lifecourse work to systematically and negatively affect health throughout the lifespan. Consistent with the concept of cumulative disadvantage, older blacks are expected to demonstrate increased risk of diabetes incidence relative to older whites. In addition, epidemiological evidence for diabetes disease rates suggest that rates for black adults are higher than white Americans (Brancati et al., 2000; McBean, et al., 2004; Wray et al., 2006).

Hypothesis 3 (H_3): The odds of developing adult-onset diabetes will be higher for our sample of older Mexican Americans relative to white older Americans (H_{3a}). Even after accounting for changing health profiles, Mexican Americans will continue

to have higher incident diabetes in comparison to whites (H_{3b}).

In line with hypothesized processes of minority health disadvantages, Hispanic Americans have also demonstrated persistent health disparities for various health outcomes. However, the state of health research on Hispanic populations is somewhat mixed. Recent work examines the health and mortality outcomes for Hispanic populations, and specifically addresses the Hispanic Health Paradox (Markides & Eschbach, 2005; Crimmins, Kim, Alley, et al., 2007). This epidemiological paradox refers to the finding that in some instances, Hispanics demonstrate outcomes comparable to white Americans despite being socioeconomically similar to black Americans. There are several proposed explanations for this occurrence. Among them are poor reporting of mortality statistics, protective effects of cultural behaviors on health, healthy migrant effects and self-selection with respect to out-migration so that individuals are no longer captured by U.S. morbidity or mortality statistics (Markides & Eschbach, 2005).

However, the Hispanic Health Paradox has not been demonstrated for all health outcomes and for all Hispanic sub-populations (Palloni & Morenoff, 2001). While studies utilizing several nationally-representative data sources find evidence for a Hispanic mortality advantage (Markides & Coreil, 1986; Franzini, Ribble & Keddie, 2001), other studies find no such advantage (Carrasquillo, Lantigua & Shea, 2000). One key finding is the confounding of many heterogeneous Hispanic sub-populations (Palloni & Morenoff, 2001). The balance of the evidence suggests that the Hispanic Paradox is most strongly supported by a mortality advantage for males, older individuals and Mexican-born persons (Crimmins, et al., 2007). It is likely that the Hispanic Paradox is

more aptly described as a Mexican-born Paradox, and only for a specific list of health outcomes, most notably mortality.

Despite the potential health advantages accruing to Mexican-origin individuals, several epidemiological studies have found Mexican Americans at much greater risk of incident type-II diabetes, complications related to diabetes, and other comorbidities (Wu, Haan & Liang, et al., 2003). In addition, Maskarinec and colleagues (2009) find that accounting for body mass index did not diminish the observed disparity of prevalent diabetes between white and minority ethnic groups observed. Mexican Americans in our study sample are expected to also demonstrate higher risk of developing type-II diabetes, and this risk will not be expected to diminish completely even after adjusting for changes in physical and mental health status or BMI.

4.3 Methods

4.3.1 Data

This study uses data from the Health and Retirement Study (HRS), collected at the University of Michigan's Institute for Social Research. The HRS respondents are a nationally-representative sample of community-based adults aged 51 and over and identified through screening of an area probability sample of households. The study involves individuals from several birth cohorts, including: the Asset and Health Dynamics of the Oldest Old (AHEAD; born prior 1924), the Children of the Depression Age (CODA; born 1924-1930), the Health and Retirement Study cohort (HRS, born 1931-1941) and War Babies (WB, born 1942-1947). In 1998, the Health and Retirement Study added the CODA and WB cohorts to the study and consolidated data collection

efforts for all birth cohorts. Prior to 1998, data collection on the original HRS cohort occurred on even years and in odd years for AHEAD data collection.

Due to wave incompatibility of key independent variables, these analyses use seven waves of data from the HRS (1995-2006). Data management and analysis is conducted with Stata 10.0 (Stata Corp., College Station, Texas).

4.3.2 Measures

4.3.2.1 Diabetes

An indicator variable for self-reported diabetes mellitus is used to measure disease status in the analyses. Self-reported health and disease status have been well established and validated in earlier studies (Johnson & Wolinsky, 1993; Ferraro & Wilmoth, 2000; Mensah et al., 2005), and is widely used in aging research. In addition, nationally representative data collection instruments provide consistent estimates of incidence when compared to clinical studies (Glymour & Avendano, 2009).

The Health and Retirement Study asks respondents if they have been diagnosed with diabetes each interview year. In subsequent interviews, individuals were given the option to dispute their preloaded responses from the previous interview. In order to deal with responses that offer conflicting information, we examine additional information reported by respondents. Consultations with geriatric physicians provided the clinical criteria for satisfying the burden of proof for each of the seven reported diseases. For each disease, a dispute was corroborated by examining the evidence variables from the previous interview. For example, if an individual gives conflicting reports of diabetes diagnosis, we utilize information on use of oral or injectible medication to verify the diagnosis.

4.3.2.2 Ethnicity

The principal covariates of interest in the analyses are indicators for self-reported black and Mexican ethnicity. Ethnicity is constructed as mutually-exclusive indicator variables for non-Hispanic white, non-Hispanic black and Mexican-ethnicity individuals. Consequently, dummy variables for black and Mexican are included in the analytic models and are each interpreted relative to white study participants. Only Hispanic respondents self-reporting as having Mexican ancestry are included in these analyses. Ideally, we would analyze other Hispanic subgroups, however the HRS is not able to track each specific Hispanic subgroup for the time period examined. Other race and other Hispanic types are excluded from these analyses.

4.3.2.3 Health status

Several covariates are used to mark the physical and mental health status of respondents in accounting for onset of diabetes. Health status is operationalized by covariates for self-rated health, functional limitations and depressive symptoms. These are included in the analyses as time-varying lagged values as well as change scores. Both, previous period health information and net health change are conceptualized as contributing unique information in models of incident diabetes.

Lagged covariates are constructed as health status at time (t-1) and predict diabetes risk at time t. Lagged self-rated ill health (SRH) is measured with a 5-item scale (1=excellent, 2=very good, 3=good, 4=fair and 5=poor). Lagged functional status (0-11) incorporates both, activities of daily living (ADL, 0-6) and instrumental activities of daily living (IADL, 0-5), with higher scores reflecting increasing number of difficulties with any of the ADL or IADL activities. Lagged depressive symptoms are measured with the

Center for Epidemiological Studies Depression Scale (CES-D, 0-9) with a higher score reflecting higher depressive symptoms.

Change scores are constructed as the difference in health status between time t and time (t-1). Change in self-rated ill health (SRH) reflects current wave SRH minus previous wave SRH. Analogously, changes in functional status and depressive symptoms also represent current minus previous wave values. Positive change scores reflect an increase in the burden of health grievances, while negative scores denote improvement via divestment of health problems. A change score of zero denotes no change in health conditions across adjacent waves.

Time-varying health covariates are intended to provide some control of population heterogeneity in health status. We conceptualize health status as multidimensional, where physical and mental health limitations work in concert to influence chronic disease emergence. Specifically, health status in previous time periods has a bearing on current period chronic disease development. That is, global self assessment of health, functional limitations and depressive symptoms are conceptualized as confounding variables in an individual's future experience with chronic disease.

4.3.2.4 Health behaviors

In addition to physical and mental health measures, we also incorporate several behavioral risk factors for diabetes into our model. We include body mass index (BMI) as lagged and change score covariates. BMI is calculated using respondents' self-reported weight for each interview year and height reported at baseline. We also include previous wave smoking status as well as a change score covariate.

4.3.2.5 Social stratification and social support

Various controls for demographic and socioeconomic factors are included as independent, time-constant and time-varying covariates in the analysis. Age is measured as age in 1995 for all individuals in the study, regardless of entry cohort. Education is measured as a continuous variable denoting years of schooling (range 0-17). Lagged and change in income is inflation-adjusted to 2006 levels, and was also re-scaled (reported per 1000s of dollars) to facilitate its estimation. Marital status is conceptualized as an indicator of social support for individuals, and is constructed as a lagged time-varying covariate. The change in marital status (range -1 to 1) reflects dissolution/widowhood, no change, and acquisition of partners at each point in time over the study period.

4.3.3 Data analysis

Discrete-time survival analysis is used to model the onset of diabetes. The discrete-time hazard represents the risk of event occurrence in each discrete time period among people in the risk set. An important feature of the analysis is that the probability is conditional—the risk of diabetes is estimated for those respondents who have not been previously diagnosed (Singer and Willett, 1993). Consequently, persons already diagnosed with diabetes were excluded from the analyses. 13% (n=2195) of the baseline HRS sample was previously diagnosed with diabetes and excluded from our analyses. Of these, black diabetics comprise 20% (n=543) and Mexican diabetics 6% (n=168) of the excluded cases.

We use Stata macros developed by Dinno (2002) to estimate the model using the logit link and adjust for clustering within households. In estimating discrete time hazard

models, the logistic transformation yields the conditional log-odds of the risk of diabetes onset:

$$\log \frac{h(t_{ij})}{1-h(t_{ij})} = [\alpha_1 D_{1ij} + \alpha_2 D_{2ij} + \ldots + \alpha_J D_{Jij}] + [\beta_1 X_{1ij} + \beta_2 X_{2ij} + \ldots + \beta_P X_{Pij}] \quad (1)$$

Equation (1) describes the conditional probability that individual i will experience a diabetes diagnosis in time period j, given that individual i is free of diabetes in time period j-1. D_{1ij}- D_{Jij} represent indicators for each time period. The associated parameters $\alpha_1 - \alpha_J$ represent multiple intercepts for each time period. Taken together, these form the log-odds of the baseline hazard of diabetes occurrence. The slope term parameters β_1- β_P assess the unit-difference effects of predictors X_{1ij}-X_{Pij} on the risk of developing diabetes.

A consideration in using longitudinal data is the possibility of missing data at follow-up due to item non-response, survey non-response, and mortality (Little & Rubin, 1987). Selection bias may occur if any of these situations results in a nonrandom subset of the study population, affecting both internal and external validity (Berk, 1983). We employ multiple imputation (Schafer & Graham, 2002) of incomplete multivariate data under a normal model software (NORM) to deal with missing data inherent in longitudinal data collection efforts. This represents a significant improvement to the use of older procedures, such as case deletion and single imputation, or assuming data are missing at random (MAR). Specifically, three complete data sets were imputed, and analyses are replicated on each of these data sets, following the standard algorithms to compute point estimates and standard errors. Estimates are then averaged across multiple imputations to generate a single point-estimate. In recent analyses of longitudinal health data, multiple imputation was employed (Liang, Quiñones & Bennett, et al., 2010).

4.3.4 Mortality

In order to account for competing risks to disease incidence, separate discrete-time survival models for mortality are also examined. A full model of the hazard of mortality is estimated for the same population of respondents free of diabetes at the outset, and includes controls for demographic, socioeconomic and health status changes over time. The risk of dying is examined to determine whether selective mortality is occurring for black and Mexican Americans relative to white Americans in our sample.

4.4 Findings

Table 4.1 details sample descriptive statistics collected at the baseline interview. Similarly, Table 4.2 presents descriptive statistics for the sample at each time interval. The mean age for our total sample is 64 years, and 57% are female. The ethnic composition of our sample shows 13% of respondents are black and 4% are of Mexican origin.

Table 4.3 presents the discrete-time hazard model results for the risk of developing diabetes. We first present the unconditional model ($M_{3.0}$) to ascertain the shape of the baseline diabetes hazard. This is followed by progressively more complex models. Table 4.3 reports the model results as fitted odds ratios. Model coefficients are transformed into predicted probabilities of the hazard using the formula, $\frac{1}{e^{-(\alpha jDj+\beta pXp)}}$ (Dinno, 2002). Figures 4.1 and 4.2 present plots detailing the predicted probability of developing diabetes by race/ethnic group for each time period for the ethnic-only model ($M_{3.1}$) and the time-varying health model ($M_{3.3}$), respectively.

Hypothesis H_1 proposes that the incidence of diabetes increases with time. The baseline hazard for diabetes in $M_{3.0}$ (unadjusted by any predictors) demonstrates a steady increase in the risk of incident diabetes over time. The probability of being diagnosed with diabetes in the first period is 0.028 and rises to 0.037 by the final time period. Consequently, H_1 is supported by the model results.

Hypothesis H_{2a} states that the odds of developing diabetes are higher for black relative to white Americans. In examining models $M_{3.1}$ through $M_{3.3}$ in Table 4.3, we are able to determine race/ethnic differences in incident diabetes. $M_{3.1}$ details significantly higher odds of diabetes for black individuals (OR=1.619, $p<.001$). These findings persist once we account for differences in demographic and socioeconomic covariates in $M_{3.2}$ (OR=1.367, $p<.001$). These findings provide support for H_{2a}. As an extension, hypothesis H_{2b} states that even after accounting for time-varying health status black Americans will still have greater odds of developing diabetes relative to whites. However, once we adjust for changing health status, black respondents no longer demonstrate significantly higher odds relative to whites (in $M_{3.3}$, OR=1.095, $p>.05$). Black Americans do not have significantly different odds of incident diabetes relative to whites in the model accounting for demographic and health differences. Given these results, we find no support for hypothesis H_{2b}.

Hypothesis H_{3a} states that the odds of developing diabetes for Mexican Americans in the HRS are elevated relative to white Americans. Overall, the increased odds of newly diagnosed diabetes are pronounced for Mexican Americans compared to whites. $M_{3.1}$ details significantly higher odds of diabetes for Mexican-ethnicity individuals (in $M_{3.1}$, OR=2.104, $p<.001$). These findings persist once we account for differences in

demographic and socioeconomic covariates, although they are substantially attenuated (in $M_{3.2}$, OR=1.494, p<.001). Both of these model findings provide support for H_{3a}. In hypothesis H_{3b}, we propose that including time-varying health covariates will not completely account for the gap in incidence between Mexican and white Americans. Even after we adjust for changing health status, Mexican origin individuals still have significantly higher odds relative to whites (in $M_{3.3}$, OR=1.795, p<.001). These model results provide support for H_{3b}. What is most interesting is that the higher odds accruing to Mexican Americans are not attenuated after including time-varying health covariates; in fact the odds increase between $M_{3.2}$ and $M_{3.3}$. Accounting for changing health status and BMI magnifies the increase in odds of receiving a diabetes diagnosis for Mexican Americans.

Figure 4.1 presents $M_{3.1}$ results graphically. Without adjusting for any other covariates, the conditional probability of developing diabetes differs for Mexican, black and white Americans for all time periods. However, figure 4.2, which shows $M_{3.3}$ results for all three race/ethnic groups evaluated at the means of other covariates, demonstrates only a significant difference between Mexican and white Americans. After accounting for changing health status, we no longer see a significant difference in the development of diabetes between black and white Americans; however, the difference between Mexican and white Americans persists.

Figure 4.2 also offers interesting dynamics over time. The figure shows that in the time-varying health model, the conditional probability of developing diabetes for black, Mexican and white Americans increases for the first few time periods then plateaus for this population of middle and older-aged adults. Overall, the risk of newly

diagnosed diabetes increased between 1995 and 2006 for all Americans over age 50. The probability of incident diabetes among Mexican Americans was 0.02 during the period of 1995-1998, which increased to 0.05 during 2004-2006. In contrast, among white Americans the risk was 0.01 during 1995-1998 and 0.03 during 2004-2006. Similarly, the probability for black Americans increased from 0.01 during 1995-1998 to 0.03 during 2004-2006.

We examine the competing risk of mortality for the same sample of HRS respondents that report being diabetes free at the baseline through separate discrete-time survival models. The results of the progressively more complex discrete-time survival models are presented in Table 4.4. In estimating these discrete-time hazard models of mortality, we include diabetes diagnosis among the comorbidities measure in $M_{4.3}$. Black Americans do not demonstrate significantly different odds of dying compared to whites in any of the models examined (in $M_{4.3}$, OR=0.893, $p>.05$). Relative to white Americans, Mexican-Americans have significantly lower odds of dying in the ethnic-only model (in $M_{4.1}$, OR=0.813, $p<.01$) as well as in the socioeconomic and demographic model (in $M_{4.2}$, OR=0.713, $p<.001$). However, once time-varying health measures are included, Mexican ethnicity individuals no longer demonstrate significantly different risk of dying from whites (in $M_{4.3}$, OR=0.761, $p>.05$).

4.5 Discussion

This study moves beyond the cross-sectional design of diabetes prevalence as well as longitudinal studies that only consider transitions between two points in time, and examines diabetes incidence for middle-aged and older individuals in the U.S. In addition, it involves the comparison of diabetes incidence for both black and Mexican to

white Americans while considering changes in health status over time. This research also examines differential rates of mortality by race/ethnic group. In examining competing mortality risks, both Mexican and black participants in the HRS had similar mortality risk with respect to whites during this same time period.

This research is able to determine how interval-by-interval changes in diabetes risk differ for black, Mexican, and white Americans over and 11-year period. The cumulative incidence of developing diabetes over the entire period for the aggregate sample is 11%, with cumulative incidence at 11% for white Americans, 12% for black Americans, and 19% for Mexican Americans. The baseline risk of incident diabetes increases modestly over time for all individuals in the HRS. One potential explanation for a secular increase in risk of receiving a diagnosis of diabetes could stem from the reduction of diagnostic criteria set by the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. In 1997, the fasting blood glucose levels considered sufficient for a diabetes diagnosis were reduced to >125 mg/dL from >140 mg/dL (McBean, et al., 2004). Although this new criterion would not influence the relative ethnic disparities in risk between Mexican and white participants, it could affect the overall shape of the baseline hazard of diabetes. This would be largely governed by the length of time physicians take to adopt the new standard. It is possible that the jump observed between the 1995/96-1998 to the 1998-2000 time intervals could be at least partially explained by the change in diagnostic criteria.

This study was able to examine diabetes risk for one Hispanic subgroup in the U.S. Consistent with the literature, pronounced elevated risks accrue to Mexican relative to white Americans. The results indicate that diabetes incidence for Mexican ethnicity

individuals have nearly double the risk accruing to their white counterparts. These variations persisted even after differences in time-varying health characteristics were adjusted. For black Americans the risk of developing diabetes was markedly different. Although blacks demonstrate higher risk of incident diabetes compared to whites, once we account for changing health attributes, the difference attenuated completely. These results extend beyond the literature by tracing ethnic-specific trends in diabetes incidence after accounting for age-related changes in health profiles.

There are important limitations that need to be acknowledged. First, it is difficult to ascertain a clear picture of minority health with the HRS sample since the study tracks individuals from middle age into old age and is not able to capture earlier-life selective mortality and diabetes onset. Health disadvantages and differential mortality that may have occurred before middle age are not traced here, and may result in underestimating the risk of incident diabetes for Mexican and black Americans. This is an important consideration when interpreting the findings. The analyzed sample of black, Mexican and white Americans that survive to middle and old age without diagnosed diabetes mellitus is very different than a representative U.S. population due to differential morbidity and mortality that occurs earlier in the lifecourse. In addition, further efforts to disentangle age-period-cohort effects in our analyses and should be explored in future analyses (Jacobs, Hannan & Wallace et al., 1999). Still, these results indicate that diabetes risk for Mexican Americans in the U.S. is substantially higher than that for white Americans.

Second, an important concern to using self-reported health measures, and specifically, self-reported disease indicators is the reliability of responses in subsequent

respondent re-interviews (Beckett et al., 2000). Specific procedures to explore the extent of inconsistencies across an individual's longitudinal record and provide more time-consistent disease indicators is reflected in these analyses. In addition, measures of diagnosed disease from self-report and clinical records depend greatly on the health-seeking behavior of individuals. Differences in behavior could drive differences between ethnic groups. Individuals may also be unaware or underreport diagnoses of diabetes due to age biases and variations in clinical practitioner quality. NHANES reports crude rates of undiagnosed diabetes based on biologically collected information. According to the 2005 NHANES surveillance data, 6.2 of 20.8 million total diabetics have undiagnosed diabetes (Centers for Disease Control and Prevention, 2005). This amounts to approximately one-third of Americans of all ages as having undiagnosed diabetes. These rates of underdiagnosis introduce some concern for bias, particularly if underdiagnosis varies by race/ethnic group.

Third, there could be informative censoring other than death that might bias results. Specifically, if minorities in the HRS are more likely to drop out and not return to the study, the differences between ethnic groups could be understated. Although we are not able to account for absorbing attrition competing risks, we do consider death as a competing risk and find no significant differences in the risk of dying for either black or Mexican Americans compared to white Americans. Consequently, our findings reflect the risk of developing diabetes among those HRS respondents that are alive. There is no evidence that differential mortality is occurring during the period of observation.

The analyses on competing mortality risks offer some additional insights. We examine the sample of HRS participants that are free of diabetes diagnosis at the

baseline. Among these respondents, we see no race/ethnic differences in subsequent mortality once we account for time-varying health changes in the model. However in additional analyses of mortality that include HRS respondents with diabetes mellitus at the baseline, we are able to discern morality advantages accruing to Mexican Americans compared to whites (analyses not shown). It is interesting to note that Mexicans without diabetes at the baseline of our study are no longer characterized by these mortality advantages. Moreover, Mexicans free of diagnosed diabetes at age 51 and over are much more likely to be diagnosed with the disease relative to whites.

The results suggest that increasing diabetes incidence for Mexican Americans into old age remain demonstrable after adjustment for differences in health behaviors, socioeconomic status, demographic characteristics and changing health status. Relative to white Americans, Mexican Americans have a significantly elevated risk of diabetes throughout the 11-year period of observation. In contrast, increases in incident risk for black Americans relative to white Americans operate largely through changes in time-varying health status.

Our research focuses on incidence of diabetes mellitus for older adults in the U.S. A second complementary piece to this research would be to examine changing prevalence of diagnosed diabetes mellitus for older white and minority Americans. Although we do not explicitly model changes in prevalence, we are able to determine that the cumulative incidence of hypertension for the observation period of 1995-2006. We estimate this to be 11% for the entire sample. In addition, we are able to determine that 13% of the baseline sample was previously diagnosed with diabetes mellitus, which represents prevalence in the HRS study population for the period up to 1995. Still, future studies are

needed to examine changing prevalence of diabetes mellitus for older Americans given its binary absorbing state specification.

Our results corroborate the need for early detection and prevention efforts into old age, particularly for Mexican ethnicity individuals in the U.S. Monitoring and controlling diabetes through medication adherence and lifestyle change is a resource-intensive endeavor for both, individuals diagnosed with the disease as well as the health system charged to provide care for them (Black, 2002). Early detection of the disease, particularly for minorities most at risk of developing diabetes is imperative.

These results confirm that older Americans with new diagnoses of diabetes also contend with multiple chronic conditions that require coordinated efforts for managing comorbidities (Paez, Zhao & Hwang, 2009). Potential complications arising from a new diabetes diagnosis has vast implications on the management of multiple diseases for elderly Americans. This is of particular concern given complicated conditions associated with diabetes progression, including cardiovascular disease, peripheral vascular disease, cerebrovascular disease, hypertension, and lower extremity amputations (Black, 2002). Understanding diabetes acuity and co-occurring disease case mix, as well as the extent to which left truncation influences ethnic differences in diabetes incidence and selective mortality are important areas for further study.

Table 4. 1 Sample Descriptive Statistics at the Baseline by Ethnic Group

	Total		**White**		**Black**		**Mexican**	
	N= 14,783		**N= 12,256**		**N= 1918**		**N= 609**	
	Mean	s.d	Mean	s.d	Mean	s.d	Mean	s.d
Proxy, time (t-1)	0.062	0.241	0.057	0.231	0.072	0.259	0.129	0.335
Married, time (t-1)	0.729	0.445	0.756	0.429	0.543	0.498	0.771	0.420
Household income, time (t-1)	61.248	84.779	66.859	89.895	37.594	50.016	25.589	27.475
Functional status, time (t-1)	0.378	1.266	0.326	1.165	0.639	1.665	0.586	1.549
Self-rated health, time (t-1)	2.592	1.111	2.517	1.094	2.928	1.116	3.012	1.137
Depressive symptoms, time (t-1)	1.640	1.874	1.550	1.805	2.003	2.056	2.257	2.297
Comorbidities, time (t-1)	1.179	1.028	1.141	1.020	1.350	1.057	0.700	0.675
Δ Proxy	0.020	0.210	0.019	0.198	0.032	0.247	0.002	0.290
Δ Marital status	-0.021	0.143	-0.020	0.142	-0.025	0.157	-0.016	0.127
Δ Household income	-0.814	82.565	-0.893	89.054	-1.170	42.765	1.863	25.689

Δ Functional status	0.169	1.285	0.160	1.223	0.212	1.560	0.202	1.494
Δ Self-rated health	0.262	0.935	0.270	0.903	0.217	1.046	0.249	1.142
Δ Depressive symptoms	0.380	2.000	0.362	1.930	0.445	2.213	0.520	2.558
Δ Comorbidities	0.170	0.427	0.151	0.401	0.245	0.510	0.100	0.316
Age in 1995	64.225	10.355	64.486	10.342	63.236	10.475	62.077	9.813
Female	0.573	0.495	0.568	0.495	0.617	0.486	0.527	0.500
Education (in years)	12.115	3.276	12.595	2.813	10.657	3.684	7.034	4.639

Notes:
(1) Household income is inflation-adjusted and reported per 1000s of dollars
(2) Baseline interview varies by study entry cohort: 1995 for AHEAD, 1996 for HRS, and 1998 for WB and CODA

Table 4. 2 Sample Descriptive Statistics by Time Interval

	1995/1996-1998		**1998-2000**		**2000-2002**		**2002-2004**		**2004-2006**	
	N= 11,615		**N= 13,311**		**N= 11,836**		**N= 10,890**		**N= 9926**	
	Mean	s.d	Mean	s.d	Mean	s.d	Mean	s.d	Mean	s.d
Proxy, time (t-1)	0.062	0.241	0.063	0.243	0.066	0.249	0.068	0.251	0.055	0.229
Married, time (t-1)	0.729	0.445	0.719	0.450	0.684	0.465	0.673	0.469	0.664	0.472
Household income, time (t-1)	61.248	84.779	65.392	126.539	65.547	108.885	63.704	92.848	74.167	74.394
Functional status, time (t-1)	0.378	1.266	0.373	1.284	0.381	1.293	0.514	1.384	0.536	1.401
Self-rated health, time (t-1)	2.592	1.111	2.732	1.116	2.645	1.083	2.694	1.068	2.744	1.067
Depressive symptoms, time (t-1)	1.640	1.874	1.859	2.044	1.836	2.030	1.805	2.074	1.729	2.051
Comorbidities, time (t-1)	1.179	1.028	1.452	1.126	1.607	1.150	1.773	1.168	1.924	1.177
Δ Proxy	0.020	0.210	0.015	0.198	0.011	0.201	-0.001	0.201	-0.011	0.201
Δ Marital status	-0.021	0.143	-0.049	0.264	-0.027	0.206	-0.021	0.203	-0.025	0.200
Δ Household income	-0.814	82.565	-1.911	126.961	-3.804	92.377	9.191	109.600	-3.309	94.481

Δ Functional status	0.169	1.285	0.108	1.198	0.286	1.287	0.171	1.203	0.124	1.238
Δ Self-rated health	0.262	0.935	-0.021	0.918	0.109	0.901	0.113	0.882	0.086	0.881
Δ Depressive symptoms	0.380	2.000	0.057	1.996	0.045	1.976	0.002	1.954	0.107	1.913
Δ Comorbidities	0.170	0.427	0.200	0.452	0.221	0.473	0.200	0.456	0.189	0.441

Note: Household income is inflation-adjusted and reported per 1000s of dollars

Table 4. 3 Discrete-Time Hazard Model Results for Diabetes Diagnosis

	Model 3.0 ($M_{3.0}$)		**Model 3.1 ($M_{3.1}$)**		**Model 3.2 ($M_{3.2}$)**		**Model 3.3 ($M_{3.3}$)**	
Covariates	**OR**	**p-value**	**OR**	**p-value**	**OR**	**p-value**	**OR**	**p-value**
Time period D_1	0.029	***	0.026	***	0.151	***	0.002	***
Time period D_2	0.032	***	0.028	***	0.161	***	0.003	***
Time period D_3	0.035	***	0.032	***	0.184	***	0.003	***
Time period D_4	0.037	***	0.033	***	0.195	***	0.004	***

Time period D_5	0.038***	0.034***	0.208***	0.004***
Non-Hispanic black		1.619***	1.367***	1.095
Mexican		2.104***	1.494***	1.795***
Proxy (lagged)			0.929	0.933
Δ Proxy			0.934	0.882
Age in 1995			0.984***	0.982***
Education			0.964***	1.002
Female			0.774***	0.766***
Married (lagged)			1.008	1.000
Δ Married			1.119	1.270
Income (lagged)			0.998***	0.999
Δ Income			0.999	1.000
BMI (lagged)				1.090***
Δ BMI				0.967**

SRH (lagged)				1.306***
Δ SRH				1.394***
CES-D (lagged)				0.979
Δ CES-D				0.962
Functional (lagged)				0.963
Δ Functional				0.989
Comorbidities (lagged)				1.206***
Δ Comorbidities				1.358***
Smoking (lagged)				0.849
Δ Smoking				0.590***
Goodness-of-fit LL	-7,810.13	-7762.84	-7116.33	-4875.71
Wald Chi-squared	13202.62	-10560.04	-9739.32	-6887.80
Probability>chi-squared	0.000	0.000	0.000	0.000

*p<.05, **p<.01, ***p<.001

Table 4. 4 Discrete-Time Hazard Model Results for All-Cause Mortality

	Model 4.0 ($M_{4.0}$)		Model 4.1 ($M_{4.1}$)		Model 4.2 ($M_{4.2}$)		Model 4.3 ($M_{4.3}$)	
Covariates	**OR**	**p-value**	**OR**	**p-value**	**OR**	**p-value**	**OR**	**p-value**
Time period D_1	0.093	***	0.092	***	0.000	***	0.009	***
Time period D_2	0.089	***	0.089	***	0.000	***	0.006	***
Time period D_3	0.074	***	0.074	***	0.000	***	0.005	***
Time period D_4	0.072	***	0.072	***	0.000	***	0.005	***
Time period D_5	0.072	***	0.071	***	0.000	***	0.009	***
Non-Hispanic black			1.085		0.987		0.893	
Mexican			0.813	**	0.713	***	0.761	
Proxy, time (t-1)					2.531	***	1.428	***
Δ Proxy					2.891	***	1.617	***
Age in 1995					1.092	***	1.025	***
Education					0.988		1.028	**

Female	0.635***	0.529***
Married, time (t-1)	0.739***	0.768***
Δ Married	0.857*	0.837
Income, time (t-1)	0.996***	0.998***
Δ Income	0.998***	0.999
BMI, time (t-1)		0.957***
Δ BMI		0.894***
SRH, time (t-1)		1.554***
Δ SRH		1.456***
CES-D, time (t-1)		1.000
Δ CES-D		1.010
Functional, time (t-1)		1.208***
Δ Functional		1.176***
Comorbidities, time (t-1)		1.124***

Δ Comorbidities				1.255***
Smoking, time (t-1)				1.655***
Δ Smoking				0.979
Goodness-of-fit LL	-15389.496	-15384.510	-12388.031	-5838.611
Wald Chi-squared	25141.130	25123.900	18156.530	12340.290
Probability>chi-squared	0.000	0.000	0.000	0.000

*p<.05, **p<.01, ***p<.001

Figure 4. 1 Probability of Developing Diabetes by Ethnic Group ($M_{3.1}$)

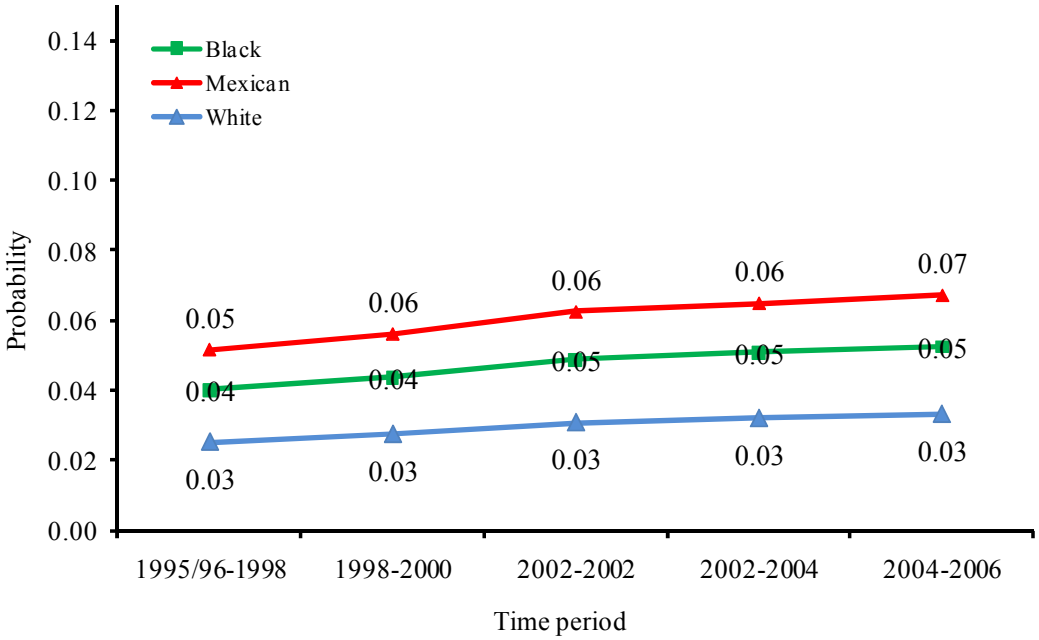
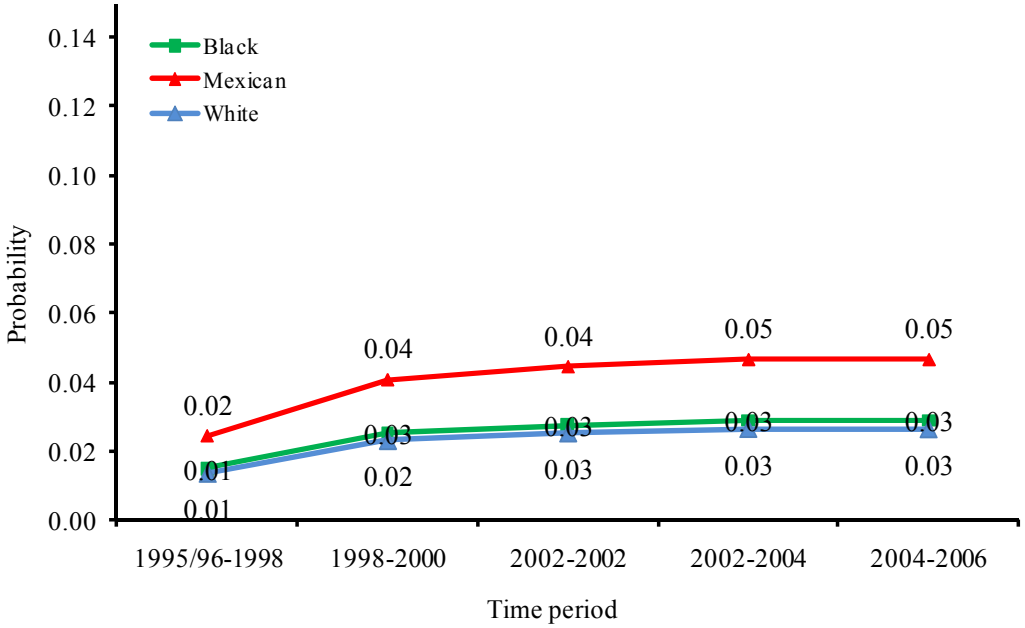


Figure 4. 2 Probability of Developing Diabetes by Ethnic Group ($M_{3.3}$)†



†Conditional hazard probabilities evaluated at the means of other $M_{3.3}$ model covariates

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CHAPTER 5:

Conclusion

This dissertation examines ethnic differences in chronic disease dynamics as detailed in the three empirical papers presented in Chapters 2-4. In these papers we are able to explore the overall level of comorbidity burden and disease dynamics for two pervasive chronic conditions for a population of older adults. These papers allow us to explore time-related changes and trends in chronic disease burden over a time period of 11 years for three race/ethnic groups in the U.S. These longitudinal data afford us with the unique opportunity to assess individual changes over time as well as analyze race and ethnic differences in chronic disease burden and onset.

Chapter 2 examines the differences in changes of comorbidity burden for a nationally-representative population of middle age and older black, white and Mexican American adults in the U.S. Here we focused on the intra- and interpersonal differences in comorbidity and how these vary across white, black and Mexican Americans. In this paper, we find that middle-aged and older Americans have on average nearly two chronic diseases at the baseline, which increased to almost three conditions over 11 years. White Americans differ from black and Mexican Americans in terms of level and rate of change of comorbidity. Mexican Americans demonstrate lower initial levels and slower accumulation of comorbidities relative to whites. In contrast, blacks showed an elevated level of comorbidity throughout the 11-year period of observation, although their rate of

change decelerated over time relative to whites. These results suggest that health disparities between black Americans and white and Mexican Americans apply to the trajectory of concurrent chronic conditions as well. Compared to whites, Mexican Americans report fewer comorbidities, whereas black Americans contend with a higher level of comorbidity. However, the trajectory of comorbidity for black Americans is converging with the trajectory for whites over time.

Specific dynamics in individual disease disparities are explored in the two subsequent chapters. Chapters 3 and 4 analyze race/ethnic differences in disease onset for two pervasive chronic conditions, hypertension and diabetes mellitus. Each of these represents a considerable burden to the U.S. population. Consequently, they also represent substantial costs to the health care system. Improved understanding of disease onset in middle and old age allows for better disease management and the expansion of prevention efforts for more affected population subgroups.

Chapter 3 goes on to examine ethnic differences in the onset of hypertension diagnosis. This paper examines the risk of developing hypertension for Americans over age 50 for a period of up to 11 years. It focuses on how incidence in self-reported hypertension varies across white, black and Mexican Americans in each of the discrete time periods examined. We find the risk of newly diagnosed hypertension increased between 1995 and 2006 for all Americans over 50: cumulative incidence for this time period is 44%. The results also suggest that the observed increase in incidence was attributable to changing demographic and health attributes for the entire population.

We find that onset of hypertension diagnosis varies by race/ethnic group. The probability of incident hypertension among black Americans increases steadily during the time period observed: from 0.10 during 1995-1998, to 0.16 in the final period of 2004-2006. In contrast, white Americans have hypertension risk that increases more slowly. White Americans start with a risk of 0.08 during 1995-1998 which increases to 0.13 in the final period of 2004-2006. For Mexican Americans, the probability also increased more slowly and mirrors the rates for whites: 0.08 during 1995-1998 to 0.13 during 2004-2006. The cumulative incidence for each of the three groups for the period examined differed accordingly. Black cumulative incidence over the 11-year period is 51%, whereas cumulative incidence for whites is 43%, and cumulative incidence for Mexican Americans is very similar to whites at 42%. While the incidence of being diagnosed with hypertension varies significantly between white and black Americans, the rates are very similar between white and Mexican Americans. These variations persisted even when differences in health behaviors, socioeconomic status, demographic, and time-varying health characteristics were adjusted.

We were also interested in exploring to what extent competing risks of dying might affect our observed disparities in incident hypertension diagnosis. It is possible that we are not able to observe disease onset due to disproportionate mortality for any of the race/ethnic groups examined. Over the 11-year period, Mexican Americans that report being hypertension-free at the baseline demonstrated a mortality advantage relative to white Americans. Black and white Americans had very similar mortality risk in the time period studied. These results suggest that these minority elderly groups are not at an increased risk of dying relative to whites. However, we are still cautious of potential bias

introduced by permanent study drop-out. If Mexican-Americans are more likely to attrit from the study, they would not be included in either hypertension morbidity or mortality statistics recorded by the HRS. Although the HRS does cross-reference mortality information with national death records, if return-migration is occurring, then these individuals would also not be captured by national mortality statistics. It is important to further this research by examining drop-out patterns for Mexican elderly in the U.S.

Finally, in Chapter 4 we estimated the onset of diabetes mellitus diagnosis for a population of middle aged and older adults that was not previously diagnosed with diabetes. This paper examines the risk of developing diabetes for a sample of older Americans for a period of up to 11 years. Differences in self-reported diabetes incidence were examined for white, black and Mexican American individuals age 51 and older. Data came from the Health and Retirement Study with up to five time intervals (1995-2006). Discrete-time survival models are used to analyze ethnic variations in the probability of developing diabetes for middle aged and older adults.

We find the risk of newly diagnosed diabetes increased between 1995 and 2006, with 11% cumulative incidence for all study participants. The probability of incident diabetes among black Americans was 0.01 during the period of 1995/96-1998, which increased steadily to 0.03 during 1998-2000 and remained at 0.03 throughout subsequent periods, and cumulative incidence at 12%. In contrast, among Mexican Americans the risk more than doubled from 0.02 during 1995/96-1998 to 0.05 during 2004-2006 and cumulative incidence at 19%. During the same period, there were no significant differences in mortality risk for either Mexican or black relative to white Americans.

These results suggest that increasing diabetes incidence for Mexican Americans into old age persists through adjustment for differences in health behaviors, socioeconomic status, demographic characteristics and changing health status. Relative to white Americans, Mexican Americans have a significantly elevated risk of diabetes throughout the 11-year period of observation. Concretely, Mexican Americans have nearly double the risk of being diagnosed with diabetes mellitus throughout the 11-year period of observation. These results are not explained by differences in health behaviors, socioeconomic status, demographic characteristics and changing health status.

In contrast, blacks demonstrate higher risk of incident diabetes compared to whites in models that account for socioeconomic and demographic differences. However, black Americans no longer have significantly different odds of incident diabetes in the full model accounting for socioeconomic, demographic and health differences. These results suggest that increases in incident risk for black Americans relative to white Americans operate largely through changes in time-varying health status.

The analysis on competing mortality risks for HRS respondents that report being diabetes-free at the baseline is also very interesting. In contrast to the mortality advantages we observe for Mexican Americans who report being hypertension-free at the baseline, we see no race/ethnic differences in subsequent mortality once we account for time-varying health changes in the model. Mexicans with diabetes at the baseline are no longer characterized by these mortality advantages. This subsample of Mexican Americans is just as likely to die and are much more likely to be diagnosed with diabetes relative to whites.

It is clear from the analyses performed in these three papers that older Americans are contending with increasing chronic disease burden as they age. Americans are increasingly hampered with chronic disease over time. Black Americans are at a substantial disadvantage with respect to whites in two of the three measures examined in these analyses. Not only are blacks burdened with greater initial levels of comorbidity (albeit this level remains relatively stable over time), but they are also at increased risk of being diagnosed with incident hypertension. Only in the measure of diagnosed diabetes mellitus are black Americans at similar risk compared to whites. In contrast, Mexican Americans are at a severe disadvantage relative to black and white Americans in their risk of diagnosed diabetes mellitus. Older Mexican Americans accrue new diagnoses of diabetes at nearly double the rate of whites.

In light of our findings of continued racial and ethnic disparities in hypertension and diabetes mellitus incidence, there is also an argument to be made for improving prevention efforts aimed at middle and older aged Americans. Prevention efforts need not only be targeted at young and middle ages, but should also be focused on older minority groups in order to reduce differentials in the risk of developing hypertension and diabetes mellitus. Although this entails increased accesss to medical care and improved awareness among medical providers, our studies also indicate that socioeconomic factors play a large role in the risk of chronic disease. Improving upstream fundamental causes of disease, such as access to resources and education, could vastly reduce excess risk for older black and Mexican individuals. Chapters 3 and 4 highlight the importance of social and structural factors as critical policy levers for mitigating elevated risk of two pervasive and important chronic diseases for minorities in the U.S.

The policy implications common to all three papers relate to improved disease coordination for all older Americans. These analyses suggest that Americans are increasingly managing multiple chronic conditions that require substantial efforts of management on the part of individuals as well as better system-wide approaches to chronic disease care and control. The U.S. healthcare system-wide benefits are clear. Given high costs associated with increasingly more complex (or uncontrolled) and co-occurring conditions, aggressive improvements in processes of care are warranted. Better understanding chronic disease acuity and co-occurring case mix are important areas for improving health care system responses to the aging U.S. population.