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

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Objectives. This research examines intra- and interpersonal differences in multiple chronic conditions reported by Americans aged 51 and older for a period up to 11 years. It focuses on how changes in multimorbidity vary across White, Black, and Mexican Americans.

Methods. Data came from 17,517 respondents of the Health and Retirement Study (1995–2006) with up to 5 repeated observations. Hierarchical linear models were employed to analyze ethnic variations in temporal changes of reported comorbidities.

Findings. Middle-aged and older Americans have on average nearly 2 chronic diseases at the baseline, which increased to almost 3 conditions in 11 years. White Americans differ from Black and Mexican Americans in terms of level and rate of change of multimorbidity. Mexican Americans demonstrate lower initial levels and slower accumulation of comorbidities relative to Whites. In contrast, Blacks showed an elevated level of multimorbidity throughout the 11-year period of observation, although their rate of change slowed relative to Whites.

Discussion. These results suggest that health differences between Black Americans and other ethnic groups including White and Mexican Americans persist in the trajectory of multimorbidity even when population heterogeneity is adjusted. Further research is needed concerning the impact of health disadvantages and differential mortality that may have occurred before middle age as well as exploring the role of nativity, the nature of self-reported diseases, and heterogeneity underlying the average trajectory of multimorbidity for ethnic elders.

Key Words: Chronic disease multimorbidity—Hierarchical linear modeling—Race/ethnic differences.

OVER the last forty 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; Vogeli et al., 2007), which is commonly referred to as comorbidity (Fried, Ferrucci, Darer, Williamson, & Anderson, 2004; Verbrugge, Lepkowski, & Imanaka, 1989). Recently, further differentiations have emerged, with multimorbidity defined as coexisting diseases that occur without one central or index disease (Boyd & Fortin, 2010). In 2005, 45.3% of the community-residing Americans aged 65–79 years and 54.2% of those aged 80 years and older reported multiple chronic diseases (Paez et al., 2009). In addition, Medicare claims data have documented that two thirds of all beneficiaries age 65 years and older have two or more chronic conditions, and one third have four or more (Fried et al., 2004). Multimorbidity 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 attributable to individual diseases (Fried et al., 2004).

There is an extensive literature documenting a disproportionate share of chronic disease morbidity and mortality for ethnic minorities (Cooper et al., 2000; Hayward, Miles, Crimmins, & Yang, 2000; Lantz, Lepkowski, Williams, Mero, & Chen, 1998; Wong, Shapiro, Boscardin, & Ettner, 2002). 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 United States (Freedman et al., 2002; Lynch & Smith, 2005). Various putative mechanisms such as double jeopardy—where minorities are subject to poor health from aging processes but also because they occupy a low status social position (Ferraro & Farmer, 1996)—as well as lifestyle choices and discrimination (Williams & Collins, 1995) have been proposed to account for these observed ethnic differences. Although 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.

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Even when longitudinal studies are undertaken, investigators tend to focus either on multiple diseases in a single minority group or on a single disease across different ethnic groups. For instance, Otiniano, Ottenbacher, Markides, Ray, and Du (2003) examine longitudinal rates of heart attack mortality for Mexican elders and find that patients are more likely to be male, older, and have co-occurring diabetes mellitus, hypertension, and stroke. Wray, Alwin, McCammon, Manning, and Best (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, current research has examined transitions in morbidity between two points in time. Often, this approach does not accurately reflect the dynamic nature of health as it provides no basis for distinguishing among alternative growth curves or trajectories (Rogosa, 1988). Connected by health transitions across successive years, examining health trajectories imparts a form and meaning distinct from those of health transitions (Clipp, Pavalko, & Elder, 1992). Accordingly, a more complete understanding of ethnic differences in multimorbidity requires an analysis of health trajectories in terms of both the level as well as the 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 multimorbidity by using longitudinal data derived from a national sample of Americans aged 51 and older for a period of up to 11 years (1995–2006). Second, we examine how the level and rate of change associated with multimorbidity differ among Black, Mexican, and White middle-aged and older adults. Finally, research that attempts to sort out the time ordering of predictors of morbidity and mortality has been sparse. We explore how ethnic differences in the trajectory of multimorbidity interface with socioeconomic and health changes. In this vein, we specify time sequencing of health changes in order to isolate predictive pathways to the development of chronic disease.

Background and Hypotheses

Although the increase in the prevalence of multimorbidity at the population level has been documented (Fried et al., 2004; Paez et al., 2009; Vogeli et al., 2007), there is very limited understanding of intrapersonal changes in coexisting chronic conditions over time. Even when repeated observations are available, the focus has been on transitions in morbidity between two points in time, which offers little information on how the level of and rate of change in multimorbidity vary over time.

H_1: Multimorbidity increases linearly over time for middle-aged and older Americans

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 differences 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 crossover with rates of more advantaged social groups) and selective mortality due to the accumulation of health disadvantages over the lifecourse (Beckett, 2000). These accumulated disadvantages represent systematic assaults to health throughout the life span. Consistent with the concept of cumulative disadvantage, Blacks are expected to demonstrate disease earlier in the life span and are hypothesized to experience greater levels of co-occurring disease in middle and old age relative to Whites. However, the trajectory for White Americans is expected to approach the Black trajectory as individuals age.

H_2: Black middle-aged and older adults have higher initial levels of multimorbidity but a slower rate of increase over time compared with 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, healthy migrant effects, where more robust individuals self-select in migrating; advantages stemming from residing in ethnic enclaves that may yield protective effects on health; poor data quality with respect to the reporting of age and the ascertainment of mortality statistics; 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). Whereas studies utilizing several nationally representative data sources find evidence for a Hispanic mortality advantage (Franzini, Ribble, & Keddie, 2001; Markides & Coreil, 1986), 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. Bearing in mind that Mexican-origin individuals comprise the largest Hispanic subgroup in the United States, we tentatively hypothesize that Mexican Americans will exhibit health advantages relative to White Americans.

H_3: Mexican-origin adults have lower initial levels of multimorbidity and a slower rate of increase relative to White adults.

In addition to depicting racial/ethnic differences in the trajectory of cormorbidity, we are interested in how these variations are influenced by socioeconomic status (SES) and evolving health status. Indeed, SES is not merely a confounder of racial/ethnic differences but part of the causal pathway through which race/ethnicity affects multimorbidity (Williams, 1997). Disadvantages afforded by social inequalities lead to differences in health through divergent employment and occupational experiences, income and wealth streams, lifestyles, and health behaviors (Bulatao & Anderson, 2004; Hayward et al., 2000; Hertzman, 2004).

Although disability and poor self-rated health are often conceptualized as outcomes of disease, they could also be predictors of disease. Differences in various dimensions of health (e.g., self-rated health and disability) may partially explain the linkages between race/ethnicity and the trajectory of multimorbidity. For instance, self-rated health has a biologic basis, and it can be a sensitive barometer of physiologic states. Many biomarkers have shown a graded relationship with self-rated health, including blood levels of albumin, white blood cell count, hemoglobin, high-density lipoprotein cholesterol, and creatine (Jylha, Volpato, & Guralnik, 2006). An individual's understanding and reporting of their own self-rated health includes not only direct information of their disease diagnoses and severity communicated through health care providers but also personal experience with daily functional difficulties and self-perceptions of mental and physical well-being in the form of fatigue, pain, and physiologic condition (Jylha, 2009; Jylha et al., 2006). Hence, self-rated health can serve as an indicator of preclinical states, which may subsequently lead to the diagnosis of disease.

Fried and colleagues (2004) suggest that disability, multimorbidity, and frailty are three distinct but interconnected concepts in describing health in old age. Disability entails difficulty or dependency in carrying out activities essential to independent living, whereas multimorbidity is the aggregation of clinically manifest diseases present in an individual, and frailty refers to the aggregate of subclinical losses of reserve across multiple physiologic systems. The presence of disability or frailty could contribute to development or progression of chronic diseases, possibly through the lower activity levels associated with the former two conditions or through other pathways affecting some basic biological mechanism essential to the maintenance of homeostasis, such as inflammation or sympathetic–parasympathetic equilibrium. Accordingly, racial/ethnic differences in multimorbidity could be affected by disability and frailty.

Method

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 years and older 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 HRS cohort (born 1931–1941), and War Babies (WB; born 1942–1947). In our data, the AHEAD cohort is interviewed in 1995 and the HRS cohort in 1996. CODA and WB cohorts do not enter into the study until 1998. From 1998 onward, data are collected for all four birth cohorts. More extensive documentation of HRS birth cohorts as well as a detailed description of the HRS study design have been published elsewhere (Hauser & Willis, 2004; Heeringa & Connor, 1995) and can be found on the HRS website (http://hrsonline.isr.umich.edu).

Earlier waves of the HRS/AHEAD surveys (1992–1994) yielded differences in the questionnaires for some of the health status variables. Due to wave incomparability 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, TX) and HLM 6.05 (Scientific Software Int., Lincolnwood, IL).

Measures

Self-reported disease.—The HRS asks respondents about a variety of diseases each interview year, including physician-diagnosed hypertension, heart disease, diabetes, cancer, lung disease, arthritis, and stroke as reported by respondents. 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 the year cancer was diagnosed or receipt of cancer therapies (radiation, surgery, and chemotherapy) to verify the diagnosis of cancer.

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

For the purpose of this study, the outcome measure is a total count of multimorbidity, ranging from 0 to 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 six to seven conditions. The distribution of the multimorbidity variable is sufficiently normal to treat it as a continuous variable (M = 2.079, SD = 1.355). The skewness (0.444) indicates that the data are slightly right or positive skewed. The kurtosis (2.833) closely approximates 3, which is the kurtosis of a standard normal distribution.

Additional analyses were undertaken to determine the sensitivity of our simple multimorbidity count with one akin to a Charlson Comorbidities Index adjustment. We constructed separate indices weighting each disease with coefficient values derived from logistic regressions that capture each disease's predictive contribution to mortality (not shown), a similar procedure to other studies (Bravo, Dubois, Hébert, De Wals, & Messier, 2002). In this way, a disease that is more predictive of death has a greater weight in the total multimorbidity score. We then examined the correlation between this alternate multimorbidity index with our simple sum (ρ = .98). Given the high correlation between the indices, we are satisfied that using the sum of conditions is a sufficient measure of the multimorbidity burden assumed by the study population.

Race and ethnicity.—The principal covariates of interest in the analyses are indicators for self-reported Black race and Mexican ethnicity. Race/ethnicity is used to construct 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 American 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 individuals in these analyses.

Social stratification and social support.—Various demographic and socioeconomic factors are included as time-constant and time-varying covariates in the analysis. Baseline age is measured as age in 1995 for all individuals in the study, regardless of entry cohort (range 48–103). Education is measured as a continuous variable denoting years of schooling (range 0–17). Income is included as a time-varying covariate in the analyses. Lagged (reported values at time $t - 1$) and change in income are inflation adjusted to 2006 levels and was also rescaled (reported per 1,000s of dollars) to facilitate its interpretation in the multilevel models. Marital status is conceptualized as an indicator of social support for individuals (House, 2001) 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 between each two points in time over the study period.

Health status and health care utilization.—Increases in multimorbidity may result from lowered activity levels stemming from mobility limitations associated with disability (Fried et al., 2004). In addition, existent depressive symptoms may limit an individual's ability to adhere to healthful practices related to disease prevention. Therefore, time-varying health covariates are included in the analytical models to adjust for population heterogeneity in health status as well as to examine changes in multimorbidity when evolving health profiles are taken into account. Modeling these dynamic processes enables us to better capture changes accruing differentially to individuals with various health profiles at each point in the study and to assess racial/ethnic variations, given these dynamic changes for individuals over an extended period of time.

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 is conceptualized as confounding variables in an individual's future development of chronic disease. Several covariates are used to mark the physical and mental health status of respondents and are included in the analyses as time-varying covariates. Self-rated ill health is measured with a five-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 ADL or IADL. Previous studies have noted the increased range and sensitivity gained from measuring functional status using a single additive measure (Spector & Fleishman, 1998). Body mass index (BMI) is calculated using respondent's self-reported weight and height measurements. Depressive symptoms are measured with an abbreviated version of the Center for Epidemiological Studies-Depression scale (0–9) that has been validated in previous studies (Turvey, Wallace, & Herzog, 1999), with a higher score reflecting more elevated depressive symptoms. Lagged covariates and covariates denoting the change (current minus previous wave) for all these health status variables are included in the analyses.

In addition, we include number of physician visits as the most direct measure of access to diagnoses. The HRS asks respondents about the number of physician visits they have had over the last two years (since the previous interview). We include covariates for both lagged physician visits and change in physician visits in the analyses.

Data Analysis

One of the limitations of using longitudinal data is the possibility of missing data at follow-up due to item nonresponse,

survey nonresponse, 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). To deal with this missing data issue, we employed multiple imputation approach (Schafer & Graham, 2002). 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. Similar approaches have been used in recent studies of health trajectories (Liang et al., 2005).

Models are estimated by growth curves—multilevel models of longitudinal data, also referred to as repeated measures longitudinal models (Raudenbush & Bryk, 2002). 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). We employ a two-level multilevel model, where Level 1 consists of repeated observations for all individuals over time and Level 2 models the interpersonal differences in the intercept as well as the slope of the trajectory. In this way, models yield estimates of both within-individual changes across time (Level 1) and between-individual differences at the baseline (Level 2).

$$\text{Level-1}: Y_{it} = \pi_{0i} + \pi_{1i}\text{TIME}_{it} + \sum \pi_k X_{kit} + \varepsilon_i \quad (1)$$

$$\text{Level-2}: \pi_{0i} = \beta_{00} + \beta_{01}\text{Mexican} + \beta_{02}\text{Black} + \sum \beta_{pq} X_{qi} + U_{0i} \quad (2)$$

$$\pi_{1i} = \beta_{10} + \beta_{11}\text{Mexican} + \beta_{12}\text{Black} + \sum \beta_{pq} X_{qi} + U_{1i} \quad (3)$$

In Equation 1, Y_{it} is the count of comorbidities for individual i at time t; π_{0i} is the intercept of the multimorbidity trajectory for individual i; and π_{1i} is the rate of change (slope) in the number of comorbidities for individual i across different time periods. X_{kit} indicates the kth time-varying covariate for individual i at time t. ε_i represents random error in health status for individual i. 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, and race/ethnic group) associated with individual i, and β_{pq} represents the effect of X_q on the pth growth parameter. U_{0i} and U_{1i} are random effects with mean of zero.

In the proposed analysis, both linear and nonlinear changes in disease were considered. Disease is modeled as a linear, quadratic, and cubic function of time. Based on significance levels of the linear, quadratic, and cubic slopes, the linear functional form of time was selected as most appropriate. In our model, the time variable is centered so that the estimated intercept reflects the level of the trajectory at the sample mean time of follow-up. In addition, time-varying covariates are included in the Level 1 equations of this model. The Level 2 equations, Equations 2 and 3, allow for the random modeling of the intercept, π_{0i}, and slope parameters, π_{1i}. Both time-varying covariates in the Level 1 equation and baseline covariates in the Level 2 equations are centered at their respective grand means.

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 complete the 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 completed by proxy respondents. Excluding proxy interviews was not considered a viable option, given that it could lead to serious selection bias. Additionally, Beckett, Weinstein, Goldman, and Yu-Hsuan (2000) note that including proxy interviews in analyses examining self-reported disease of older respondents is imperative when proxy caregivers are in a position to provide a more accurate reporting of conditions that cause cognitive or physical impairment. Consequently, proxy interviews with imputed data are included in the analyses.

Findings

Table 1 details descriptive statistics for the total sample as well as by race/ethnicity. On average, the respondents have 2.08 diseases, whereas the sample mean duration of follow-up is 5.64 years. At the person level, 14% of respondents are Black and 4% are of Mexican origin. Additionally, 26% of respondents died during the observation period and 9% missed at least one interview at some point between baseline and 2006.

Table 2 offers descriptive statistics for the time-varying covariates by survey year. Because the 1995, 1996, and 1998 data were used to provide the lagged measures to time-varying covariates for the AHEAD, HRS, and WB/CODA cohorts, respectively, we had five data points (i.e., 1998, 2000, 2002, 2004, and 2006).

Table 3 offers the hierarchical linear model results for multimorbidity burden. Progressively complex models are explored from the unconditional growth model to the growth model incorporating time-varying covariates. By order of presentation in Table 3, the unconditional model (M_0) findings are used to test hypothesis H_1. Model M_1 follows and provides controls for indicators of proxy response, death, and attrition. We control for attrition and mortality early on to get more appropriate estimates for the effects of race/ethnicity

Table 1. Descriptive Statistics for Hierarchical Linear Models, HRS 1995–2006

	Total		White		Black		Mexican	
Measures	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
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
Proxy (at baseline)	0.06	0.24	0.06	0.23	0.07	0.26	0.12	0.33
Married (at baseline)	0.72	0.45	0.74	0.43	0.53	0.50	0.75	0.43
Household income (at baseline)	61.52	112.20	67.06	88.27	37.13	46.17	40.72	357.57
BMI (at baseline)	27.50	5.23	27.11	4.98	29.22	6.03	28.71	5.39
Functional status (at baseline)	0.42	1.35	0.34	1.21	0.74	1.80	0.69	1.78
Self-rated health (at baseline)	2.70	1.14	2.60	1.12	3.09	1.14	3.16	1.16
Depressive symptoms (at baseline)	1.73	1.94	1.60	1.85	2.19	2.16	2.43	2.35
Level 1 (intrapersonal changes)	*n* = 67,358		*n* = 55,051		*n* = 9,220		*n* = 3,087	
Multimorbidity	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 $_{t-1}$	0.06	0.25	0.06	0.23	0.08	0.28	0.13	0.33
Married $_{t-1}$	0.69	0.46	0.72	0.45	0.51	0.50	0.72	0.45
Household income $_{t-1}$	63.74	97.66	69.39	95.5	40.90	53.9	34.87	182.93
BMI $_{t-1}$	27.71	5.34	27.32	5.09	29.55	6.19	28.85	5.42
Physician visits $_{t-1}$	9.44	16.10	9.20	15.47	11.06	20.25	8.87	12.26
Functional status $_{t-1}$	0.49	1.43	0.41	1.27	0.87	1.93	0.82	1.92
Self-rated health $_{t-1}$	2.78	1.11	2.67	1.09	3.17	1.10	3.32	1.09
Depressive symptoms $_{t-1}$	1.86	2.06	1.73	1.98	2.34	2.24	2.58	2.43
ΔProxy $_{t-1,t}$	0.01	0.21	0.01	0.19	0.01	0.24	0.01	0.29
ΔMarital status $_{t-1,t}$	–0.03	0.21	–0.03	0.21	–0.03	0.22	–0.03	0.20
ΔHousehold income $_{t-1,t}$	0.04	102.03	–0.48	100.6	2.27	64.3	2.19	185.18
ΔPhysician visits $_{t-1,t}$	0.59	19.43	0.61	18.47	0.27	24.81	1.17	17.81
ΔBMI $_{t-1,t}$	–0.03	2.06	–0.02	1.91	–0.07	2.65	–0.02	2.47
ΔFunctional status $_{t-1,t}$	0.19	1.31	0.18	1.21	0.22	1.66	0.21	1.68
ΔSelf-rated health $_{t-1,t}$	0.10	0.91	0.11	0.89	0.08	0.99	0.10	0.97
ΔDepressive symptoms $_{t-1,t}$	0.12	2.00	0.13	1.94	0.09	2.18	0.08	2.48

Notes: Level 1 is associated with repeated observations for survey participants. Level 2 is associated with differences between individuals at the baseline (i.e., 1995 for the Asset and Health Dynamics of the Oldest Old, 1996 for HRS, and 1998 for War Babies and the Children of the Depression Age). BMI = body mass index; HRS = Health and Retirement Study.

and other covariates in subsequent models. Model M_2 incorporates demographic covariates except education, where model M_3 presents the model with education controlled. These models are included to analyze the effect of education in addition to race/ethnicity. Finally, model M_4 includes time-varying covariates of marital status and health. Models M_2–M_4 allow for the evaluation of racial/ethnic differences in multimorbidity trajectories while taking into account

Table 2. Time-Varying Covariates and Year of Survey, Health and Retirement Study Data 1995–2006

	1998		2000		2002		2004		2006	
Measures	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Proxy $_{t-1}$	0.07	0.25	0.07	0.25	0.07	0.25	0.07	0.26	0.06	0.23
Married $_{t-1}$	0.72	0.45	0.71	0.45	0.68	0.47	0.67	0.47	0.66	0.47
Household income $_{t-1}$	58.45	82.76	62.08	119.35	62.40	103.86	60.81	89.89	72.6	73.92
Physician visits $_{t-1}$	8.83	14.68	9.45	16.12	9.00	14.30	9.51	15.85	9.94	18.39
Body mass index $_{t-1}$	27.51	5.27	27.58	5.22	27.76	5.30	27.79	5.37	27.75	5.49
Functional status $_{t-1}$	0.44	1.39	0.44	1.42	0.45	1.42	0.60	1.51	0.62	1.52
Self-rated health $_{t-1}$	2.71	1.14	2.85	1.14	2.75	1.11	2.79	1.09	2.85	1.09
Depressive symptoms $_{t-1}$	1.76	1.96	1.97	2.11	1.94	2.09	1.89	2.12	1.83	2.12
ΔProxy $_{t-1,t}$	0.02	0.22	0.02	0.20	0.01	0.21	–0.00	0.20	–0.01	0.20
ΔMarital status $_{t-1,t}$	–0.02	0.14	–0.05	0.26	–0.03	0.21	–0.02	0.20	–0.03	0.20
Δ Household income $_{t-1,t}$	–0.30	91.0	–1.96	118.02	–3.68	88.63	10.42	106.92	–3.36	93.80
ΔPhysician visits $_{t-1,t}$	1.55	21.41	0.08	18.75	0.93	19.34	0.64	18.44	0.76	21.30
ΔBMI $_{t-1,t}$	0.08	1.84	0.12	1.96	–0.04	2.09	2.09	2.04	–0.10	2.20
ΔFunctional status $_{t-1,t}$	0.18	1.37	0.12	1.28	0.32	1.37	0.19	1.27	0.127	1.31
ΔSelf-rated health $_{t-1,t}$	0.26	0.94	–0.03	0.92	0.11	0.90	0.11	0.88	0.076	0.88
ΔDepressive symptoms $_{t-1,t}$	0.38	2.05	0.06	2.03	0.03	2.02	0.03	1.99	0.106	1.95

Table 3. Hierarchical Linear Model Results for Total Comorbidities, Health and Retirement Study Data 1995–2006

	Model 0 (M_0)		Model 1 (M_1)		Model 2 (M_2)		Model 3 (M_3)		Model 4 (M_4)	
Covariates	Coefficient	*p* Value	Coefficient	*p* Value	Coefficient	*p* Value	Coefficient	*p* Value	Coefficient	*p* Value
Fixed effect										
For intercept, π_0										
Intercept	2.168	***	2.185	***	2.188	***	2.188	***	2.046	***
Death			0.842	***	0.630	***	0.600	***	0.567	***
Ever attritted			–0.122	***	–0.111	***	–0.121	***	–0.115	***
Proxy			0.021		0.020		–0.056			
Age (in 1995)					0.203	***	0.172	***	0.281	***
Female					0.008		–0.008		–0.018	
Non-Hispanic Black					0.301	***	0.199	***	0.097	***
Mexican					0.003		–0.293	***	–0.329	***
Education							–0.053	***	–0.024	***
For time slope, π_1										
Intercept	0.114	***	0.120	***	0.120	***	0.120	***	0.103	***
Death			0.035	***	0.030	***	0.030	***	0.028	***
Ever attritted			–0.004		–0.002		–0.002		0.004	
Proxy			0.000		–0.003		–0.004			
Age (in 1995)					0.003	*	0.003	*	0.000	
Female					–0.012	***	–0.012	***	–0.012	***
Non-Hispanic Black					–0.009	**	–0.010	**	–0.008	*
Mexican					–0.004		–0.008		–0.017	**
Education							–0.001		–0.001	**
Time-varying covariates										
$Proxy_{t-1}$									0.006	
$Married_{t-1}$									–0.009	
$Household\ income_{t-1}$									–0.000	**
$Physician\ visits_{t-1}$									0.004	***
BMI_{t-1}									0.029	***
$Functional\ status_{t-1}$									0.039	***
SRH_{t-1}									0.228	***
$Depressive\ symptoms_{t-1}$									0.022	***
$\Delta Proxy_{t-1,t}$									0.015	
$\Delta Marital\ status_{t-1,t}$									0.007	
$\Delta Household\ income_{t-1,t}$									–0.000	
$\Delta Physician\ visits_{t-1,t}$									0.003	***
$\Delta BMI_{t-1,t}$									0.009	***
$\Delta Functional\ status_{t-1,t}$									0.023	***
$\Delta SRH_{t-1,t}$									0.134	***
$\Delta Depressive\ symptoms_{t-1,t}$									0.013	***
Random effect										
Intercept	1.673	***	1.551	***	1.506	***	1.483	***	1.054	***
Linear time slope	0.012	***	0.012	***	0.012	***	0.012	***	0.013	***
Level 1, R	0.109		0.109		0.109		0.109		0.096	

Notes: 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 rescaled; it is reported per 1,000s of dollars. Age is rescaled and reported as age in decades in order to present results within visible range. BMI = body mass index; SRH = self-rated ill health.

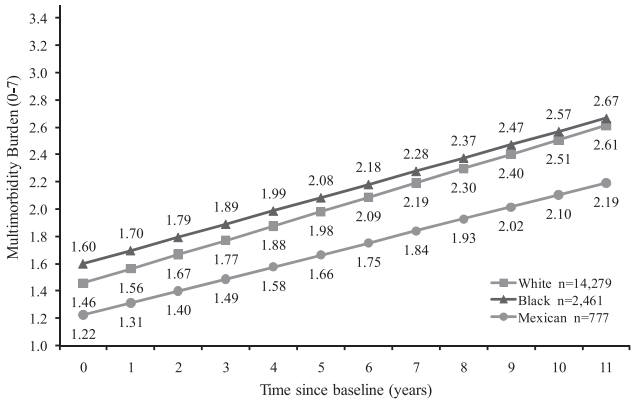
*$p < .05$; **$p < .01$; ***$p < .001$.

the confounding effects of other demographic and health variables (H_2 and H_3). Figure 1 offers graphical results of M_4 by ethnic group for the trajectories of total multimorbidity burden.

Hypothesis H_1 proposes that multimorbidity burden increases over time. From the unconditional model, M_0, we see that the unadjusted multimorbidity trajectory increases linearly with time (in M_0, $b = 0.114$, $p = .000$), offering support to H_1. First and foremost, our findings for H_1 establish that the average trajectory for multimorbidity is increasing over time. In addition, the significant random effect on the linear slope in Table 3 also indicates that there is significant individual variability around this average slope.

In support for hypothesis H_2, there are significant differences between Blacks and Whites in both the intercept (in M_4, $b = 0.097$, $p = .001$) and the slope (in M_4, $b = –0.008$, $p = .027$) of the trajectory. Specifically, Blacks exhibit significantly higher initial levels of multimorbidity relative to Whites and a slower rate of disease accumulation over time. The negative sign on the estimated difference in the slope of the trajectory between Black and White Americans indicates that multimorbidity burden in Black Americans is increasing at a slower rate relative to White Americans (Figure 1).

There are also significant differences in the multimorbidity trajectories between Mexican-origin individuals and Whites, supporting hypothesis H_3. Specifically, after adjusting for



†Trajectories are calculated using estimates from model M_4

Figure 1. Trajectories of multimorbidity burden (Trajectories are calculated using estimates from M_4).

time-varying covariates, the intercept (in M_4, $b = -0.329$, $p < .001$) demonstrates a multimorbidity advantage, and the slope (in M_4, $b = -0.017$, $p = .008$) denotes a slower rate of disease accumulation for Mexican-origin adults compared with White Americans. In Figure 1, we see that the trajectory of multimorbidity for Black individuals is higher than the trajectories for Mexican and White individuals, with Mexican individuals displaying the lowest trajectory of multimorbidity burden.

The inclusion of education moderately attenuates the already significant relationship between the intercept of multimorbidity and Black and significantly alters the direction of the relationship between multimorbidity and Mexican ethnicity. In comparing M_2 and M_3, we found that the significant Black–White differences in the intercept and linear slope of the disease trajectory persist even after accounting for varying education levels. In contrast, after including education in the model, the multimorbidity trajectory of Mexican American individuals now demonstrates significantly lower intercepts (in M_2, $b = 0.003$, $p = .957$; in M_3, $b = -.293$, $p < .001$). Moreover, higher education is associated with fewer reported diseases (in M_4, $b = -0.024$, $p < .001$) as well as a slower rate of change (in M_4, $b = -0.001$, $p = .011$) after accounting for health status and sociodemographic changes.

Lagged and change in health covariates are also consistently significant when influencing the multimorbidity trajectory. Higher functional impairment, worse self-rated health, greater depressive symptoms, and higher BMI in the previous period are all associated with higher trajectories of multimorbidity (i.e., an upward shift of the multimorbidity trajectory). In addition, increases in these health covariates in adjacent time periods also contribute significantly to greater multimorbidity. In particular, greater time-varying health limitations consistently contribute toward upward shifts in the multimorbidity trajectory.

Surprisingly, we do not see a decrease in the heterogeneity in the random slope after the inclusion of the time-varying covariates in M_4. Further exploration parsing out the baseline and time component of each of these time-varying covariates (not shown) demonstrates that the extent of the change in the time-varying covariates is similar between participants. Moreover, these covariates explain within-subject changes in multimorbidity accumulation over time (rather than between-subject differences). Although the inclusion of time-varying covariates does not decrease the variance of the random slope across models, adding these covariates allows us to evaluate how multimorbidity changes within individuals as they age and examine the racial/ethnic differences in multimorbidity trajectories after accounting for changing health profiles of individuals.

Discussion

This research provides new information concerning ethnic variations in health changes by quantitatively depicting the trajectory of multimorbidity in Black, White, and Mexican Americans. On average, the within-individual change in multimorbidity is increasing over time. This supports previous findings in the literature (Paez et al., 2009; Vogeli et al., 2007) and forms an important preliminary step in examining multimorbidity trajectory trends by race/ethnic group. Middle-aged and older Americans have on average two chronic diseases at the baseline, with an increase of 0.10 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 multimorbidity. After accounting for demographic differences as well as evolving health status, Mexican Americans demonstrate lower initial levels and slower accumulation of comorbidities relative to Whites. In contrast, Blacks showed an elevated level of multimorbidity throughout the 11-year period of observation, although their rate of increase was slower relative to Whites.

Complementing prior observations of ethnic differences in mortality and single diseases (R. J. Angel & Angel, 2006; Hummer, Benjamins, & Rogers, 2004; Mensah et al., 2005), our research extends our understanding of differences in multimorbidity 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 multimorbidity among Blacks, this differentiation is diminishing as individuals age. Specifically, multimorbidity among Blacks was 9% higher than that of Whites (1.6/1.46) at the outset, which narrowed to 2% (2.67/2.61) by 2006 (Figure 1). If this rate of change persists for another decade, multimorbidity between Blacks and Whites may fully converge. This would appear to be consistent with the prediction of the age-as-leveler hypothesis. However, the near convergence may be reflective of approaching a ceiling of comorbid conditions. Further analyses are warranted.

According to our findings, the multimorbidity trajectory for Mexican Americans is lower than that for White Americans, hence aligning broadly with the concept of the Hispanic paradox or perhaps more specifically, the Mexican paradox (Crimmins, Kim, Alley, Karlamangla, & Seeman, 2007). Interestingly, once educational differences are accounted for (M_2 vs. M_3), the disease trajectory for Mexican Americans is significantly lower at the outset relative to Whites. However, it is not until time-varying covariates are included (M_4) that we also see a significantly divergent slope of the trajectory and consequently a slower accumulation of diseases compared to Whites. Although the lower level of the trajectory seems to be related to educational differences, the significant slope appears to be related to time-varying covariates. Which exact time-varying covariates are at play, however, remains to be determined. That Mexican American adults have the same rate of disease accumulation than Whites without accounting for time-varying covariates suggests that their risk of developing additional conditions is less sensitive to changes in time-varying covariates. It is possible that Mexican Americans may be operating with greater resiliency into old age, potentially deriving from a healthy immigrant effect. However, the implications of these findings are not fully clear and warrant further study. These results should be regarded as a preliminary step in understanding disease trajectories for Mexican Americans.

Multimorbidity was measured in this research by a composite of seven chronic conditions. This amalgamated diagnosis information collected by the HRS serves to synthesize chronic disease multimorbidity for a large representative sample of the U.S. population. The inclusion of both interrelated and independent chronic conditions as well as general categorizations of disease with a ceiling of seven possible surveyed chronic diseases warrants consideration in interpreting the findings. In view of the fact that these diseases differ substantially in their etiologies and health consequences, parallel analyses focusing on single diseases or clusters of interdependent diseases are needed to further understand the ethnic differences in trajectories of chronic conditions.

Furthermore, for a given level of multimorbidity, 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, making use of the linked Medicare claims data to the general HRS data would provide more diagnostic precision in calculating disease multimorbidity for age eligible and program eligible study participants. This is a promising area for future study.

In addition to ethnicity, this research also sheds some light on the influences of other dimensions of social stratification (e.g., age, gender, and SES) on multimorbidity. 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 SES and prior health. Although women did not differ from men in their initial level of multimorbidity, they did differ from men in the rate of change. Those with more education experienced fewer comorbidities as well as slower rates of multimorbidity change. It is therefore important to take these factors into account when examining ethnic differences in multimorbidity. Furthermore, how various dimensions of social stratification interact in affecting multimorbidity 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 older. 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 analyses including individuals less than the age of 51 years would be extremely useful.

Second, nativity has consistently been an important predictor when examining Hispanic disease profiles in the United States (J. L. Angel, Buckley, & Sakamoto, 2001; Crimmins et al., 2007). Covariates for foreign-born Mexican and age at immigration were included in exploratory analyses (not shown) to address some of these concerns; however, 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. Parsing out trajectories for Mexican-origin adult immigration dynamics (nativity status, generational status, time since migration, and education level at the time of migration) to test the healthy migrant hypothesis may provide further insight into the findings of lower multimorbidity trajectory for Mexican versus White Americans. Further research is necessary to disentangle the complex relationship between immigration and health over time for Mexican-origin individuals.

Third, our findings should be interpreted while keeping in mind the nature of self-reported disease indicators in large longitudinal population-based surveys. Our data are not clinical, and results should not be interpreted as clinical findings. This is particularly salient with regard to the extent of inconsistencies in reporting diagnoses over time (Beckett et al., 2000). However, as mentioned previously, we take several steps to minimize the inconsistencies in diagnoses reporting over time for individuals in the HRS. That said, the HRS provides a rich set of data that rely on widely used and standardized self-reported disease diagnosis as well as providing information from social, economic, and behavioral domains.

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 health care have important implications to this

work. It is possible that underdiagnosis may obscure even greater differences between ethnic groups. Hence, limited access to diagnosis by Mexican-origin respondents, particularly recent immigrants with fewer resources and less familiarity with available health care options, may lead to the underestimation of differences in the burden of multimorbidity. 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, & Espino, 1997, p. 295). In addition, we examine the extent to which having access to health care via physician visits affects our substantive findings. Although access to a physician is indeed associated with a higher level of multimorbidity, it does not alter the substantive findings by race and ethnic group.

Fourth, there might be significant heterogeneity in distinct classes of trajectories of multimorbidity, which is not explored in this research. For instance, recent analysis of data from the HRS 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, 2010). Similar heterogeneity may exist for the trajectory of multimorbidity.

Many middle-aged and older Americans face multiple chronic conditions simultaneously that are increasing with time and age. Black Americans showed an elevated level of multimorbidity throughout the 11-year period of observation, whereas Mexican Americans show a favorable trajectory of coexisting chronic conditions relative to White Americans. Further research is needed concerning the impact of health disadvantages and differential mortality that may have occurred before middle age as well as exploring the role of nativity, the nature of self-reported diseases, and heterogeneity underlying the average trajectory of multimorbidity for ethnic elders.

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References

Angel, J. L., Buckley, C. J., & Sakamoto, A. (2001). Duration or disadvantage? Exploring nativity, ethnicity and health in midlife. *The Journals of Gerontology, Series B: Psychological Sciences and Social Sciences*, *56*, 275–284.

Angel, R. J., & Angel, J. L. (2006). Minority group status and healthful aging: Social structure still matters. *American Journal of Public Health*, *96*, 1152–1159.

Beckett, M. (2000). Converging health inequalities in later life—An artifact of mortality selection? *Journal of Health and Social Behavior*, *41*, 106–119.

Beckett, M., Weinstein, M., Goldman, N., & Yu-Hsuan, L. (2000). Do health interview surveys yield reliable data on chronic illness among older respondents? *American Journal of Epidemiology*, *151*, 315–323.

Berk, R. A. (1983). An introduction to sample selection bias in sociological data. *American Sociological Review*, *48*, 386–398.

Boyd, C. M., & Fortin, M. (2010). Future of multimorbidity research: How should understanding of multimorbidity inform health system design? *Public Health Reviews*, *32*, 451–474.

Bravo, G., Dubois, M. F., Hébert, R., De Wals, P., & Messier, L. (2002). A prospective evaluation of the Charlson Comorbidity Index for use in long-term care patients. *Journal of the American Geriatrics Society*, *50*, 740–745.

Bulatao, R. A., & Anderson, N. B. (Eds.). (2004). *Understanding racial and ethnic differences in health in late life: A research agenda*. Washington, DC: National Academies Press.

Carrasquillo, O., Lantigua, R. A., & Shea, S. (2000). Differences in functional status of Hispanic versus non-Hispanic White elders: Data from the Medical Expenditure Panel Survey. *Journal of Aging and Health*, *12*, 342–361.

Clipp, E. C., Pavalko, E. K., & Elder, G. H. (1992). Trajectories of health: In concept and empirical pattern. *Behavior, Aging and Health*, *2*, 159–179.

Cooper, R., Cutler, J., Desvigne-Nickens, P., Fortmann, S. P., Friedman, L., Havlik, R. & Thom, T. (2000). Trends and disparities in coronary heart disease, stroke, and other cardiovascular diseases in the United States: Findings of the National Conference on Cardiovascular Disease Prevention. *Circulation*, *102*, 3137–3147.

Crimmins, E. M. (2004). Trends in the health of the elderly. *Annual Review of Public Health*, *25*, 79–98.

Crimmins, E. M., Kim, J. K., Alley, D. E., Karlamangla, A., & Seeman, T. (2007). Hispanic paradox in biological risk profiles. *American Journal of Public Health*, *97*, 1305–1310.

Ferraro, K. F., & Farmer, M. M. (1996). Double jeopardy, aging as leveler, or persistent health inequality? A longitudinal analysis of White and Black Americans. *The Journals of Gerontology, Series B: Psychological Sciences and Social Sciences*, *51*, 319–328.

Ferraro, K. F., & Wilmoth, J. M. (2000). Measuring morbidity: Disease counts, binary variables, and statistical power. *The Journals of Gerontology, Series B: Psychological Sciences and Social Sciences*, *55*, 173–189.

Franzini, L., Ribble, J. C., & Keddie, A. M. (2001). Understanding the Hispanic paradox. *Ethnicity & Disease*, *11*, 496–518.

Freedman, V., Martin, L. G., & Schoeni, R. F. (2002). Recent trends in disability and functioning among older adults in the United States, a systematic review. *Journal of the American Medical Association*, *288*, 3137–3146.

Fried, L. P., Ferrucci, L., Darer, J., Williamson, J. D., & Anderson, G. (2004). Untangling the concepts of disability, frailty, and comorbidity: Implications for improved targeting and care. *Journal of Gerontology: Medical Sciences*, *59*, 255–263.

Glymour, M. M., & Avendano, M. (2009). Can self-reported strokes be used to study stroke incidence and risk factors?: Evidence from the Health and Retirement Study. *Stroke*, *40*, 873–879.

Hauser, R. M., & Willis, R. J. (2004). Survey design and methodology in the Health and Retirement Study and the Wisconsin Longitudinal Study. *Population and Development Review*, *30*, 209–235.

Hayward, M. D., Miles, T. P., Crimmins, E. M., & Yang, Y. (2000). The significance of socioeconomic status in explaining the racial gap in chronic health conditions. *American Sociological Review*, *65*, 910–930.

Heeringa, S. G., & Connor, J. (1995). Technical description of the Health and Retirement Study sample design. HRS/AHEAD Documentation Report DR-002. Retrieved from http://hrsonline.isr.umich.edu/sitedocs/userg/HRSSAMP.pdf

Hertzman, C. (2004). The life-course contribution to ethnic disparities in health. In N. B. Anderson, R. Bulatao, & B. Cohen (Eds.), *Critical perspectives on racial and ethnic differences in health in late life* (pp. 145–170). Washington, DC: National Academies Press.

House, J. S. (2001). Understanding social factors and inequalities in health: 20th century progress and 21st century prospects. *Journal of Health and Social Behavior*, *43*, 125–142.

Hummer, R. A., Benjamins, M. R., & Rogers, R. G. (2004). Racial and ethnic disparities in health and mortality among the U.S. elderly population. In N. B. Anderson, R. A. Bulatao, & B. Cohen (Eds.), *Critical perspectives on racial and ethnic differences in health and late life* (pp. 53–94). Washington, DC: National Academies Press.

Johnson, R. J., & Wolinsky, F. D. (1993). The structure of health status among older adults: Disease, disability, functional limitation, and perceived health. *Journal and Health and Social Behavior*, *34*, 105–121.

Jylha, M. (2009). What is self-rated health and why does it predict mortality? Towards a unified conceptual model. *Social Science & Medicine*, *69*, 307–316.

Jylha, M., Volpato, S., & Guralnik, J. M. (2006). Self-rated health showed a graded association with frequently used biomarkers in a large population sample. *Journal of Clinical Epidemiology*, *59*, 465–471.

Lantz, P. M., Lepkowski, J. M., Williams, D. R., Mero, R. P., & Chen, J. (1998). Socioeconomic factors, health behaviors and mortality. *Journal of the American Medical Association*, *279*, 1703–1708.

Liang, J., Shaw, B. A., Krause, N., Bennett, J. M., Kobayashi, E., Fukaya, T., & Sugihara, Y. (2005). How does self-assessed health change with age? A study of older adults in Japan. *The Journals of Gerontology, Series B: Psychological Sciences and Social Sciences*, *60*, 224–232.

Liang, J., Xu, X., Bennett, J. M., Ye, W., & Quiñones, A. R. (2010). Ethnicity and changing functional health in middle and late life: A person-centered approach. *The Journals of Gerontology, Series B: Psychological Sciences and Social Sciences, 65B*, 470–481.

Little, R. J. A., & Rubin, D. R. (1987). *Statistical analysis with missing data*. New York, NY: Wiley.

Lynch, J., & Smith, G. D. (2005). A life course approach to chronic disease epidemiology. *Annual Review of Public Health*, *26*, 1–35.

Markides, K. S., & Coreil, J. (1986). The health of Hispanics in the southwestern United States: An epidemiologic paradox. *Public Health Reports*, *101*, 253–265.

Markides, K. S., & Eschbach, K. (2005). Aging, migration and mortality: Current status of research on the Hispanic paradox. *The Journals of Gerontology, Series B: Psychological Sciences and Social Sciences*, *60* (Special Issue II), 68–75.

Markides, K. S., Rudkin, L., Angel, R. J., & Espino, D. V. (1997). Health status of Hispanic elderly. In L. G. Martin, & B. J. Soldo (Eds.), *Racial and ethnic differences in the health of older Americans*. Washington, DC: National Academy Press.

Mensah, G. A., Mokdad, A. H., Ford, E. S., Greenlund, K. J., & Croft, J. B. (2005). State of disparities in cardiovascular health in the United States. *Circulation*, *111*, 1233–1241.

Otiniano, M. E., Ottenbacher, K. J., Markides, K. S., Ray, L. A., & Du, X. L. (2003). Self-reported heart attack in Mexican-American elders: Examination of incidence, prevalence, and 7-year mortality. *Journal of the American Geriatrics Society*, *51*, 923–929.

Paez, K. A., Zhao, L., & Hwang, W. (2009). Rising out-of-pocket spending for chronic conditions: A ten-year trend. *Health Affairs*, *28*, 15–25.

Palloni, A., & Morenoff, J. D. (2001). Interpreting the paradoxical in the Hispanic paradox: demographic and epidemiologic approaches. *Annals of the New York Academy of Sciences*, *954*, 140–174.

Raudenbush, S. W., & Bryk, A. S. (2002). *Hierarchical linear models: Applications and data analysis methods* (2nd ed.). Thousand Oaks, CA: Sage Publications.

Rogosa, D. R. (1988). Myths about longitudinal research. In K. W. Schaie, R. T. Campbell, W. Meredith, & S. C. Rawlings (Eds.), *Methodological issues in aging research* (pp. 171–210). New York, NY: Springer Publishing Company.

Schafer, J. L., & Graham, J. W. (2002). Missing data: Our view of the state of the art. *Psychological Methods*, *7*, 147–177. doi:10.1037/1082-989X.7.2.147

Spector, W. D., & Fleishman, J. A. (1998). Combining activities of daily living with instrumental activities of daily living to measure functional disability. *The Journals of Gerontology, Series B: Psychological Sciences and Social Sciences*, *53*, 46–57.

Turvey, C. L., Wallace, R. B., & Herzog, A. R. (1999). A revised CES-D measure of depressive symptoms and a DSM-based measure of major depressive episodes in the elderly. *International Psychogeriatrics*, *11*, 139–148.

Verbrugge, L. M., Lepkowski, J. M., & Imanaka, Y. (1989). Comorbidity and Its Impact on Disability. *The Milbank Quarterly*, *67*, 450–484.

Vogeli, C., Shields, A. E., Lee, T. A., Gibson, T. B., Marder, W. D., Weiss, K. B., & Blumenthal, D. (2007). Multiple chronic conditions: prevalence, health consequences, and implications for quality, care management and costs. *Journal of General Internal Medicine*, *22* (Suppl. 3), 391–395.

Williams, D. R. (1997). Race and health: Basic questions, emerging directions. *Annals of Epidemiology*, *7*, 322–333.

Williams, D. R., & Collins, C. (1995). U.S. socioeconomic and racial differences in health: Patterns and explanations. *Annual Review of Sociology*, *21*, 349–386.

Wong, M. D., Shapiro, M. F., Boscardin, W. J., & Ettner, S. L. (2002). Contribution of major diseases to disparities in mortality. *New England Journal of Medicine*, *347*, 1585–1592.

Wray, L. A., Alwin, D. F., McCammon, R. J., Manning, T., & Best, L. E. (2006). Social status, risky behaviors, and diabetes in middle-aged and older adults. *The Journals of Gerontology, Series B: Psychological Sciences and Social Sciences*, *61*, 290–298.