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### Race-Ethnicity and Health Trajectories: Tests of Three Hypotheses across Multiple Groups and Health Outcomes

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#### Abstract

Racial-ethnic disparities in static levels of health are well documented. Less is known about racialethnic differences in age trajectories of health. The few studies on this topic have examined only single health outcomes and focused on black-white disparities. This study extends prior research by using a life course perspective, panel data from the Health and Retirement Study, and multilevel growth curve models to investigate racial-ethnic differences in the trajectories of serious conditions and functional limitations among blacks, Mexican Americans, and whites. We test three hypotheses on the nature of racial-ethnic disparities in health across the life course (aging-as-leveler, persistent inequality, and cumulative disadvantage). Results controlling for mortality selection reveal that support for the hypotheses varies by health outcome, racial-ethnic group, and life stage. Controlling for childhood socioeconomic status, adult social and economic resources, and health behaviors reduces but does not eliminate racial-ethnic disparities in health trajectories.

#### Keywords

aging; cumulative disadvantage; functional limitations; health disparities; life course; morbidity; race-ethnicity

An abundance of research shows that racial-ethnic minorities experience poorer health than whites on a wide array of health outcomes (Williams and Sternthal 2010), but less is known regarding the temporal development of racial-ethnic differences across health conditions, particularly for lesser studied groups such as Mexican Americans. Although disparities in static health *levels* are well documented, our understanding of racial-ethnic differences in *age trajectories of health* (i.e., long-term, intraindividual rates of stability and change in health with age) remains poor. Do health disparities shrink, persist, or grow with age, and by how much? Three alternative hypotheses on the nature of racial-ethnic inequalities in health across the life course address these questions: the *aging-as-leveler* (Kim and Miech 2009), *persistent inequality* (Ferraro and Farmer 1996a), and *cumulative disadvantage* (Dannefer 1987; DiPrete and Eirich 2006; O'Rand 1996; Willson, Shuey, and Elder 2007) hypotheses. These hypotheses posit that with age, racial-ethnic disparities in health decrease, remain stable, or increase, respectively. In this study, we investigate whether trajectories of serious

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conditions and functional limitations differ by race-ethnicity in ways consistent with these hypotheses.

Recent studies of health disparities across selected race and class categories have found evidence that cumulative disadvantage processes operate through midlife but that health inequality decreases in late life (Shuey and Willson 2008; Willson et al. 2007). However, these prior studies have tended to examine only single health outcomes and to focus primarily on black-white differences. We build on these prior studies in two important ways. First, given that many studies have treated health as a monolithic concept, a systematic investigation of multiple health outcomes is necessary for refining the theories that underlie these processes (Kim and Miech 2009). Different health conditions vary in their etiologies; therefore, risk factors for the accumulation of serious conditions may differ from those that lead to declines in functional health. Investigating multiple health outcomes is likely to have considerable utility for identifying different social processes. Second, we broaden the focus on race-ethnicity by including Mexican Americans, the largest subset of Hispanics in the United States. By combining these two objectives with the application of longitudinal methods to capture life course processes, we add to the understanding of health processes across important subgroups of the U.S. population.

This study extends previous research on health disparities by drawing on life course perspectives to conceptualize and model age trajectories of serious conditions and functional limitations as dynamic life course processes among blacks, Mexican Americans, and whites. Specifically, we investigate (1) whether racial-ethnic differences in health trajectories (between 51 and 73 years of age) are consistent with the aging-as-leveler, persistent health inequality, or cumulative disadvantage hypothesis; (2) whether support for the hypotheses varies across health outcomes; and (3) the extent to which racial-ethnic differences in social and behavioral factors (e.g., childhood socioeconomic status [SES], adult social and economic resources, and health behaviors) account for racial-ethnic disparities in health levels and rates of change.

#### BACKGROUND

#### **Racial-Ethnic Health Disparities**

Racial-ethnic disparities in health are well documented. Blacks experience worse health than whites on an array of health outcomes, including serious conditions and functional limitations. For example, black adults have a higher prevalence of diabetes, hypertension, strokes, and heart disease than their white counterparts (Farmer and Ferraro 2005; Pleis, Ward, and Lucas 2010). Furthermore, blacks have substantially higher rates of disability (Kelley-Moore and Ferraro 2004; Taylor 2008) and functional limitations (Kahn and Fazio 2005; Haas and Rohlfsen 2010) and shorter overall life expectancy (Warner and Hayward 2006).

The picture of Mexican Americans' health and well-being is more complex. On one hand, Mexican Americans exhibit higher rates of diabetes and kidney and liver disease (Markides, Coreil, and Rogers 1989; Pleis et al. 2010) and have poorer functional health than whites (Markides et al. 1997). On the other hand, Mexican Americans have been shown to have a health advantage over whites in heart disease and cancer (Pleis et al. 2010), although Mexican Americans born in the United States have mortality rates comparable with those of U.S.-born whites (Hummer et al. 2000). The relatively good health of U.S. residents of Mexican origin, despite their socioeconomic disadvantage, has been attributed to sociocultural "buffering" that results in lower rates of smoking and alcohol abuse (Abraido-Lanza et al. 1999; Hummer et al. 2000), healthy migrant selection (Landale, Oropesa, and Gorman 2000), and return migration of migrants in poor health (Palloni and Arias 2004). To

reduce the potential for biases resulting from healthy immigrant or return migration effects, this study focuses on U.S.-born blacks, Mexican Americans, and whites.

#### Hypothesizing Race-Ethnicity and Health Trajectories

This study draws on three hypotheses addressing patterns of intracohort inequality with age. First, the aging-as-leveler hypothesis posits that aging involves negative consequences for both advantaged and disadvantaged populations, and that those with advantages earlier in life have the most to lose in later life (Dowd and Bengtson 1978). This hypothesis predicts that senescence and mortality operate in constant ways across advantaged and disadvantaged groups, leading to diminishing disparities at older ages, especially after age 60 (Haas and Rohlfsen 2010; House, Herd, and Lantz 2005; Shuey and Willson 2008). Therefore, controlling for early mortality selection, racial-ethnic disparities in health should attenuate later in life. Second, the persistent inequality hypothesis asserts that intracohort stratification is constant as the cohort ages (Henretta and Campbell 1976), with sociodemographic and human capital factors having persistent effects on well-being across the life course (Ferraro and Farmer 1996a). This hypothesis predicts that racial-ethnic inequalities in health remain stable with age.

Third, the cumulative disadvantage hypothesis posits that intracohort inequality increases with age (Dannefer 1987; DiPrete and Eirich 2006; O'Rand 1996; Willson et al. 2007). Relative dis/advantages are amplified with age through a "cumulative process of differentiation" (Dannefer 1988:16), whereby individuals with an initial advantage have increasing access to resources and opportunities and hence a greater capacity to avoid or abate risks (Ferraro, Shippee, and Schafer 2009). Similarly, disadvantages early in life anchor social and developmental pathways that lead to subsequent disadvantages and risks (Ferraro et al. 2009; O'Rand 2001; O'Rand and Hamil-Luker 2005). The cumulative disadvantage hypothesis predicts that initial advantages and disadvantages compound and produce diverging health trajectories as individuals age. Given the structural disadvantages that people of color face across multiple domains of the life course, the cumulative disadvantage hypothesis predicts that racial-ethnic health disparities increase with age.

Although the aging-as-leveler, persistent inequality, and cumulative disadvantage hypotheses are typically framed as competing, the foregoing review of the health disparities literature suggests that support for each of these hypotheses may vary across racial-ethnic groups and health outcomes. Almost everything we know on the topic comes from studies of trajectories of functional limitations because, to our knowledge, prior research has not tested these hypotheses as they relate to racial-ethnic differences in age trajectories of serious conditions. Moreover, extant studies on black-white differences in functional impairment trajectories provide conflicting evidence for the three hypotheses. Whereas some have found evidence of leveling (Kim and Miech 2009), results from others suggest persistent inequality (Clark and Maddox 1992; Warner and Brown 2011), and still others provide support for cumulative disadvantage (Kelley-Moore and Ferraro 2004; Kim and Durden 2007; Taylor 2008). These conflicting results may be because prior studies have examined different life stages. Indeed, Kim and Miech (2009) found support for cumulative disadvantage in early and midlife and patterns consistent with the leveling hypothesis in later life.

Prior studies have not tested these hypotheses in relation to the Mexican American–white gap in serious conditions, and only a handful have examined them in relation to Mexican American–white differences in functional impairment trajectories. A recent study by Warner and Brown (2011) found that although Mexican Americans had higher initial levels of functional limitations vis-à-vis whites, they had similar rates of change in functional limitations as they aged, consistent with the persistent inequality hypothesis. Two additional studies that examined differences in functional impairment trajectories between whites and

Hispanics/Latinos found conflicting evidence for persistent inequality (Liang et al. 2008) and aging-as-leveler hypotheses (Haas and Rohlfsen 2010). However, the extent to which these findings can be generalized to U.S.-born Mexican Americans is unclear, because these studies examined Hispanics/Latinos as a whole, despite subgroup differences in health on the basis of country of origin or nativity status (see Markides et al. 2007; Read and Gorman 2006). Moreover, these studies explored single health outcomes; therefore, it is unclear whether life course processes operate similarly across health conditions. In this study, we aim to address these gaps in the literature by examining trajectories of multiple health outcomes during the transition from midlife to late life among blacks, Mexican Americans, and whites.

#### Mechanisms Responsible for Racial-Ethnic Health Disparities

Both theory and empirical research suggest that racial-ethnic minorities have poor health relative to whites because of differential and cumulative exposure to both institutional and interpersonal racism (Du Bois [1899] 1967; Williams and Sternthal 2010). Race-ethnicity and social class are closely related but distinct social dimensions that produce inequality in access to resources, exposures to risks, and, consequently, health (LaVeist 2005). An abundance of research has shown that good health is positively associated with social and economic resources, including education (Dupre 2007), income (Mirowsky and Hu 1996), wealth (Willson et al. 2007), and marriage (Umberson and Liu 2008). These resources shape health by influencing health risk behaviors, access to health care and nutritious foods, and exposure to stressful life events and toxins. In addition, circumstances in early life such as childhood adversity have long-term consequences for health. They affect adult health both directly (Haas 2008; O'Rand and Hamil-Luker 2005) and indirectly via adult socioeconomic achievement processes (Warner and Hayward 2006). Socio-economic conditions and raceethnicity are considered "fundamental causes" of disease and illness because of their persistent association with health over time regardless of changing intermediate mechanisms (Link and Phelan 1995).

Racial-ethnic health disparities are often attributed to group differences in socioeconomic resources, owing to the well-established SES-health gradient and large racial-ethnic inequalities in SES. Compared with whites, blacks and Hispanics/Latinos are more likely to experience early life socioeconomic disadvantage (Haas and Rohlfsen 2010; Warner and Brown 2011), have lower educational attainment (Snyder and Dillow 2011), have lower household earnings (U.S. Census Bureau 2004), and possess far less wealth (Oliver and Shapiro 2006); are less likely to be married (Cherlin 1992); and have limited access to quality health care (Kirby and Kaneda 2010). Although some studies suggest that racialethnic socioeconomic disadvantages explain racial-ethnic health disparities (Yao and Robert 2008), the vast majority of studies suggest that socioeconomic inequality does not fully account for racial-ethnic health disparities (Hayward et al. 2000; Kim and Miech 2009; Williams and Sternthal 2010). In addition to socioeconomic inequality, it may be useful to consider the roles that other factors, such as unequal and cumulative exposure to stressors and discrimination across the life course, may play in generating racial-ethnic health disparities (Brown 2003; Williams and Mohammed 2009). If the health gap between whites and racial-ethnic minorities is not closed after accounting for racial-ethnic differences in social and economic resources and other measured personal attributes, this suggests that differential exposure to unobserved stressors and discrimination may play an important, underrecognized role in generating health disparities (Bratter and Gorman 2011).

#### Serious Illness and Functional Limitations

Another pattern of disparity introduced in the earlier discussion is that racial-ethnic differences in health trajectories, and probably in the specific effects of explanatory factors,

vary by the health condition under study. Within individuals, various body systems and functions differ in their rates of decline and dysregulation with age, as well as in their reactions to social conditions and stressors. Thus, the selection of health outcomes for study is a nontrivial decision that should be guided by the consideration of the etiology and relevant developmental mechanisms of each outcome. Our focus on both serious conditions and functional limitation trajectories is informed by Verbrugge and Jette's (1994) sociomedical model of the disablement process, which explicates the pathway that links pathology (diagnoses of disease), impairment (dysfunctions and structural abnormalities in body systems), functional limitations (restrictions in basic physical and mental actions, such as ambulation, reaching, stooping, climbing stairs, etc.), and disability (difficulty doing activities of daily life). The disablement process model posits that pathology onset is typically observed in midlife or late life and leads to progressive functional declines, though the pace of this process is driven by variation in resource access and exposure to risks over the variable life course (O'Rand 2001).

Risk factors for serious conditions include demographic, social, economic, psychological, biological, and physical environmental conditions and their interactions. Racial-ethnic minorities experience higher rates of many chronic diseases than whites because of greater cumulative exposure to risk factors (Farmer and Ferraro 2005; Hayward et al. 2000; Kahn and Fazio 2005). In addition to serious conditions, we focus on functional limitations because they are manifestations of underlying disease processes (Kelley-Moore and Ferraro 2004; Verbrugge and Jette 1994) and are important indicators of total morbidity burden in the middle-aged and older population, detectable regardless of clinical diagnosis (Hayward and Warner 2005).

One question not answered by previous research is whether patterns of racial-ethnic disparities are consistent across different health outcomes. To address this question, we examine racial-ethnic inequality in trajectories of both serious conditions and functional limitations and consider racial-ethnic variation in the role of a selected array of risk factors across health outcomes. This approach is consistent with three general notions. First, factors contributing to these conditions may vary because of differences in their etiologies. For instance, serious conditions, which are known to be affected by stress (Stam 2007), may be particularly sensitive to discrimination stress (Gee et al. 2007; Williams and Mohammed 2009). Second, factors that contribute to the onset of serious conditions or functional limitations may differ from those that orient their trajectories following onset; that is, the accumulation of serious conditions and the progression of functional decline (see Kim and Miech 2009; Williams and Mohammed 2009) may respond differently to the same set of factors. Third, different health conditions that develop over the life course may affect the onset and progression of others. These considerations are consistent with the socio-medical model of disablement, which posits that risk factors affect downstream health outcomes, such as functional limitations, primarily via upstream pathologies such as serious illnesses. Thus, we may expect a less direct and/or more delayed impact of stressors on functional health relative to serious conditions. Finding patterns consistent with the aging-as-leveler, persistent inequality, or cumulative disadvantage hypothesis in both serious conditions and functional limitations would suggest a common social process driving racial-ethnic disparities in overall health across midlife to later life, whereas finding divergent patterns across health outcomes would indicate the presence of condition-specific social processes.

#### DATA AND METHODS

Data from waves 1 through 7 of the Health and Retirement Study (HRS) were analyzed. The HRS uses a multistage area probability sample, with a target population of all English- or Spanish-speaking adults in the contiguous United States aged 51 to 61 years in 1992

(spouses of respondents were interviewed regardless of age eligibility), who reside in households. Blacks and Hispanics were oversampled (1.86:1 and 1.72:1, respectively) to allow independent analysis of racial groups. Respondents were reinterviewed in 1994, 1996, 1998, 2000, 2002, and 2004 (response rates were between 82% and 89%). Only a minor proportion of individuals were institutionalized at the target ages of this study, and respondents remained in the study in the event that they were institutionalized between 1992 and 2004. Nonetheless, levels of morbidity and disability may be somewhat understated given the exclusion of institutionalized populations at baseline. To reduce the potential bias from the healthy immigrant effect or from return migration (Palloni and Arias 2004), the analytic sample excluded foreign-born respondents. Analyses were based on 1,558 blacks, 290 Mexican Americans, and 6,551 whites aged 51 to 61 years in 1992. Other racial-ethnic groups were excluded because of small sample sizes.

#### Serious Conditions

Respondents were asked, "Has a doctor ever told you that you have (had a) [condition]." Serious conditions examined in this study include cancer, diabetes, heart disease, hypertension, and stroke. We follow the conventional practice in research on racial-ethnic differences in morbidity of using a summary measure of the total number of serious conditions ever diagnosed, ranging between zero and five (Haas and Rohlfsen 2010; Kahn and Fazio 2005; Kelley-Moore and Ferraro 2004). We use a summary count of serious conditions on the basis of previous research showing that this measure provides a more parsimonious approach to understanding broad dimensions of health than analyzing single items (Farmer and Ferraro 2005; Ferraro and Farmer 1996b). Moreover, Ferraro and Farmer (1999) found that self-reported summary counts of serious conditions have considerable predictive validity for health, even better than information from general medical examinations, especially among minorities. Supplementary analyses (available on request) indicated that the main results of this study are robust to the exclusion of each of the lifethreatening conditions, and thus, the results are not being driven by a single condition. To account for measurement error in the evaluation of serious illnesses among respondents with limited access to care, analyses control for whether respondents have visited a doctor's office or hospital in the past year and whether they have health insurance.

#### **Functional Limitations**

In waves 2 through 7 (1994 to 2004), respondents were asked whether they had some difficulty performing a set of tasks including walking several blocks; walking one block; walking across the room; sitting for two hours; getting up from a chair after having sat for a while; climbing several flights of stairs; climbing a single flight of stairs; stooping, kneeling, or crouching; lifting or carrying 10 pounds; picking up a dime off of a table; raising their arms above their shoulders; and pushing or pulling large objects such as furniture. A summary measure of the total number of limitations ranging from 0 to 12 was constructed (Cronbach's  $\alpha = .84$ ), consistent with prior studies using the HRS to model functional limitation trajectories (Haas and Rohlfsen 2010). The functional limitation measure in wave 1 is not comparable with measures in subsequent waves and thus is not included (see Haas 2008).

#### Sociodemographic and Control Variables

Three dummy variables index self-reported race-ethnicity: white (omitted), black, and Mexican American. Respondents are classified as white or black if they indicated that they considered themselves, respectively, as "primarily White or Caucasian" or "Black or African American" and did not report any Hispanic/Latino ethnicity. Individuals are classified as Mexican American if they reported that they considered themselves to be "Hispanic or Latino" and then indicated that they were "Mexican American" or "Chicano" on a follow-up

question concerning detailed Hispanic ethnicity. Although Hispanics/Latinos were oversampled in the HRS, it contains a very small number of respondents of Cuban, Puerto Rican, or other origins, rendering the reliable detection of differences within the Hispanic/ Latino population by ethnicity unfeasible (see Brown and Warner 2008). Given their relatively small numbers and the fact that Hispanic subgroups have different health profiles (Hummer et al. 2000; Markides et al. 2007), Mexican Americans are the only Hispanics/ Latinos included in this study. Both age and  $age^2$  are included in the analysis to capture health changes with age. Because health is known to vary across cohorts (Alwin et al. 2008), dummy variables indicate the birth cohorts of respondents (cohorts 0 to 10, corresponding to birth cohorts 1931 to 1941, respectively). Gender is measured by a dummy variable (1 =female, 0 = male). To account for different rates of dropout and death attrition, a measure of the number of waves interviewed (one to seven) and a dummy indicator of whether the respondent died during the observation are included in the models (see Thomas 2011; Warner and Brown 2011). In addition, models of serious condition trajectories include dummy variables indicating whether respondents have visited a doctor's office (1 = yes) or hospital (1 = yes) in the past 12 months.

Childhood SES is indicated by whether the family was poor, and the respondents' mothers' and fathers' educational attainment. Measures of adult social and economic resources include educational attainment, household earnings, household social security income, household net worth, health insurance coverage, and marital status. Indicators of respondents' health behaviors include measures of obesity, smoking history, whether they currently smoke, and whether they drink heavily. See the online supplement for detailed descriptions of the operationalization of these variables (part A).

#### **Analytic Strategy**

Random coefficient growth curves were estimated within a mixed-model (i.e., hierarchical linear model) framework to investigate racial-ethnic differences in health trajectories between midlife and late life. These models are well suited for the assessment of individual change with age (Raudenbush and Bryk 2002). A hierarchical strategy was used, whereby repeated observations (level 1) are nested within respondents (level 2). The growth curve models generate individual trajectories that are based on estimates of person-specific intercepts (initial value) and slopes (rate of change) that describe intraindividual patterns of change in health as a function of age. We rescaled age, setting earliest observed age to 0 (age 51 for serious conditions, age 53 for functional limitations), to aid in parameter interpretation. Comparisons of nested likelihood ratio tests of various shapes of health trajectories (e.g. linear, quadratic, or cubic models) suggested that for serious conditions, a quadratic growth curve with random intercepts and random linear and quadratic age slopes provided the best fit to the data; a linear growth curve with random intercepts and random linear slopes provided the best fit for functional limitations.

We formally tested for convergence in this accelerated longitudinal design to address the potential biases associated with confounding age-related changes and cohort differences (Alwin et al. 2008; Miyazaki and Raudenbush 2000). Specifically, we estimated both a full model that included cohort-specific intercepts and slopes and a reduced model that constrained cohort-specific trajectories to be equal (convergence model) and then tested the null hypothesis of convergence by comparing the fit of the full and reduced models (see Supplemental Tables S1 and S2). Supplemental Table S1 contains the cohort-specific coefficients for the trajectory intercepts and slopes.

The joint significance of the cohort effects on the age trajectories was tested by comparing the log likelihoods (LLs) of the full and reduced models, because the reduced model is nested in the full model (Miyazaki and Raudenbush 2000). The difference in the deviance

was calculated as  $S = -2(LL_{reduced} - LL_{full})$ , where *S* is distributed as a central  $\chi^2$  statistic with degrees of freedom equal to the difference in the number of parameters in the models. Thus, for the comparison of the full and reduced models for serious conditions, S = 219.09, with df = 30, yielding p < .01, and for the functional limitations models, S = 58.93, with df =20, yielding p < .01 (Supplemental Table S2). Therefore, we reject the null hypothesis that the cohort effects are ignorable and include them in all subsequent models to account for cohort variation in age trajectories. For the sake of concision, cohort-specific coefficients are not shown. The random coefficient growth curves for serious conditions are modeled as follows:

$$Y_{ti} = \left(\beta_{00} + \sum_{j=1}^{10} \beta_{0j} d_{ji}\right) + \left(\beta_{10} + \sum_{j=1}^{10} \beta_{1j} d_{ji}\right) (a_{ti} - \overline{a}_{ji}) \\ + \left(\beta_{20} + \sum_{j=1}^{10} \beta_{2j} d_{ji}\right) (a_{ti} - \overline{a}_{ji})^{2} \\ + \sum_{k=1}^{2} \beta_{3k} r_{ki} + \sum_{k=1}^{2} \beta_{4k} r_{ki} (a_{ti} - \overline{a}_{ji}) + \sum_{k=1}^{2} \beta_{5k} r_{ki} (a_{ti} - \overline{a}_{ji})^{2} \\ + \sum_{l=1}^{5} \beta_{6l} C_{li} + u_{0i} + u_{1i} (a_{ti} - \overline{a}_{ji}) + u_{2i} (a_{ti} - \overline{a}_{ji})^{2} + \varepsilon_{ti},$$

where *Y* is the health outcome; *t* indexes time, nested within subjects *i*; *d* is a dummy variable set representing cohorts, indexed  $j = 1 \dots 10$ ;  $(a_{ti} - _{ji})$  represents age, centered within each cohort; and thus,  $(a_{ti} - _{ji})^2$  is cohort-centered age<sup>2</sup>. The earliest cohort (cohort 0) is the reference group. Therefore,  $\beta_{00}$ ,  $\beta_{10}$ , and  $\beta_{20}$  specify the mean intercept, linear age slope, and quadratic age slope, respectively, for the reference cohort. Similarly,  $\beta_{0j}$ ,  $\beta_{1j}$ , and  $\beta_{2j}$  specify the mean difference in intercept, linear age slope, and quadratic age slope, respectively, for the reference cohort. Similarly,  $\beta_{0j}$ ,  $\beta_{1j}$ , and  $\beta_{2j}$  specify the mean difference in intercept, linear age slope, and quadratic age slope, respectively, for cohort *j*. The terms of greatest substantive interest are included in the second line of the equation and define the mean trajectories for each racial-ethnic group *r* indexed  $k = 1 \dots 2$  (with reference group specify the specified as white); thus,  $\beta_{3k}$ ,  $\beta_{4k}$ , and  $\beta_{5k}$  mean difference in intercept, linear age slope, and quadratic age slope, respectively, for racial-ethnic group *r*.  $\beta_{6l}$  specifies the coefficients for five control covariates *c*, indexed  $l = 1 \dots 5$ . Random effects (subject-specific deviations) for the intercept, linear age slope, and quadratic age slope, and quadratic age slope are specified  $u_{0j}$ ,  $u_{1j}$ , and  $u_{2j}$  respectively. Finally, e is the model residual for subject *i* at time *t*. We impose no constraints on the random-effect covariance matrix (i.e., "unstructured"). Thus, it is specified:

$$\sum_{\rm RE} = \begin{bmatrix} \sigma_{u0}^2 & & \\ \sigma_{01} & \sigma_{u1}^2 & \\ \sigma_{02} & \sigma_{12} & \sigma_{u2}^2 \end{bmatrix}.$$

The equation for functional limitation growth curve models is similar to the one for serious conditions except that there are no quadratic terms. Various sets of covariates (i.e., childhood social origins, adult social and economic resources, and health behaviors) are added to the base model in a stepwise manner before estimating the full models with all of the covariates. Serious conditions are also added as an independent variable in the models predicting functional limitation trajectories because the disablement process model specifies diseases as antecedents of functional decline (Verbrugge and Jette 1994) and because racial-ethnic differences in morbidity may underlie disparities in functional limitation (Haas and Rohlfsen 2010; Kelley-Moore and Ferraro 2004).

All variables are time varying except measures of demographics, childhood SES, educational attainment, and smoking history. And continuous variables, with the exception

of age and age<sup>2</sup>, are mean centered to facilitate model interpretation (Singer and Willett 2003). Supplemental analyses (available on request) modeled health trajectories separately for blacks, Mexican Americans, and whites to determine whether the association between the covariates and health vary by race-ethnicity. Results suggested that all three racial-ethnic groups have similar health returns to social, economic, and behavioral factors, consistent with prior research (Haas and Rohlfsen 2010; Hayward et al. 2000).

To avoid biases due to racial-ethnic differences in death rates, we use hierarchical linear models in tandem with maximum likelihood estimation, which has the advantage of being able to incorporate all respondents who have been observed at least once, including those who die (or leave the study for other reasons) during the observation period in the sample, and is consistent with the approaches of recent robust studies on disparities in health trajectories (Yang and Lee 2009). Under these circumstances, Raudenbush and Bryk (2002) noted that (1) the data may be assumed to be missing at random, meaning that the probability of missing a time point is independent of missing data given the observed data, and (2) this is a reasonable assumption when the observed data include variables related to both missingness and the dependent variable. Moreover, we control for differential rates of dropout and death attrition by including indicators of the number of measurement occasions and whether respondents died, consistent with Warner and Brown (2011). Assuming that the data are missing at random, because all of the data are used in the analysis and a fully efficient estimation procedure (maximum likelihood) is used, estimates from the growth curve models are asymptotically unbiased (Raudenbush and Bryk 2002). See the online supplement for additional details on the analytic strategy (part B).

#### RESULTS

#### Sample Characteristics

Table 1 presents the baseline descriptive statistics by race-ethnicity. As expected, blacks and Mexican Americans have more serious conditions and functional limitations than whites. Compared with whites, racial-ethnic minorities are also disadvantaged in terms of childhood SES, education, earnings, employment, wealth, health insurance, obesity, smoking, and marriage. Moreover, blacks participated in fewer interviews, are more likely to die during the observation period and are more likely to visit the doctor or a hospital. Mexican Americans are less likely than whites to have seen a doctor within the past year.

#### **Race-Ethnicity and Serious Condition Trajectories**

Table 2 presents growth curve models of serious conditions between 51 and 73 years of age. Model 1 in Table 2 includes the effects of demographic characteristics on the intercept, as well as the linear and quadratic slopes, controlling for attrition and health care utilization. On average, respondents have few serious conditions at age 51 and accumulate health problems with age at an accelerating rate, though there is considerable variability within and between racial-ethnic groups. Blacks have very different serious condition intercepts and slopes than whites, as evidenced by the significant and positive black intercept and black  $\times$ linear age slope coefficients, as well as the negative black  $\times$  quadratic age slope coefficient. These results reveal that blacks have .187 or 21% (.187/.892) more conditions than whites at age 51, and that gap increases with age, at a decelerating rate, peaking at .297 at age 62 before declining to .165 by age 73. Interestingly, although the diverging serious condition trajectories of blacks and whites between the ages of 51 and 62 are consistent with processes of cumulative disadvantage, the convergence of their trajectories between ages 63 and 73 supports the aging-as-leveler hypothesis. Mexican Americans and whites also experience different morbidity trajectories. Mexican Americans have .126 or 14% (.126/.892) more serious conditions than whites at age 51, and this gap is stable with age, consistent with the

persistent inequality hypothesis. The magnitude and shape of the racial-ethnic disparities in serious condition trajectories is illustrated in Figure 1, which visualizes model-implied group-specific morbidity trajectories, on the basis of model 1 in Table 2 (controlling for cohort, gender, visits to the doctor and hospital, and dropout and mortality attrition).

Models 2, 3, and 4 in Table 2 add childhood SES, adult social and economic resources, and health behaviors to the base model, respectively; model 5 includes all of these measures. Surprisingly, a majority of blacks' excess morbidity levels—75% (.141/.187)—remains after controlling for their disadvantages across all three sets of characteristics, and black-white differences in rates of change in serious conditions were not mediated at all, suggesting that other factors underlie blacks' elevated levels of morbidity. On the other hand, the Mexican American—white disparity in serious condition intercepts is eliminated after controlling for social origins and adult social and economic resources, but not health behaviors, consistent with research showing that socioeconomic resources over the life course are strong determinants of late life health (Haas 2008; Warner and Hayward 2006).

#### Race-Ethnicity and Functional Limitation Trajectories

Growth curve models of functional limitation trajectories are presented in Table 3. Large racial-ethnic differences in functional limitation levels are observed, though all groups exhibit comparable linear increases in functional limitations with age. On average, whites have 1.058 functional limitations at the intercept age of 53 years, and they accumulate more functional limitations with age, indicated by the significant positive coefficient for their linear (.080) slope. Compared with whites, blacks and Mexican Americans have .620 and . 756 more functional limitations, respectively. The nonsignificance of the interaction between race-ethnicity and the linear slope suggests that there are no racial-ethnic differences in intraindividual rates of change in functional limitations between ages 53 and 73 years. As predicted by the persistent inequality hypothesis, blacks and Mexican Americans have worse functional health than whites, and the health gaps are constant between midlife and late life (see Figure 2, which shows model-implied functional limitation trajectories on the basis of model 1 in Table 3).

Models 2 through 5 in Table 3 suggest that the different sets of factors mediate black-white and Mexican American–white gaps in functional limitations to varying degrees. Although adjusting for childhood SES and serious conditions narrows the gaps, controlling for adult social and economic resources closes the gaps, and accounting for health behaviors does not appreciably mediate racial-ethnic health disparities. Supplemental analyses (available on request) indicated that racial-ethnic differences in education and wealth are key to explaining health disparities. Serious conditions increase the risk for developing functional limitations, consistent with the disablement process model (Verbrugge and Jette 1994). Model 6 indicates that there is racial-ethnic parity in functional impairment, net of childhood SES, adult social and economic resources, health behaviors, and serious conditions.

#### DISCUSSION

Although racial-ethnic disparities at static levels of physical and functional health are well documented, less is known about racial-ethnic differences in serious condition and functional limitation age trajectories, especially when comparing Mexican Americans to whites. This study extends prior research in three key respects. First, this is the only study to date to investigate racial-ethnic differences in age trajectories in both serious conditions and functional limitations, key outcomes and determinants of the disablement process. Examining racial-ethnic differences in trajectories of multiple health outcomes among the same sample facilitates the investigation of whether different life course processes shape different health outcomes. Second, this study is among the first to conceptualize and model

health trajectories of middle-aged and older Mexican Americans along with blacks and whites and thus is uniquely positioned to determine whether aging-as-leveler, persistent inequality, and cumulative disadvantage processes operate similarly or differently across a wider set of racial-ethnic groups. Third, this study focuses on racial-ethnic differences in health trajectories during the transition from midlife to late life, a phase of the life course over which the health trajectories of blacks and whites may shift from divergence to convergence (Shuey and Willson 2008).

The aging-as-leveler, persistent inequality, and cumulative disadvantage hypotheses are often framed as competing hypotheses; however, we find conditional support for all three. Our results reveal that support for these hypotheses varies across racial-ethnic groups, life stages, and health outcomes. For example, whereas Mexican Americans' elevated levels of serious conditions vis-à-vis whites are stable with age (persistent inequality), black-white disparities in serious conditions increase during the 50s and early 60s (cumulative disadvantage), followed by decreasing health inequalities through the early 70s (aging-as-leveler). Thus, Mexican American–white and black-white health disparities appear to follow different life course processes with respect to serious condition trajectories, suggesting that the mechanisms underlying health disparities vary by race-ethnicity. Our finding that the black-white gap in serious conditions widens until the early 60s before leveling is consistent with racial disparities in age trajectories of self-rated health (Shuey and Willson 2008). Because this study controls for differential mortality rates, it is unlikely that this finding is due to selective survival; rather, whites experience compressed morbidity until later stages of life, effectively delaying, but not forgoing, precipitous increases in serious conditions.

Our findings underscore the multidimensional nature of health. Whereas prior research examining the aging-as-leveler, persistent inequality, and cumulative disadvantage hypotheses with regard to racial-ethnic differences in health trajectories has focused on single health outcomes, we find that investigating disparities in trajectories of multiple health outcomes provides leverage for examining similarities and differences in life course processes shaping racial-ethnic disparities in a broader set of health problems. In the case of functional limitations, results are congruent with the persistent inequality hypothesis: both Mexican Americans and blacks have more functional limitations than whites in their early 50s, and all three groups follow parallel trajectories through their early 70s, such that the health disparities are neither accentuated nor diminished with age. A key finding of this study is that whereas the black-white gap in functional limitations was stable (persistent inequality), the black-white disparity in serious conditions increased (cumulative disadvantage) before declining with age (aging-as-leveler), suggesting that the life course processes underlying racial-ethnic health disparities are health condition specific.

There are several plausible explanations for different processes operating across health outcomes that are consistent with the sociomedical model of disablement. For example, there may be a less direct and/or more delayed impact of social factors and stressors on functional health compared with serious conditions. Given the expected lag time between the onset of serious illnesses and subsequent functional impairment, the widening and contracting black-white gap in serious conditions during the 50s and 60s, respectively, may manifest as similar functional limitation patterns much later in the life course (beyond the ages observed). Alternatively, the magnitudes of the rise and fall in serious condition disparities may not have been large enough to translate into comparable functional limitation trajectories. Furthermore, the effects of the increasing, then decreasing gap in serious conditions may have canceled each other out, resulting in long-term stability in black-white disparities in functional impairment. Moreover, resources stemming from social insurance programs available in the 60s and beyond, such as Medicare and extensions of Social Security, may have had an ameliorative effect, preventing blacks' earlier rapid increase in

serious conditions from translating into steep increases in functional impairment. Although identifying the specific processes behind differences in black-white disparities across health outcomes is beyond the scope of this study, this important topic clearly warrants future research.

The present study draws on the social stratification of health and aging literature that highlights the effects of childhood SES and adult structural positions on later life chances and outcomes. We find that various socioeconomic factors are related to health in later life and that racial-ethnic inequalities in these areas account for a portion, but not all, of disparities in health trajectories. For example, social and economic resources in adulthood are, by far, the dominant mediators of racial-ethnic functional health disparities, followed by childhood SES; yet only a small portion of the black-white gap in serious conditions is explained by racial inequalities in socioeconomic and behavioral factors. The fact that black-white disparities in serious conditions are not eliminated after accounting for blacks' disadvantages in childhood SES, adult social and economic resources, and health behaviors suggests that other unmeasured factors such as discrimination and unequal levels of exposure to stressors may play a major role.

Racial-ethnic minorities are more likely than their white counterparts to experience interpersonal discrimination (Forman, Williams, and Jackson 1997), and a growing body of robust empirical research shows that minorities' higher levels of perceived discrimination result in elevated levels of stress that lead to health disparities (Williams and Mohammed 2009). Indeed, Bratter and Gorman (2011) found that health disparities between blacks and whites persisted after controlling for SES but were eliminated after adjusting for perceived discrimination. In addition, Williams and Collins (2001) argued that racial residential segregation is a fundamental cause of health disparities because it affects living conditions, leading to differential exposure to societal risks and resources. They noted that compared with residents of white neighborhoods, those who reside in predominantly minority neighborhoods have less access to health-promoting resources such as quality education, job opportunities, quality medical care, nutritious foods, and green spaces, and are more likely to be exposed to environmental toxins, criminal victimization, and advertising for tobacco and alcohol. Therefore, the residual black-white disparity in serious conditions may be due to differential exposure to discrimination (Bratter and Gorman 2011) and/or deleterious neighborhood characteristics (Yao and Robert 2008).

The finding that adjusting for social and economic resources mediates the Mexican American–white gap in serious conditions but not the black-white gap is not surprising given that different racial-ethnic minority groups experience distinct historical and social conditions (Bobo and Charles 2009). Despite racial progress, blacks remain on the bottom rung as a consequence of social distance arising from whites' fears and doubts about blacks' innate intelligence and work ethic (Charles 2006; Massey and Denton 1993; Watters 1999), all attitudes that lead to stereotyping and discrimination (Jackman 1994). Indeed, blacks perceive the greatest racial-ethnic alienation and the least fairness and opportunity, followed by Hispanics and then whites (Bobo 2001). Although the HRS does not include the requisite information to investigate the influences of discrimination and neighborhood context on health disparities, it should be a primary aim of future research on health inequality.

Our finding that socioeconomic factors accounted for black-white inequality in functional limitations, but not serious conditions, may be indicative of the variability across neurophysiological systems and functions in response to social factors and stressors. Notably, the lifelong exposure to interpersonal racism (e.g., perceived discrimination) as a cognitive stressor tends to have the greatest impact on health conditions that are most directly affected by stress (Williams and Mohammed 2009). Much literature has

documented the negative effects of stress on various body systems, such as the neuroendocrine, cardiovascular, gastrointestinal, and immune systems (Stam 2007; Wilson, Finch, and Cohen 2002), leading to chronic diseases. In fact, perceived discrimination is known to increase the risks for serious conditions, including heart attack, stroke, hypertension, diabetes, and cancer (Gee et al. 2007). However, it remains unclear whether and to what extent perceived discrimination affects functional health. Thus, it is possible that perceived discrimination may have a greater or more direct impact on chronic conditions than functional limitations. Future research should investigate whether the impact of perceived discrimination is health condition specific and whether this explains why social structural factors mediate racial disparities in some health outcomes but not others.

Although the primary focus of this study is on racial-ethnic disparities in health, it is important for future studies to consider how racism intersects with other systems of domination, such as social class and sexism, to shape health. Intersectionality theorists and an emerging body of research suggest that racial-ethnic, class, and gender inequalities have simultaneous and multiplicative effects on health (Schulz and Mullings 2006; Warner and Brown 2011). Future research should integrate life course and intersectionality perspectives to investigate how social factors combine to shape long-term health trajectories.

A limitation of this study is that the HRS does not observe respondents until late midlife, thereby resulting in left censoring. Results presented here are likely to understate racialethnic health disparities given that racial-ethnic minorities have higher mortality rates prior to midlife, and inclusion in the HRS sample is conditional on survival to ages 51 to 61 years. Therefore, findings should be interpreted as conservative and conditional on survival to midlife.

Although an analysis of racial-ethnic differences in health trajectory variability is beyond the scope of this study, future research on this topic may improve our understanding of the processes that lead to aged heterogeneity. While the primary focus of this study is on the average trajectories of health for different racial-ethnic groups, Kelley-Moore and Lin (2011) highlighted the importance of also examining patterns of within-group variability in health as individuals and groups age. Studies rarely examine variance in age trajectories of health; rather, they tend to focus on average health trajectories (Markides et al. 2007; Warner and Brown 2011), which, as Kelley-Moore and Lin (2011) noted, has several limitations. First, estimates of average trajectories assume that different racial-ethnic groups have similar degrees of variability in intraindividual change with age. Second, these methods tend to "smooth" changes in health over time, potentially obscuring intraindividual variability and treating deviations from the predicted average trajectories may inadvertently reify "normal" aging experiences. Therefore, the conclusions of this study should be considered within the context of these caveats.

Future research should investigate whether health trajectory inequalities differ across cohorts in response to structural and contextual changes. The 1931 to 1941 birth cohort attended segregated schools and came of age prior to the profound social changes of the 1960s. These historical experiences may uniquely situate this cohort's biographical experiences. Levels of educational attainment among minorities increased over the twentieth century, and their educational outcomes, relative to those of whites, have improved among more recent cohorts. Given the strong link between education and health, one might expect racial-ethnic health disparities to decline among successive cohorts (Yang and Lee 2009). However, such optimism should be tempered by the fact that high levels of discrimination and residential segregation persist (Charles 2006) and that racial-ethnic stratification in the

economic sphere remains large and has been relatively stable or increasing in recent decades (Kochhar, Fry, and Taylor 2011).

Increasing the quality and years of healthy life and eliminating health disparities are important public health goals. To achieve these goals, it is necessary to understand patterns of intraindividual changes in health with age, and whether and how these processes differ across racial-ethnic groups and health outcomes. Empirical studies of racial-ethnic differences in health trajectories continue to lag behind theories on the matter. Specifically, aspects of temporality have been neglected in research on health disparities. Greater attention to racial-ethnic inequalities in intraindividual health changes is warranted. We find that blacks, Mexican Americans, and whites have distinctly variable experiences of health and illness as they age. Importantly, this study shows that dramatic health disparities between racial-ethnic minorities and whites have emerged by midlife. To better understand and eliminate health disparities, future research should investigate racial-ethnic differences in health trajectories and the mechanisms responsible for them earlier in life.

#### Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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#### Biographies

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## Table 1

Means and Standard Deviations for Baseline (1992) Study Variables by Race-Ethnicity

	4W	ites	Bls	acks	Mexican	Americans
Variable	W	sp	М	SD	Μ	sp
Health outcomes						
Number of serious conditions <sup>ab</sup>	.57	LT.	.91	89.	.64	.81
Number of functional limitations $^{ab}$	1.94	.56	2.85	3.25	2.92	3.12
Age (years)	55.77	3.13	55.72	3.09	55.44	3.08
Early-life social origins						
Family was poor <sup>ab</sup>	.24		.32		.37	
Mother had more than a high school education $^{ab}$	.11		.04		.01	
Father had more than a high school education $^{ab}$	.12		.03		.01	
Adult socioeconomic status						
Years of education <sup>ab</sup>	12.66	2.65	11.23	3.15	9.21	3.93
Earnings <sup>a</sup> b	\$39,091	\$43,247	\$24,009	\$26,425	\$17,564	\$19,180
Social Security income <sup>ab</sup>	\$1,049	\$2,964	\$1,217	\$2,811	\$1,135	\$2,912
Net worth <sup>ab</sup>	\$286,917	\$541,574	\$75,932	\$188,615	\$76,292	\$140,539
In the labor force <sup>ab</sup>	.71		.62		.59	
Uninsured <sup>ab</sup>	.13		.21		.32	
Unmarried <sup>ab</sup>	.21		.48		.28	
Health-related behaviors						
Obese (body mass index 30 kg/m <sup>2</sup> ) $^{ab}$	.21		.35		.28	
Ever smoked	.65		.64		.65	
Currently smokes <sup>a</sup>	.27		.31		.26	
Heavy drinker (3 drinks/day)	.05		.05		.07	
Controls						
Female <sup>a</sup>	.51		.58		.50	
Doctor visit in past year <sup>ab</sup>	67.		.83		.70	
Hospital visit in past year <sup>a</sup>	.10		.16		.11	

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	Wh	ites	Bla	ıcks	Mexican A	Americans
Variable	Μ	SD	Μ	SD	W	SD
Measurement occasions <sup>a</sup>	5.86	1.84	5.46	1.99	5.73	1.80
Died during observation period <sup><math>a</math></sup>	.13		.23		.17	
n	6,551		1,558		290	

 $^{a}$ Statistically significant difference between whites and blacks at the .05 level.

b Statistically significant difference between whites and Mexican Americans at the .05 level.

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Fixed effects <sup>4</sup>	Model 1	Model 2	Model 3	Model 4	Model 5
Intercept	.892	.884	.931 ***	.845 ***	.931 ***
Race-ethnicity b					
Black	.187 ***	.163***	.149***	.191 ***	.141 ***
Mexican American	.126*	.103	.050	.121 *	.039
Linear slope (age)	.037 ***	.037 ***	.036***	.036***	.035
Black	.021 ***	.021	.020	.021	.020 <sup>***</sup>
Mexican American	015	014	016	015	016
Quadratic slope (age <sup>2</sup> )	.002	.002	.001 ***	.002	.001
Black	$001^{**}$	$001^{***}$	001 **	001 ***	001 **
Mexican American	.001	.001	.001	.001	.001
Early life social origins					
Family was poor		.028			.012
Mother had more than a high school education		120 ***			093 **
Father had more than a high school education		017			.005
Adult socioeconomic status					
Years of education			020 ***		018 ***
Earnings (ln)			001*		$001^{*}$
Social Security income (ln)			.002**		.002 **
Net worth (ln)			003 ***		003 ***
In the labor force			054 ***		052
Uninsured			018 <sup>**</sup>		015 *
Unmarried			.007		600.
Health-related behaviors					
Obese (body mass index 30 kg/m <sup>2</sup> )				.011	600.
Ever smoked				.102***	.091 ***

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Brown	et	al.

Variable	Model 1	Model 2	Model 3	Model 4	Model 5
Currently smokes				142 ***	143 ***
Heavy drinker ( 3 drinks/day)				.006	005
Controls					
Female	029	028	049 **	012	035 *
No doctor visit	356 ***	340 ***	365 ***	344 ***	353 ***
No hospital visit	423 ***	418 ***	400 ***	417 ***	–.392 <sup>***</sup>
Measurement occasions	026	014	024 ***	026 ***	014
Died during observation	.441 ***	.446***	.410 <sup>***</sup>	.453 ***	.432
Random effects					
Level 1 residual	.240 ***	.240 <sup>***</sup>	.240 <sup>***</sup>	.239 ***	.239 ***
Level 2 age	.117***	.117 ***	.117 ***	.116***	.116***
Level 2 age <sup>2</sup>	.000 ***	.000 ***	,000 ***	.000 ***	.000 ***
Level 2 intercept	.713 ***	.711 <sup>***</sup>	.707 ***	.710 <sup>***</sup>	.702 ***
Log likelihood	-27,294.6	-27,272.6	-27,135.5	-27,062.1	-26,891.4
<i>a</i>					

 $^{\it a}$  Models control for cohort differences in trajectory intercepts and slopes.

 $b_{\rm White serves as the reference group.}$ 

p < .05.p < .01.p < .001.p < .001.

# Table 3

Race-Ethnicity and Trajectories of Functional Limitations among Adults Aged 53 to 73 Years: Random Coefficient growth Curve Models (n = 7,965)

ariable	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
ixed effects <sup>a</sup>						
Intercept	1.058***	.971 ***	2.250 ***	.596***	.750***	$1.442^{***}$
Race-ethnicity b						
Black	.620 <sup>***</sup>	.450***	.171	.578***	.416***	057
Mexican American	.756***	.562**	.003	.719***	.702 ***	033
Linear slope (age)	.080 ***	.080 ***	.067 ***	.075 ***	.044	.032
Black	.002	.002	.004	.002	.002	.006
Mexican American	006	005	007	006	004	003
Early life social origins						
Family was poor		.458				.270 ***
Mother had more than a high school education		365 ***				015
Father had more than a high school education		415				129
Adult socioeconomic status						
Years of education			179 ***			144 ***
Earnings (In)			010 ***			011 ***
Social Security income (ln)			.025 ***			.024 ***
Net worth (ln)			037 ***			034 ***
In the labor force			–.593 <sup>***</sup>			548 ***
Uninsured			059			027
Unmarried			.118**			.120**
Health-related behaviors						
Obese (body mass index 30 kg/m <sup>2</sup> )				.404		.344 ***
Ever smoked				.542 ***		.346 <sup>***</sup>
Currently smokes				176***		089
Heavy drinker (3 drinks/day)				315 ***		237*

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.840 \*\*\*

.958\*\*\*

 $1.044^{***}$ 

.721 \*\*\*

.953 \*\*\*

.935 \*\*\*

Controls Female

Model 6

Model 5 .631 \*\*\*

Model 4

Model 3

Model 2

Model 1

Serious conditions

Variable

.562 \*\*\*

 $1.403^{***}$ 

 $1.404^{***}$ 

 $1.403^{***}$ 

 $1.405^{***}$ 

 $1.404^{***}$ 

 $1.404^{***}$ 

Random effects Level 1 residual

Level 2 age

.184

.180\*\*\*

.184 <sup>\*\*\*</sup>

.185 <sup>\*\*\*</sup>

2.092 \*\*\*

.181 <sup>\*\*\*</sup> 2.313 <sup>\*\*\*</sup>

2.382 \*\*\*

2.199 <sup>\*\*\*</sup>

2.389 \*\*\*

2.417 \*\*\*

Level 2 intercept

Log likelihood

-79,972.1

-81,292.3

-81,157.2

-81,036.5

-81,713.4

-81,799.3

.176\*\*\*

 $1.262^{***}$ 

 $1.616^{***}$ 

 $1.929^{***}$ 

 $1.602^{***}$ 

2.013 \*\*\*

2.010<sup>\*\*\*</sup>

Measurement occasions Died during observation

.063 \*

.014

-.012

.013

.044

-.005

bWhite serves as the reference group.

\* p<.05.

p < .00.

p < .001.

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