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The Hispanic Paradox and Predictors of Mortality in an Aging Bi-ethnic Cohort of Mexican Americans and European Americans: The San Antonio Longitudinal Study of Aging

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Abstract

OBJECTIVES—To examine predictors of mortality in aging Mexican Americans (MAs) and European Americans (EAs).

DESIGN—Longitudinal, observational cohort study.

SETTING—Socioeconomically diverse neighborhoods in San Antonio, Texas.

PARTICIPANTS—Three hundred and ninety-four MA and 355 EA community-dwelling older adults (65+) who completed the baseline examination (1992–96) of the San Antonio Longitudinal Study of Aging (SALSA) and for whom vital status was ascertained over an average 8.2 years of follow-up.

MEASUREMENTS—Ethnic group was classified using a validated algorithm. Hazards ratios (HR) for mortality were estimated using Cox proportional hazards models with age, sex, ethnic group, education, income, frailty, diabetes with and without complications, comorbidity, cognition, depressive symptoms, and body mass index included as predictors in sequential models.

RESULTS—At baseline, MAs had higher prevalence of diabetes and frailty and lower socioeconomic status (SES) compared to EAs. The age- and sex-adjusted ethnic HR (MA vs. EA)

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for mortality was 1.54 (95% CI: 1.17–2.03). After adjusting for SES, the ethnic HR was no longer significant (HR = 1.16, 95% CI: 0.83–1.61). In the final model, comorbidity, diabetes with complications, depressive symptoms, and cognitive impairment were significant independent risk factors for mortality.

CONCLUSION—Contrary to the Hispanic paradox, MAs were at increased risk of mortality. Moreover, this ethnic disparity was largely explained by SES differences. Significant independent predictors of mortality, regardless of ethnic group, included diabetes with complications, comorbidity, depressive symptoms and cognitive impairment. Mortality reduction in older MAs requires attention to both socioeconomic disparities and disease factors.

Keywords

hispanic paradox; mortality; frailty

BACKGROUND

The U.S. population is aging rapidly and becoming increasingly more ethnically diverse. According to some estimates, approximately one-fifth of the US population will be over age 65 by the year 2050,¹ and the proportion of Hispanic older adults will increase from 4% to 16% of the population.² Mexican Americans (MAs) are the largest subgroup of U.S. Hispanics³ and have different rates of risk factors for mortality compared to European Americans (EAs). For example, both diabetes and frailty are more prevalent in MAs compared to EAs, and both have been associated with increased mortality risk.^{4,5} MAs as a whole also have lower socioeconomic status (SES) compared to EAs.^{6,7}

Based on the Hispanic paradox, a term used to describe the finding that Hispanics in the U.S. have similar mortality rates to those of EAs in spite of worse disease profiles and lower SES, one would predict a lower mortality rate in older MAs compared to EAs.⁸ The validity of the Hispanic paradox is, however, controversial. Because the San Antonio Longitudinal Study of Aging (SALSA) provides the opportunity for direct comparison of mortality and predictors of mortality in an aging, bi-ethnic cohort of MAs and EAs, the purpose of this study was to examine whether or not ethnic differences in mortality in the SALSA cohort were consistent with the Hispanic paradox.

METHODS

Subjects

Subjects were 394 MAs and 355 EAs, aged 65 to 80, who participated in the baseline examination (1992–1996) of SALSA, a community-based study of the disablement process in older MAs and EAs. The cohort comprised the oldest persons who participated previously in the baseline examination of the San Antonio Heart Study (SAHS), a population-based study of diabetes and cardiovascular disease in MAs and EAs conducted in two phases between 1979 and 1988. Details of the SAHS design, sampling approach, recruitment and field procedures have been previously reported; subjects were randomly sampled from low-, middle-, and high-income neighborhoods in order to provide a cohort with comparable numbers of MAs and EAs and to maximize sociocultural variation among MAs in the study. Response rate to the SALSA baseline examination among age eligible SAHS cohort members was 70.5%, with no evidence of major attrition bias between the initial SAHS survey and the SALSA survey.

A SALSA follow-up study was carried out from 2000 to 2005 and included three study visits. Follow-up 1 occurred an average of 7.0 ± 1.3 years (range: 4.4–9.7 years) after baseline; follow-ups 2 and 3 occurred at 18-month intervals thereafter. Vital status of

SALSA baseline participants was ascertained over an average period of 8.2 ± 2.9 years (range: 0–12.5 years) from baseline to the end of Follow-up 3. All measures were translated into Spanish using established techniques to assure cross-cultural equivalence of the English and Spanish versions, and were administered by trained bilingual staff in English or Spanish based on the participants' stated language preference. The study was approved by the Institutional Review Board of the University of Texas Health Science Center, and all subjects gave informed consent.

Measures

Ethnic Group—Ethnic group was classified as MA or EA using a validated, standardized algorithm, which considers parental surnames (maiden name of mother), birthplace of both parents, self-declared ethnic identity, and ethnic background of grandparents.⁹

Frailty Characterization—Validated CHS criteria and standardization procedures⁴ were applied to the pooled SALSA sample; standardized SALSA cutpoints have been published previously.⁶ CHS frailty criteria are the presence of five frailty characteristics: 1) walking speed standardized based on median height and sex, 2) grip strength standardized based on body mass index (BMI) and sex, 3) energy expenditure standardized based on sex, 4) exhaustion based on self-report, and 5) weight loss. Frailty was classified as a dichotomous variable. Individuals with less than 3 frailty characteristics were classified as non-frail, and those with 3 or more characteristics were classified as frail.

Vital status: Vital status was monitored through the end of Follow-up 3. Deaths and date of occurrence were ascertained by regular review of local newspaper obituaries, San Antonio Metropolitan Health District vital statistics records, and searches of the Social Security Death Index, the National Death Index, and Lexis Nexis.

Covariates

Chronic Disease and Obesity—Body mass index (BMI) was calculated as weight, in kilograms, divided by height, in meters squared. Clinical measures were used to assess diabetes (American Diabetes Association, ADA, criteria),¹⁰ and hypertension (Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure 6, JNC 6, guidelines).¹¹ ADA criteria for diabetes are fasting blood glucose ≥ 126 mg/dL and/or currently taking glucose lowering medications. JNC6 criteria for hypertension are systolic blood pressure ≥ 140 mm Hg or diastolic blood pressure ≥ 90 mm Hg or currently taking antihypertensive medications. Myocardial infarction (MI) was assessed by evaluation of 12-lead electrocardiogram (ECG) for the presence of ischemic ECG abnormalities. These included the presence of Q-waves of at least 0.04 seconds in duration in leads II, III, and aVF, and/or Q-waves in the precordial leads (V1–V6).¹² Angina pectoris was assessed using the Rose questionnaire.¹³ Chronic obstructive pulmonary disease (COPD) was assessed by Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria as previously described.¹⁴ Arthritis, cancer (non-skin), and stroke were assessed by self-report of physician-diagnosed disease. Comorbidity was calculated as presence of two or more of these diseases excluding diabetes, which was considered separately. Diabetes with complications was defined as the presence of both diabetes and one or more of the following conditions: stroke, MI, angina, proteinuria, or peripheral arterial disease (PAD). Proteinuria was assessed as an early indicator of nephropathy based on a spot urine sample using the Albustix test (Ames, Elkhart, IN) and was defined as a value of $\geq 1+$.¹⁵ Ankle-brachial indices (ABI) were calculated as the ratio of the higher systolic pressure of the dorsalis pedis or posterior tibia for each leg over the mean of the right and left brachial systolic blood pressure. PAD was defined as an ABI < 0.9 in either leg and was considered

absent if an ABI of 0.9 – 1.3 was present in both legs. An ABI > 1.3 was defined as high and non-compressible and considered as indicating presence of PAD.¹⁶

Cognitive impairment—Cognitive impairment was assessed using the Folstein Mini-Mental State Examination (MMSE).¹⁷

Depressive Symptoms—Depressive symptoms were assessed using the Geriatric Depression Scale (GDS).¹⁸

Socioeconomic Status (SES)—Household income and number of years of formal education were assessed by self-report.

Statistical Analysis

Ethnic differences in baseline characteristics were compared using two sample t-tests for continuous variables and the chi-squared statistic for categorical variables. The mortality rate at each follow-up unadjusted for variation in follow-up interval was calculated by ethnic group and sex; overall ethnic differences in mortality as well as sex differences in mortality within ethnic groups were tested at each time point using the chi-squared statistic. Subjects lost to follow-up at the time of each follow-up exam were categorized as censored, i.e., vital status was unknown. Multivariable models of mortality predictors were estimated using a series of Cox proportional hazards models in which sets of covariates were added sequentially to the models. Subjects who died were censored at date of death, and those lost to follow-up were censored at date of last contact. Model 1 included age, sex and ethnic group. Model 2 added SES (education and household income). Model 3 added frailty. Model 4 added diabetes with and without complications; and, the final Model 5 added comorbidity, MMSE, GDS, BMI and an ethnic*sex interaction term, in order to examine whether observed unadjusted sex differences in mortality risk varied by ethnic group.

RESULTS

Sample Characteristics

Table 1 summarizes the participant characteristics at baseline by ethnic group. MAs compared to EAs were significantly younger, less educated, less affluent, and more likely to have diabetes mellitus (with or without complications), and proteinuria. MAs had poorer performance on the MMSE and higher scores on the GDS compared to EAs. No ethnic difference was observed with respect to gender distribution, presence of angina, MI, hypertension, stroke, COPD, PAD, or arthritis. EAs were more likely than MAs to have non-skin cancer, but there was no ethnic difference in the prevalence of comorbidity.

Table 2 shows vital status information by ethnic group and sex at the three follow-up exams. Males in both ethnic groups had a higher number of deaths compared with females at all three time points; however, this sex difference achieved greater statistical significance among MAs compared with EAs across all three time points. Deaths were more prevalent in MAs compared to EAs at all follow-ups, but the ethnic difference was significant at follow-ups 1 and 2 only. The proportion of censored individuals (lost to follow-up) was similar among MAs and EAs, and there was no significant ethnic difference in censorship at any of the three follow-ups. Multivariable Cox proportional hazards models are presented in Table 3. In Model 1, after adjustment for age and sex, MAs were at 54% increased risk of death compared to EAs; the ethnic hazards ratio (HR) was 1.54, 95% confidence interval (CI): 1.17–2.03 ($p=0.002$). Men were at two times greater risk of mortality compared with women (HR = 2.01, 95% CI = 1.54–2.62, $p < .001$), and there was a 12% increased risk of mortality with each increasing year of age (HR = 1.12, 95% CI = 1.08–1.16, $p < .001$). When SES was

added in Model 2, age and sex remained significantly associated with mortality, but ethnic group was no longer statistically significant. Each increase in household income category was associated with an 8% lower risk of mortality (HR=0.92, 95% CI: 0.87–0.97; $p=.002$), but education was not significantly associated with mortality. When added in Model 3, frailty was significantly associated with greater than two-fold increased risk of mortality (HR=2.14, 95% CI=1.46–3.15, $p<.001$); and, although the association was somewhat attenuated, frailty remained statistically significant when diabetes and diabetes with complications were added in Model 4 (HR=1.78, 95% CI=1.16–2.73, $p=.008$). Diabetes with complications was significantly associated with an almost three-fold increased risk of mortality (HR=2.92, 95% CI: 1.36–6.25, $p=.006$) in Model 4, but diabetes without complications was not a significant predictor of mortality. In the final Model (Model 5) in which comorbidity, cognitive impairment, depressive symptoms, and BMI were added, frailty was no longer a significant predictor of mortality. Diabetes with complications remained statistically significant, and the magnitude of the HR was only slightly attenuated (2.92 to 2.71). Comorbidity, cognitive impairment, and depressive symptoms were also significant predictors of mortality.

Because the mortality difference between males and females in the unadjusted analyses appeared to be more pronounced in MAs compared to EAs, a sex*ethnic interaction term was included in each of the Cox proportional hazards models; however, the interaction term was statistically significant in Models 4 and 5 only. In Model 4, the risk of death in MA men compared to MA women (HR=3.66, 95% CI: 2.34–5.72, $p<.001$) was over twice as high as the corresponding risk for EA men compared to EA women (HR=1.67, 95% CI: 1.04–2.67, $p=.033$). The p -value for the interaction term was .0149. The sex*ethnic interaction effect observed in Model 5 was similar to that observed in Model 4. Among MAs the HR for mortality in men compared with women was 3.89, 95% CI: 2.42–6.26, $p<.001$; among EAs the HR for mortality in men compared with women was 1.74 (95% CI: 1.08–2.79, $p=.023$). The p -value for the interaction term was .0148.

DISCUSSION

The purpose of this study was to directly examine ethnic differences in mortality between MAs and EAs in a community-based longitudinal study of older adults. Mortality was significantly higher in MAs compared to EAs in an age- and sex-adjusted model, but the ethnic difference was not significant after adjustment for SES. Frailty was a significant predictor of mortality in model adjusted for age, sex, SES, and diabetes with or without complications; but was not significantly associated with mortality after adjustment for comorbidity, cognition, depression, and BMI. In this final model, significant predictors of mortality were diabetes with complications, cognitive impairment, and depressive symptoms. The finding that frailty was not significantly associated with mortality after adjustment for cognitive impairment and depressive symptoms may suggest that the latter two factors play a role in mediating the association between frailty and mortality.

Our analyses were based on direct observation in a bi-ethnic cohort, which allowed for comparison of mortality between older MAs and EAs. The findings are contrary to what would have been predicted based on the Hispanic paradox, a term that has been used to describe the paradoxical finding that Hispanics in the U.S. have similar mortality rates compared to those of non-Hispanic whites in spite of worse disease profiles and lower SES.⁸ Many studies that support the Hispanic paradox are based on data from national and state vital statistics and national health surveys. Some research has suggested that the apparent Hispanic paradox based on these data can be largely explained by misclassification of ethnic group in death records, and/or incomplete ascertainment of death information.^{19,20} Other hypothesized explanations include migration of a relatively healthier population to the U.S.

(“healthy migrant effect”), return migration of less healthy immigrants (“salmon bias”), or possible sociocultural factors unique to Hispanics.²¹ Some studies have included various applied methods to attempt to adjust for these possible sources of bias, with mixed results. While some studies suggest that the bulk of this observation is artifact,²² others report findings that continue to support the Hispanic paradox.^{19,23,24} Further, it has been suggested that the mortality advantage is particularly apparent for older Hispanic adults, as compared to those of young adult and middle age groups.^{23,25}

The findings from our study do not support a mortality advantage in older MAs and in fact showed a mortality *disadvantage* prior to covariate adjustment. Moreover, adjustment for age, sex, and SES, indicated that the MA mortality disadvantage was explained primarily by the lower SES of MAs compared with EAs, particularly lower household income (Model 2). Two prior studies in the SAHS examined ethnic differences in mortality between middle-aged MAs and EAs. These studies had nearly 100% ascertainment of vital status, and showed that mortality was higher in MAs relative to EAs.^{26,27} The lack of a mortality advantage in Hispanics was also shown by Borrell and Crawford;²⁸ however, that study included younger adults, aged 25–44 years, as well as Hispanics from other subgroups. Eschbach et al. also found that mortality among younger (aged 15 – 44 years) U.S. born Hispanics in California was higher compared to that in non-Hispanic whites.²⁹

The prevalence of diseases known to increase mortality risk vary across ethnic groups and within ethnic subgroups, which may make it difficult to compare results across studies. Pandey et al. directly studied coronary heart disease-related mortality in MAs compared to non-Hispanic whites in Corpus Christi, TX.³⁰ They found that, among women, coronary heart disease mortality for MAs exceeded that for EAs (relative risk [RR]=1.32, 95% CI: 1.09–1.63); and, while there was a trend toward increased mortality in MA men relative to EA men, the RR did not reach statistical significance (RR=1.11, 95% CI: 0.96–1.28). In the present study, we measured all-cause mortality. It is possible that our finding of increased age- and sex-adjusted mortality in MAs relative to EAs is related to the significantly higher prevalence of diabetes observed among MAs in SALSA, as well as its parent study, the SAHS. In addition, we found that diabetes with complications, but not diabetes without complications, was a significant risk factor for mortality. Diabetes with complications was over two-fold more prevalent at baseline in MAs compared to EAs (19.8% vs. 7.9%, $p < .001$), which likely plays a major role in explaining the increased unadjusted all-cause mortality in MAs compared to EAs in this study. Indeed, it has been well demonstrated that MAs have significantly higher prevalence and incidence rates of diabetes compared with EAs, and the mortality rate in diabetics is higher than that in non-diabetics.^{5,31}

Because the MA mortality disadvantage was no longer present after adjustment for SES, lower SES in MAs is also a significant reason for the observed difference in mortality. Backlund et al. found mortality to increase with decreasing income in a study using data from the National Longitudinal Mortality Study.³² This finding was replicated by Suresh et al. in a study using National Health and Nutrition Examination Survey (NHANES) data, which showed lower SES was associated with increased mortality in all race and ethnic groups.³³ Along these lines, Turra et al. showed that the Hispanic paradox was present in Hispanics of lower SES, but not in Hispanics of higher SES.³⁴ We also found that while mortality was higher in males compared to females overall, which has been shown by others,³⁵ male MAs had almost 4 times the mortality risk compared to female MAs, while the mortality risk of male EAs compared to female EAs was increased by only 74%.

With regard to predictors of mortality in this study, diabetes is a major risk factor for death.⁵ It is estimated that diabetes is the fifth leading cause of death world-wide and contributes to over 8% of the deaths in the U.S.^{36,37} Prior studies show that adults with diabetes are at

increased risk of death due to all causes; and in most studies, the increased risk approaches and often exceeds twice the risk of adults without diabetes.^{37,38} It has been shown that cardiovascular diseases are largely responsible for premature death in older adults with diabetes.³⁹ The results from the present study are consistent with the latter findings, and show that older adults who have diabetes with cardiovascular complications are at 2.5 times increased risk of death compared to older adults in which diabetes with complications is absent.

The latter finding suggests that tight glycemic control to prevent diabetic complications may reduce the risk of mortality; however, the issue of whether tight glycemic control lowers the rate of diabetic complications or mortality has been controversial.⁴⁰ While there is evidence to suggest that improved glycemic control decreases the risks of microvascular complications (retinopathy, neuropathy, and nephropathy), there is less strong evidence that glycemic control has substantial impact on macrovascular complications, such as coronary heart disease,⁴¹ or mortality after MI in diabetics.⁴² Recent meta-analyses have suggested, however, that glycemic control does improve cardiovascular outcomes in adults with diabetes. Selvin et al. found that the risk for coronary heart disease in adults with type 2 diabetes increased by 13% for each 1-percentage point increase in glycosylated hemoglobin;⁴³ and Tkac (2009) more recently found a reduction in the incidence of myocardial infarction in patients with intensive glycemic control (OR=0.84, 95% CI: 0.75–0.93).⁴⁴ Further, there is recent evidence in older adults that better glycemic control is associated with better maintenance of lower-extremity function over time as measured by the Short Physical Performance Battery.⁴⁵ Because the issue remains controversial, however, and because aggressive glycemic control is not without potential adverse events, such as hypoglycemia, weight gain, and worsened quality of life,⁴¹ careful thought should be given when deciding about glycemic targets for older adults. Generally, decisions should be based on a comprehensive assessment of the whole patient, including medical comorbidities, level of independence in instrumental activities of daily living, which will impact ability to comply with diabetic medications and testing, as well as presence of social support.⁴⁰

Depressive symptoms were also found to be predictive of mortality in our study; each additional 1-point increase on the GDS was associated with a 4% increase in mortality. Consistent with other publications, GDS scores indicating depressive symptoms were significantly higher in MAs compared with EAs. Interestingly, whether depression leads to premature death has been debated, with some studies showing increased risk and others finding none.⁴⁶ The inconsistency across studies is thought to be related to differences in the populations studied, methods used to assess depression, and length of follow-up.⁴⁷ Our findings are consistent with those from another population-based cohort study, the Cardiovascular Health Study (CHS), which found that depression at baseline was predictive of higher mortality.⁴⁷ Specifically, the risk of mortality was 25–34% higher among individuals with depressive symptoms. We also demonstrated that cognition is associated with mortality; mortality risk decreased by 6% for each 1-point increase in score on the MMSE. Similar findings have been reported in other studies of cognition and mortality.⁴⁸ Our finding that depressive symptoms and poorer cognition are independent risk factors for mortality is consistent with prior work by Mehta et al. suggesting that depression and cognition are additive risk factors for mortality.⁴⁹

Limitations of this study include the fact it was carried out in a single major city in south Texas and included only Mexican-origin Hispanics. Therefore, the findings may not be generalizable to other Hispanic subgroups or to Hispanics living in other areas of the U.S. It should also be noted that the proportion of participants for whom vital status could not be ascertained increased from 1.6% at Follow-up 1 to 6.3% and 8.3% at Follow-up 2 and 3,

respectively. However, there was no significant ethnic difference in vital status ascertainment at any of these time points, suggesting that ascertainment bias was not a major factor influencing our results.

In summary, contrary to the Hispanic Paradox, ethnic differences in mortality in SALSA were primarily explained by differences in SES and disease factors in MAs relative to EAs. Significant predictors of mortality regardless of ethnic group were diabetes with complications, cognitive impairment, and depression. Given the increasing rates of diabetes in older Hispanics in particular as well as the obesity epidemic in the U.S.,⁵⁰ increasing attention should be paid to diabetes prevention and prevention of diabetic complications in order to promote healthy lifespan in older adults.

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Table 1

Characteristics of Participants at Baseline by Ethnicity

	Mexican Americans n=394	European Americans n=355	Total n=749	P-value for Ethnic Difference
	n(%) or mean(SD) ^a	n(%) or mean(SD) ^a	n(%) or mean(SD) ^a	
Age , years (range: 65–80)	69.3 (3.2)	70.2 (3.6)	69.8 (3.4)	<.001
Female	227 (57.6)	205 (57.7)	432 (57.7)	.97
Education , years (range: 0–23)	8.9 (4.6)	13.4 (2.6)	11 (4.4)	<.001
Income , category ^b (range: 1–15)	10.4 (3.2)	12.8 (2.3)	11.6 (3)	<.001
Married or living as married	252 (65.6)	243 (69.0)	495 (67.3)	.33
Hypertension	193 (49)	185 (52.1)	378 (50.5)	.39
Myocardial Infarction	55 (14.0)	58 (16.4)	113 (15.2)	.36
Angina	33 (8.5)	23 (6.6)	56 (7.6)	.32
Stroke	42 (10.8)	25 (7.1)	67 (9.0)	.08
Arthritis	173 (44.3)	170 (48.7)	343 (46.4)	.22
Cancer (non-skin)	24 (6.1)	50 (14.2)	74 (10.0)	<.001
Comorbidity , ^c excluding diabetes	202 (52.5)	197 (56.4)	399 (54.4)	.28
Diabetes	122 (33.5)	35 (11.9)	157 (23.9)	<.001
Diabetes with complications ^d	68 (19.8)	23 (7.9)	91 (14.3)	<.001
Chronic Obstructive				
Pulmonary Disease	120 (32.0)	128 (37.2)	248 (34.5)	.14
Proteinuria	49 (14.9)	12 (4.2)	61 (9.9)	<.001
Peripheral arterial disease	91 (24.0)	74 (21.3)	165 (22.7)	.38
Geriatric Depression Scale	6.5 (5.5)	5.1 (4.4)	5.9 (5.1)	<.001
Mini-mental State Exam	24.8 (4.0)	27.7 (2.4)	26.2 (3.6)	<.001
Body Mass Index, kg/m²	29.3 (5.2)	27.4 (5.2)	28.4 (5.3)	<.001
Frailty status				.04
Non-frail	329 (88.7)	320 (93)	649 (90.8)	
Frail	42 (11.3)	24 (7)	66 (9.2)	

^a Abbreviations: SD = standard deviation, kg/m² = kilograms per meters squared^b Monthly household income categories: 1=\$0–49, 2=\$50–99, 3=\$100–149, 4=\$150–199, 5=\$200–299, 6=\$300–399, 7=\$400–499, 8=\$500–749, 9=\$750–999, 10=\$1,000–1,249, 11=\$1,250–1,499, 12=\$1,500–1,999, 13=\$2,000–2,499, 14=\$2,500–2,999, 15=\$3,000+.^c Comorbidity was defined as the presence of 2 or more of the following chronic diseases: hypertension, myocardial infarction, angina, stroke, arthritis, cancer (non-skin), and chronic obstructive pulmonary disease.^d Diabetes with complications was defined as the presence of diabetes and one or more of: angina, myocardial infarction, stroke, peripheral arterial disease, or proteinuria.

Table 2

Vital Status of Participants at Each Follow-Up by Gender within Each Ethnic Group

	Mexican Americans ^a				European Americans ^a			
	Male n (%)	Female n (%)	Total n (%)	P-value for Sex Difference	Male n (%)	Female n (%)	Total n (%)	P-value for Sex Difference
Follow-up 1								
Alive	112 (67.1)	184 (81.1)	296 (75.1)	<.001	113 (75.3)	178 (86.8)	291 (82.0)	.014
Dead	54 (32.3)	35 (15.4)	89 (22.6)		36 (24.0)	25 (12.2)	61 (17.2)	
Censored ^b	1 (0.6)	8 (3.5)	9 (2.3)		1 (0.7)	2 (1.0)	3 (0.8)	
Total n	167	227	394		150	205	355	
Follow-up 2								
Alive	88 (52.7)	166 (73.1)	254 (64.5)	<.001	106(70.7)	155 (75.6)	261 (73.5)	.092
Dead	70 (41.9)	44 (19.4)	114 (28.9)		36 (24.0)	25 (12.2)	61 (17.2)	
Censored	9 (5.4)	17 (7.5)	26 (6.6)		6 (4.0)	15 (7.3)	21 (5.9)	
Total n	167	227	394		150	205	355	
Follow-up 3								
Alive	79 (47.3)	150 (66.1)	229 (58.1)	<.001	94 (62.7)	142 (69.3)	236 (66.5)	.049
Dead	76 (45.5)	54 (23.8)	130 (33.0)		48 (32.0)	44 (21.5)	92 (25.9)	
Censored	12 (7.2)	23 (10.1)	35 (8.9)		8 (5.3)	19 (9.3)	27 (7.6)	
Total n	167	227	394		150	205	355	

^a P-value for ethnic differences between Mexican Americans and European Americans were .044 at Follow-up 1, .022 at Follow-up 2, and .06 at Follow-up 3.

^b Censored refers to individuals who were lost to follow-up, i.e., vital status was unknown.

Table 3
Hazard Ratios^a for Mortality by Ethnic Group from Sequential Cox Proportional Hazards Models

	Model 1		Model 2		Model 3 ^b		Model 4 ^c		Model 5 ^d	
	HR (95% CI)	P-value	HR (95% CI)	P-value	HR (95% CI)	P-value	HR (95% CI)	P-value	HR (95% CI)	P-value
Ethnicity (MAs vs. EAs)	1.54 (1.17–2.03)	.002	1.16 (0.83–1.61)	.39	1.09 (0.77–1.54)	.63	1.13 (0.77–1.66)	0.52	1.06 (0.71–1.59)	.77
Age (1-year increment)	1.12 (1.08–1.16)	<.001	1.12 (1.07–1.16)	<.001	1.10 (1.05–1.14)	<.001	1.11 (1.06–1.16)	<.001	1.09 (1.04–1.14)	<.001
Sex (male vs. female)	2.01 (1.54–2.62)	<.001	2.38 (1.80–3.15)	<.001	2.63 (1.95–3.55)	<.001	2.55 (1.84–3.54)	<.001	2.64 (1.87–3.72)	<.001
Education 1-category increment)			0.99 (0.95–1.03)	.48	0.99 (0.96–1.04)	.89	1.03 (0.98–1.08)	.28	1.06 (1.001–1.113)	.046
Income 1-category increment)			0.92 (0.87–0.97)	.002	0.93 (0.88–0.98)	.012	0.93 (0.87–0.99)	.018	0.94 (0.88–1.007)	.078
Frailty frail vs. non-frail)					2.13 (1.45–3.14)	<.001	1.78 (1.16–2.73)	.008	1.44 (0.91–2.28)	.12
Diabetes							0.77 (0.37–1.58)	.47	0.75 (0.35–1.63)	.47
Diabetes (with complications) ^e							2.92 (1.36–6.25)	.006	2.71 (1.20–6.12)	.017
Comorbidity (not including diabetes) ^f									1.38 (0.99–1.92)	.056
Mini-mental Status Exam (1-point increment)									0.94 (0.88–0.995)	.033
Geriatric Depression Scale (1-point increment)									1.04 (1.004–1.069)	.026
Body Mass Index									1.002 (0.97–1.03)	0.91

^a Abbreviations: HR = hazards ratios, CI = confidence interval, MA = Mexican Americans, EA = European Americans

^b N reduced to 715 due to available data to classify frailty status.

^c N reduced to 619 due to available data to classify diabetes status.

^d N reduced to 600 due to available data for mini-mental state examination, geriatric depression scale, or body mass index.

^e Diabetes with complications was defined as the presence of diabetes and one or more of: angina, myocardial infarction, stroke, peripheral arterial disease, or proteinuria.

^f Comorbidity was defined as the presence of 2 or more of the following chronic diseases: hypertension, myocardial infarction, angina, stroke, arthritis, cancer (non-skin), and chronic obstructive pulmonary disease.