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Depressive symptoms and Cardiovascular Mortality in Older African-American and White Adults: Evidence for a Differential Association by Race

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Abstract

Background—An emerging body of research suggests that depressive symptoms may confer an “accelerated risk” for cardiovascular disease (CVD) in African-Americans, compared with whites. Research in this area has been limited to cardiovascular risk factors and early markers; less is known about black-white differences in associations with important clinical endpoints.

Methods—The authors examined the association between depressive symptoms and overall CVD mortality, ischemic heart disease (IHD) mortality, and stroke mortality in a sample of 6,158 (62% African-American; 61% female) community-dwelling older adults. Cox proportional hazards models were used to model time-to-CVD, IHD and stroke death over follow-up.

Results—In race-stratified models adjusted for age and sex, elevated depressive symptoms were associated with CVD mortality over follow-up in African-Americans (HR=1.95, 95% CI=1.61-2.36, $p<.001$), but were not significantly associated with CVD mortality in whites (HR=1.26, 95% CI=.95-1.68, $p=.11$; race by depressive symptoms interaction $p=.03$). Similar findings were observed for IHD mortality (African-American HR=1.99, 95% CI=1.49-2.64, $p<.001$; white HR=1.28, 95% CI=.86-1.89, $p=.23$); and stroke mortality (African-American HR=2.08, 95% CI=1.32-3.27, $p=.002$; white HR=1.32, 95% CI=.69-2.52, $p=.40$). Findings for total CVD mortality and IHD mortality were attenuated, but remained significant after adjusting for standard risk factors. Findings for stroke were reduced to marginal significance.

Conclusions—Elevated depressive symptoms were associated with multiple indicators of CVD mortality in older African-Americans, but not whites. Findings were not completely explained by standard risk factors. Efforts aimed at reducing depressive symptoms in African-Americans may ultimately prove beneficial for their cardiovascular health.

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Keywords

Depressive Symptoms; cardiovascular disease (CVD); race; psychosocial risk factors; epidemiology

Background

Depression and elevated depressive symptomatology have been linked to increased cardiovascular disease morbidity (CVD) and mortality across a wide variety of studies.¹⁻⁵ An emerging body of research suggests that the impact of depressive symptoms on CVD may differ by race, with depressive symptoms conferring an “accelerated risk” for CVD in African-Americans, compared with whites.⁶⁻⁸ African Americans frequently report more severe and untreated major depressive disorder and a higher prevalence of depressive symptomatology than their white counterparts.⁹⁻¹² Compared with whites, African Americans also suffer from disproportionately high rates of CVD morbidity and mortality.¹³

Until recently, however, few studies have examined black-white differences in the association between depressive symptoms and indices of CVD. In a recent analysis of middle-aged women, Lewis et al.,⁶ found that depressive symptoms were associated with early atherosclerotic disease in African-Americans, but did not observe a similar association in whites. A few additional studies of both men and women have observed a similar pattern of results, with stronger associations between depressive symptoms and cardiovascular risk factors found in African-Americans, compared with whites.^{7, 10, 14-16} To date, findings in this area have primarily been limited to early atherosclerotic disease, hypertension, and diabetes.

Despite the potential relevance of depressive symptoms as a risk factor for accelerated CVD in African-American populations, very little is known about black-white differences in the association between depressive symptoms and important clinical endpoints, such as CVD mortality. To our knowledge, only one study has explicitly examined racial differences in the association between depressive symptoms and any form of CVD mortality, and the primary outcome in that study was stroke.⁸ We are aware of no studies that have focused on overall CVD mortality or mortality due to ischemic heart disease (IHD), the single leading cause of death¹ in the United States^{17, 18}.

The primary aim of the current study was to examine associations among race, elevated depressive symptoms and CVD mortality over a 12-year follow-up in a population-based cohort of community-dwelling older African-American and white adults. We were particularly interested in determining whether the association between depressive symptoms and CVD mortality would be more pronounced in older African-Americans, compared with whites. We examined IHD and stroke as separate subtypes of overall CVD mortality, in order to determine whether associations were consistent across outcomes.

Methods

Study Population

Data are from the Chicago Health and Aging Project (CHAP), an ongoing, population-based longitudinal study of risk factors for Alzheimer's disease and other common conditions of aging. Details of the CHAP study design and procedures have been previously reported¹⁹. Briefly, CHAP was designed to include a large number of older African-American and non-

¹Based on age-adjusted rates, compared to deaths from all causes.

Hispanic white adults living in three adjacent areas on the south side of Chicago, IL. These areas were chosen because they included an approximately equal number of African-Americans and whites, with a broad representation of socioeconomic status (SES) within both racial groups. The CHAP study began in 1993 with a complete census of all households in these areas. All residents aged 65 and older were asked to participate. Of the 7,813 eligible residents, 6,158 enrolled, for an overall participation rate of 78.9% (African-Americans, 81.4%; whites, 75.1%). Between late 1993 and 1996, all participants were interviewed in person in their home. The baseline interviews included questions on sociodemographic characteristics, psychosocial variables, medical history and current health status. CHAP participants are interviewed every three years, and the cohort is currently in its fifth wave of data collection (over 12 years of follow-up). The current analyses are limited to those 5990 participants with complete data on depressive symptoms and information on vital status through 12/31/2005.

Assessment of Depressive Symptoms

Depressive symptoms were assessed at baseline in CHAP with the 10-item version of the Center of Epidemiologic Studies-Depression (CES-D) scale.²⁰ This scale asks respondents to endorse whether or not they have felt various symptoms of depression within the past week. It has been widely used in both clinical and community samples of young and middle-aged adults.²¹⁻²³ The shorter 10-item CES-D was developed to reduce participant burden in older adults, and its correspondence to the original version has been established.²⁴ The 10-item version has good internal consistency and test-retest reliability in older adult samples.²⁵ Coefficient alpha for this scale in CHAP is 0.75. Scores of 4 or greater on this scale are considered to be indicative of major depression.²⁵ Because we were particularly interested in clinically relevant depressive symptoms, the CES-D was modeled as a dichotomous “high” (4 or greater) or “low” (below 4) variable in all analyses.

Assessment of CVD Mortality

Information on vital status was obtained at each follow-up interview and ascertainment of mortality, obtained from the National Death Index, newspaper obituaries and informants, was complete through 12/31/2005. Causes of death prior to 1999 were classified using the *International Classification of Diseases, Ninth Revision (ICD-9)*; while deaths occurring after 1999 were classified using the *International Statistical Classification of Diseases, 10th Revision (ICD-10)*. Total CVD mortality was defined as ICD-9 codes 390-459 or ICD-10 codes I00-I99.9. IHD mortality was defined as ICD-9 codes 410-414 and ICD-10 codes 120-125, and stroke mortality was defined as ICD-9 codes 430-438 and ICD-10 codes 160-169.

Additional Variables

Race was self-reported as (non-Hispanic) African-American or white and sex was self-reported as male or female. Age was assessed via self-reported date of birth. Education was measured as years of schooling completed. Anti-depressant use was determined by visual inspection of medications taken and coded as a presence/absence variable. Two consecutive readings of systolic blood pressure (SBP) were taken with 30 seconds between measurements, using standard sphygmomanometers on seated subjects with the arm resting at heart level.²⁶ The average of the two SBP measurements was recorded. Height and weight were measured using standardized methods appropriate for an elderly population.²⁷ Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared, and categorized as normal, underweight, overweight, or obese, using standard criteria²⁸. Physical activity and smoking status were assessed via self-report using a series of questions from the Established Populations for Epidemiologic Studies of the Elderly (EPESSE) project.^{24, 29} Cigarette smoking was further categorized as never, current or ever.

Chronic conditions in the form of heart disease, stroke, hypertension, diabetes, cancer and/or hip fracture were self-reported using standardized questions from EPESE.²⁴ The number of chronic conditions was used as an indicator of overall physical health status. Cardiovascular and lipid-lowering medication use was determined by visual inspection of all medications taken. Because beta-blocker use has been associated with reduced CVD in animal models of “psychosocial stress” and CVD,^{30, 31} we created a separate medication variable to indicate beta blocker use. Hence, there were three CVD medication use indicator variables: 1) Lipid-lowering medication use, 2) CVD medication use with beta-blockers and 3) CVD medication use without beta-blockers.

Data Analysis

Descriptive statistics were calculated for baseline sociodemographic characteristics, depressive symptoms, anti-depressant use, and health status variables. T-tests and Chi-square tests were used to examine black-white differences in these variables. Cox proportional hazards models were conducted to model time-to-CVD death as a function of depressive symptoms and other covariates. All models examining the association between depressive symptoms and indices of CVD mortality were run separately by race. This was done for two reasons: 1) in preliminary models, we observed a significant race by depressive symptoms interaction for total CVD mortality. Because the main effect of depressive symptoms in race-specific models is often easier to interpret than the interaction coefficient in the pooled models, we chose to stratify models by race; also 2) Race-specific models allow us to examine the potential confound due to other CVD risk factors in the association between depressive symptoms and CVD mortality separately for African-Americans and whites. In addition to variation in the association between depressive symptoms and CVD mortality, there may also be racial differences in the pathways through which depressive symptoms impact CVD mortality. Race-specific models allow us to examine potential confounds *within* each racial group.³²

Following confirmation that assumptions of the proportional hazards model were met, the first set of Cox models were run to examine the association between depressive symptoms and total CVD mortality for African-Americans and whites separately, after adjusting for age and sex. We then added additional adjustments for education and potential confounds of the association between depressive symptoms and total CVD mortality, such as SBP, smoking status, BMI, physical activity, chronic health conditions, lipid-lowering medication use, and CVD medication use. Following these initial models, a second and third series of Cox proportional hazards models were conducted using the exact same sequence, with IHD mortality and stroke mortality as outcomes. Each of our race-stratified Cox models was followed by a formal test of the race by depressive symptoms interaction in models with both African-Americans and whites.

We did not include a term for anti-depressant use in our fully-adjusted models because the prevalence of anti-depressant use was particularly low in the CHAP cohort (2%), and including a term for this variable resulted in unstable estimates in some of our multivariate models. In order to gauge the impact of anti-depressant use on our results, we ran models including and excluding participants on anti-depressants. Results with and without these individuals were comparable; thus all participants were retained in final models.

Because depressive symptoms are known to differ by sex, we also ran our models stratified by race-gender group. This was done for CVD mortality only because of the relatively small number of events for IHD and stroke within each race/sex group. Findings within each racial group did not differ for men and women, thus only race-stratified models are presented. All analyses above were conducted using SAS (SAS Institute Inc, Cary, NC).

Results

Participant characteristics

Respondent characteristics at baseline for the full sample and by race are presented in Table 1. On average, participants were 75 years old (standard deviation (SD) = 7.0) with 11.8 years (SD = 3.7) of education. Participants had an average systolic blood pressure of 139.4 (SD=20.3), an average BMI of 26.7 (SD=5.5), and approximately 15% were current smokers. Approximately 17% of participants had high depressive symptoms, with a CES-D score ≥ 4 . Only 2% of participants were taking anti-depressants. Compared with whites, African-Americans were slightly younger and less educated. African-Americans also had higher BMIs, were more likely to be current smokers, and had lower levels of physical activity and more chronic health conditions than their white counterparts. African-Americans had higher levels of depressive symptomatology compared with whites, but were less likely to be on anti-depressant medication.

Over the 12-year follow-up, there were 925 deaths from CVD overall, including 449 deaths from IHD and 163 deaths from stroke. African-Americans had 523 total CVD deaths, 236 deaths due to IHD and 90 deaths from stroke. Numbers for whites were 402, 213, and 73, respectively.

Depressive Symptoms and Cardiovascular Mortality

Table 2 presents the age and sex-adjusted hazard ratios (HRs) and 95% confidence intervals (CIs) for each outcome separately by race. Among African-Americans, elevated depressive symptoms were significantly associated with CVD mortality, IHD mortality and stroke mortality, such that the rate of CVD, IHD and stroke death among African-Americans with high depressive symptoms was almost twice that of African-Americans with low depressive symptoms. There were no significant associations among high depressive symptoms and any type of cardiovascular mortality in whites, although the HRs were all greater than 1 (Table 2). A formal test of the race by depressive symptoms interaction in models with both African-Americans and whites adjusted for age, race, sex and depressive symptoms revealed a significant race by depressive symptoms interaction for total CVD mortality (HR=1.44, 95% CI=1.03-2.02, $p=.03$), a marginally significant interaction for IHD mortality (HR=1.52, 95% CI=.94-2.45, $p=.09$), and no significant interaction for stroke mortality (HR=1.40, 95% CI=.64-3.05, $p=.40$).

Findings were somewhat attenuated, but remained significant, after additional adjustments for education, SBP, smoking status, BMI, physical activity, CVD medication use, lipid-lowering drug use and chronic health conditions. As shown in Table 3, the HR for high depressive symptoms and overall CVD mortality remained significant for African-Americans (HR=1.66, 95% CI=1.33-2.07, $p<.001$), and non-significant for whites (HR=1.15, 95% CI=.84-1.56, $p=.38$) in fully-adjusted models. HRs for several of the other covariates in the fully-adjusted models (e.g. SBP, chronic conditions) were comparable for African-Americans and whites. However, there were a few notable differences. The HR for smoking was 1.20 and nonsignificant in the model for African-Americans; but was 1.75 and significant in the model for whites (Table 3). Similarly, the HR for CVD medications (without beta-blockers) was lower in African-Americans compared to whites (.93 compared to 1.53) and was non-significant in models for African-Americans and significant for whites. Obesity was protective in both groups, although only significantly so for African-Americans. HRs for age and male sex were slightly lower in African-Americans compared to whites, although age and male sex were significantly associated with CVD mortality in both racial groups. Despite these differences, the overall patterning of results for high

depressive symptoms and CVD mortality by race was similar in minimally and fully-adjusted models.

In fully-adjusted models examining the association between depressive symptoms and CVD mortality in both African-Americans and whites, after adjusting for age, race, sex, depressive symptoms and covariates in Table 3, the race by depressive symptoms interaction was slightly reduced to marginal significance (HR= 1.41 (95% CI =.97 - 2.04, p=.07), indicating that CVD risk factors may partially, but not fully, explain racial differences in the association between depressive symptoms and CVD mortality.

In fully-adjusted models with IHD as the outcome, high depressive symptoms remained significantly associated with IHD mortality in models for African-Americans (HR=1.62, 95% CI=1.15-2.27, p= .006), but not whites (HR=1.18, 95% CI=.77-1.80, p=.45) (full models not shown). The association between elevated depressive symptoms and stroke mortality remained marginally significant in fully-adjusted models for African-Americans (HR=1.65, 95% CI=.97-2.79, p= .06) and non-significant in whites (HR=1.22, 95% CI=.61-2.43, p=.57) (full models not shown). In fully-adjusted models the race by depressive symptoms interaction remained nonsignificant for both IHD (p=.18) and stroke (p=.56).

Exploratory Analyses: Competing Risk Models

We re-ran our final, fully-adjusted models examining the association between depressive symptoms and CVD mortality over follow-up, treating deaths due to other causes as “competing risks”.³³ This was done because of the age of our sample – given the relatively high mortality rate in elderly populations we wanted to account for the possibility of death due to causes other than CVD. The HR for high depressive symptoms and CVD mortality for African-Americans was slightly lower in the competing risks model (HR=1.57, 95% CI=1.26-1.96, p< .001), albeit significant, and associations between high depressive symptoms and CVD mortality remained non-significant for whites (HR= 1.11, 95% CI=.80-1.53, p=.53). These analyses were repeated for IHD and stroke mortality, respectively. Similar to results for CVD mortality, for African-Americans the HRs were slightly reduced for associations between high depressive symptoms and IHD (HR=1.48, 95% CI=1.06-2.07, p=.02) and stroke (HR=1.44, 95% CI=.84-2.46, p=.18) mortality, although associations were only significant for IHD mortality (perhaps due to the small number of stroke deaths). Again, there were no significant associations observed between high depressive symptoms and IHD (p=.57) or stroke (p=.67) mortality in whites. Thus, the overall pattern of results remained the same.

Discussion

The current study was designed to examine the associations among race, elevated depressive symptoms and CVD mortality over a 12-year follow-up in a sample of older African-American and white adults. Although we only observed a significant race by depressive symptoms interaction for total CVD mortality, in age and sex-adjusted models stratified by race, we found that high levels of depressive symptoms were significantly associated with total CVD mortality, IHD mortality and stroke mortality among African-Americans only. Among older African-Americans, those with high levels of depressive symptomatology were almost twice as likely to die from CVD-related causes than those with low levels of depressive symptomatology. These associations persisted after adjustment for demographic covariates and multiple CVD risk factors. We did not observe statistically significant associations between depressive symptoms and any indicator of CVD mortality in older whites.

Although non-significant, the HRs in whites were positive (i.e. greater than 1). This suggests that the direction of the association between depressive symptoms and indicators of CVD mortality may be somewhat similar in African-Americans and whites in our cohort. Differences appear to be primarily in the magnitude of the association, with depressive symptoms exerting a greater impact on indicators of CVD mortality in African-Americans compared with whites.

It is important to note that we did not observe *statistical* evidence of a race difference in the impact of depressive symptoms on IHD or stroke, as the race by depressive symptoms interactions for these outcomes were not significant. Because these are subcategories of overall CVD mortality, where we did see a significant race by depressive symptoms interaction, it is likely that the lack of a significant interaction for IHD and stroke is primarily due to insufficient power. Replication of these results in a larger sample may be warranted.

Several prior studies have documented significantly stronger associations between depressive symptoms and indices of CVD in African-Americans, compared to whites.^{10, 14-16} The current study provides further evidence in support of this patterning of results, and extends these findings to CVD mortality and mortality due to IHD. Despite the growing body of work in this area, however, we know very little about the factors underlying these associations.

It is possible that the pathways through which depressive symptoms impacted indicators of CVD mortality differed for African-Americans compared to whites. We did not formally test the interactive effects of race and depressive symptoms on each possible confound (e.g. BMI, SBP, smoking, chronic conditions), however, we did obtain HRs for these potential confounds in race-specific models. Although there were some differences in HR for potential confounds in race-specific models for African-Americans and whites – particularly for smoking and one type of CVD medication use -- these differences did not change the overall patterning of our results. This suggests that standard CVD risk factors may not completely explain our observed associations. However, other factors may play a role.

Increased inflammation,^{34, 35} platelet aggregation,³⁶ and the hypersecretion of cortisol^{37, 38} have all been identified as possible mechanisms through which depressive symptoms influence CVD. However, to date, there has been limited research on whether the association between depressive symptoms and these more novel indicators of CVD risk differ for African-Americans compared to whites. Recently, Boyle et al.¹⁵ found significant black-white differences in the association between depressive symptoms and cortisol in a sample of African-American and white male veterans. Depressive symptoms were positively associated with cortisol in both racial groups; however, the magnitude of the effect was considerably stronger in African-Americans.¹⁵ Cortisol has also been associated with indices of CVD,^{39, 40} and could be one possible physiological explanation for black-white differences in the association between depressive symptoms and indicators of CVD mortality. Emerging evidence suggests that Depressive symptoms may also exert a stronger effect on C-Reactive Protein (CRP) in African-Americans versus whites.⁴¹ Additional research on physiologic pathways through which depressive symptoms might differentially impact CVD risk for African-Americans compared to whites is warranted.

Unmeasured psychosocial factors may also play a role. Older African-American adults in our cohort reported significantly higher levels of depressive symptomatology compared to whites, which may be due to higher exposure to lifetime and/or current psychosocial stress burden. A number of studies have found higher reports of lifetime negative life events, traumatic stressors, and chronic stressors in African-Americans compared to whites.⁴²⁻⁴⁴

These background stressors may limit the ability of older African-Americans to effectively cope with high levels of depressive symptoms, which could subsequently increase their vulnerability to CVD. Other psychosocial factors that may enhance vulnerability to CVD mortality in older African-Americans with elevated depressive symptomatology include dispositional traits such as low optimism and high pessimism –both of which have been linked to increased risk of overall (although not CVD) mortality in African-Americans relative to whites.⁴⁵ These background stressors and dispositional traits were not assessed in the CHAP cohort, but may be important to consider in future studies.

It is also possible that the stronger association between depressive symptoms and indicators of CVD mortality observed in older African-Americans compared to whites is simply a function of a longer lifetime exposure to depression and depressive symptomatology. We do not have data on history of depression/depressive symptoms for our cohort; however, research in younger adult samples suggests that African-Americans are more likely to have severe, chronic, and untreated depression than their white counterparts.¹² Thus, the higher level of depressive symptoms observed in our cohort of older African-Americans compared to whites may be reflective of prior symptoms that have persisted into older age. In this respect, levels of depressive symptomatology at baseline may be a proxy for length of exposure to depressive symptoms over the lifecourse.

The overall prevalence of anti-depressant use was fairly low in this sample, at 2.1%. This is only slightly lower than the rate of 3.5% reported in the Cardiovascular Health Study,⁴⁶ a similarly aged cohort. However, African-Americans in our cohort had an even lower prevalence of anti-depressant use than whites, despite their higher levels of elevated depressive symptomatology. This is consistent with national data where there are significant black-white disparities in anti-depressant use, even at older ages.⁴⁷ However, our findings suggest that the overall burden of untreated depressive symptoms may be greater in older African-Americans compared to whites.

This study has limitations that should be noted. First, although widely used in epidemiologic cohort studies,^{10, 34, 46} the CES-D is not designed to assess clinical depression. Our findings may have been stronger with the inclusion of actual DSM-IV diagnosed depression.⁴ Second, we relied on self-reported measures of chronic conditions (heart disease, stroke, diabetes), which tend to be less valid than actual clinical diagnoses. Finally, our findings are based on a sample of African-Americans and whites living in an urban context in the Midwest, and may not be generalizable to populations in other parts of the country.

Nevertheless, our study has several strengths. To our knowledge, this study is the first to examine the associations among race, depressive symptoms, and total CVD mortality or mortality due to IHD. Our cohort is fairly unique, in that it features a large number of African-Americans and whites who are living in a similar environment (i.e. three adjacent neighborhoods). It is also population-based, with a high participation rate in both racial groups.

In summary, we found a significant, independent association between depressive symptoms and total CVD mortality, and more specifically, IHD and stroke mortality in older African-Americans, even after controlling for a number of potential explanatory variables. This association was positive, but non-significant in whites, providing strong support for the notion that African-Americans may be particularly vulnerable to the effects of depressive symptoms on CVD. The mechanisms underlying these associations remain to be determined. Nonetheless, future research should determine whether reducing depressive symptomatology in African-Americans could ultimately prove beneficial for their cardiovascular health.

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Table 1
Baseline Participant characteristics for the total sample and by Race

| Variable | Total (N=5990) | African-American (N=3691) | Whites (N=2299) | P-value |
|------------------------------|-------------------|------------------------------|--------------------|---------|
| Age | 74.79 ± 7.03 | 73.70 ± 6.65 | 76.53 ± 7.27 | <.001 |
| Female, % | 60.62 | 59.63 | 62.20 | 0.048 |
| Education | 11.78 ± 3.70 | 10.72 ± 3.49 | 13.49 ± 3.39 | <.001 |
| Depressive symptoms ≥4, % | 16.54 | 19.89 | 11.18 | <.001 |
| Antidepressant Use, % | 2.1 | 1.37 | 3.26 | <.001 |
| Systolic Blood pressure | 139.41 ± 20.31 | 139.27 ± 20.28 | 139.64 ± 20.35 | 0.49 |
| BMI | 26.70 ± 5.51 | 27.39 ± 5.80 | 25.64 ± 4.83 | <.001 |
| BMI 4 categories, % | | | | <.001 |
| BMI<18.5 | 3.94 | 3.69 | 4.33 | |
| 18.5≤BMI<25 | 37.01 | 32.43 | 44.06 | |
| 25≤BMI<30 | 36.02 | 36.24 | 35.69 | |
| BMI≥30 | 23.03 | 27.65 | 15.92 | |
| Smoking status, % | | | | <.001 |
| Never smoker | 46.46 | 44.36 | 49.83 | |
| Ever smoker | 38.79 | 38.56 | 39.16 | |
| Current smoker | 14.75 | 17.07 | 11.01 | |
| Physical activity | 3.28 ± 5.48 | 2.99 ± 5.50 | 3.76 ± 5.43 | <.001 |
| Chronic conditions | .99 ± .92 | 1.03 ± .95 | .91 ± .88 | <.001 |
| Lipid-Lowering Drug Use | 5.41 | 4.80 | 6.39 | .008 |
| CVD meds (Beta-Blockers) | 12.45 | 9.81 | 16.7 | <.001 |
| CVD meds (w/o Beta-Blockers) | 46.61 | 49.66 | 39.10 | <.001 |

* Note: Values are mean ± SD or percentage

Table 2
Age and sex-adjusted Hazard Ratios (95% Confidence Intervals) for associations between High Depressive Symptoms and Types of Cardiovascular Mortality for African-Americans and Whites

| | African-Americans | | | Whites | | |
|------------------------------|-------------------|-----------|---------|--------|----------|---------|
| | HR | 95% CI | P-value | HR | 95% CI | P-value |
| Total Cardiovascular Disease | 1.95 | 1.61-2.36 | <.001 | 1.26 | .95-1.68 | .11 |
| Ischemic Heart Disease | 1.99 | 1.49-2.64 | <.001 | 1.28 | .86-1.89 | .23 |
| Stroke | 2.08 | 1.32-3.27 | .002 | 1.32 | .69-2.52 | .40 |

* P-value for race by depressive symptoms interaction is .03 for CVD mortality, p=.09 for IHD mortality and p=.40 for stroke mortality.

Table 3
Fully-adjusted Models of the association between Elevated Depressive Symptoms and Cardiovascular Disease Mortality for African-Americans and Whites

| | African-Americans | | | Whites | | |
|------------------------------|-------------------|-----------|---------|--------|-----------|---------|
| | HR | 95% CI | P-value | HR | 95% CI | P-value |
| Depressive symptoms ≥ 4 | 1.66 | 1.33-2.07 | <.001 | 1.15 | .84-1.56 | .38 |
| Age | 1.07 | 1.05-1.09 | <.001 | 1.12 | 1.11-1.14 | <.001 |
| Male | 1.59 | 1.28-1.96 | <.001 | 1.96 | 1.55-2.48 | <.001 |
| Education | .96 | .94-.99 | .01 | .98 | .95-1.02 | .28 |
| Systolic BP | 1.00 | 1.00-1.01 | .05 | 1.00 | 1.00-1.01 | .85 |
| BMI (18.5-25; normal) | | | | | | |
| <18.5, underweight) | 1.43 | .93-2.22 | .11 | 1.43 | .88-2.32 | .15 |
| (25 \leq <30, overweight) | .67 | .53-.84 | .0005 | .73 | .57-.94 | .01 |
| (≥ 30 , obese) | .55 | .42-.72 | <.001 | .93 | .67-1.30 | .68 |
| Physical Activity | .97 | .95-.99 | .02 | .96 | .94-.99 | .004 |
| Smoking Status (never) | | | | | | |
| Past | 1.16 | .93-1.45 | .18 | 1.11 | .87-1.40 | .40 |
| Current | 1.20 | .90-1.61 | .21 | 1.75 | 1.21-2.52 | .003 |
| Chronic Conditions | 1.47 | 1.33-1.64 | <.001 | 1.41 | 1.24-1.60 | <.001 |
| Lipid-Lowering Drug Use | .81 | .50-1.30 | .38 | .91 | .58-1.44 | .68 |
| CVD meds (Beta-Blockers) | 1.05 | .75-1.47 | .79 | 1.29 | .91-1.83 | .14 |
| CVD meds (w/o Beta-Blockers) | .93 | .73-1.18 | .55 | 1.53 | 1.16-2.02 | .003 |

* Note: P-value for race by depressive symptoms interaction =.07.