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Does the Association Between Depressive Symptoms and Cardiovascular Mortality Risk Vary By Race? Evidence from the Health and Retirement Study

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Abstract

Objective—To test whether the association between depressive symptoms and cardiovascular disease (CVD) mortality is stronger among blacks than whites. **Design, Setting and Participants:** 2,638 black and 15,132 white participants from a prospective, observational study of community-dwelling Health and Retirement Study participants (a nationally representative sample of U.S. adults age 50+). Average follow-up was 9.2 years. **Outcome Measure:** Cause of death (per ICD codes) and month of death were identified from National Death Index linkages.

Methods—The associations between elevated depressive symptoms and mortality from stroke, ischemic heart disease (IHD), or total CVD were assessed using Cox proportional hazards models to estimate adjusted hazard ratios (HRs). We used interaction terms for race by depressive symptoms to assess effect modification (multiplicative scale).

Results—For both whites and blacks, depressive symptoms were associated with a significantly elevated hazard of total CVD mortality (whites: HR=1.46; 95% CI: 1.33, 1.61; blacks: HR=1.42, 95% CI: 1.10, 1.83). Adjusting for health and socioeconomic covariates, whites with elevated depressive symptoms had a 13% excess hazard of CVD mortality (HR=1.13, 95% CI: 1.03, 1.25) compared to whites without elevated depressive symptoms. The HR in blacks was similar, although the confidence interval included the null (HR=1.12, 95% CI: 0.86, 1.46). The hazard

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associated with elevated depressive symptoms did not differ significantly by race (p>0.15 for all comparisons). Patterns were similar in analyses restricted to respondents age 65+.

Conclusions—Clinicians should consider the depressive state of either black or white patients as a potential CVD mortality risk factor.

Keywords

depression; stroke; cardiovascular disease; mortality; race

Introduction

Racial disparities in cardiovascular disease among U.S. adults are well established: blacks have a higher risk of both cardiovascular disease (CVD) incidence and mortality than whites.[1] The association between psychosocial factors such as depression and cardiovascular disease is also well documented.[2-5] For example, meta-analyses suggest depression is associated with a 69% elevated relative risk of coronary heart disease (CHD) mortality[6], a 55% elevated relative risk of fatal CVD,[3] and a between a 35%[7] and 50% elevated odds of fatal stroke.[8] However, racial differences in the association between depression and CVD mortality are not well understood. Because racial differences in CVD attenuate with age, it is important to compare how race may modify the association between depressive symptoms and CVD mortality risk from middle to older age.

As Everson-Rose and Lewis suggested in their review of the literature on this issue, few prior studies have sufficient representation of blacks to adequately examine this question.[9] The few available studies on racial differences in the association between depression and CVD report mixed results. A recent study by Lewis et al.[10] stands out because of the diverse participant sample providing an opportunity to address this question. They found that depressive symptoms were associated with elevated odds of CVD mortality for blacks but not whites. However, this finding contrasts with some prior work showing associations between depressive symptoms and stroke incidence in both blacks and whites.[11-13] These new results are also somewhat inconsistent with several earlier studies that reported associations between depressive symptoms and CVD mortality in predominantly white general population samples.[14,15]

Our study assessed differences in CVD mortality associated with elevated depressive symptoms using a large cohort of U.S. black and white adults aged 50+. We hypothesized that depressive symptoms would predict CVD mortality in blacks and whites, with no significant variation in the magnitude of association by race. We also assessed whether this association varied by age.

Methods

Study Population

The Health and Retirement Study (HRS) is a nationally-representative longitudinal survey of U.S. adults aged 50+ years and their spouses. Details of the study are provided elsewhere.[16,17] Enrollments occurred in 1992, 1993, 1998 (based on respondent's birth year) with assessments occurring approximately every two years. However, in order to have a consistent measure of depressive measures the following years were considered baselines: 1996, 1995, and 1998, respectively. The 2004 and subsequent HRS enrollees were not included in this analysis. We followed participants from baseline through the 2008 wave mortality assessment; retention rates through 2008 were above 80%. HRS was approved by the University of Michigan Health Sciences Human Subjects Committee and these analyses

were approved by the Harvard School of Public Health Office of Human Research Administration.

Of the full HRS sample enrolled in 1992, 1993 or 1998 (n=27,473), we excluded 8,536 who were age ineligibe or with a sampling weight of 0 (31.1%); 1,386 (5.1%) who were Hispanic; 1,284 (4.7%) missing exposure assessment; and 426 (1.6%) who were missing key covariate information to ensure complete case analysis; leaving 15,845 respondents for the analyses.

Assessment of Depressive Symptoms

We defined elevated depressive symptoms based on the baseline measure of a modified, 8-item version of the Centers for Epidemiologic Studies-Depression (CES-D) scale. Our outcome variable, hereafter called "elevated depressive symptoms," was a dichotomized indicator of whether the respondent reported experiencing 3 or more of the following symptoms in the last week: feeling depressed, feeling everything was an effort, restless sleep, feeling happy (reverse coded), feeling lonely, enjoying life (reverse coded), feeling sad, and could not get going. The 3+ cutpoint was based on prior psychometric validation studies.[18,19]

Assessment of Mortality

HRS respondents' deaths were confirmed via linkage to the National Death Index conducted in May, 2009. For deaths prior to 1999, cause of death was characterized using the International Classification of Diseases, Ninth Revision (ICD-9). Total CVD deaths included ICD codes 390 to 459; strokes included 430 to 438 and IHD included 410 to 414. For deaths after 1999, cause of death was characterized using the International Classification of Diseases, Tenth Revision (ICD-10). Total CVD deaths were classified as those with ICD-10 codes I00-99.9; strokes comprised I60-I69 and IHD comprised I20-I25.

Covariates

We considered groups of demographic covariates and health risk factors as potential confounders. All covariates were self-reported. We used the earliest reported values for each covariate. Demographics included race (black/white), age (continuous), and sex (male/female). Socioeconomic status was measured as years of education (0-17). The health risk factors included body mass index (BMI) as a categorical variable (<18.5; 18.5-24.9, reference; 25-29.9; ≥30); smoking status (current and ever, never as reference); physical activity, measured as an indicator variable of regular (1+ times per week) physical activity in the previous two weeks (yes/no); a summary score of self-reported history of a doctor's diagnosis of chronic conditions (high blood pressure, diabetes, cancer, lung disease, and arthritis; range 0-5), consistent with prior literature[10]; heart medication usage ("Are you now taking or carrying medication for your heart problem?", yes/no); and psychiatric medication usage ("Do you now take tranquilizers, antidepressants, or pills for nerves?", yes/no).

Methods of Statistical Analysis

The hazard of CVD mortality associated with elevated depressive symptoms was assessed using age- and sex-adjusted Cox proportional hazard models stratified by race. Two additional models included covariates to adjust for CVD risk factors: first, socioeconomic and health status variables (education, comorbidity summary score, body mass index) and then health behaviors (smoking, physical activity). These factors likely partially mediate the causal pathway, so the results may understate the full causal effect of disease. Person-time was calculated from the initial interview date until the date of their death. These estimates were calculated using PROC PHREG in SAS version 9.3 (SAS Institute, Inc., Cary, NC),

with the sampling weight from the first interview applied. We tested for effect modification on a multiplicative scale by examining the interaction between race and elevated depressive symptoms. Sensitivity analyses included the same models estimated in a sample restricted to respondents age 65+ at baseline (n=8,680). HRS has a complex survey design; however, based on prior work suggesting minimal design effects, we treated the data as a simple random sample in these analyses (design effect estimates available from authors).

Results

There were 13,444 white and 2,401 black respondents in this analysis. There was an average of 9.2 years of follow-up in the total sample and 8.4 among those age 65+ at enrollment. The mean age of our cohort was similar for blacks (65.3, SD=10.0) and whites (67.1, SD=10.3) (Table 1). Prevalence of elevated depressive symptoms was higher in blacks (27.6%) than whites (18.1%). Whites had more years of education and generally better heath than blacks: whites had lower BMI, lower prevalence of current smoking, higher prevalence of physical activity, and higher prevalence of heart medication usage than blacks. Blacks and whites had similar prevalence of chronic diseases and psychiatric medications (Table 1).

In the total sample, elevated depressive symptoms were associated with a significant elevation in hazard of total CVD mortality after adjusting for sex and age in both blacks (HR=1.42, 95% CI: 1.10, 1.83) and whites (HR=1.46, 95% CI: 1.33, 1.61) (Table 2). The estimated associations for IHD and stroke outcomes were similar between whites and blacks. The estimated association did not vary significantly by race for total CVD (p=0.76 for test of interaction), IHD (p=0.97) or stroke mortality (p=0.76).

Results were similar in analyses restricted to respondents age 65+ at baseline (Table 2, bottom panel). For example, restricting to those age 65+, elevated depressive symptoms were associated with a 37% excess hazard of total CVD mortality (HR=1.37, 95% CI: 1.02, 1.85) among blacks and 38% excess hazard among whites (HR=1.38, 95% CI: 1.25, 1.53). Among those aged 65+, the estimated associations between elevated depressive symptoms and IHD and stroke mortality were similar to those for the total sample aged 50+ for both blacks and whites, although the association for strokes among blacks had a wide confidence interval that crossed the null (HR=1.24, 95% CI: 0.54, 2.85). Like those aged 50+, the hazard of mortality associated with depressive symptoms did not vary significantly by race among those aged 65+ for total CVD (p=0.99), IHD (p=0.91) or stroke mortality (p=0.72).

After adjusting for demographics and potential CVD risk factors, the estimated association between depressive symptoms with total CVD mortality was attenuated: whites with depressive symptoms had a 23% elevation in hazard of CVD mortality (HR=1.23, 95% CI: 1.11, 1.35); and blacks with depressive symptoms had an 18% elevation (HR=1.18, 95% CI: 0.91, 1.54) with a confidence interval that included the null. The associations for other outcomes and among those 65+ were also attenuated; among blacks, adjustment for covariates led to wider confidence intervals that crossed the null (Table 2, Model 2).

Models were then adjusted for health behaviors (smoking and physical activity), which further attenuated the associations. For example, the estimated association between depressive symptoms and total CVD mortality was a 12% increased hazard of mortality for blacks (HR=1.12, 95% CI: 0.86, 1.46) and 13% increased hazard for whites (HR=1.13, 95% CI: 1.03, 1.25) (Table 2, Model 3). The association between depressive symptoms and CVD mortality did not vary significantly by race in this adjusted model (p=0.56 for test of interaction of total sample; for 65+, p=0.58). There were similar patterns of attenuation for the IHD and stroke; and for all outcomes among those aged 65+. Results were almost identical when controlling for use of psychiatric and heart medications (results not shown).

Models stratified by follow-up time suggested the associations attenuated very little with longer duration of follow-up; for example in pooled analyses the HR for years 0-8 was 1.55 (95% CI: 1.38-1.73), while for years 9-15, the association was 1.47 (95% CI: 1.16, 1.87).

Discussion

This study found that elevated depressive symptoms were associated with an increased hazard of CVD mortality among blacks and whites after statistically controlling for age and sex. The magnitude of the age- and sex-adjusted relative association between elevated depressive symptoms and CVD mortality was similar for blacks (total sample: HR=1.42, 65+: HR=1.37) and whites (total sample: HR=1.46, 65+: HR=1.38) and there was no significant difference by race in the hazard associated with higher depressive symptoms for total CVD mortality, stroke mortality, or IHD mortality. The age and sex-adjusted HR point estimates indicated blacks had similar magnitudes of association between depression and both stroke and IHD mortality compared to whites. None of the differences were statistically significant. Patterns differed little when considering the population age 50+ compared to age 65+.

Adjustment for additional covariates attenuated the associations for all outcomes and groups. The associations remained statistically significant in whites after adjustment for socioeconomic, health status, and health behavior variables. Although the point estimates in the models were similar for blacks and whites, the CIs for blacks were wider and included the null. However, the health covariates and health behaviors included in these models likely partially mediate the association between depression and CVD outcomes. Adjusting for such mediators likely underestimates the causal effect of depressive symptoms on CVD mortality.

Study Limitations and Strengths

Although using a brief symptom scale (CES-D) rather than diagnosis of clinical depression is a potential limitation of this study, our measure is very similar to that used by prior studies.[10] Furthermore, our cut-point for elevated depressive symptoms has also been shown to have high sensitivity and specificity for clinical depression.[19]

We controlled for particular health variables to facilitate comparisons with prior literature;[6,10] however, we recognize that without better knowledge of the temporal order of covariates these models may yield biased results because of mediation. Although controlling for psychiatric medication use did not change our findings, this result should be interpreted cautiously because the measure available in HRS was limited.

The wide confidence intervals of estimates for blacks reflect low sample size in this stratum; this is particularly true for IHD and stroke otucomes as well as in multivariable adjusted models. We may be underpowered to detect a result for this group. However, the interaction term test indicated no significant difference in effect sizes for blacks and whites. We examined interactions by race on a multiplicative rather than additive scale because the Cox model is naturally multiplicative. Our results support the use of a multiplicative model in that we found no evidence for deviation from multiplicativity in our interaction tests. However, because CVD mortality is generally higher among younger blacks than whites, the absolute excess event rate induced by depressive symptoms may be larger among blacks, even if the relative effects are the same.

This study has a number of important strengths. The cause of death was linked from the National Death Index. Secondly, we used identical analytic design, measures, and analyses to prior research in this area, which improves the comparability of our findings to the extant

literature. Lastly, we use a nationally-representative sample, enhancing the generalizability of findings.

Comparisons to prior literature

These results are consistent with prior literature that suggests depressive symptoms are associated with increased CVD-related mortality and incidence.[5,6,11,20-23] Furthermore, our results are of a similar magnitude to those found in prior studies.[24] Our estimates are lower than results from meta-analyses[3,6,7,25] of the association between depression and incident cardiovascular events; however, differences in the study designs, age of the population, control variables, and exposure and outcome variables may account for these differences. As van der Kooy and colleagues[3] note in their meta-analysis of this association among the elderly, depressive symptoms do not predict CVD onset as strongly as clinical diagnosis of depression.

We do not find differences in this association by race among either those aged 50+ or among the older age cohort, which contributes to a somewhat mixed literature on the racial differences in the association between depression and CVD. Some studies report no racial difference in the association between depression and stroke onset[11,13]. However, our results contrast with recent findings by Lewis et al[10] who found that the effects of depressive symptoms on cardiovascular mortality among those aged 65+ varied significantly by race in the Chicago Health and Aging Project (CHAP) cohort. For example, they found depressive symptoms were associated with a more modest and non-significant elevation in age- and sex-adjusted hazard of CVD mortality among whites in CHAP (HR=1.26; 95% CI: 0.95, 1.68) than we find in HRS (HR=1.38; 95% CI: 1.25, 1.53), but a higher elevation among blacks in CHAP (HR=1.95; 95% CI: 1.61, 2.36) than HRS (HR=1.37; 95% CI: 1.02, 1.85). The divergence in these hazard ratios may reflect distinct features of a geographically defined, urban cohort, such as shared life experiences or exposure to other contextual or environmental risk factors[26] that exacerbated the hazard of CVD mortality associated with depression that would not be present in a nationally representative sample. Consistent with this possibility, chronic life stress was associated with carotid intima-media thickness in blacks but not whites in the Pittsburgh site of the Study of Women's Health across the Nation (SWAN).[27] In the Midlife in the United States (MIDUS) study, which is enriched with African Americans from Milwaukee, early life adversity was associated with elevated inflammatory markers among blacks but not whites;[28] the authors suggest that racial interactions may be most evident in populations drawn from high-poverty urban areas.

Implications and Conclusions

Clinicians should be aware of the elevated hazard for cardiovascular mortality associated with depressive symptoms, regardless of a patient's race. However, there are important racial differences in prevalence and treatment of depressive symptoms: in this sample, although blacks had almost 10% higher prevalence of depressive symptoms than whites, blacks and whites reported similar rates of psychiatric medication usage. A critical, unanswered question remains regarding whether CVD mortality risk declines when depressive symptoms resolve or are successfully treated. Further studies of this association are needed in samples adequately powered to test differences between whites and non-white samples to replicate the results of a limited number of studies. In addition, future studies should explicitly assess differences in this association by race to see how the risk of mortality changes into older age.

In sum, our results indicate depressive symptoms increased the hazard of cardiovascular mortality for whites and blacks, and the effect does not vary significantly by race. The patterns were similar among those in middle age (50+) and those in older age (65+).

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References

1. Mensah GA, Mokdad AH, Ford ES, Greenlund KJ, Croft JB. State of disparities in cardiovascular health in the United States. Circ. Mar. 2005; 111(10):1233–1241.
2. Sher Y, Lolak S, Maldonado J. The Impact of Depression in Heart Disease. Current Psychiatry Reports. 2010; 12(3):255–264. [PubMed: 20425289]
3. Van der Kooy K, van Hout H, Marwijk H, Marten H, Stehouwer C, Beekman A. Depression and the risk for cardiovascular diseases: systematic review and meta analysis. International Journal of Geriatric Psychiatry. 2007; 22(7):613–626. [PubMed: 17236251]
4. Wulsin LR, Singal BM. Do Depressive Symptoms Increase the Risk for the Onset of Coronary Disease? A Systematic Quantitative Review. Psychosomatic Medicine. Mar 1; 2003 65(2):201–210. 2003. [PubMed: 12651987]
5. Reiner R. Depression as a predictor for coronary heart disease: a review and meta-analysis. American Journal of Preventive Medicine. 2002; 23(1):51–61. [PubMed: 12093424]
6. Nicholson A, Kuper H, Hemingway H. Depression as an aetiologic and prognostic factor in coronary heart disease: a meta-analysis of 6362 events among 146 538 participants in 54 observational studies. European Heart Journal. 2006; 27(23):2763–2774. [PubMed: 17082208]
7. Dong JY, Zhang YH, Tong J, Qin LQ. Depression and risk of stroke: a meta-analysis of prospective studies. Stroke. Jan; 2012 43(1):32–37. [PubMed: 22020036]
8. Pan A, Sun Q, Okereke OI, Rexrode KM, Hu FB. Depression and risk of stroke morbidity and mortality: a meta-analysis and systematic review. JAMA. Sep 21; 2011 306(11):1241–1249. [PubMed: 21934057]
9. Everson-Rose SA, Lewis TT. Psychosocial factors and cardiovascular diseases. Annu Rev Public Health. 2005; 26:469–500. [PubMed: 15760298]
10. Lewis TT, Guo H, Lunos S, et al. Depressive Symptoms and Cardiovascular Mortality in Older Black and White Adults. Circulation: Cardiovascular Quality and Outcomes. 2011; 4(3):293–299. [PubMed: 21505153]
11. Glymour M, Yen J, Kosheleva A, Moon J, Capistrant B, Patton K. Elevated depressive symptoms and incident stroke in Hispanic, African-American, and White older Americans. Journal of Behavioral Medicine. 2011 1-10-10-11-10-10.
12. Jonas BS, Mussolino ME. Symptoms of Depression as a Prospective Risk Factor for Stroke. Psychosomatic Medicine. 2000; 62(4):463–471. [PubMed: 10949089]
13. Ostir GV, Markides KS, Peek MK, Goodwin JS. The Association Between Emotional Well-Being and the Incidence of Stroke in Older Adults. Psychosomatic Medicine. 2001; 63(2):210–215. [PubMed: 11292267]
14. Whang W, Kubzansky LD, Kawachi I, et al. Depression and Risk of Sudden Cardiac Death and Coronary Heart Disease in Women: Results From the Nurses' Health Study. Journal of the American College of Cardiology. 2009; 53(11):950–958. [PubMed: 19281925]
15. Wyatt SB, Williams DR, Calvin R, Henderson FC, Walker ER, Winters K. Racism and Cardiovascular Disease in African Americans. The American Journal of the Medical Sciences. 2003; 325(6):315–331. [PubMed: 12811228]
16. Heeringa, SG. Technical description of the Asset and Health Dynamics (AHEAD) survey sample. Institute for Social Research, University of Michigan; Ann Arbor, MI: 1995.
17. Juster FT, Suzman R. An Overview of the Health and Retirement Study. The Journal of human resources. 1995; 30(Special Issue on the Health and Retirement Study: Data Quality and Early Results):S7–S56. S57–S56.

18. Wallace, RB.; Herzog, AR.; Ofstedal, MB.; Steffick, D.; Fonda, S.; Langa, K. Documentation of Affective Functioning Measures in the Health and Retirement Study. University of Michigan Survey Research Center; Ann Arbor, MI: 2000.

19. Turvey CL, Wallace RB, Herzog R. A Revised CES-D Measure of Depressive Symptoms and a DSM-Based Measure of Major Depressive Episodes in the Elderly. International Psychogeriatrics. 1999; 11(02):139–139. [PubMed: 11475428]

20. Schulz R, Beach SR, Ives DG, Martire LM, Ariyo AA, Kop WJ. Association Between Depression and Mortality in Older Adults: The Cardiovascular Health Study. Archives of Internal Medicine. 2000; 160(12):1761–1768. [PubMed: 10871968]

21. Ariyo AA, Haan M, Tangen CM, et al. Depressive symptoms and risks of coronary heart disease and mortality in elderly Americans. Cardiovascular Health Study Collaborative Research Group. Circulation. Oct 10; 2000 102(15):1773–1779. [PubMed: 11023931]

22. Hemingway H, Marmot M. Evidence based cardiology: psychosocial factors in the aetiology and prognosis of coronary heart disease. Systematic review of prospective cohort studies. BMJ. May 29; 1999 318(7196):1460–1467. [PubMed: 10346775]

23. Wassertheil-Smoller S, Shumaker S, Ockene J, et al. Depression and Cardiovascular Sequelae in Postmenopausal Women: The Women's Health Initiative (WHI). Archives of Internal Medicine. 2004; 164(3):289–298. [PubMed: 14769624]

24. Gump BB, Matthews KA, Eberly LE, Chang YF. Depressive symptoms and mortality in men: results from the Multiple Risk Factor Intervention Trial. Stroke. Jan; 2005 36(1):98–102. [PubMed: 15569872]

25. Pan A, Okereke OI, Sun Q, et al. Depression and Incident Stroke in Women. Stroke. 2011

26. Diez-Roux AV, Link BG, Northridge ME. A multilevel analysis of income inequality and cardiovascular disease risk factors. Social Science & Medicine. 2000; 50(5):673–687. [PubMed: 10658848]

27. Troxel WM, Matthews KA, Bromberger JT, Sutton-Tyrrell K. Chronic stress burden, discrimination, and subclinical carotid artery disease in African American and Caucasian women. Health Psychology. 2003; 22(3):300. [PubMed: 12790258]

28. Slopen N, Lewis TT, Gruenewald TL, et al. Early life adversity and inflammation in African Americans and whites in the midlife in the United States survey. Psychosom Med. 2010; 72(7): 694–701. [PubMed: 20595419]

Table I

Baseline Participant Characteristics of Total Sample and by Race

Variable	Full Sample			Age ≥ 65		
	Total (n=15,845)	Blacks (n=2,401)	White (n=13,444)	Total (n=7,460)	Blacks (n=917)	White (n=6,543)
Average years of follow-up	9.1±3.7	9.0±3.7	9.1±3.7	8.1±3.9	7.9±3.9	8.1±3.9
Age	66.8±10.3	65.3±10.0	67.1±10.3	76.3±6.3	76.4±6.5	76.3±6.2
Male, %	42.3	36.2	43.4	38.9	33.9	39.6
Education	12.2±3.0	10.7±3.6	12.5±2.8	11.7±3.3	9.2±4.0	12.0±3.0
Depressive Symptoms ≥3, %	19.6	27.6	18.1	21.2	28.2	20.3
BMI	26.8±5.1	28.6±5.9	26.4±4.9	25.8±4.7	27.4±5.4	25.6±4.5
BMI 4 categories, %						
BMI <18.5	2.1	1.6	2.2	3.2	2.3	3.3
18.5≤BMI<25	36.4	24.7	38.4	42.7	31.6	44.3
25≤BMI<30	39.8	40.2	39.7	38.2	40.1	38.0
BMI≥30	21.7	33.5	19.6	15.9	26.0	14.5
Smoking Status, %						
Never-smoker	38.1	38.6	38.0	40.2	42.3	39.9
Ever-smoker	41.9	36.5	42.9	45.0	38.3	46.0
Current smoker	16.8	21.3	16.0	10.2	14.1	9.7
Chronic conditions, median±IQR	1±2	1±1	1±2	1±1	2±1	1±1
Physically Active, %	45.0	37.4	46.4	37.5	26.7	39.0
Heart Medications, %	13.7	11.3	14.1	20.2	16.3	20.7
Psychiatric Medications, %	5.6	5.4	5.7	5.0	5.1	5.0

Note: BMI indicates body mass index, or kg/m^2. Values are mean ± SD, median ± inter-quartile range, or percentage.

Table II

Adjusted Hazard Ratios (95% Confidence Intervals) for Associations Between High Depressive Symptoms and Types of Cardiovascular Mortality for Blacks and Whites

	Blacks				Whites			
	Events	Model 1 HR 95%CI	Model 2 HR 95%CI	Model 3 HR 95%CI	Events	Model 1 HR 95%CI	Model 2 HR 95%CI	Model 3 HR 95%CI
Full Sample								
Total CVD	280	1.42 1.10, 1.83	1.18 0.91, 1.54	1.12 0.86, 1.46	1563	1.46 1.33, 1.61	1.23 1.11, 1.35	1.13 1.03, 1.25
IHD	132	1.65 1.15, 2.37	1.37 0.95, 1.99	1.26 0.87, 1.83	820	1.48 1.30, 1.69	1.24 1.08, 1.42	1.14 0.99, 1.30
Stroke	40	1.31 0.64, 2.69	1.20 0.57, 2.53	1.17 0.55, 2.47	251	1.60 1.29, 2.00	1.45 1.15, 1.81	1.32 1.05, 1.67
Age ≥ 65								
Total CVD	194	1.37 1.02, 1.85	1.19 0.88, 1.62	1.14 0.83,1.55	1341	1.38 1.25, 1.53	1.18 1.06, 1.30	1.10 0.99, 1.21
IHD	95	1.61 1.06,2.45	1.38 0.89, 2.12	1.28 0.84, 1.97	685	1.45 1.26, 1.67	1.23 1.07, 1.43	1.15 0.99, 1.33
Stroke	28	1.24 0.54, 2.85	1.22 0.52, 2.88	1.25 0.53, 2.96	223	1.58 1.25, 1.99	1.43 1.12, 1.81	1.32 1.04, 1.68

Notes: HR indicates Hazard Ratio, CI indicates Confidence Interval

Model 1 adjusts for demographics: age and sex. Model 2 adjusts for demographics and CVD risk factors: education, summary score of chronic conditions, and body mass index. Model 3 adjusts for demographics, CVD risk factors, and health behaviors: smoking and physical activity.

p-values for race*depression interactions are $p>0.05$ for each outcome, in the full sample and of those age ≥ 65