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Racial Differences in the Predictive Role of High Depressive Symptoms on Incident Heart Disease Over 18 Years: Results From the Health and Retirement Study

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Abstract

Background: Studies have investigated racial differences in the relationship between depression and CVD mortality.
Objectives: This study tested the hypothesis that race moderates the effect of baseline depressive symptoms on subsequent heart disease among a nationally representative sample of Black and White older Americans.
Patients and Methods: Data came from ten waves of the health and retirement study (HRS), a nationally representative longitudinal study of US adults over age 50. The present study followed 7,444 Black and White individuals without a diagnosis of heart disease at baseline for up to 18 years for incident heart disease. Elevated depressive symptoms at baseline was the independent variable, time to incident heart disease was the dependent variable, while baseline sociodemographics, health risk behaviors, obesity, and chronic medical conditions were controls. We used Cox proportional hazards models in the pooled sample and stratified by race to test the effect of elevated depressive symptoms on the outcome net of other risk factors.
Results: In the pooled sample, a significant positive interaction was found between the effect of elevated depressive symptoms and Black race (hazard ratio, 1.29; 95% CI = 1.01 - 1.65), suggesting a stronger effect for Blacks compared to Whites. In fully adjusted race-stratified models, elevated depressive symptoms increased the risk of developing heart disease for Blacks (hazard ratio, 1.47; 95% CI = 1.04 - 2.07) but not Whites (hazard ratio, 1.13; 95% CI = 0.97 - 1.32).
Conclusions: Black and White older adults differ in the effect of depressive symptoms on subsequent heart disease over a long period of follow up. Elevated depressive symptoms are associated with a larger risk of incident heart disease among Black but not White older individuals.

Keywords: Depressive Symptoms, Heart Disease, Racial Health Disparities, Longitudinal, Epidemiology

1. Background

Racial and ethnic differences in heart disease in the United States (US) are well established, with Blacks experiencing a higher risk of cardiovascular disease (CVD) compared to Whites in the general population (1) and higher conditional mortality. For example, Blacks die several years sooner than Whites from CVD, and the age-adjusted CVD death rate is 33 % higher for Blacks compared to the overall US population (2). Increasingly, health disparities research seeks to understand mechanisms behind these differences with a goal of helping to close the gaps.

A considerable body of population-based research suggests that depression and depressive symptoms increase subsequent risk of CVD and heart disease. A systematic quantitative review found the overall relative risk of heart disease associated with depression was 1.64 (3). Other research has shown that depression and elevated depressive symptoms increase the risk of morbidity and mortality among patients who have been diagnosed with heart disease (4). Physiological mechanisms such as genetic, inflammatory, and vascular pathways in addition to behaviors such as smoking and lack of exercise may explain the increased risk associated with depression (5).

Recent research has drawn attention to the possibility that the relationship between depression and CVD may vary by race. Several studies have investigated racial differences between depression and risk factors for CVD. Lewis et al. (6) studied the risk of depressive symptoms on aortic calcification among 508 (38% Black, 62% White) women. In regression models stratified by race, they found a significant association between depressive symptoms and aortic calcification in Black but not White women. The authors concluded that Black women may be particularly vulnerable to the effects of depressive symptoms on early atherosclerotic disease. Another recent study examined stroke risk, finding that remitted depressive symptoms predicted increased stroke among Whites (adjusted haz-

ard ratio 1.66, 95% CI = 1.18 to 2.33) and was marginally associated among Hispanics (adjusted hazard ratio 2.36, 95% CI = 0.98 to 5.67) but not Blacks (7).

Other studies have investigated racial differences in the relationship between depression and CVD mortality. Lewis et al. (8) examined racial patterns in the association between baseline depressive symptoms and subsequent overall CVD mortality, ischemic heart disease mortality, and stroke mortality using community data from the Chicago health and aging project over 12 years. They found increased risk of all three CVD mortality outcomes associated with elevated depressive symptoms for Blacks but not for Whites. Capistrant et al. (9) examined these associations in a large national sample of older Americans, the Health and Retirement Study (HRS). Their research confirmed the well-documented association between elevated depressive symptoms and later cardiovascular disease mortality but no significant racial differences in the relationship.

2. Objectives

In this context, the present study investigated the potential moderating effect of race on the relationship between baseline depressive symptomatology and incident heart disease, instead of mortality, using 18 year follow-up data from the HRS. The higher chronicity of depression as well as higher burden of heart disease among Blacks compared to Whites (10, 11) makes further research exploring the racial patterning of this complex interrelationship paramount. Based on the literature (8), we expected a larger risk of subsequent heart disease associated with baseline depressive symptoms for Black compared to White older individuals.

3. Patients and Methods

3.1. The Study

We used data from ten waves (1994 - 2012) of the HRS, a longitudinal biennial survey of a nationally representative sample of adults over the age of 50 in the US. The sampling involves a multi-stage area probability design with geographic stratification and clustering. HRS oversamples Black and Hispanic households at about twice the rate of Whites. The study produces sample weights that can be applied analytically in order to account for the differential probability of selection into the study and differential non-response. The follow-up rate is around 90%. Further detail is provided elsewhere (12). HRS is funded by the National Institute on Aging (NIA) and housed at the University of Michigan's Institute for Social Research (ISR). All participants have provided written consent, and the study protocol has been approved by the University of Michigan Institutional Review Board (IRB).

3.2. Analytic Sample

Participants of the original HRS cohort enrolled at baseline in 1992 included non-institutionalized men and women 51 to 61 years of age (born between 1931 and 1941) and their spouse or partner of any age. We used the RAND version of the HRS (13), which was downloaded from the HRS website (http://hrsonline.isr.umich.edu/). The HRS measure of depression is available in the RAND version from wave 2 onward. Therefore, we begin with 11,596 respondents interviewed at baseline (wave 2) in 1994, when they were 53 to 63 years old. In order to study incident heart disease, we selected those who did not have heart disease at baseline and had data on heart disease in at least one other wave during the next 18 years. Following Capistrant et al. (9), we also limited the sample to Black and White participants only. The resulting analytic sample size at baseline was 7,444. The rate of drop out (lost to follow up) ranged between 12.5 - 15 percent across different waves. The overall death rate was 26.9% over the 18 year follow up period.

3.3. Measures

3.3.1. Depressive Symptoms

A modified 8-item version of the center for epidemiologic studies depression scale (CES-D) was used for measurement of depressive symptoms (14). Participants reported on the extent to which in the previous week, they felt: a) depressed, b) everything was an effort, c) sleep was restless, d) that he or she could not get going, e) lonely, f) that he or she enjoyed life, g) sad, and h) happy. The calculated internal consistency was good (Cronbach's alpha = .80). In line with prior research (15), we used a cut-off score of 4 or greater to indicate substantially elevated depressive symptoms comparable to a cut-off score of 16 on the full CES-D.

3.3.2. Incident Heart Disease

In each wave, participants were asked "Has a doctor told you that you had a heart attack, coronary artery disease, angina, congestive heart failure, or other heart problem?" Respondents answered yes or no. Type of heart disease was not available.

3.3.3. Sociodemographics

Self-reported data were collected on age, race (reference: White), and gender (reference: male). Data were also collected on education and marital status. Marital status

was coded into four categories for married/partnered, divorced, separated/widowed, and never married (reference: married).

3.3.4. Obesity

HRS obtains self-reported height and weight. The calculation of body mass index (BMI) was based on these self-reports using the standard formula: (weight (in kg)/height^2 (in m)). "We used a cut-off of BMI of 30 or greater to indicate obesity (reference: BMI less than 30)".

3.3.5. Health Behaviors

Smoking behavior at baseline was measured using two items: 1) have you ever smoked? 2) Are you currently smoking? Based on these items, we created an indicator dummy variable indicating never smokers/previous smokers versus current smokers (reference: not current smoker). Participants were also asked how often they participated in vigorous physical exercise or sports such as aerobics, running, swimming, or bicycling. We used a variable created for the RAND version for participating 3 or more times per week (reference: not vigorously active). A four category quantity/frequency variable of alcohol use indicated consumption of less than 1, 1 to 2, 3 to 4, and 5 or more drinks per day (reference: non-drinker).

3.3.6. Chronic Medical Conditions

At entry into the study, HRS assesses history of diabetes, cancer, lung disease, stroke, arthritis, and high blood pressure. At each follow-up wave, participants are asked if they have had a new diagnosis of one of these chronic medical conditions. We created a count variable that not only included history of other chronic conditions at baseline but also occurrence of new disease over the period of follow-up (reference: no chronic conditions).

3.4. Analysis

Descriptive statistics were generated for the study population. Cox proportional hazards models stratified by race were used to determine factors associated with the development of heart disease over 18 years. Two variables were required for the Cox models: incident heart disease and time to event since baseline. Incident heart disease was coded as one for those who answered positively to the heart disease question any time during follow-up after baseline and zero if heart disease was not reported. Time to event was defined as the number of years from baseline to the first wave that the respondent or proxy reported heart disease. Participants were censored at the time of first heart disease, death, or lost to follow-up.

Table 1. Sample Characteristics for Continuous Variables[a,b]

	White (n = 6,087)	Black (n = 1,357)	All (n = 7,444)
Age (wave 2)	57.3 (0.05)	57.4 (0.11)	57.3 (0.04)
Number of chronic conditions (wave 2)	0.88 (0.01)	1.16 (0.04)	0.91 (0.01)
Years of education (wave 2)	12.9 (0.08)	11.46 (0.14)	12.8 (0.07)

[a]Values are expressed as Mean (SE).
[b]Results are weighted with baseline HRS sample weights. N's reflect the unweighted sample distributions; Source: health and retirement study, 1994.

To test the hypothesis of a racial difference in the effect of high depressive symptoms on incident heart disease, we fitted a set of Cox proportional hazards models in the full sample that also included a main effect for race, depression, and all our covariates. Our final model also included an interaction term with race and elevated depressive symptoms (Model VI).

A series of race-stratified models were also fitted to evaluate the effect of gender, education, and marital status (Model 1), adding obesity (Model 2), adding smoking status, vigorous physical activity, and alcohol use (Model 3), adding chronic medical conditions (Model 4), and adding elevated depressive symptoms at baseline (Model 5). Hazard ratios with 95% confidence intervals (CI) are reported. Sample weights were applied in the analysis. Stratification and clustering in the estimation of standard errors was accounted for using Taylor series linearization. Sample sizes reflect the unweighted sample distributions. All statistical analyses were performed using Stata version 13 (Stata Corp., College Station, TX).

4. Results

Tables 1 and 2 report descriptive statistics at baseline for the total sample and for Blacks and Whites separately. In the full sample, a majority of participants were women (52%). The average age at wave 2 was 57 years with about 12 years of education. Seventy-seven percent were married, 24% were current smokers, and 22% reported vigorous physical activity at baseline. About 85% of the sample reported little to no alcohol use. About 14.4 % reported elevated depressive symptoms at wave 2, and 24.28 % developed new heart disease during the 18 years of follow up. Eighty-nine percent of the sample was White and 11% was Black.

A slightly higher percentage of Blacks were females.

Table 2. Sample Characteristics for Categorical Variables[a,b]

	White (n = 6,087)	Black (n = 1,357)	All (n = 7,444)
Gender			
Men	3,009 (47.37)	593 (42.23)	3,602 (48.04)
Women	3,078 (52.63)	764 (57.77)	3,842 (51.96)
Marital Status			
Married/partnered	5,001 (79.05)	798 (58.36)	5,799 (76.86)
Divorced	540 (10.75)		719 (11.12)
Separated/widowed	392 (7.04)	289 (20.75)	681 (8.47)
Never married	154 (3.17)	91 (7.04)	245 (3.55)
Obesity (wave 2)			
BMI < 29	4,855 (80.22)	908 (66.85)	5,763 (72.02)
BMI > = 30	1,168 (19.78)	437 (33.15)	1,605 (27.98)
Current smoker (wave 2)			
No	4,627 (75.84)	1,005 (74.09)	5,632 (75.74)
Yes	1,460 (24.16)	350 (25.91)	1,810 (24.26)
Physical vigorous activity (wave 2)			
No	4,539 (77.00)	1,081 (80.66)	5,620 (77.09)
Yes	1,353 (23.00)	243 (19.34)	1,596 (22.91)
Drinking (wave 2)			
None	2,410 (37.19)	738 (53.71)	3,148 (41.79)
< 1/day	2,667 (45.56)	457 (34.01)	3,124 (43.36)
1 - 2/day	687 (11.93)	112 (8.82)	799 (9.75)
3 - 4/day	238 (4.14)	30 (2.19)	268 (4.39)
5 + /day	84 (1.17)	16 (1.27)	100 (0.71)
Depressive symptoms (wave 2)			
CES-D < 4	5,233 (86.22)	1,042 (77.27)	6,275 (85.29)
CES-D > = 4	854 (13.78)	315 (22.73)	1,169 (14.71)
Prevalence of heart disease 1994	1,703 (18.62)	325 (19.32)	1,378 (18.46)
Incident heart disease 1994 - 2012	1561 (24.78)	272 (19.96)	1833 (24.28)

[a] Values are expressed as No. (%).
[b] Percentages are weighted using sampling weights as described in the text. Sample sizes reflect the unweighted sample distributions; Source: health and retirement study, 1994.

Whites had a much higher rate of being married (79% versus 58%). While there were no differences between the samples in rates of current smokers and vigorous physical activity, Blacks were more likely to be nondrinkers (53% versus 37%) but more likely to be obese (33% versus 20%). The rate of elevated depressive symptoms in Blacks at wave 2 was 22% compared to 14% for Whites. About 15% of Whites had heart disease compared to about 17% of Blacks.

Table 3 reports the same set of models in the full sample, with the main effect for race included in all models and Model VI testing the race × depressive symptoms interaction term. Model I shows that, net of gender, age, education, and marital status, Blacks were at higher risk of heart disease over 18 years compared to Whites (hazard ratio: 1.11; 95% CI = 1.01 - 1.22, P < 0.05). Model II showed the elevated risk associated with obesity, and this diminished the risk associated with Black race. In the next model, with the addition of health behaviors, the effect of race on risk of heart disease was marginally significant. In Model IV which included baseline chronic medical conditions, race was no longer associated with the onset of heart disease. Although in Model V, net of all other risks, elevated depressive symptoms at baseline did not predict risk of heart disease over 18 years, in the final model, the interaction term for race and elevated depressive symptoms was significant and positive (hazard ratio: 1.29; 95% CI = 1.01 - 1.65, P < 01), suggesting that the effect of depressive symptoms on heart disease is greater for Blacks than Whites.

Tables 4 and 5 provide the results of hierarchical (Cox) regressions in Blacks and Whites, respectively. According to Model V, net of other controls variables, the effect of elevated depressive symptoms on risk of heart disease over 18 years was significant for Blacks (hazard ratio: 1.47; 95% CI: 1.04 - 2.07, P < 0.01) (Table 3). Model V among Whites (Table 4), however, did not show a significant association between baseline depression and subsequent risk of heart disease (hazard ratio: 1.13; 95% CI: 0.97 - 1.32).

5. Discussion

Using data from a nationally representative sample of older adults, this study found that elevated depressive symptoms at baseline were a stronger predictor of subsequent heart disease over an 18-year follow-up period among Blacks than Whites, after controlling for demographics, socioeconomics, obesity, health risk behaviors, and medical conditions at baseline. Findings were consistent between models in the pooled sample and race-stratified models.

These findings extend the existing literature that has shown an effect of elevated depressive symptoms or depression on subsequent heart disease. Indeed, in the present study, elevated depressive symptoms at baseline were a risk factor for incident heart disease. Fewer studies have examined the moderating effect of race on this relationship using longitudinal data. Our results are in line with the findings of Lewis et al. but not Capistrant et al. (9). Lewis et al. (8) hypothesize that depressive symptoms confer an "accelerated risk for cardiovascular disease in Blacks compared with Whites" (p. 293). Our findings support Lewis' argument. On the other hand, Capistrant et al. found no racial differences in the effects of depressive symptoms on cardiovascular mortality between Blacks and Whites in the HRS. Employing a similar set of controls but considering heart disease rather than mortality, the present study detected a racial difference in the effect of depressive symptoms. While mortality selection is a concern, it seems unlikely that this explains differences across these studies. Rather, Capistrant et al. noted that the sample of Blacks was quite small given the mortality outcome, and that study may have lacked the power to detect a significant racial difference in these relationships (9).

Our finding of a stronger effect of elevated depressive symptoms on heart disease for Blacks compared to Whites can be at least partially explained by racial differences in the natural history of depression potentially due to differential treatment (16, 17). Although major depressive disorder may be less prevalent among Blacks than Whites, depression is more chronic and disabling for Blacks (chronicity: 56.5% for Blacks and 38.6% for Whites) (11). Blacks who endorse criteria for depression are less likely to receive treatment for the disorder. Furthermore, compared to Whites, Blacks are less likely to find antidepressant medications acceptable and are less likely to be open to counseling and more likely to have negative views of treatment for depression (18). Thus, race differences in access to care, presentation of depression, time of diagnosis, or severity of disease may contribute to different vulnerabilities of Blacks and Whites to medical consequences of depression (19). In short, a variety of sociocultural and structural differences in access and treatment may be at play in influencing the response to depressive episodes and the consequent impact on the development of heart disease. Exactly how these factors may influence the relationship between depression and heart disease is a fertile topic for future research. We argue that race should be seen as a contextual factor that shapes correlates, causes, and consequences of medical and mental health problems.

Our findings should be viewed in light of several limitations. As others have noted, use of a brief symptom scale for depressive symptoms is not ideal. This is particularly important because it has been shown that clinical depression has a stronger effect on heart disease compared to depressive symptoms (20). We used a cut-off score that has

Table 3. Adjusted Hazard ratios for Heart Disease in the Full Sample (N = 7,444)[a]

	Model 1		Model 2		Model 3		Model 4		Model 5		Model 6	
	Exp (B)	95%CI	Exp (B)	95% CI	Exp (B)	95% CI	Exp (B)	95% CI	Exp (B)	95% CI	Exp (B)	95% CI
Race	1.11[b]	1.01 - 1.22	1.09	0.98 - 1.21	1.10[c]	0.99 - 1.22	1.07	0.97 - 1.19	1.09	0.98 - 1.2	1.03	0.92 - 1.16
Gender	0.86[d]	0.78 - 0.94	0.86[d]	0.79 - 0.94	0.86[d]	0.78 - 0.94	0.85[e]	0.78 - 0.94	0.86[e]	0.79 - 0.94	0.86[e]	0.79 - 0.94
Age	1.01	1.00 - 1.02	1.01	0.99 - 1.02	1.01	1.00 - 1.03	1.01	0.99 - 1.03	1.01	0.99 - 1.03	1.01	1.00 - 1.03
Education	0.99	0.97 - 1.01	0.99	0.98 - 1.01	1.00	0.98 - 1.02	1.00	0.98 - 1.02	1.00	0.98 - 1.02	1.00	0.98 - 1.03
Marital status												
Divorce	1.20[b]	1.02 - 1.41	1.20[b]	1.02 - 1.40	1.19[b]	1.01 - 1.39	1.17[c]	1.00 - 1.38	1.17[c]	0.99 - 1.38	1.18[b]	1.00 - 1.39
Separated/widowed	1.03	0.89 - 1.19	1.02	0.89 - 1.18	1.00	0.87 - 1.15	0.99	0.86 - 1.14	1.00	0.86 - 1.15	0.99	0.86 - 1.14
Never married	1.08	0.88 - 1.32	1.06	0.87 - 1.29	1.05	0.84 - 1.33	1.06	0.85 - 1.32	1.08	0.86 - 1.37	1.08	0.85 - 1.36
Obesity			1.08[c]	0.99 - 1.19	1.09[c]	0.99 - 1.21	1.06	0.96 - 1.18	1.05	0.96 - 1.16	1.06	0.96 - 1.17
Smoking					1.14[b]	1.02 - 1.27	1.14[b]	1.02 - 1.28	1.12[c]	1.00 - 1.26	1.13[b]	1.00 - 1.26
Vigorous physical activity					0.88[c]	0.78 - 1.00	0.90[c]	0.79 - 1.01	0.90	0.79 - 1.03	0.90	0.79 - 1.03
Drinking												
< 1/day					0.95	0.87 - 1.03	0.95	0.87 - 1.04	0.95	0.87 - 1.04	0.95	0.87 - 1.04
1 - 2/day					1.00	0.87 - 1.15	1.01	0.88 - 1.17	1.01	0.88 - 1.16	1.01	0.88 - 1.16
3 - 4/day					0.91	0.71 - 1.16	0.89	0.70 - 1.14	0.89	0.69 - 1.15	0.88	0.68 - 1.15
5 + /day					1.38	0.76 - 2.48	1.33	0.76 - 2.34	1.45	0.79 - 2.68	1.46	0.79 - 2.7
Chronic medical conditions							1.09[e]	1.04 - 1.14	1.07[d]	1.02 - 1.12	1.07[d]	1.02 - 1.12
CES-D									1.11	0.92 - 1.33	1.06	0.87 - 1.29
CES-D Black											1.29[b]	1.01 - 1.65

[a] Reference group of race: White; reference group of gender: male; reference group of marital status: married; reference group of obesity: non-obese; reference group of smocking: non-smoker; reference group of vigorous physical activity: not active; reference group of drinking: non-drinker; reference group of chronic medical conditions : no chronic conditions; reference group of CES-D: CES-D < 4. Source: health and retirement study, 1994 - 2012.
[b] P < 0.1.
[c] P < 0.05.
[d] P < 0.01.
[e] P < 0.001.

Table 4. Adjusted Hazard Ratios for Heart Disease in Blacks (N = 1,357)[a]

	Model 1		Model 2		Model 3		Model 4		Model 5	
	Exp (B)	95% CI	Exp (B)	95% CI	Exp (B)	95% CI	Exp (B)	95% CI	Exp (B)	95% CI
Gender	1.02	0.85 - 1.24	1.03	0.86-1.22	0.93	0.77 - 1.13	0.94	0.77 - 1.15	0.96	0.79 - 1.17
Age	1.04[b]	1.00 - 1.08	1.04[b]	1.00-1.08	1.04[b]	1.00 - 1.08	1.04[b]	1.00 - 1.08	1.05[b]	1.01 - 1.10
Education	0.99	0.97 - 1.02	0.99	0.96-1.02	1.00	0.97 - 1.04	1.01	0.98 - 1.04	1.03	0.99 - 1.07
Marital status										
Divorced	1.27[c]	0.98 - 1.64	1.27[c]	0.99-1.65	1.45[d]	1.11 - 1.90	1.45[d]	1.11 - 1.88	1.41[d]	1.09 - 1.82
Separated/widowed	0.84	0.61 - 1.14	0.84	0.62-1.14	0.85	0.62 - 1.16	0.82	0.57 - 1.17	0.77	0.55 - 1.09
Never married	0.62[d]	0.44 - 0.88	0.62[b]	0.43-0.90	0.60[d]	0.42 - 0.85	0.61[d]	0.43 - 0.86	0.58[d]	0.41 - 0.83
Obesity			0.99	0.71-1.39	1.03	0.76 - 1.39	1.01	0.73 - 1.37	1.07	0.79 - 1.46
Smoking					1.06	0.78 - 1.42	1.07	0.79 - 1.44	1.12	0.79 - 1.57
Vigorous physical activity					0.74[c]	0.54 - 1.03	0.76[c]	0.57 - 1.02	0.80	0.58 - 1.10
Drinking										
< 1/day					0.97	0.75 - 1.27	1.00	0.74 - 1.35	1.01	0.75 - 1.37
1 - 2/day					0.63[d]	0.47 - 0.86	0.67[d]	0.51 - 0.88	0.66[d]	0.52 - 0.85
3 - 4/day					1.32	0.89 - 1.96	1.42[c]	0.95 - 2.12	1.37	0.84 - 2.23
5 + /day					-	-	-	-	-	-
Chronic medical conditions							1.11	0.98 - 1.25	1.10	0.98 - 1.25
CES-D									1.47[b]	1.04 - 2.07

[a] Reference group of race: White; reference group of gender: male; reference group of marital status: married; reference group of obesity: non-obese; reference group of smocking: non-smoker; reference group of Vigorous Physical Activity: not active; reference group of Drinking: non-drinker; reference group of chronic medical conditions : no chronic conditions; reference group of CES-D: CES-D < 4. Source: Health and Retirement Study, 1994 - 2012.
[b] P < 0.05
[c] P < 0.1
[d] P < 0.01

Table 5. Adjusted Hazard Ratios for Heart Disease for Whites (N = 6,087)[a]

	Model 1		Model 2		Model 3		Model 4		Model 5	
	Exp (B)	95% CI	Exp (B)	95% CI	Exp (B)	95% CI	Exp (B)	95% CI	Exp (B)	95% CI
Gender	0.85[b]	0.77 - 0.93	0.86[c]	0.78 - 0.94	0.85[c]	0.77 - 0.93	0.84[c]	0.77 - 0.93	0.85[c]	0.77 - 0.93
Age	1.01	0.99 - 1.02	1.01	0.99 - 1.02	1.01	0.99 - 1.02	1.01	0.99 - 1.02	1.01	0.99 - 1.02
Education	0.99	0.97 - 1.01	0.99	0.97 - 1.01	1.00	0.98 - 1.02	1.00	0.98 - 1.02	1.00	0.99 - 1.02
Marital status										
Divorced	1.18[d]	1.00- 1.41	1.18[d]	1.00 - 1.40	1.16[d]	0.98 - 1.38	1.15	0.97 - 1.37	1.13	0.95 - 1.35
Separated/widowed	1.07	0.90-1.27	1.06	0.90-1.26	1.03	0.87 - 1.23	1.03	0.87 - 1.23	1.03	0.87 - 1.23
Never Married	1.15	0.89 - 1.49	1.12	0.87 - 1.44	1.13	0.84 - 1.51	1.13	0.85 - 1.51	1.14	0.87 - 1.51
Obesity			1.09[d]	0.99 - 1.20	1.10[d]	0.99 - 1.23	1.07	0.95 - 1.20	1.07	0.96 - 1.20
Smoking					1.15[e]	1.02 - 1.30	1.16[e]	1.02 - 1.31	1.15	1.02 - 1.30
Vigorous physical activity					0.90	0.79 - 1.03	0.91	0.80 - 1.04	0.92	0.80 - 1.06
Drinking										
< 1/day					0.95	0.86 - 1.04	0.95	0.86 - 1.04	0.96	0.87 - 1.06
1 - 2/day					1.04	0.89 - 1.21	1.05	0.90 - 1.22	1.06	0.91 - 1.23
3 - 4/day					0.88	0.68 - 1.14	0.87	0.67 - 1.12	0.87	0.67 - 1.13
5 + /day					1.37	0.76 - 2.49	1.33	0.75 - 2.35	1.30	0.73 - 2.32
Chronic medical conditions							1.09[c]	1.03- 1.15	1.09[c]	1.03 - 1.15
CES-D									1.13	0.97 - 1.32

[a] Reference group of race: White; reference group of gender: male; reference group of marital status: married; reference group of obesity: non-obese; reference group of smocking: non-smoker; reference group of vigorous physical activity: not active; reference group of drinking: non-drinker; reference group of chronic medical conditions : no chronic conditions; reference group of CES-D: CES-D < 4. Source: health and retirement study, 1994 - 2012.
[b] P < 0.001.
[c] P < 0.01.
[d] P < 0.1.
[e] P < 0.05.

been used to indicate an elevated level of depressive symptoms that are indicative of clinical depression (15). This was a higher cut-off than that used by Capistrant et al. (9) (4 rather than 3 on the 8 point scale), but this only makes the test of our hypothesis more conservative. Although we utilized a proportional hazards framework, other analytic procedures could be used that can account for the time-varying nature of some covariates. For example, we utilized depressive symptoms in wave 2, but future research could evaluate the impact of sustained depression. Future research could also test bidirectional links between depression and heart disease (21). Finally, our findings on a lack of effect of depressive symptoms on risk of incident heart disease for Whites and also a positive effect of depressive symptoms on risk of incident heart disease for Blacks should be interpreted with caution, as although the 95% CI included 1.00, the lower bound was very close to 1.00. Although Blacks were more prone to heart disease due to depressive symptoms, the lower limit of CI was 1.04, which is very close to 1.00. Nonetheless, this study utilized data from a large, nationally representative sample, which allows us to generalize these findings to the general US population over the age of 50. The longitudinal data allow us to report estimates for risk of incident heart disease.

5.1. Conclusions

Results of the present study suggest that, compared to Whites, elevated depressive symptoms are a better predictor of incident heart disease, net of a set of potential confounders, for Blacks compared to Whites. Beyond the clear value of potentially reducing the burden of depression, detection, prevention, and treatment of depression in Black Americans may have the added benefit of reducing risk of CVD. Our findings contribute to informing policies and programs that aim to reduce racial disparities in morbidity and mortality due to CVD in the US. Addressing depression is an important step that public health officials and clinicians can take to mitigate the risk of heart disease among Blacks. Given the interaction effects identified in this study, developing culturally tailored approaches designed to engage Blacks in depression care is a highly relevant goal.

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