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Long-Term Reciprocal Associations Between Depressive Symptoms and Number of Chronic Medical Conditions: Longitudinal Support for Black–White Health Paradox

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Abstract
Background Previous research has identified a Black–White health paradox, which can be defined as less frequent depression despite a higher prevalence of chronic medical conditions among Blacks compared to Whites in the USA. Based on this paradox, we would expect weaker associations between chronic medical conditions and depression among Blacks than Whites. However, the literature on this topic is mostly cross-sectional and has provided findings that contradict the Black–White health paradox. The present longitudinal study extends prior research by assessing Black–White differences in reciprocal associations between number of chronic medical conditions and depressive symptoms over a 25-year period.

Methods Data came from the Americans' Changing Lives Study that followed 1034 surviving Black and White respondents for 25 years from 1986 to 2011. Chronic medical conditions were measured based on a count of self-reported physician diagnoses including hypertension, diabetes, chronic lung disease, heart disease, stroke, cancer, and arthritis at baseline (1986) and follow-up (2011). Depressive symptoms were also measured at baseline and follow-up using a 10-item Center for Epidemiological Studies-Depression (CES-D) scale. Multi-group structural equation modeling was used to assess reciprocal associations between baseline and subsequent depressive symptoms and baseline and subsequent chronic medical conditions comparing Black and White respondents.
Results Among White but not Black respondents, a higher number of chronic medical conditions at baseline predicted a greater increase in depressive symptoms over 25 years of follow-up. Among Whites but not Blacks, individuals with more depressive symptoms at baseline developed more chronic medical conditions over time.
Conclusion Findings documented Black–White differences in reciprocal associations between chronic medical conditions and depressive symptoms over time. Our study provides longitudinal evidence for the Black–White health paradox across mid and later life, as reciprocal associations between depression and chronic medical conditions were weaker for Blacks compared to Whites.

Keywords Ethnic groups · African Americans · Depression · Chronic medical conditions

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Background

Compared to Whites, Blacks have a higher prevalence of chronic medical conditions (CMCs) [1, 2] but lower levels

of clinical and subclinical depression [1–8], a phenomenon which has been labeled the Black–White health paradox [9, 10]. In line with this phenomenon, a number of studies have shown that Blacks and Whites differ in the magnitude of the association between various CMCs and depression or depressive symptoms [11–14] suggesting that race may moderate the reciprocal associations between CMCs and depression.

There remains a need for more longitudinal research examining moderating effects of race on the reciprocal associations between CMCs and depression in the United States. First, most prior studies were cross-sectional in design [12–15], limiting causal inferences regarding the association between depression and CMCs. This is particularly important because the optimal design for studying the reciprocal associations is a longitudinal design [16–18]. Second, literature has provided inconsistent [14, 15] and even unexpected findings [13, 14, 19]. If Blacks have more CMCs and with a lower prevalence of depression than Whites, we would expect to observe a stronger association between depressive symptoms and CMCs among Whites than Blacks, with each incremental increase in number of chronic medical conditions associated with a smaller increase in depressive symptoms among Blacks compared to Whites. However, Hankerson and colleagues documented a stronger association between major depressive disorder (MDD) and hypertension, obesity, and liver disease among Blacks compared to Whites [19], and Assari found a positive cross-sectional association between MDD and cardiovascular diseases among Blacks that could not be replicated for Whites [13]. Further, using the National Survey of American Life (NSAL) data, Watkins, Assari, and Johnson-Lawrence (2015) showed that lifetime MDD was associated with at least one CMC among Blacks, but not Whites [15]. A few existing studies have failed to show any moderating effect of race on the association between CMCs and depression [14, 20]. As several of the above findings contradict the expected stronger association between depression and CMCs among Whites than Blacks, there is still a need for research to better understand how race moderates the longitudinal association between CMCs and depression [14, 15].

Longitudinal studies have shown that not only do baseline CMCs predict subsequent depression [16, 21–24], but baseline depression also increases risk of CMCs over time [17, 18]. However, most of this literature has not studied US Blacks and Whites [16–18, 24], suggesting the need for further s studies, given the importance of context-dependent racial experiences of populations [25]. Despite our knowledge of the bidirectional relationship between depression and CMCs over time, very few studies have specifically compared this reciprocal relationship among Blacks and Whites. Thus, to build on the existing literature, we examined the reciprocal associations between number of CMCs and depressive symptoms over 25 years among Blacks and Whites.

Methods

Design and Setting Data come from the Americans' Changing Lives (ACL), a nationally representative US cohort study conducted from 1986 until 2011. Detailed information on the study design is available elsewhere [26, 27].

Sampling and Participants The ACL enrolled a stratified multistage probability sample of adults ages 25 or above who lived in the continental USA in 1986. The study included 3617 non-institutionalized respondents (representing 70 % of sampled households and 68 % of sample individuals at baseline) with an oversampling of those age 60 and older and African Americans. Wave 1 included 70 % of sampled households and 68 % of sample individuals. Wave 2 included re-interviews of 83 % (n= 2867) of survivors of wave 1. Waves 3 to 5 included 83, 74, and 81 % of survivors in 1994, 2001/2002, and 2011/2012, respectively. Current analysis is limited to Blacks and Whites who were followed from 1986 to 2011/2012. This strategy was chosen because we wanted to study long-term reciprocal associations between depression and CMCs regarding Black–White health paradox.

Process Data were collected via face-to-face interviews in the first two waves, while waves 3 to 5 were conducted via telephone or face-to-face interviews. In a small number of cases, when participants were unavailable for a given wave, data were collected from proxy reporters in waves 3 through 5.

Measures Information on socio-demographic characteristics was obtained from wave 1. Baseline and subsequent depressive symptoms were measured at 1986 and 2011, in wave 1 and wave 5 of the survey. Data from all waves, however, were used to measure CMCs over the 25-year follow-up period.

Socio-Demographics Demographic data included gender (a dichotomous variable with male as the referent category) and age (a continuous variable), while socioeconomic status was measured with an indicator of education (less than 12 years of education, high school degree or some college [referent category], and college degree or higher). Race was the moderator, defined as Black vs. White (White respondents as the referent category).

Depressive Symptoms Depressive symptoms were measured with 10 items from the Center for Epidemiological Studies-Depression scale (CES-D) [28]. Items measured the extent to which respondents felt depressed, happy, lonely, sad, that everything was an effort, that their sleep was restless, that people were unfriendly, that they did not feel like eating, that

people dislike them, that they could not get going, and that they enjoyed life. Positively worded items were reverse-coded. This abbreviated CES-D has shown acceptable reliability and a similar factor structure compared to the original version. Item responses were 1 ("hardly ever") to 3 ("most of the time"). The total score was computed across the 10 items [29–31], resulting in a continuous measure of depressive symptoms for baseline and follow-up, potentially ranging from 10 to 30. Higher scores indicated greater severity of depressive symptoms.

Number of CMCs Number of CMCs was measured using self-report data in all waves. All participants were asked whether a health-care provider had ever told them they had each of seven focal conditions including hypertension, diabetes, chronic lung disease, heart disease, stroke, cancer, and arthritis. Participants were also asked if they were currently taking medication for such conditions. Responses were dichotomized, and a sum score was calculated, ranging from 0 to 7. A detailed description on the measurement of CMCs is provided elsewhere in House and colleagues [27]. We used interim waves to identify CMCs during the 25-year follow-up period. As chronic medical conditions evaluated in this study do not have cure, we carried positive responses forward to compute the CMCs in our next wave. For instance, if an individual had reported that a condition was present in a prior wave, we assumed that the condition is present in all subsequent waves even if the condition was not reported in a later wave.

Statistical Analysis

In this study, univariate and bivariate analyses were performed using the SPSS statistical package (IBM Corp, Armonk, NY). We used Amos 20 (IBM Corp, Armonk, NY) for multivariable analysis. $P<0.05$ was considered as statistically significant. For bivariate associations, we used Pearson's correlations and independent sample *t* tests. For multivariable analysis, we used multi-group structural equation modeling to test if baseline depression and CMCs predict subsequent depression and CMCs, while age, education, and gender were held constant. In our multi-group analysis, group was defined based on race [32].

The Amos software computes maximum likelihood estimates in the presence of missing data [33, 34]. The adequacy of model fit was assessed by examining the chi-square statistic, the comparative fit index (CFI), and the root mean square error of approximation (RMSEA). A non-significant chi-square statistic, a chi-square to degrees of freedom ratio of less than 4, a CFI above .90, and a RMSEA value of .06 or less are indicators of a good fitting model to the data [35, 36].

Results

A total of 715 White (69.1 %) and 319 Black (30.9 %) respondents who completed surveys in wave 1 and wave 5 were entered in this analysis. As Table 1 suggests, females were over represented and the mean age was 41 (SD=11) years at baseline. Most participants did not have any CMCs at baseline but developed at least one CMC during the follow-up period (Table 1).

Black respondents were slightly younger and had lower educational attainment compared to their White counterparts. Among Blacks and Whites, depressive symptoms slightly declined over time, while CMCs increased (Table 2).

In the pooled sample, baseline age was negatively associated baseline depressive symptoms and positively associated with baseline and follow-up CMCs. Among Whites, women reported significantly higher baseline and follow-up depressive symptoms and CMCs than men. Among Blacks, compared to men, women reported significantly higher baseline depressive symptoms, but not baseline CMCs and follow-up depressive symptoms and CMCs (Table 3).

Table 1 Frequency of demographic characteristics and chronic medical conditions at baseline and follow-up for the sample overall and separately for surviving White and Black respondents from 1986 to 2011

	All (n=1034) n (%)	Whites (n=715) n (%)	Blacks (n=319) n (%)
Gender			
Male	379 (36.7)	277 (38.7)	102 (32.0)
Female	655 (63.3)	438 (61.3)	217 (68.0)
Race			
White	715 (69.1)	– (–)	– (–)
Blacks	319 (30.9)	– (–)	– (–)
Chronic medical conditions at baseline			
0	651 (63.0)	468 (65.5)	183 (57.4)
1	276 (26.7)	184 (25.7)	92 (28.8)
2	86 (8.3)	50 (7.0)	36 (11.3)
3	16 (1.5)	10 (1.4)	6 (1.9)
4	4 (0.4)	3 (0.4)	1 (0.3)
5	1 (0.1)	0 (0.0)	1 (0.3)
Chronic medical conditions at follow-up			
0	87 (8.4)	68 (9.5)	19 (6.0)
1	410 (39.7)	297 (41.5)	113 (35.4)
2	338 (32.7)	230 (32.2)	108 (33.9)
3	149 (14.4)	89 (12.4)	60 (18.8)
4	39 (3.8)	24 (3.4)	15 (4.7)
5	10 (1.0)	7 (1.0)	3 (0.9)
6	1 (0.1)	0 (0.0)	1 (0.3)

Sampling weights have not been applied as analysis is limited to 1034 surviving Black and White respondents for 25 years from 1986 to 2011 (from a total number of 3617 participants)

Table 2 Distribution of demographic and socio-economic characteristics, number of chronic medical conditions, and depressive symptoms at baseline and follow up for the sample overall and separately for surviving White and Black respondents from 1986 to 2011

	All (n=1034)		Whites (n=715)		Blacks (n=319)	
	Min–max	Mean±SD	Min–max	Mean±SD	Min–max	Mean±SD
Age	24.00–72.00	41.07±11.34	25.00–72.00	41.67±11.72	24.00–68.00	39.72±10.34
Education	0.00–17.00	12.90±2.53	0.00–17.00	13.23±2.52	5.00–17.00	12.17±2.41
Baseline depression	9.00–29.00	14.03±3.81	9.00–29.00	13.44±3.52	9.00–29.00	15.37±4.08
Follow-up depression	10.00–30.00	13.47±3.67	10.00–30.00	12.97±3.46	10.00–26.00	14.57±3.89
Baseline CMCs	0.00–5.00	0.50±0.76	0.00–4.00	0.46±0.72	0.00–5.00	0.60±0.83
Follow-up CMCs	0.00–6.00	1.69±1.02	0.00–5.00	1.62±1.00	0.00–6.00	1.85±1.04

CMCs chronic medical conditions

Our model showed an excellent fit to the data [chi-square = .550, probability level = .760, CMIN/DF = .275, CFI=1.000, RMSEA=0.001] (Figs. 1 and 2). Baseline age was associated with a greater increase in depressive symptoms for Whites (b=0.078, p=0.025) but not Blacks (b=−.036, p=.557). Baseline age was also predictive of the increase in number of CMCs over time among Whites (b=0.189, p<0.001) but not Blacks (b=.039, p=.530). Similarly among Whites and Blacks, high education was associated with a smaller increase in depressive symptoms (b=− 0.155, p<0.001 for Whites vs. b=−.368, p<.001 for Blacks). Education was not associated with an increase in the number of CMCs over time for Whites (b=−0.035, p=.229) and marginally significant for Blacks (b=.103, p=.055) (Table 4).

Number of baseline CMCs was associated with worsening of depressive symptoms over time among Whites (b=0.169,

Table 3 Correlation between demographic, socio-economic, chronic medical conditions, and depressive symptoms at baseline and follow-up for the sample overall and separately for surviving White and Black respondents from 1986 to 2011 (n=1034)

	1	2	3	4	5	6	7
All (n=1034)							
Age	1	.066*	−.141**	−.208**	0.01	.343**	.262**
Female		1	−.131**	.164**	.115**	.089**	.080**
Education			1	−.174**	−.291**	−.267**	−.189**
Baseline depression				1	.424**	.108**	.099**
Follow-up depression					1	.198**	.250**
Baseline CMC						1	.479**
Follow-up CMC							1
Whites (n=715)							
Age	1	.094*	−.132**	−.209**	0.031	.305**	.279**
Female		1	−.139**	.162**	.121**	.089*	.079*
Education			1	−.134**	−.239**	−.227**	−.175**
Baseline depression				1	.414**	.110**	.136**
Follow-up depression					1	.212**	.276**
Baseline CMC						1	.507**
Follow-up CMC							1
Blacks (n=319)							
Age	1	0.014	−.232**	−.172**	0.019	.465**	.260**
Female		1	−0.074	.136*	0.071	0.074	0.062
Education			1	−.140*	−.313**	−.317**	−.168**
Baseline depression				1	.364**	0.057	−0.034
Follow-up depression					1	.137*	.156**
Baseline CMCs						1	.413**
Follow-up CMCs							1

CMCs chronic medical conditions

*p<0.05; **p<0.01

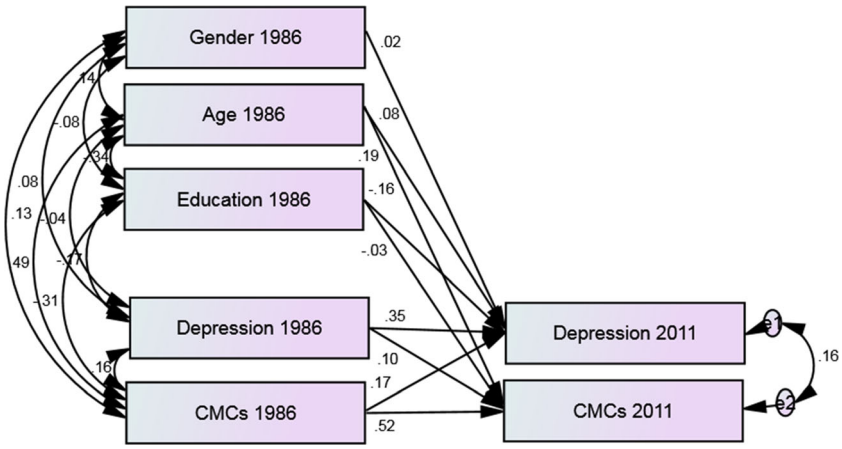

Fig. 1 Standardized regression weights for longitudinal associations between chronic medical conditions (CMCs) and depressive symptoms (CES-D) over 25 years among surviving 715 White respondents from 1986 to 2011. Chi-square = .Standardize, probability level = .760, CMIN/DF = .275, CFI = 1.000, RMSEA = 0.001

$p<0.001$) but not Blacks ($b=0.050$, $p=0.375$). Higher depressive symptoms at baseline were also predictive of a larger increase in number of CMCs over time among Whites ($b=0.099$, $p<0.001$) but not Blacks ($b=-0.052$, $p=0.265$) (Table 4).

Discussion

According to our findings, among Whites but not Blacks, number of baseline CMCs predicts worsening of depressive symptoms over 25 years. Higher depressive symptoms at baseline were also predictive of incident CMCs among Whites but not Blacks. Findings suggest that patterns of longitudinal association between CMCs and depressive symptoms may depend on race in the USA.

Our study provides longitudinal evidence supporting the Black–White health paradox [8, 9], defined as a higher rate of CMCs [1, 2] but lower prevalence of depression or depressive symptoms [1–10] among Blacks compared to Whites. Our findings are supported by the results reported by Capistrant and colleagues who showed that although among Whites and Blacks depressive symptoms are associated with cardiovascular mortality, the association between baseline depressive symptoms and subsequent cardiovascular mortality was only significant for Whites, but not Blacks, after adjusting for covariates [37].

Jackson and colleagues have argued that Blacks' low rates of depression despite of high rates of medical conditions compared to Whites may be due to their engagement in negative health behaviors (e.g., smoking or overeating) that may be associated with low psychological but high physiological damages [38–41]. Jackson's hypothesis suggests that the association between psychological and medical conditions among Blacks may not be as strong as Whites. Although these behaviors may protect individuals' mental health, such ineffective coping behaviors will have numerous negative physical health consequences [39–42]. Our findings could lend support for Jackson's hypothesis. Additional evidence has been provided by studies that have documented racial differences in depression associated with obesity [41, 43, 44].

Our findings do not, however, support the findings of Lewis et al. in 2011, who suggested that depressive symptoms confer an "accelerated risk" for cardiovascular disease in

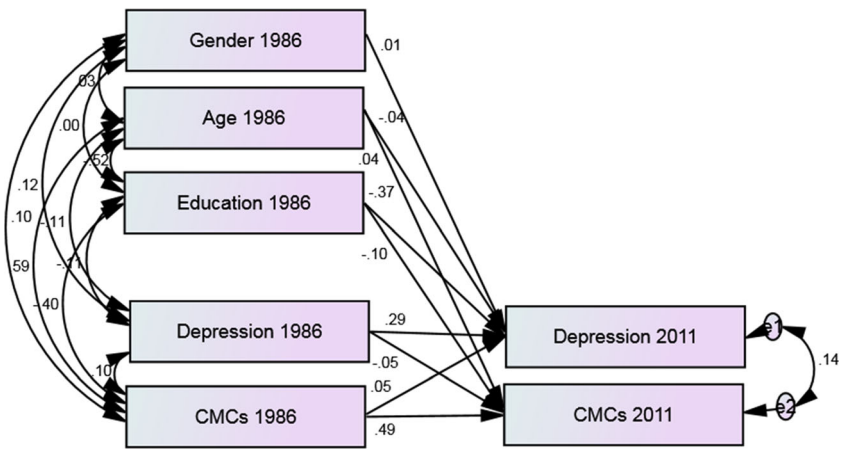

Fig. 2 Standardized regression weights for longitudinal associations between chronic medical conditions (CMCs) and depressive symptoms (CES-D) over 25 years among 319 surviving Black respondents from 1986 to 2011. Chi-square = .550, probability level = .760, CMIN/DF = .275, CFI = 1.000, RMSEA = 0.001

Table 4 Longitudinal associations between chronic medical conditions (CMC) and depressive symptoms (CES-D) over 25 years among surviving White and Black respondents from 1986 to 2011 (n=1034)

			Whites (n=715)					Blacks (n=319)				
			Unstandardized Estimate	S.E.	C.R.	Standardized Estimate	P	Unstandardized Estimate	S.E.	C.R.	Standardized Estimate	p
Baseline CMCs	→	Follow-up depression	.563	.114	4.955	.169	<0.001	.183	.207	.887	.050	.375
Baseline depression	→	Follow-up CMCs	.032	.009	3.620	.099	<0.001	−.015	.013	−1.115	−.052	.265
Education	→	Follow-up CMCs	−.013	.011	−1.204	−.035	.229	−.031	.016	−1.919	−.103	.055
Baseline CMCs	→	Follow-up CMCs	.576	.034	16.778	.524	<0.001	.487	.056	8.649	.489	<0.001
Age	→	Follow-up CMCs	.012	.002	5.991	.189	<0.001	.003	.004	.627	.039	.530
Baseline depression	→	Follow-up Depression	.350	.030	11.783	.353	<0.001	.302	.050	6.096	.286	<0.001
Age	→	Follow-up depression	.015	.007	2.249	.078	.025	−.009	.015	−.588	−.036	.557
Female	→	Follow-up depression	.142	.207	.687	.020	.492	.107	.393	.273	.012	.784
Education	→	Follow-up depression	−.174	.035	−4.929	−.155	<0.001	−.406	.059	−6.869	−.368	<0.001

CMCs chronic medical conditions, *S.E.* standard error, *C.R.* critical ratio

Blacks compared with Whites. The authors examined the association between depressive symptoms and overall cardiovascular mortality, ischemic heart disease mortality, and stroke mortality, using Cox's proportional hazards models. A sample of 6158 community-dwelling older adults was enrolled in the study. In race-stratified models adjusted for age and sex, elevated depressive symptoms were associated with cardiovascular mortality in blacks (hazard ratio [HR], 1.95) but were not significantly associated with cardiovascular mortality in whites (HR, 1.26), with significant interaction for the effect of race by depressive symptoms (p=0.03). Similar findings were observed for IHD mortality (Black: HR, 1.99; White: HR, 1.28) and stroke mortality (Black: HR, 2.08; White: HR, 1.32). In sum, Lewis and colleagues found that elevated depressive symptoms are associated with multiple indicators of cardiovascular mortality in older Blacks but not in Whites [11].

Different from our findings, there is literature suggesting that depression is more chronic and disabling for Blacks compared to Whites [13, 15]. Williams and colleagues reported that frequency of 12 month MDD among those with lifetime MDD is higher for Blacks (56 %) than Whites (39 %) [45], but we believe this could be explained by later diagnosis of lifetime MDD among Blacks as compared to Whites. If depression is more disabling among Blacks than Whites, we would expect a larger effect of depression among Blacks, a hypothesis which was not supported by our findings.

Our findings among Whites in the USA are in support of previous longitudinal studies documenting effects of baseline CMCs on subsequent depression [16, 21–24] and vice versa [17, 18] in other countries. In a population-based cohort of more than 38,000 participants in Taiwan, baseline CMCs were a strong predictor of increased risk of subsequent depression [17]. Based on the Canadian Community Health Survey, in which 17,000 participants were followed for 10 years, individuals with CMCs were 1.32 times more likely to subsequently use mental health services (HR=1.32) relative to controls [16]. In the Canadian National Population Health Survey (NPHS), baseline major depressive episode predicted subsequent heart disease (RR=1.7), arthritis (RR=1.9), asthma (RR=2.1), back pain (RR=1.4), chronic bronchitis or emphysema (RR=2.2), hypertension (RR=1.7), and migraines (RR=1.9). As baseline depression did not predict subsequent cataracts and glaucoma, peptic ulcers, and thyroid disease, authors concluded the effect of baseline major depressive episode on subsequent CMCs is specific to a subset of conditions characterized particularly by inflammation, autonomic reactivity, and pain [18].

In our study, reciprocal associations between depression and medical conditions among Whites could not be found among Blacks, supporting the Black–White health paradox [8, 9, 46, 47]. Our findings, however, do contrast with the hypothesis that depression is more consequential for Blacks than Whites [13, 15, 45]. Given low access and trust to the health-care system and high stigma, depression is expected to be more consequential for Blacks than Whites [13, 15, 45]. Compared to Whites, Blacks endorse more negative beliefs regarding pharmaceutical treatment of depression [45], are more likely to prefer non-pharmacologic approaches (e.g., counseling and prayer), are less frequently to believe that depression medications are effective, and are frequently to believe that antidepressants are addictive [48]. These beliefs may operate as barriers against promotion of depression treatment among Blacks. Thus, improvement of screening, diagnoses, and treatment of depression among Blacks may require enormous efforts [45, 49, 50].

Comorbid medical conditions add to the complexity of diagnosis of depression, particularly among Blacks [1, 2, 51, 52], particularly due to the belief that depression is more somatic among Blacks [53]. Despite this complexity, the

primary care setting provides a unique opportunity for screening and detection of depression among Blacks who suffer chronic medical conditions [54–56]. However, quality of psychiatric services in primary care settings for Blacks with medical conditions is not optimal and may require some improvement [15, 57].

Although this study makes a unique contribution to the literature by enhancing our understanding of Black–White differences in reciprocal associations between depression and CMCs over the life course, the results should be only interpreted in the light of study limitations. First, this study did not include data on type of CMCs and only used a total number of CMCs. In addition, measurement of CMCs was based on self-reported data. Furthermore, our list of medical conditions was not very comprehensive. Future research should test if race only moderates the association between depression and particular CMCs. In addition, symptoms of depression, not clinical depression, were assessed in this study. The study did not control for neighborhood, family structure, and access to health care that may influence trajectories of depression and CMCs over time. Females also were over-represented in the study sample. Finally, the duration of follow-up was 25 years, which may have caused selective deaths in the population, particularly among Blacks and those with high-baseline chronic medical conditions. Future reports may consider replication of the current findings using shorter follow-up periods. Researchers should also test if clinical diagnosis of depression is differently associated with CMCs than depressive symptoms. Despite these limitations, this study is one of very few nationally representative studies with 25 years of follow-up and a large sample of Black respondents.

The current study provides potential support for the design and implementation of mental health screening of individuals with CMCs in primary health-care settings and considering the race of the target population. Given the importance of race in shaping determinants of physical and mental health, future research should investigate the mechanisms by which race changes the pattern of comorbidity between depression and CMCs.

To conclude, our findings documented reciprocal associations between CMCs and depressive symptoms over time among Whites but not Black Americans. These results indicate that physical and emotional health problems may be more distinct in their course over time among Black than White persons, suggesting that screening and prevention in Black populations should address both of these conditions without assuming universal pattern of co-occurring between these health problems.

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Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no competing interests.

Informed Consent All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) with the Helsinki Declaration of 1975, as revised in 2000. Informed consent was obtained from all participants included in the study.

Animal Studies No animal studies were carried out by the authors for this article.

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