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Bidirectional associations between chronic medical conditions, disability, and depressive symptoms over 25 years: Black- White differences

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ABSTRACT

Background and aims: Blacks have a higher rate of chronic medical conditions (CMC), disability, and depressive symptoms compared to Whites. The present study compared Blacks and Whites for bidirectional associations between CMC, disability, and depressive symptoms over a 25-year period.
Methods: Data came from all five waves (1986, 1989, 1994, 2001, and 2011) of the Americans' Changing Lives Study (ACL). This study followed 3,497 Whites and Blacks for up to 25 years. Number of CMC, disability, and depressive symptoms [Center for Epidemiological Studies-Depression (CES-D)] were measured at all waves. Multi-group crossed lag modeling was used to test four models, where race defined the groups.
Results: Based on Models 1 and Model 2, cross-sectional associations between CMC and depressive symptoms at 1989, 2001, and 2011 were significant among Whites but not Blacks. Based on Model 3, lagged effect of CMC in 2001 on depressive symptoms in 2011 was only significant for Whites. Based on Model 4, cross-sectional associations between disability and depressive symptoms in 1989 were found among Whites but not Blacks.
Conclusion: Compared to Whites, Blacks show fewer cross-sectional and longitudinal associations between CMC, disability, and depressive symptoms over time. These findings are robust, regardless of whether we conceptualize disability or depressive symptoms as the outcome.

Keywords: Ethnic Groups, Blacks, Depressive Symptoms, Medical Conditions, disability, flourishing.

Original article

INTRODUCTION

In the United States, Blacks experience higher rates of chronic medical conditions (CMC), disability, and depressive symptoms than do Whites.[1-6] How the reciprocal associations between depressive symptoms, CMC, and disability differ between Blacks and Whites are, however, unknown. The related literature has shown mixed results; most existing studies are cross-sectional, with very few longitudinal studies having ever compared Blacks and Whites for the reciprocal associations between CMC and

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depression over time.[7-20] A recent longitudinal study suggested that Blacks and Whites differ in the link between medical conditions and depression.[7] The study, which followed Whites and Blacks for 25 years, provided longitudinal support for the Black-White health paradox; however, the study was prone to selective attrition bias, as it was limited to the survivors who completed the 25 year follow-up period. Selective attrition due to deaths in a long term follow up is known to be higher for Blacks than Whites.[7]

Surprisingly, some of the studies on the topic have documented a stronger association between depression and CMC among Whites than Blacks.[8,10,13] These findings contradict the Black-White health paradox, defined as better mental health despite a higher prevalence of CMC and social and economic adversities among Blacks compared to Whites in the USA.[1] A better understanding of the mechanisms behind race differences in the depression-CMC link requires additional longitudinal studies.[7] Although CMC predicts subsequent depression, ideal study design requires a longitudinal design with multiple observations over a long period of time.[21,25-27]

Flourishing is one of the main hypotheses used to explain racial differences in the links between CMC, disability, and depressive symptoms.[26,27] Serious medical conditions such as cancer and heart disease may result in post-traumatic growth, which is associated with better psychological well-being and lower depressive symptoms.[28,33-36] This phenomenon has been explained by positive psychologists who suggest facing life-threatening problems and trauma may result in psychological growth instead of distress.[26,37-40] Thus, developing a chronic medical condition may change one's values and initiate personal transformation, resulting in an unexpectedly low rate of depression in the patient.[41,42] As suggested by research on post-traumatic growth, Blacks may have a higher likelihood of experiencing growth after trauma. Flourishing has been hypothesized as a possible explanation behind the Black-White health paradox, which explains the lower prevalence of clinical depression despite a higher rate of CMC and associated disability among Blacks compared to Whites.[7,26,27,36,41-46] It is not known why racial minorities such as Blacks are more likely to experience growth after trauma.[36]

In a step forward to advance the available literature on Black-White differences in the links between physical and mental health, and to better understand the Black-White health paradox,[7,26,27] we used a crossed lagged model to compare Blacks and Whites on the links between CMC, disability, and depressive symptoms. By using a nationally representative dataset with 25 years of follow up, the study provides generalizable findings to the United States population.

METHODS

We used data from all five waves (1986, 1989, 1994, 2001, and 2011) of the Americans' Changing Lives (ACL) study, 1986- 2011, a nationally-representative United States cohort of adults ages 25 or older. Detailed information on the design, sampling, and methods of the ACL is available elsewhere.[7,49,50]

The ACL study enrolled a stratified multistage probability sample of adults ages 25 or above who lived in the continental

United States in 1986. The original study enrolled 3,617 non-institutionalized respondents (representing 70% of sampled households and 68% of sample individuals at baseline) with an oversampling of older adults and Blacks. Wave 1 included 70% of sampled households and 68% of sample individuals. Wave 2 included re-interviews of 83% (n=2867) of survivors in wave 1. Waves 3 to 5 included 83%, 74% and 81% of survivors in 1994, 2001/02, and 2011/12, respectively. Data were only used for Blacks or Whites (n=3,497).

Data were collected via face-to-face interviews in the first two waves. Data were conducted via telephone or face-to-face interviews in waves 3, 4, and 5. In a small number of cases in waves 3 to 5, when participants were unavailable for a given wave, a proxy reporter was interviewed.

Information on socio-demographic characteristics was obtained from wave 1 (1986). Depressive symptoms and number of CMC were measured at all waves (1986, 1989, 1994, 2001, and 2011).

Demographic data included gender (a dichotomous variable with male as the referent category) and age (a continuous variable). Socio-economic factors included education (less than 12 years of education, high school degree or some college [referent category], and college degree or higher), and race (a dichotomous variable, Black vs. White).

Depressive symptoms were measured using the 10 items from the Center for Epidemiological Studies-Depression Scale (CES-D).[51] Respondents were asked the extent to which they 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 CES-D version. Item responses were 1 (“hardly ever”) to 3 (“most of the time”). The total score was computed across the 10 items, resulting in a continuous measure of depressive symptoms for baseline and follow up, with a potential range from 10 to 30.[7,52-54] Higher scores indicated more depressive symptoms.

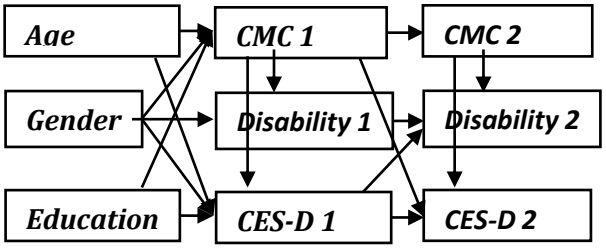
The number of CMC was also measured using self-report data. All participants were asked whether a health care provider had ever told them that they had each of seven focal conditions including hypertension, diabetes, chronic lung disease, heart disease, stroke, cancer, and arthritis. Participants were also asked if they were currently taking medication for such conditions. Responses were dichotomized, and a sum score was calculated, with a potential range from 0 to 7.[7,50]

Disability was measured as physical function. Individuals were asked whether they were bedbound, whether they had difficulty bathing, climbing stairs, walking, or doing heavy housework, and the degree of difficulty of these tasks. Higher scores reflected worse physical function and more disability.[55].

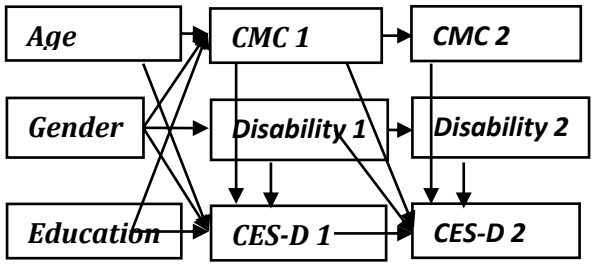
Regarding statistical analysis, all univariate and bivariate analyses were conducted in the SPSS statistical package (IBM Corp, Armonk, NY). Multivariable analysis was performed in the Amos 20 (IBM Corp, Armonk, NY). For bivariate associations, we used Spearman's correlations

and independent samples t-tests. For multivariable analysis, we used path analysis to conduct a multi-group crossed lagged model (Figure 1-a to 1-d) to test associations between depressive symptoms, CMC, and disability among Blacks and Whites. In our model, age, education, and gender were covariates, and group was defined based on self-reported race.[7,56]

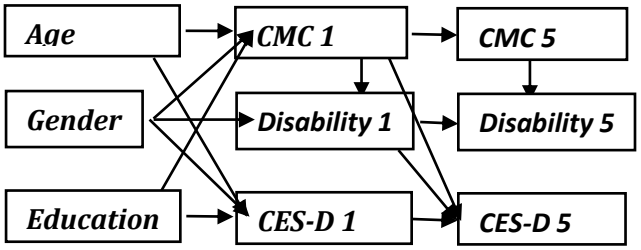
Cross-lagged analysis is one of the effective statistical approaches to detect sequences of effects longitudinally in the presence of multiple observations. This approach allows for the consideration of the stability of any construct over time by adding autoregressive paths within the same constructs. The model also estimates cross-lagged effects of variables in a reciprocal manner. The unique feature of a cross-lagged model is the ability to investigate reciprocal associations between variables; this is the main assumption when associations are bidirectional, not unidirectional. As age, gender, and education are associated with baseline depressive symptoms, CMC, and disability, we controlled for all these factors in our model.[57]



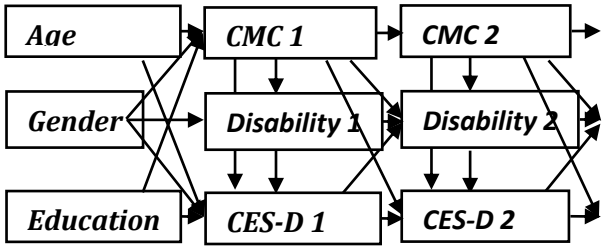
1-a) Model 1



1-b) Model 2



1-c) Model 3



1-d) Model 4

Figure 1: Cross lagged models to test the longitudinal associations between CMC, depressive symptoms (CES-D), and disability among Blacks and Whites [Center for Epidemiological Studies-Depression (CES-D)]

In total, four models were tested. Model 1 explores paths from CMC to disability and depressive symptoms at each time, from CMC to the subsequent depressive symptoms, and from depressive symptoms to subsequent disability. In Model 2, paths from CMC to disability and depressive symptoms at each time, and from CMC and disability to subsequent depressive symptoms were explored. Model 3 was the most parsimonious model, with Model 4 being the most saturated of the models. Model 3 proposed a path from CMC to disability at each time, and from CMC and

disability to the subsequent depressive symptoms. In Model 4, paths from CMC to disability and depressive symptoms at each time, and from CMC to subsequent depressive symptoms, and from depressive symptoms to subsequent disability were explored.

The AMOS software uses Full Information Maximum Likelihood (FIML) to handle missing data.[58,59] The adequacy of model fit was assessed by examining the comparative fit index (CFI), Chi square to degree of freedom ratio (CMIN/DF) which reflects minimum discrepancy divided by its degrees of freedom, and the root mean square error of approximation (RMSEA).[60] A CMIN/DF of less than 4, a CFI above 0.95, and a RMSEA value of 0.05 or less are indicators of an excellent fit of the model to the data.[61,62] Considering the large sample in the present study, and the over-sensitivity of the Chi square measure to sample size, significant Chi square was not considered as an indicator of poor fit.[63]

RESULTS

This study included 3497 White and Black individuals who had participated at Wave 1 of the ACL Study. From these individuals, 66% were Whites (n=2323) and 34% were Blacks (n=1174). Most participants were female (n=2194, 62%). Compared to Blacks, Whites were older, and had higher education at baseline. Compared to Blacks, Whites had lower depressive symptoms, had lower CMC, and lower disability at all time points (Table 1).

Table 1: Distribution of socio-demographics, number of chronic medical condition (CMC), depressive symptoms, disability among Blacks and Whites from 1986 to 2011

Variable	Whites	Mean(SD)	Blacks	SD	P
Age	2323	54.61(17.69)	1174	52.60(17.33)	0.001
Education	2318	12.05(3.11)	1168	10.40(3.75)	<0.001
CES-D 1986	2314	13.57(3.51)	1172	14.89(3.91)	<0.001
CES-D 1989	1903	13.28(3.30)	869	14.70(3.81)	<0.001
CES-D 1994	1646	12.63(3.01)	681	14.31(3.80)	<0.001
CES-D 2001	1226	12.66(3.14)	411	13.78(3.49)	<0.001
CES-D 2011	893	12.78(3.35)	378	14.55(3.98)	<0.001
CMC 1986	2323	0.91(1.05)	1174	1.14(1.14)	<0.001
CMC 1989	1906	0.93(1.06)	874	1.17(1.14)	<0.001
CMC 1994	1751	1.04(1.10)	737	1.30(1.16)	<0.001
CMC 2001	1228	0.97(0.99)	414	1.18(1.12)	0.001
CMC 2011	784	1.39(1.03)	345	1.65(1.01)	<0.001
Disability 1986	2323	3.60(0.82)	1174	3.51(.92)	0.004
Disability 1989	1906	3.62(0.80)	874	3.46(1.00)	<0.001
Disability 1994	1751	3.52(0.95)	737	3.29(1.12)	<0.001
Disability 2001	1291	3.49(0.96)	444	3.32(1.12)	0.003
Disability 2011	969	3.34(1.08)	409	3.03(1.26)	<0.001

P: Independent t- test, CES-D: Center for Epidemiological Studies-Depression; CMC: Chronic Medical Condition.

Table 2 presents bivariate correlations between age, education, gender, depressive symptoms, CMC, and disability among Blacks and Whites. Gender and education were associated with baseline depressive symptoms among Blacks and Whites. Age was associated with baseline depressive symptoms among Blacks, but not Whites. Among Blacks and Whites, age, gender, and education were associated with baseline CMC. Blacks and Whites were similar in correlates of baseline disability (Table 2).

Table 2: Correlation between demographic, socio-economic, chronic medical condition (CMC), depressive symptoms, and disability at baseline among Whites and Blacks in 1986

Variable	1	2	3	4	5	6
1 Age (Yrs)	1	-0.34	0.14	-0.04	0.49	0.36
2 Education (Yrs)	-0.52	1	-0.08	-0.16	-0.31	-0.26
3 Gender (Female)	0.03	0.01	1	0.08	0.13	0.09
4 CES-D	-0.11	-0.11	0.12	1	0.16	0.27
5 CMC	0.59	-0.40	0.10	0.10	1	0.44
6 Disability	0.39	-0.31	0.13	0.23	0.50	1

CMC: Chronic Medical Condition, CES-D: Center for Epidemiological Studies-Depression.

Figures 1-a to 1-d show diagrams of Models 1 to 4 to test the longitudinal associations between CMC, depressive symptoms (CES-D), and disability over a 25 year follow up period from 1986 to 2011.

In Model 1, paths from CMC to disability and depressive symptoms at each time, and from CMC to the subsequent depressive symptoms, and from depressive symptoms to subsequent disability are shown. Based on Model 1, increases in CMC from 1986 to 1989, from 1994 to 2001, and from 2001 to 2011 were only predictive of increase in depressive symptoms at the same time periods among Whites, but not Blacks (Table 3).

Table 3: Results of Model 1; Black and White differences in longitudinal associations between chronic medical conditions (CMC), disability, and depressive symptoms (CES-D) over 25 years from 1986 to 2011

Predictor		Outcome	Estimate (S.E.) Whites	P	Estimate (S.E.) Blacks	P
CMC 1986	→	CES-D 1986	0.11(0.17)	0.030	0.17(0.29)	0.039
CMC 1989	→	CES-D 1989	**0.16**(0.16)	**0.002**	0.16(0.32)	0.108
CMC 1994	→	CES-D 1994	**0.18**(0.16)	**0.002**	0.04(0.33)	0.685
CMC 2001	→	CES-D 2001	**0.22**(0.22)	**0.002**	0.09(0.47)	0.577
CMC 2011	→	CES-D 2011	**0.46**(0.50)	**0.002**	0.24(1.66)	0.587
CMC 1986	→	Disability 1986	0.28(0.02)	<0.001	0.37(0.03)	<0.001
CMC 1989	→	Disability 1989	0.06(0.02)	0.004	0.18(0.03)	<0.001
CMC 1994	→	Disability 1994	0.16(0.02)	<0.001	0.21(0.03)	<0.001
CMC 2001	→	Disability 2001	0.14(0.02)	<0.001	0.20(0.04)	<0.001
CMC 2011	→	Disability 2011	0.10(0.03)	<0.001	0.12(0.06)	0.007
CMC 1986	→	CES-D 1989	-0.05(0.14)	0.204	-0.04(0.25)	0.646
CMC 1989	→	CES-D 1994	**-0.12**(0.13)	**0.006**	-0.01(0.26)	0.951
CMC 1994	→	CES-D 2001	-0.06(0.15)	0.207	0.03(0.34)	0.803
CMC 2001	→	CES-D 2011	**-0.17**(0.30)	**0.054**	-0.13(0.80)	0.595
Disability 1986	→	CES-D 1989	-0.00(0.11)	0.974	-0.02(0.17)	0.612
Disability 1989	→	CES-D 1994	-0.01(0.11)	0.823	**-0.12**(0.19)	**0.023**
Disability 1994	→	CES-D 2001	-0.03(0.10)	0.33	**-0.10**(0.16)	**0.043**
Disability 2001	→	CES-D 2011	0.06(0.13)	0.143	0.08(0.21)	0.235
CES-D 1986	→	Disability 1989	-0.04(0.01)	0.097	0.02(0.01)	0.537
CES-D 1989	→	Disability 1994	-0.03(0.01)	0.104	**-0.09**(0.01)	**0.01**
CES-D 1994	→	Disability 2001	-0.01(0.01)	0.621	**-0.10**(0.01)	**0.011**
CES-D 2001	→	Disability 2011	**-0.10**(0.01)	**<0.001**	0.07(0.02)	0.143
CMC 1986	→	CMC 1989	1.01(0.03)	<0.001	0.98(0.04)	<0.001
CMC 1989	→	CMC 1994	0.94(0.03)	<0.001	0.89(0.04)	<0.001
CMC 1994	→	CMC 2001	0.85(0.03)	<0.001	0.87(0.05)	<0.001
CMC 2001	→	CMC 2011	0.80(0.05)	<0.001	0.69(0.06)	<0.001
CES-D 1986	→	CES-D 1989	0.83(0.07)	<0.001	0.87(0.10)	<0.001
CES-D 1989	→	CES-D 1994	0.94(0.05)	<0.001	0.98(0.09)	<0.001
CES-D 1994	→	CES-D 2001	0.87(0.05)	<0.001	0.9(0.08)	<0.001
CES-D 2001	→	CES-D 2011	0.89(0.06)	<0.001	0.85(0.10)	<0.001
Disability 1986	→	Disability 1989	1.02(0.05)	<0.001	0.65(0.08)	<0.001
Disability 1989	→	Disability 1994	0.83(0.04)	<0.001	0.71(0.07)	<0.001
Disability 1994	→	Disability 2001	0.92(0.04)	<0.001	0.89(0.08)	<0.001
Disability 2001	→	Disability 2011	1.01(0.05)	<0.001	0.89(0.08)	<0.001
Female	→	Disability 1986	0.04(0.03)	0.011	0.10(0.05)	<0.001
Age	→	Disability 1986	0.21(0.00)	<0.001	0.12(0.00)	<0.001
Education	→	Disability 1986	-0.10(0.00)	<0.001	-0.12(0.01)	<0.001
Female	→	CMC 1986	0.05(0.03)	<0.001	0.09(0.05)	<0.001
Age	→	CMC 1986	0.43(0.00)	<0.001	0.51(0.00)	<0.001
Education	→	CMC 1986	-0.15(0.01)	<0.001	-0.15(0.01)	<0.001
Female	→	CES-D 1986	0.07(0.13)	<0.001	0.09(0.21)	<0.001
Age	→	CES-D 1986	-0.16(0.01)	<0.001	-0.33(0.01)	<0.001
Education	→	CES-D 1986	-0.20(0.03)	<0.001	-0.23(0.03)	<0.001

CMCs: Chronic Medical Conditions, CES-D: Center for Epidemiological Studies-Depression.

In Model 2, paths are shown from CMC to disability and depressive symptoms at each time, and also from CMC and disability to subsequent depressive symptoms. Based on Model 2, increases in CMC from 1986 to 1989, from 1994 to 2001, and from 2001 to 2011 were only predictive of increase in depressive symptoms at the same time periods among Whites, but not Blacks (Table 4).

Table 4: Results of Model 2; Black and White differences in longitudinal associations between chronic medical conditions (CMC), disability, and depressive symptoms (CES-D) over 25 years from 1986 to 2011

Predictor		Outcome	Estimate (S.E.)	P	Estimate (S.E.)	P
			Whites		Blacks	
CMC 1986	→	CES-D 1986	0.03(0.16)	0.510	0.09(0.29)	0.267
CMC 1989	→	CES-D 1989	**0.17**(0.17)	**0.002**	0.11(0.33)	0.293
CMC 1994	→	CES-D 1994	**0.15**(0.16)	**0.013**	-0.01(0.35)	0.909
CMC 2001	→	CES-D 2001	**0.14**(0.22)	**0.045**	-0.02(0.49)	0.912
CMC 2011	→	CES-D 2011	**0.30**(0.46)	**0.028**	-0.12(1.39)	0.731
Disability 1986	→	CES-D 1986	0.16(0.12)	<0.001	0.22(0.20)	<0.001
Disability 1989	→	CES-D 1989	0.16(0.14)	<0.001	0.36(0.22)	<0.001
Disability 1994	→	CES-D 1994	0.13(0.11)	<0.001	0.20(0.17)	<0.001
Disability 2001	→	CES-D 2001	**0.32**(0.12)	**<0.001**	0.14(0.22)	0.087
Disability 2011	→	CES-D 2011	0.29(0.21)	<0.001	0.50(0.41)	0.001
CMC 1989	→	CES-D 1994	**-0.15**(0.13)	**0.002**	-0.02(0.27)	0.781
CMC 1986	→	CES-D 1989	**-0.10**(0.14)	**0.021**	-0.07(0.26)	0.365
CMC 1994	→	CES-D 2001	-0.08(0.15)	0.106	0.04(0.34)	0.708
CMC 2001	→	CES-D 2011	-0.12(0.28)	0.152	-0.02(0.68)	0.922
Disability 1986	→	CES-D 1989	-0.11(0.14)	0.002	-0.21(0.20)	<0.001
Disability 1989	→	CES-D 1994	-0.08(0.12)	0.013	-0.21(0.20)	<0.001
Disability 1994	→	CES-D 2001	-0.19(0.12)	<0.001	-0.17(0.19)	0.007
Disability 2001	→	CES-D 2011	-0.11(0.22)	0.112	-0.13(0.35)	0.245
CMC 1986	→	CMC 1989	1.01(0.03)	<0.001	0.98(0.04)	<0.001
CMC 1989	→	CMC 1994	0.94(0.03)	<0.001	0.89(0.04)	<0.001
CMC 1994	→	CMC 2001	0.85(0.03)	<0.001	0.85(0.05)	<0.001
CMC 2001	→	CMC 2011	0.79(0.05)	<0.001	0.70(0.06)	<0.001
CES-D 1986	→	CES-D 1989	0.86(0.08)	<0.001	0.87(0.10)	<0.001
CES-D 1989	→	CES-D 1994	0.95(0.05)	<0.001	1.00(0.09)	<0.001
CES-D 1994	→	CES-D 2001	0.84(0.05)	<0.001	0.90(0.08)	<0.001
CES-D 2001	→	CES-D 2011	0.88(0.06)	<0.001	0.78(0.10)	<0.001
Disability 1986	→	Disability 1989	1.27(0.06)	<0.001	1.10(0.08)	<0.001
Disability 1989	→	Disability 1994	0.96(0.04)	<0.001	0.87(0.06)	<0.001
Disability 1994	→	Disability 2001	1.06(0.04)	<0.001	1.14(0.08)	<0.001
Disability 2001	→	Disability 2011	1.04(0.04)	<0.001	0.97(0.07)	<0.001
Female	→	Disability 1986	0.05(0.02)	<0.001	0.12(0.04)	<0.001
Age	→	Disability 1986	0.32(0.00)	<0.001	0.29(0.00)	<0.001
Education	→	Disability 1986	-0.12(0.00)	<0.001	-0.18(0.01)	<0.001
Female	→	CMC 1986	0.05(0.03)	<0.001	0.09(0.05)	<0.001
Education	→	CMC 1986	-0.16(0.01)	<0.001	-0.14(0.01)	<0.001
Age	→	CMC 1986	0.43(0.00)	<0.001	0.51(0.00)	<0.001
Female	→	CES-D 1986	0.06(0.13)	<0.001	0.07(0.21)	0.004
Age	→	CES-D 1986	-0.17(0.01)	<0.001	-0.36(0.01)	<0.001
Education	→	CES-D 1986	-0.19(0.02)	<0.001	-0.21(0.03)	<0.001

CMCs: Chronic Medical Conditions; CES-D: Center for Epidemiological Studies-Depression
Numbers in bold were significant among one race.

Model 3 proposed a path from CMC to disability at each time, and from CMC and disability to the subsequent depressive symptoms. In Model 3, an increase in CMC from 1994 to 2001 only predicted an increase in depressive symptoms form 2001 to 2011 among Whites, but not Blacks (Table 5).

Table 5: Results of Model 3; Black and White differences in longitudinal associations between chronic medical conditions (CMC), disability, and depressive symptoms (CES-D) over 25 years from 1986 to 2011

Predictor		Outcome	Estimate (S.E.) Whites	P	Estimate (S.E.) Blacks	P
CMC 1986	→	Disability 1986	0.32(0.02)	<0.001	0.38(0.03)	<0.001
CMC 1989	→	Disability 1989	0.09(0.02)	<0.001	0.22(0.03)	<0.001
CMC 1994	→	Disability 1994	0.15(0.02)	<0.001	0.26(0.04)	<0.001
CMC 2001	→	Disability 2001	0.15(0.03)	<0.001	0.21(0.05)	<0.001
CMC 2011	→	Disability 2011	0.12(0.04)	<0.001	0.10(0.06)	0.041
CMC 1994	→	Disability 2001	0.00(0.03)	0.910	**-0.15**(0.07)	**0.022**
CMC 1986	→	Disability 1989	-0.21(0.04)	<0.001	-0.27(0.07)	<0.001
CMC 1989	→	Disability 1994	0.03(0.02)	0.293	-0.04(0.04)	0.328
CMC 2001	→	Disability 2011	-0.06(0.05)	0.066	0.14(0.07)	0.029
CMC 1986	→	CES-D 1989	0.07(0.07)	<0.001	0.06(0.11)	0.073
CMC 1994	→	CES-D 2001	0.10(0.07)	<0.001	0.08(0.14)	0.071
CMC 1989	→	CES-D 1994	0.03(0.07)	0.32	0.03(0.13)	0.377
CMC 2001	→	CES-D 2011	**0.08**(0.11)	**0.011**	0.02(0.19)	0.783
Disability 1986	→	CES-D 1989	0.01(0.11)	0.628	0.01(0.17)	0.817
Disability 1989	→	CES-D 1994	0.01(0.10)	0.821	**-0.10**(0.19)	**0.042**
Disability 1994	→	CES-D 2001	-0.02(0.09)	0.441	**-0.10**(0.15)	**0.048**
Disability 2001	→	CES-D 2011	**0.08**(0.12)	**0.032**	0.08(0.19)	0.21
CES-D 1986	→	Disability 1989	**-0.09**(0.01)	**0.003**	-0.05(0.01)	0.207
CES-D 1989	→	Disability 1994	**-0.03**(0.01)	**0.129**	-0.09(0.01)	0.01
CES-D 1994	→	Disability 2001	-0.01(0.01)	0.680	**-0.14**(0.02)	**0.002**
CES-D 2001	→	Disability 2011	-0.11(0.01)	<0.001	0.09(0.02)	0.044
CMC 1986	→	CMC 1989	0.99(0.03)	<0.001	0.96(0.04)	<0.001
CMC 1989	→	CMC 1994	0.93(0.03)	<0.001	0.89(0.04)	<0.001
CMC 1994	→	CMC 2001	0.86(0.03)	<0.001	0.87(0.05)	<0.001
CMC 2001	→	CMC 2011	0.79(0.05)	<0.001	0.70(0.06)	<0.001
CES-D 1986	→	CES-D 1989	0.80(0.07)	<0.001	0.83(0.10)	<0.001
CES-D 1989	→	CES-D 1994	0.94(0.05)	<0.001	0.95(0.08)	<0.001
CES-D 1994	→	CES-D 2001	0.86(0.05)	<0.001	0.90(0.08)	<0.001
CES-D 2001	→	CES-D 2011	0.90(0.06)	<0.001	0.85(0.10)	<0.001
Disability 1986	→	Disability 1989	1.34(0.11)	<0.001	1.07(0.16)	<0.001
Disability 1989	→	Disability 1994	0.81(0.05)	<0.001	0.71(0.08)	<0.001
Disability 1994	→	Disability 2001	0.92(0.05)	<0.001	1.12(0.12)	<0.001
Disability 2001	→	Disability 2011	1.08(0.06)	<0.001	0.73(0.09)	<0.001
Female	→	Disability 1986	0.04(0.02)	0.004	0.10(0.04)	<0.001
Age	→	Disability 1986	0.17(0.00)	<0.001	0.09(0.00)	0.003
Education	→	Disability 1986	-0.09(0.00)	<0.001	-0.13(0.01)	<0.001
Female	→	CES-D 1986	0.08(0.14)	<0.001	0.12(0.21)	<0.001
Age	→	CES-D 1986	-0.11(0.00)	<0.001	-0.24(0.01)	<0.001
Education	→	CES-D 1986	-0.22(0.02)	<0.001	-0.26(0.03)	<0.001
Female	→	CMC 1986	0.06(0.04)	<0.001	0.09(0.05)	<0.001

CMCs: Chronic Medical Conditions, CES-D: Center for Epidemiological Studies-Depression.

In Model 4, paths are from CMC to disability and depressive symptoms at each time, from CMC to subsequent depressive symptoms, and from depressive symptoms to subsequent disability. Based on Model 4, increase in disability from 1986 to 1989 predicted increase in depressive symptoms at the same time period among Whites, but not Blacks (Table 6).

Table 6: Results of Model 4; Black and White differences in longitudinal associations between CMC (CMC), disability, and depressive symptoms (CES-D) over 25 years from 1986 to 2011

Predictor		Outcome	Estimate (S.E.)	P	Estimate (S.E.)	P
			Whites		Blacks	
Disability 1986	→	CES-D 1986	0.05(0.21)	0.340	0.01(0.83)	0.945
Disability 1989	→	CES-D 1989	0.02(0.35)	0.835	0.17(0.70)	0.353
Disability 1994	→	CES-D 1994	0.10(0.13)	0.015	0.19(0.23)	0.008
Disability 2001	→	CES-D 2001	0.27(0.15)	<0.001	0.09(0.25)	0.322
Disability 2011	→	CES-D 2011	0.20(0.30)	0.075	0.39(0.52)	0.042
CMC 1986	→	Disability 1986	0.28(0.02)	<0.001	0.36(0.03)	<0.001
CMC 1989	→	Disability 1989	0.06(0.02)	0.004	0.17(0.03)	<0.001
CMC 1994	→	Disability 1994	0.15(0.02)	<0.001	0.21(0.03)	<0.001
CMC 2001	→	Disability 2001	0.13(0.02)	<0.001	0.19(0.04)	<0.001
CMC 2011	→	Disability 2011	0.09(0.03)	<0.001	0.11(0.06)	0.015
CMC 1986	→	CES-D 1986	0.09(0.17)	0.09	0.17(0.38)	0.121
CMC 1989	→	CES-D 1989	0.17(0.17)	0.003	0.13(0.35)	0.213
CMC 1994	→	CES-D 1994	0.16(0.17)	0.010	-0.01(0.35)	0.899
CMC 2001	→	CES-D 2001	0.15(0.23)	0.043	0.03(0.49)	0.879
CMC 2011	→	CES-D 2011	0.31(0.55)	0.052	-0.17(1.87)	0.729
CES-D 1989	→	Disability 1994	-0.05(0.01)	0.168	-0.07	0.275
CMC 1986	→	CMC 1989	1.01(0.03)	<0.001	0.98(0.04)	<0.001
CMC 1989	→	CMC 1994	0.94(0.03)	<0.001	0.89(0.04)	<0.001
CMC 1994	→	CMC 2001	0.85(0.03)	<0.001	0.87(0.05)	<0.001
CMC 2001	→	CMC 2011	0.80(0.05)	<0.001	0.70(0.06)	<0.001
CES-D 1986	→	CES-D 1989	0.84(0.07)	<0.001	0.87(0.10)	<0.001
CES-D 1989	→	CES-D 1994	0.94(0.05)	<0.001	1.00(0.09)	<0.001
CES-D 1994	→	CES-D 2001	0.84(0.05)	<0.001	0.90(0.08)	<0.001
CES-D 2001	→	CES-D 2011	0.88(0.06)	<0.001	0.81(0.10)	<0.001
Disability 1986	→	Disability 1989	1.00(0.05)	<0.001	0.67(0.08)	<0.001
Disability 1989	→	Disability 1994	0.84(0.04)	<0.001	0.71(0.07)	<0.001
Disability 1994	→	Disability 2001	0.93(0.04)	<0.001	0.87(0.08)	<0.001
Disability 2001	→	Disability 2011	0.97(0.04)	<0.001	0.96(0.08)	<0.001
CMC 1986	→	CES-D 1989	-0.06(0.14)	0.181	-0.05(0.26)	0.529
CMC 1989	→	CES-D 1994	-0.13(0.13)	0.006	-0.01(0.26)	0.890
CMC 1994	→	CES-D 2001	-0.08(0.15)	0.103	0.06(0.33)	0.610
CMC 2001	→	CES-D 2011	-0.12(0.30)	0.140	-0.02(0.80)	0.946
Disability 1986	→	CES-D 1989	-0.02(0.22)	0.667	0.12(0.41)	0.231
Disability 1989	→	CES-D 1994	-0.07(0.13)	0.033	-0.20(0.22)	<0.001
Disability 1994	→	CES-D 2001	-0.17(0.12)	<0.001	-0.16(0.19)	0.008
Disability 2001	→	CES-D 2011	-0.07(0.24)	0.363	-0.07(0.32)	0.480
Female	→	CMC 1986	0.05(0.03)	<0.001	0.09(0.05)	<0.001
Age	→	CMC 1986	0.43(0.00)	<0.001	0.51(0.00)	<0.001
Education	→	CMC 1986	-0.15(0.01)	<0.001	-0.15(0.01)	<0.001
Female	→	Disability 1986	0.04(0.03)	0.015	0.10(0.05)	<0.001
Age	→	Disability 1986	0.21(0.00)	<0.001	0.12(0.00)	<0.001
Education	→	Disability 1986	-0.10(0.00)	<0.001	-0.12(0.01)	<0.001
Female	→	CES-D 1986	0.07(0.13)	<0.001	0.09(0.26)	0.004
Age	→	CES-D 1986	-0.16(0.01)	<0.001	-0.33(0.01)	<0.001
Education	→	CES-D 1986	-0.20(0.03)	<0.001	-0.23(0.04)	<0.001

CMC: Chronic Medical Conditions; CES-D: Center for Epidemiological Studies-Depression.

In all hypothetical models, multiple cross-sectional and lagged paths from CMC and disability to depressive symptoms that were present for Whites were absent for Blacks.

DISCUSSION

This study compared Blacks and Whites for 1) additive effects of CMC and disability on depressive symptoms, and 2) additive effects of CMC and depressive symptoms on disability. For our hypotheses, four alternative hypothetical models were tested because the associations between CMC, disability, and depressive symptoms are bidirectional.[7,47,48] Regardless of the modeling approach, more cross-sectional and lagged paths were found between CMC, disability, and depressive symptoms among Whites than Blacks. For instance, based on our Models 1 and 2, increases in CMC from 1986 to 1989, from 1994 to 2001, and from 2001 to 2011 were only predictive of increase in depressive symptoms at the same time periods among Whites but not Blacks. Based on our Model 3, an increase in CMC from 1994 to 2001 only predicted an increase in depressive symptoms form 2001 to 2011 among Whites but not Blacks. Based on Model 4, an increase in disability from 1986 to 1989 predicted an increase in depressive symptoms at the same time period among Whites but not Blacks. All these models suggested that Blacks and Whites differ in the additive effects of CMC and depressive symptoms on disability.

Building on our previous work, our research has shown that differential links between depression and CMC exist between Blacks and Whites; these differential effects based on race may explain the Black-White health paradox, defined as a lower rate of depression for Blacks than Whites, despite their higher rate of CMC.[7,26,27,41,42,44,64] Using various statistical modeling for replication, CMC, depressive symptoms, and disability may have links among Whites that are absent for Blacks.[7,64] Independent longitudinal studies have also suggested that the association between baseline depressive symptoms and subsequent cardiovascular mortality is present for Whites may be difficult to find for Blacks.[13]

Our findings are in line with the positive psychology perspective that suggests flourishing may happen in response to problems in life.[37-39] Keyes argues that flourishing may explain the Black-White health paradox.[7,26,27] Research has suggested that patients with serious medical conditions such as cancer or heart disease have a higher chance of experiencing post-traumatic growth. Blacks may also be more likely to experience a higher rate of growth after trauma.[28-34,36,65] Growth after trauma is a predictor of lower psychological distress and depressive symptoms under stress.[33,36] In this view, Blacks who develop medical conditions or a related disability may experience degrees of flourishing instead of distress.[37-40] Although CMCs are risk factors for distress, such personal transformation lowers the risk of depression.[41,42] As explained above, being from an ethnic minority group has been shown to increase post-traumatic growth.[36]

Jackson and colleagues have also provided one of the explanations for the Black-White health paradox. According to their theory, Blacks may experience lower rates of depression than Whites, despite suffering multiple medical conditions, because they may tend to engage in negative health behaviors (e.g. substance use for men and comfort food eating for women) that impose physiological damage, yet may lower their psychological distress concurrently.[14-18] Jackson's hypothesis explains why the mental-physical health link

is weaker for Blacks than Whites.[15-18] Although our findings do not suggest any mechanisms, they provide additional evidence on role of race in shaping the depression-medical conditions link.[7,17,19,20]

Despite using a nationally representative sample and providing results that are generalizable to the United States adults, the current study is not free of limitations. This study did not analyze type of CMC. In addition, the list of medical conditions was not comprehensive in this study. Furthermore, CMCs were measured based on self-reported data. Fourth, the study included symptoms of depression, rather than diagnosis of clinical depression. Finally, the study used a simple measure of disability. Despite having the above limitations, this study makes a unique contribution to the health disparity literature and provides additional evidence regarding the role of race in changing the links between depression, CMC, and disability.

In line with our previous research, some of the reciprocal associations between depressive symptoms, CMC, and disability were present for Whites but absent for Blacks. Due to some negative paths from disability to depression and from CMC to depression among Blacks, flourishing may be an explanation behind the Black-White health paradox. Additional research, however, is required.[7,64]

To conclude, our findings suggest that regardless of how we conceptualize their associations, race modifies the complex relationship between CMC, disability, and depressive symptoms. No matter if we consider depression and medical conditions as additive causes of disability, or medical conditions and disability as additive causes of depressive symptoms, Blacks and Whites differ in the longitudinal associations between these health problems over time.

CONFLICT OF INTEREST

There is no Conflict of interest in this study.

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REFERENCES

1. Cabassa LJ, Humensky J, Druss B, Lewis-Fernández R, Gomes AP, Wang S, et al. Do race, ethnicity, and psychiatric diagnoses matter in the prevalence of multiple chronic medical conditions? Med Care. 2013; 51(6): 540-7.
2. Whitson HE, Hastings SN, Landerman LR, Fillenbaum GG, Cohen HJ, Johnson KS. Black-white disparity in disability: The role of medical conditions. J Am Geriatr Soc. 2011; 59(5): 844-50.
3. Clark DO, Maddox GL. Racial and social correlates of age-related changes in functioning. J Gerontol. 1992; 47(5): S222-32.
4. Kelley-Moore JA, Ferraro KF. The black/white disability gap: Persistent inequality in later life? J Gerontol B Psychol Sci Soc Sci. 2004; 59(1): S34-43.
5. Ostchega Y, Harris TB, Hirsch R, Parsons VL, Kington R. The prevalence of functional limitations and disability in older persons in the US: Data from the National Health and Nutrition Examination Survey III. J Am Geriatr Soc. 2000; 48(9): 1132-5.
6. Bromberger JT, Harlow S, Avis N, Kravitz HM, Cordal A. Racial/ethnic differences in the prevalence of depressive symptoms among middle-aged women: The Study of Women's Health Across the Nation (SWAN). Am J Public Health. 2004; 94(8): 1378-85.

7. Assari S, Burgard S, Zivin K. Long-Term Reciprocal Associations Between Depressive Symptoms and Number of Chronic Medical Conditions: Longitudinal Support for Black-White Health Paradox. J Racial Ethn Health Disparities. 2015; 2(4): 589-97.
8. Lewis TT, Guo H, Lunos S, Mendes de Leon CF, Skarupski KA, Evans DA, et al. Depressive symptoms and cardiovascular mortality in older black and white adults: evidence for a differential association by race. Circ Cardiovasc Qual Outcomes. 2011; 4(3): 293-9.
9. Gottlieb SS, Khatta M, Friedmann E, Einbinder L, Katzen S, Baker B, et al. The influence of age, gender, and race on the prevalence of depression in heart failure patients. J Am Coll Cardiol. 2004; 43(9): 1542-9.
10. Watkins DC, Assari S, Johnson-Lawrence V. Race and Ethnic Group Differences in Comorbid Major Depressive Disorder, Generalized Anxiety Disorder, and Chronic Medical Conditions. J Racial Ethn Health Disparities. 2015; 2(3): 385-94.
11. Assari S. Chronic Kidney Disease, Anxiety and Depression among American Blacks; Does Ethnicity Matter? Int J Travel Med Glob Health. 2014; 2(4): 133-9.
12. Assari S, Lankarani MM. Race and ethnic differences in associations between cardiovascular diseases, anxiety, and depression in the United States. Int J Travel Med Glob Health. 2014; 2(3): 103-9.
13. Capistrant BD, Gilsanz P, Moon JR, Kosheleva A, Patton KK, Glymour MM. Does the association between depressive symptoms and cardiovascular mortality risk vary by race? Ethn Dis. 2013; 23(2): 155-60.
14. Mezuk B, Rafferty JA, Kershaw KN, Hudson D, Abdou CM, Lee H, et al. Reconsidering the role of social disadvantage in physical and mental health: Stressful life events, health behaviors, race, and depression. Am J Epidemiol. 2010; 172(11): 1238-49.
15. Mezuk B, Abdou CM, Hudson D, Kershaw KN, Rafferty JA, Lee H, et al. "White Box" Epidemiology and the Social Neuroscience of Health Behaviors: The Environmental Affordances Model. Soc Ment Health. 2013; 3(2).
16. Jackson JS, Knight KM, Rafferty JA. Race and unhealthy behaviors: Chronic stress, the HPA axis, and physical and mental health disparities over the life course. Am J Public Health. 2010; 100(5): 933-9.
17. Assari S. Association between obesity and depression among American Blacks: Role of ethnicity and gender. J Racial Ethn Health Disparities. 2014; 1(1): 36-44.
18. Jackson JS, Knight KM, Rafferty JA. Race and unhealthy behaviors: chronic stress, the HPA axis, and physical and mental health disparities over the life course. Am J Public Health. 2010; 100(5): 933-9.
19. Assari S. Additive Effects of Anxiety and Depression on Body Mass Index among Blacks: Role of Ethnicity and Gender. Int Cardiovasc Res J. 2014; 8(2): 44-51.
20. Assari S, Caldwell CH. Gender and ethnic differences in the association between obesity and depression among black adolescents. J Racial Ethn Health Disparities. 2015; 2(4): 481-93.
21. Matheson FI, Smith KL, Fazli GS, Moineddin R, Dunn JR, Glazier RH. Physical health and gender as risk factors for usage of services for mental illness. J Epidemiol Community Health. 2014; 68(10): 971-8.
22. Patten SB. Long-term medical conditions and major depression in the Canadian population. Can J Psychiatry. 1999; 44(2): 151-7.
23. Kim JM, Stewart R, Kim SW, Yang SJ, Shin IS, Yoon JS. Insomnia, depression, and physical disorders in late life: A 2-year longitudinal community study in Koreans. Sleep. 2009; 32(9): 1221-8.

24. Patten SB. Long-term medical conditions and major depression in a Canadian population study at waves 1 and 2. J Affect Disord. 2001; 63(1-3): 35-41.
25. Husaini BA. Predictors of depression among the elderly: Racial differences over time. Am J Orthopsychiatry. 1997; 67(1): 48-58.
26. Keyes CL. The Black-White paradox in health: Flourishing in the face of social inequality and discrimination. J Pers. 2009; 77(6): 1677-706.
27. Barnes DM, Keyes KM, Bates LM. Racial differences in depression in the United States: How do subgroup analyses inform a paradox? Soc Psychiatry Psychiatr Epidemiol. 2013; 48(12): 1941-9.
28. Jim HS, Jacobsen PB. Posttraumatic stress and posttraumatic growth in cancer survivorship: A review. Cancer J. 2008; 14(6): 414-9.
29. Brix SA, Bidstrup PE, Christensen J, Rottmann N, Olsen A, Tjonneland A, et al. Post-traumatic growth among elderly women with breast cancer compared to breast cancer-free women. Acta Oncol. 2013; 52(2): 345-54.
30. Morrill EF, Brewer NT, O'Neill SC, Lillie SE, Dees EC, Carey LA, et al. The interaction of post-traumatic growth and post-traumatic stress symptoms in predicting depressive symptoms and quality of life. Psychooncology. 2008; 17(9): 948-53.
31. Svetina M, Nastran K. Family relationships and post-traumatic growth in breast cancer patients. Psychiatr Danub. 2012; 24(3): 298-306.
32. Kolokotroni P, Anagnostopoulos F, Tsikkinis A. Psychosocial factors related to posttraumatic growth in breast cancer survivors: A review. Women Health. 2014; 54(6): 569-92.
33. Barskova T, Oesterreich R. Post-traumatic growth in people living with a serious medical condition and its relations to physical and mental health: A systematic review. Disabil Rehabil. 2009; 31(21): 1709-33.
34. Teodorescu Ds, Heir T, Hauff E, Wentzel-Larsen T, Lien L. Mental health problems and post-migration stress among multi-traumatized refugees attending outpatient clinics upon resettlement to Norway. Scand J Psychol. 2012; 53(4): 316-32.
35. Devine KA, Reed-Knight B, Loiselle KA, Fenton N, Blount RL. Posttraumatic growth in young adults who experienced serious childhood illness: a mixed-methods approach. J Clin Psychol Med Settings. 2010; 17(4): 340-8.
36. Kleim B, Ehlers A. Evidence for a curvilinear relationship between posttraumatic growth and posttrauma depression and PTSD in assault survivors. J Trauma Stress. 2009; 22(1): 45-52.
37. Keyes CL, Simoes EJ. To flourish or not: positive mental health and all-cause mortality. Am J Public Health. 2012; 102(11): 2164-72.
38. Keyes CL. The mental health continuum: From languishing to flourishing in life. J Health Soc Behav. 2002; 43(2): 207-22.
39. Ryff CD, Keyes CL, Hughes DL. Status inequalities, perceived discrimination, and eudaimonic well-being: do the challenges of minority life hone purpose and growth? J Health Soc Behav. 2003; 44(3): 275-91.
40. Gershuny BS, Thayer JF. Relations among psychological trauma, dissociative phenomena, and trauma-related distress: A review and integration. Clin Psychol Rev. 1999; 19(5): 631-57.
41. Gonzalez HM, Tarraf W, Whitfield KE, Vega WA. The epidemiology of major depression and ethnicity in the United States. J Psychiatr Res. 2010; 44(15): 1043-51.
42. Williams DR, Gonzalez HM, Neighbors H, Nesse R, Abelson JM, Sweetman J, et al. Prevalence and distribution of major depressive disorder in African Americans,

Caribbean blacks, and non-Hispanic whites: results from the National Survey of American Life. Arch Gen Psychiatry. 2007; 64(3): 305-15.
43. Cabassa LJ, Humensky J, Druss B, Lewis-Fernandez R, Gomes AP, Wang S, et al. Do race, ethnicity, and psychiatric diagnoses matter in the prevalence of multiple chronic medical conditions? Med Care. 2013; 51(6): 540-7.
44. Johnson-Lawrence V, Griffith DM, Watkins DC. The effects of race, ethnicity, and mood/anxiety disorders on the chronic physical health conditions of men from a national sample. Am J Mens Health. 2013; 7(4): 58S-67S.
45. Mendes de Leon CF, Barnes LL, Bienias JL, Skarupski KA, Evans DA. Racial disparities in disability: recent evidence from self-reported and performance-based disability measures in a population-based study of older adults. J Gerontol B Psychol Sci Soc Sci. 2005; 60(5): S263-71.
46. Skarupski KA, de Leon CF, Bienias JL, Scherr PA, Zack MM, Moriarty DG, et al. Black-white differences in health-related quality of life among older adults. Qual Life Res. 2007; 16(2): 287-96.
47. Turvey CL, Schultz SK, Beglinger L, Klein DM. A longitudinal community-based study of chronic illness, cognitive and physical function, and depression. Am J Geriatr Psychiatry. 2009; 17(8): 632-41.
48. Stegenga BT, Nazareth I, Torres-Gonzalez F, Xavier M, Svab I, Geerlings MI, et al. Depression, anxiety and physical function: exploring the strength of causality. J Epidemiol Community Health. 2012; 66(7): e25.
49. House JS, Lepkowski JM, Kinney AM, Mero RP, Kessler RC, Herzog AR. The social stratification of aging and health. J Health Soc Behav. 1994; 35(3): 213-34.
50. House JS, Kessler RC, Herzog AR. Age, socioeconomic status, and health. Milbank Q. 1990; 68(3): 383-411.
51. Radloff LS. The CES-D scale a self-report depression scale for research in the general population. Appl Psychol Meas. 1977; 1(3): 385-401.
52. Amtmann D, Kim J, Chung H, Bamer AM, Askew RL, Wu S, et al. Comparing CESD-10, PHQ-9, and PROMIS depression instruments in individuals with multiple sclerosis. Rehabil Psychol. 2014; 59(2): 220-9.
53. Zhang W, O'Brien N, Forrest JI, Salters KA, Patterson TL, Montaner JS, et al. Validating a shortened depression scale (10 item CES-D) among HIV-positive people in British Columbia, Canada. PloS one. 2012; 7(7): e40793.
54. Andresen EM, Malmgren JA, Carter WB, Patrick DL. Screening for depression in well older adults: evaluation of a short form of the CES-D (Center for Epidemiologic Studies Depression Scale). Am J Prev Med. 1994; 10(2): 77-84.
55. Tsai PF, Jirovec MM. The relationships between depression and other outcomes of chronic illness caregiving. BMC Nurs. 2005; 4(1): 3.
56. Kline RB. Principles and practice of structural equation modeling: Guilford publications; 2010.
57. Read S, Grundy E. Allostatic load and health in the older population of England: a crossed-lagged analysis. Psychosom Med. 2014; 76(7): 490-6.
58. Allison PD. Missing data: Sage publications; 2001.
59. Arbuckle AJ. Amos 20 user's guide. Crawfordville, FL: Amos Development. 2012.
60. Wheaton B, Muthen B, Alwin DF, Summers GF. Assessing reliability and stability in panel models. Sociol Methodol. 1977; 8(1): 84-136.
61. Hu Lt, Bentler PM. Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. Struct Equ Modeling J. 1999; 6(1): 1-55.

62. Lei M, Lomax RG. The effect of varying degrees of nonnormality in structural equation modeling. Struct Equ Mo. 2005; 12(1): 1-27.
63. Lindwall M, Larsman P, Hagger MS. The reciprocal relationship between physical activity and depression in older European adults: A prospective cross-lagged panel design using SHARE data. Health Psychol. 2011; 30(4): 453.
64. Assari S, Lankarani MM. Chronic Medical Conditions and Negative Affect; Racial Variation in Reciprocal Associations Over Time. Front Psychiatry. 2016; 7: 140. doi: 10.3389/fpsyt.2016.00140.
65. Leung YW, Alter DA, Prior PL, Stewart DE, Irvine J, Grace SL. Posttraumatic growth in coronary artery disease outpatients: Relationship to degree of trauma and health service use. J Psychosom Res. 2012; 72(4): 293-9.
66. Tsai TY, Livneh H, Lu MC, Tsai PY, Chen PC, Sung FC. Increased risk and related factors of depression among patients with COPD: A population-based cohort study. BMC Public Health. 2013; 13: 976.
67. Patten SB, Williams JV, Lavorato DH, Modgill G, Jette N, Eliasziw M. Major depression as a risk factor for chronic disease incidence: Longitudinal analyses in a general population cohort. Gen Hosp Psychiatry. 2008; 30(5): 407-13.
68. Pudrovska T. What makes you stronger: age and cohort differences in personal growth after cancer. J Health Soc Behav. 2010; 51(3): 260-73.

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