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Residual Effects of Restless Sleep over Depressive Symptoms on Chronic Medical Conditions: Race by Gender Differences

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Abstract

Background—Sleep and depression are comorbid problems that contribute to the development of chronic medical conditions (CMC) over time. Although racial and gender differences in the bidirectional associations between sleep, depression, and CMC are known, very limited information exists on heterogeneity of the residual effects of sleep problems over depressive symptoms on CMC across race by gender groups.

Aim—Using a life-course perspective, the present study compared race by gender groups for residual effects of restless sleep over depressive symptoms on CMC.

Methods—We used data from waves 1 (year 1986), 4 (year 2001), and 5 (year 2011) of the Americans' Changing Lives Study (ACL). The study followed 294 White men, 108 Black men, 490 White women, and 237 Black women for 25 years. Restless sleep, depressive symptoms (Center for Epidemiological Studies-Depression Scale [CES-D]) and number of chronic medical conditions (hypertension, diabetes, chronic lung disease, heart disease, stroke, cancer, and arthritis) were measured in 1986, 2001, and 2011. We employed multi-group cross-lagged modeling, with chronic medical conditions as the outcome, and race by gender as the groups.

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Authors' contribution:

The study was designed and drafted by Assari. Data analysis was conducted by Assari. Sonnega, Leggett, and Pepin contributed to the draft and revision. All authors approved the last version.

Conflict of Interest

Assari, Sonnega, Leggett, and Pepin declare that they have no conflicts of interest.

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.

Results—Major group differences were found in the residual effect of restless sleep on CMC over depressive symptoms across race by gender groups. Restless sleep in 2001 predicted CMC 10 years later in 2011 among Black women (Standardized Adjusted $B=.135$, $P<.05$) and White men (Standardized Adjusted $=0.145$, $P<.01$) and White women (Standardized Adjusted $B=.171$, $P<.001$) but not Black men (Standardized Adjusted $B = .001$, $P > 0.05$).

Conclusion—Race by gender heterogeneity in the residual effect of restless sleep over depressive symptoms on CMC over 25 years suggests that comorbid poor sleep and depressive symptoms differently contribute to development of multi-morbidity among subpopulations based on the intersection of race and gender. Thus, interventions that try to prevent comorbid sleep problems and depression as a strategy to prevent medical conditions may benefit from tailoring based on the intersection of race and gender.

Keywords

Additive Effect; Residual Effects; Intersectionality; Ethnic Groups; Sleep Quality; Depressive Symptoms; Chronic Medical Conditions

Background

Sleep problems (1–3) and depressive symptoms (4) are comorbid mental health problems and both contribute to development of chronic medical conditions (CMC) across populations. Race and gender may, however, alter how poor sleep (5) and depression (6,7) increase the risk of CMC at the community level. For instance, the link between poor sleep and diabetes is stronger for Whites than Blacks (5), and baseline depressive symptoms increase the risk of CMC over 25 years among Whites but not Blacks (6).

Both cross-sectional (8,9) and longitudinal (10,11) studies have documented a link between poor sleep and CMC. Reviews have shown that sleep problems tend to co-occur with a variety of CMC including cardiovascular, pulmonary, gastrointestinal, and infectious diseases (8). In a community sample of 772 individuals, those with insomnia were significantly more likely to report a CMC (9). Among community dwelling adults aged 55 to 84, 41% of those with 4 or more CMC, 22% of those with 1 to 3 conditions, and only 10% of those with no CMC reported fair or poor sleep quality. Similarly, 69% of individuals with 4 or more CMC, 52% of those with one to 3 CMC, and 36% of those with no CMC reported sleep problems (12).

Furthermore, the effects of race on sleep (5,13,14) and depression (15,16) also depend on gender. While cross-sectional studies have suggested that Black men report less nighttime awakenings but more daytime sleepiness than Black women, White men, and White women (17,18), longitudinal research has documented a higher risk of sleep disturbance over time among Black women compared to other race by gender groups (19,20). The link between depression and medical conditions also appears to depend on race and gender (21–25). For instance, major depressive disorder (MDD) is associated with higher risk of obesity among Black women, but lower risk of obesity among Black men (22). When the additive effects of anxiety and depression on obesity are considered, among Black women, lifetime generalized anxiety disorder (GAD), but not MDD, was associated with high body mass index (BMI)

(24). In another study, among Whites but not Blacks, baseline depressive symptoms predicted an increase in CMC over 25 years of follow-up (6). In a nationally representative sample, lifetime MDD was associated with at least one CMC among Blacks, but not Whites (26). Because of these findings, Griffith and colleagues (27) suggested that the residual effects of sleep problems over depressive symptoms on medical conditions should be compared in race and gender groups simultaneously to better understand how the intersection of race and gender shapes these complex links across diverse populations.

There are several reasons to compare race by gender groups for the residual effects of poor sleep over depression on CMC. First and foremost, despite the well-known comorbidity between sleep and depression (28,29), very little is known about how race and gender modify the additive effects of sleep problems and depression on CMC over time. Due to the racial and gender differences in the distribution and pattern of comorbidities between sleep problems (5,13,14), depressive symptoms (30–37), and CMC (26,30,31), the residual effect of poor sleep (38) over depression (6,26) on CMC may also be race and gender dependent (6,26). Other support for this argument comes from studies suggesting that race, gender, and their intersection change the correlates of depression (6,22,24,25,26), as well as residual effects of other risk factors while depression is controlled (39–42). For instance, according to the Black-White health paradox (43,44), despite higher prevalence of CMC (30,31), Blacks are less likely to be depressed than Whites (15,16). Interestingly, the results of this literature are mixed (26,45,46), and some studies have even reported a stronger link between depression and CMC among Blacks than Whites (7,45,47). Finally, there is a need for long-term, longitudinal studies that examine potential mechanisms behind health disparities and begin to sort out potential causal ordering. Most of the literature on the moderating effects of race on the contribution of depression (6,7,26,45) and sleep (5) on CMC has used cross-sectional designs or only short-term follow-up (6,7,26,45), limiting causal inference.

In this study we applied a life-course perspective to better understand race by gender differences in the residual effects of sleep problems over depressive symptoms on CMC. Based on this approach, understanding the subsequent health of individuals requires information on cumulative and additive exposures to multiple risk factors over the course of life (48–50). This is particularly appropriate for studying racial and ethnic health disparities in the U.S. where Blacks experience higher levels of accumulation of exposures at different life stages that collectively contribute to disparities in racial differences in morbidity and mortality (50). According to this framework, CMC are the final product of a number of underlying processes that operate additively and multiplicatively across the lifespan even if CMC develops late in life (48). The major emphasis is on the relevance of previous and cumulative exposures that collectively explain subsequent distributions of the disease in the population. In this study, we hypothesize that poor sleep and depressive symptoms in mid-life will have long-term health effects that may be detectable decades later. Given prior research, we expect these effects to differ based on the intersection of race and gender.

Methods

Design and setting

Data were from wave 1 (1986), wave 4 (2001), and wave 5 (2011) of the Americans' Changing Lives Study (ACL), a nationally-representative longitudinal study of the United States (U.S.) population conducted at the University of Michigan. Details of the study design, sampling, and data collection methods are published elsewhere (51,52).

Participants and sampling

The ACL used a stratified multistage probability sampling strategy, with oversampling of African Americans and those who are age 60 and older. In 1986, the study enrolled 3,617 community-dwelling adults age 25 or older who lived in the continental U.S. Wave 1 included 70% of sampled households and 68% of sampled individuals. Waves 4 and 5 included 74% and 81% of survivors in 2001/02, and 2011/12, respectively. The current analysis is limited to 1,129 individuals who were followed for 25 years, composed of White men (n=294), Black men (n=108), White women (n=490), and Black women (n=237). Data were collected via face-to-face interviews in the first wave, while waves 4 to 5 were conducted via either face-to-face or telephone interviews. In waves 4 and 5, for a small number of cases, when participants were unavailable for a given wave, data were collected from a proxy interviewee.

Measures

Baseline data were collected on demographic characteristics, socioeconomic status, restless sleep, depressive symptoms, and number of CMC. Sleep problems and depressive symptoms were measured at wave 1 and wave 4. Number of CMC was measured at wave 1, wave 4, and wave 5.

Socio-demographics

Demographic variables included age (continuous measure), gender (dichotomous variable with male as the reference group), and race (Black and White, with White as the reference group). Education (less than 12 years of education, high school degree or some college [reference group], and college degree or higher) was used to measure socioeconomic status. Groups were based on the intersection of gender and race.

Depressive Symptoms

Depressive symptoms were measured with a brief version of the Center for Epidemiological Studies-Depression scale (CES-D) which included 10-items (53). Items measured the extent to which in the past week respondents felt depressed, happy, lonely, sad, that everything was an effort, 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. The sleep item was removed from the depression scale and used to indicate sleep problems (below). Item responses were 1 ("hardly ever") to 3 ("most of the time"). Positively worded items were reverse-coded, and a total score was computed across the 10 items (54–56), resulting in a continuous measure of depressive symptoms for baseline and follow-up, ranging from 10 to 30. Higher scores

indicated greater severity of depressive symptoms. This abbreviated CES-D has acceptable reliability and a similar factor structure to the original version (57).

Number of Chronic Medical Conditions (CMC)

Number of CMC was measured based on self-reported data. Participants were asked whether a health care provider had ever told them they had any of the following seven CMC: hypertension, diabetes, chronic lung disease, heart disease, stroke, cancer, and arthritis. Participants were also asked if they were currently taking medication for these conditions. Based on dichotomous responses, a summed score was calculated, ranging from 0 to 7, where a higher score indicated more CMC. Thus the count of CMC at each time point (e.g. CMC 2011) was the number of all CMC a patient had at the time of interview (e.g. 2011). A detailed description of the measurement of CMC is provided in House and colleagues (52).

Restless Sleep

We used the following item to measure sleep problems: “During the past week, my sleep was restless.” Responses ranged from 1 (“hardly ever”) to 3 (“most of the time”). The same single item measure has been previously used to measure restless sleep in the absence (58,59) or presence (60–62) of other domains of sleep quality. Burgard and Ailshire in 2009 (59) and Leggett and colleagues in 2015 (58) used the same single item measure to study sleep disturbance. Self-reported restless sleep is shown to be associated with adverse sleep symptoms, sleep burden, and high risk obstructive sleep apnea (61). Restless sleep, as an important aspect of sleep quality, is now receiving increased research attention (59–61,63). Due to the longitudinal design of the study, we were able to model changes in restless sleep from baseline to follow up.

Statistical Analysis

All statistical analyses were conducted in SPSS 20.0 (IBM Corp, Armonk, NY). For univariate analyses, we reported means or frequencies (%) when appropriate. For bivariate associations, we used the Pearson correlation test, ANOVA, and Tukey test for post-hoc comparisons. For multivariate analysis, we used multi-group cross-lagged models, where groups were defined based on the intersection of race and gender.

Cross-lagged models are effective statistical methods to detect sequential effects over long periods of follow-up in the presence of repeated observations (64, 65). This approach allows for the stability of each construct over time (autoregressive paths) and also estimation of lagged effects between the independent and dependent variables over time. In all models, age, age-squared, and education were covariates. Depressive symptoms and sleep were independent variables, and number of CMC was the dependent variable. The cross-lagged model adjusts for number of CMC at each time point, so the outcome is increase in CMC compared to the earlier wave. Groups were defined based on the intersection of race and gender (6,66). Multi-group models yielded separate estimates for each group (67). Considering age square as a confounder in addition to age is an accepted practice for long term studies which focus on trajectory of health outcomes over a long period of time (68–74). This is because particularly for long follow-up periods, influence of age on health

outcomes is not linear, and adding age square to the model can capture the non-linear effect (j shape or the reverse j shape) of age on health outcome.

To conduct these analyses, we used AMOS (Analysis of Moment Structures), a module in SPSS. AMOS uses Full Information Maximum Likelihood (FIML) to handle missing data (75,76). 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). A CMIN/DF of less than 4, a CFI above .90, and a RMSEA value of .06 or less are indicators of good fit of the model (77,78). 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 fit (79). A significance level of $p < 0.05$ was considered as statistically significant. Adjusted path coefficients with 95% confidence intervals (CI) are reported.

Results

The current analysis included 1,129 individuals who were followed for 25 years and completed surveys in wave 1, wave 4, and wave 5 of the ACL. Participants were White men (n=294), Black men (n=108), White women (n=490), and Black women (n=237).

Table 1 provides descriptive statistics for baseline age, education, CES-D, sleep problems, and CMC by race and gender at baseline and follow-up. The majority of participants (64%) were female with a mean age of 41 (SD = 11) years at baseline in 1986. Although most participants did not have any CMC at baseline, most of them had developed at least one CMC during the 25 years of follow-up. An ANOVA showed significant differences in age, age-squared, education, CES-D, sleep problems, and CMC by race and gender at baseline and follow-up. Post-hoc comparisons are shown in Table 2. Depressive symptoms were lowest among White men, with significant differences from Black men, White men and White women. The only significant difference on restless sleep was between Black women and White men (Table 2).

Table 3 shows correlations between age, education, depressive symptoms, restless sleep, and CMC for each group. Among all groups, restless sleep and depressive symptoms were correlated; however, the depressive symptoms - CMC and also restless sleep - CMC associations varied based on race and gender.

The fit of the multi-group cross-lagged model was good (Chi-square = 322.474, $p = < 0.001$, CFI = .960, $\chi^2/df = 4.243$, RMSEA = .054, 90% CI = .048–.060). Restless sleep in 2001 predicted CMC in 2011 among all race by gender groups except among Black men. Among White men only, baseline depressive symptoms at 1986 predicted CMC in 2001. Among White women, age and age-squared were predictive of CMC in 2011 (Table 4, Figure 1-a to 1-d).

Discussion

According to the findings, the intersection of race and gender modifies the residual effects of restless sleep over depressive symptoms on CMC over a 25-year period in the US. Specifically, Black men were the only group where their restless sleep in 2001 did not predict their CMC in 2011, above and beyond the effect of depressive symptoms. Thus Black men may be less vulnerable to the residual effects of restless sleep on development of CMC compared to Black women, White men, and White women, above and beyond the effect of depressive symptoms.

At baseline, poorer sleep was reported among Black compared to White men. Using data from the 2005 National Health Interview Survey (NHIS), Nunes and colleagues found that compared to Whites, Blacks less frequently report sleeping at least 7 hours and are more likely to experience short sleep duration (80). In 2011 Pigeon and colleagues found higher prevalence of sleep disturbance among Blacks compared to Whites (81). Our bivariate associations (Table 3) show a lower correlation between restless sleep and depressive symptoms (CES-D) at baseline for White men ($r = .35$) than Black men and Black women (r between .51 and .54) or White women ($r = .49$). This finding suggests that White men may not associate sleep problems with other depressive symptoms as much as other groups. That is sleep problems may be a less salient symptom in depressive episodes for White men.

The residual effect of restless sleep over depressive symptoms on CMC which was present in all other race by gender groups was absent among Black men. Based on the Black-White health paradox (6,37,43), Blacks are believed to be more resilient to the effect of mental health on physical health. According to Jackson's hypothesis, the weaker effects of psychological stressors on mental health and CMC of Black men compared to White men may be due to engagement of Black men in negative health behaviors such as smoking or drinking to self-medicate their stressful conditions (82–84). Future research should investigate whether unhealthy coping mechanisms among Black men as hypothesized by Jackson and colleagues, explains their weakened association between mental and physical health problems, a phenomenon called the Black-White health paradox (83,84). Specifically, future research should examine if risk behaviors differently attenuate the effect of restless sleep and depression on CMC among Black men than other groups.

Our finding is not in line with the literature which suggests more severe consequences of psychological problems (such as depression and sleep) for Blacks than Whites (16,26,85). Lower access to and trust in the mental health care system and higher stigma for mental health problems among Blacks than Whites are all expected to worsen the consequences of any mental health problem among Blacks (16,26,86). If they seek care, Blacks are more likely to receive mental health treatment in primary care settings than Whites, which is known to be associated with lower quality of mental health treatments (26,87). Compared to Whites, Blacks endorse more negative beliefs regarding pharmaceutical treatment of psychological problems (16), are more likely to prefer non-pharmacologic approaches (e.g., counseling and prayer), as they more commonly believe that such medications are addictive (88). Blacks are also believed to less frequently receive a prescription for sleep problems

(89). Given this background we might have expected a stronger effect of sleep on CMC for Blacks than Whites, which was not found in the current study.

Depression and poor sleep are both associated with altered inflammation and immune function (90–94). Thus, chronic inflammation and oxidative stress may be the main pathophysiological mechanism behind the effect of depression and sleep on CMC. Significant evidence has shown dysregulation of homeostatic buffering mechanisms regulating oxidation and inflammation that exist in healthy individuals in the presence of depression, sleep disorder, and CMC (95). Thus, changes in inflammation, oxidative stress and immune function may be the mechanisms that at least in part explain the effect of depression and poor sleep on CMC over time (96–100). It is still not clear if peripheral and central inflammation fully mediate the comorbidities between poor sleep, depression, and CMC (101).

The prevention, as well as identification and treatment of sleep disturbance should be considered a core element of CMC prevention strategies and also health disparity reduction programs. The absence of a relationship between restless sleep and CMC in the first follow-up period (i.e., during late mid-life) and the consistency of the restless sleep - CMC associations (except for Black men) in the second follow-up period (i.e., during older adulthood) suggests that it may be especially important to address the problem of restless sleep among older adults compared to adults at mid-life.

Unfortunately, sleep disturbance is particularly under recognized and treated among minorities. Although there are few studies on early detection and screening of poor sleep within communities, building on strategies to screen for other conditions (e.g., depressive symptoms) in community-based settings, development of similar tools to aid in the detection of sleep problems is a reasonable goal (102,103). There may be an opportunity for health coaches or lay community workers to screen, detect, and treat sleep problems in community and primary care settings, particularly for Black women. Effective interventions that improve sleep quality (104,107) may reduce the burden of CMC. Such programs in community and primary health-care settings may benefit from tailoring based on the race of the participant. Zozula et al. (108) argued that race should be considered in conducting an education program for ongoing consultation to primary care providers. The authors conducted a study to provide an educational intervention on sleep disorders for professionals at a community health clinic with high rates of minority patients. The educational intervention resulted in a four-fold increase in referrals for sleep disorder assessment (108).

The results of the current study should be interpreted in the light of four main limitations. First and foremost, restless sleep was measured using a single item with a score ranging from 1 to 3. A single sleep item may not be an adequate substitute for a multi-item measure of overall sleep disturbance (59). Future research should test the replicability of our results using multi-item measures and also other aspects of sleep quality such as sleep efficiency, latency, or disorder which may provide different results. We operationalized our predictor as increase in restless sleep from baseline to follow-up which should not be interpreted as deterioration in sleep quality over time, or chronicity of sleep problems. Such a conclusion requires more detailed information and long-term measurement of sleep problems over a

long period of time, which may better reflect chronicity of sleep problems during the follow-up period. Second, measurement of CMC was limited to self-reported data with only a limited number of CMC assessed. We also did not analyze type of CMC, and sleep is known to have different effects on various medical conditions. Third, we studied symptoms of depression, not clinical depression. Fourth, due to the long duration of follow-up (25 years), bias due to mortality selection, particularly among Blacks and those with high baseline CMC cannot be ruled out. The study has strengths as well. As we used a nationally representative study with over 25-years of follow-up, our findings are generalizable to the U.S. population as a whole. In addition, the current study is one of very few studies with long-term follow-up on race differences on the additive effects of sleep and depression on the development of CMC.

To conclude, our findings suggest a differential residual effect of restless sleep over depressive symptoms on subsequent CMC 25 years based on the intersection of race and gender. Restless sleep in 2001 predicted CMC 10 years later in 2011 among Black women and White men and women but not Black men. This finding suggests that restless sleep and depressive symptoms are differently important as risk factors for development of CMC across diverse populations based on race and gender.

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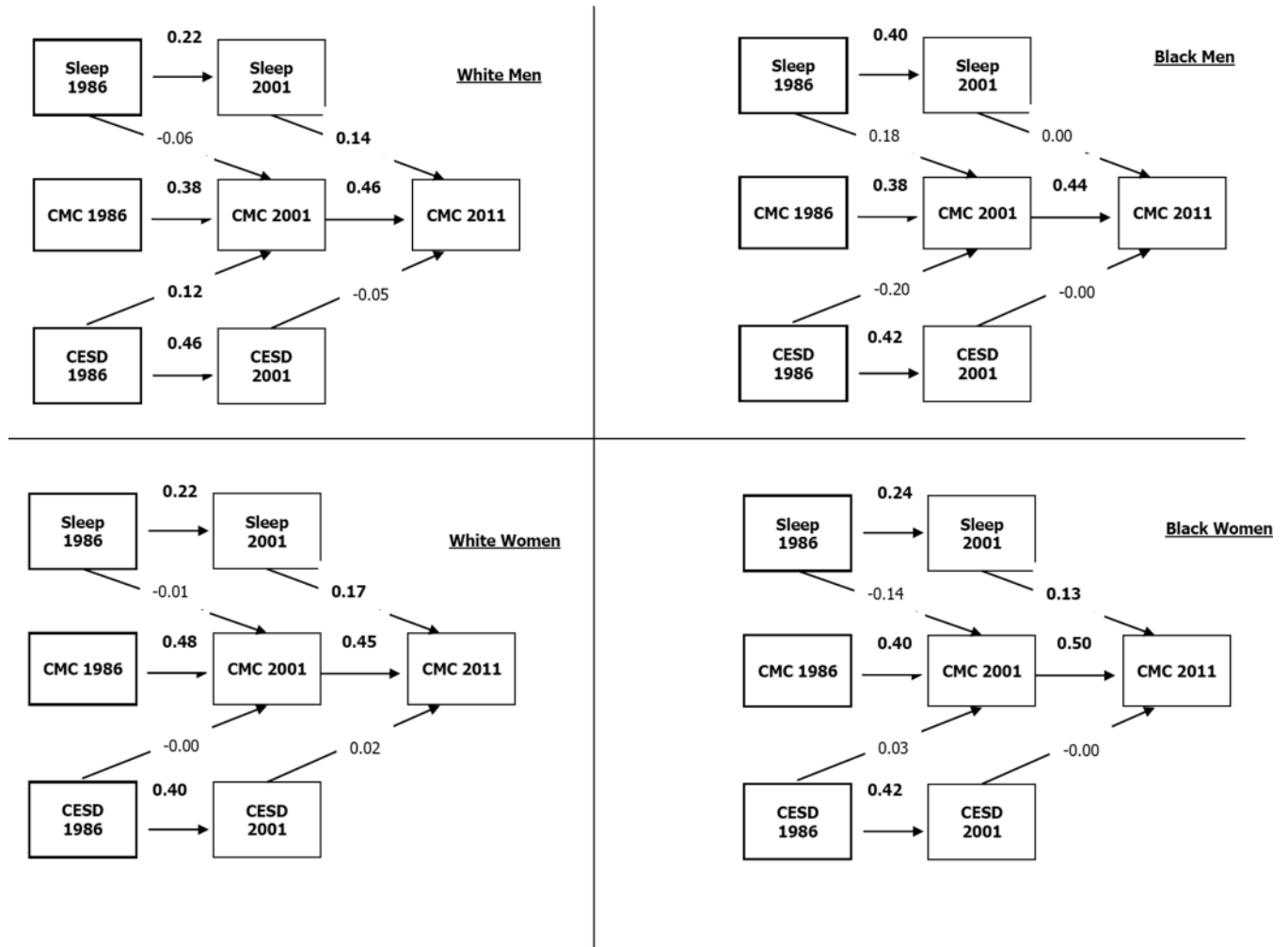
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**Figure 1.**

Summary of cross-lagged model by race and gender

Chi-square = 322.474, $P = <0.001$, CFI = .960, $\chi^2/df = 4.243$, RMSEA = .054 (90% CI = .048–.060)

Table 1

Descriptive statistics and ANOVA comparing race and gender groups

	White Male				Black Male				White Female				Black Female			
	n	Min-max	Mean(SD)	n	Min-max	Mean(SD)	n	Min-max	Mean(SD)	n	Min-max	Mean(SD)	n	Min-max	Mean(SD)	P
Age	294	25.00–70.00	41.27(11.56)	108	25.00–68.00	40.39(10.34)	490	25.00–75.00	44.59(13.11)	237	24.00–71.00	41.47(11.87)	237	24.00–71.00	41.47(11.87)	<.001
Education	294	1.00–17.00	13.50(2.73)	107	3.00–17.00	12.18(2.79)	488	0.00–17.00	12.82(2.53)	237	2.00–17.00	11.84(2.56)	237	2.00–17.00	11.84(2.56)	<.001
CESD-10 1986	294	10.00–224.00	12.75(2.80)	108	10.00–23.00	14.51(3.52)	490	9.00–29.00	13.86(3.82)	237	9.00–29.00	15.61(4.21)	237	9.00–29.00	15.61(4.21)	<.001
CESD-10 2001	256	8.00–24.00	12.11(2.61)	74	9.00–23.00	13.20(3.14)	435	9.00–25.00	12.82(3.26)	159	10.00–28.00	13.91(3.66)	159	10.00–28.00	13.91(3.66)	<.001
Sleep 1986	292	1.00–3.00	1.60(0.65)	108	1.00–3.00	1.61(0.65)	490	1.00–3.00	1.69(0.72)	236	1.00–3.00	1.78(0.72)	236	1.00–3.00	1.78(0.72)	0.023
Sleep 2001	256	1.00–3.00	1.55(0.64)	74	1.00–3.00	1.58(0.72)	435	1.00–3.00	1.65(0.71)	159	1.00–3.00	1.75(0.72)	159	1.00–3.00	1.75(0.72)	0.034
CMC 1986	294	0.00–4.00	0.39(0.66)	108	0.00–4.00	0.53(0.81)	490	0.00–4.00	0.57(0.80)	237	0.00–5.00	0.72(0.88)	237	0.00–5.00	0.72(0.88)	<.001
CMC 2001	256	0.00–4.00	0.93(0.95)	75	0.00–4.00	1.00(0.93)	436	0.00–5.00	1.05(0.93)	159	0.00–5.00	1.33(1.11)	159	0.00–5.00	1.33(1.11)	<.001
CMC 2011	294	0.00–5.00	1.29(1.02)	108	0.00–5.00	1.53(1.06)	490	0.00–5.00	1.45(1.03)	237	0.00–4.00	1.71(0.98)	237	0.00–4.00	1.71(0.98)	<.001

Note CMC: Chronic Medical Conditions, CESD: Center for Epidemiological Studies-Depression

Table 2

Post-hoc analysis for comparison of study variables by race and gender

		Mean difference (SE)	Age	Education	CESD-10 1986	Sleep 1986	CMC 1986
White Male	Black Male	0.88(1.37)	1.32(0.30)*		-1.76(0.41)*	-0.01(0.08)	-0.14(0.09)
	White Female	-3.32(0.90)*	.68(0.19)*		-1.11(0.27)*	-0.09(0.05)	-.18(0.06)*
Black Male	Black Female	-0.20(1.07)	1.67(0.23)*		-2.86(0.32)*	-.177(0.06)*	-.33(0.07)*
	White Male	-0.88(1.37)	-1.32(0.30)*		1.76(0.41)*	0.01(0.08)	0.14(0.09)
White Female	White Female	-4.21(1.30)*	-0.64(0.28)		0.65(0.39)	-0.08(0.07)	-0.04(0.08)
	Black Female	-1.08(1.42)	0.34(0.30)		-1.10(0.42)*	-0.17(0.08)	-0.19(0.09)
Black Male	White Male	3.32*(0.90)*	-.68(0.19)*		1.11(0.27)*	0.09(0.05)	.18(0.06)*
	Black Male	4.21*(1.30)*	0.64(0.28)		-0.65(0.39)	0.08(0.07)	0.04(0.08)
Black Female	Black Female	3.13*(0.97)*	.98(0.21)*		-1.75(0.29)*	-0.09(0.06)	-0.15(0.06)
	White Male	0.20(1.07)	-1.67(0.23)*		2.86(0.32)*	.177(0.06)*	.33(0.07)*
White Female	Black Male	1.08(1.42)	-0.34(0.30)		1.10(0.42)*	0.17(0.08)	0.19(0.09)
	White Female	-3.13(0.97)*	-.98(0.21)*		1.75(0.29)*	0.09(0.06)	0.15(0.06)

* p<0.05;

** p<0.01

*** ; p<0.001;

Table 3

Bivariate correlations by race and gender

	1	2	3	4	5	6
White Male						
1 Age	1	.992**	-.196**	-.076	-.031	.342**
2 Age squared		1	-.206**	-.086	-.036	.335**
3 Education			1	-.145*	-.046	-.182**
4 CESD-10 1986				1	.354**	.146*
5 Sleep 1986					1	.243**
6 CMC 1986						1
Black Male						
1 Age	1	.991**	-.262**	-.208*	-.084	.483**
2 Age squared		1	-.279**	-.192*	-.078	.495**
3 Education			1	-.091	-.147	-.171
4 CESD-10 1986				1	.537**	.091
5 Sleep 1986					1	.126
6 CMC 1986						1
White Female						
1 Age	1	.992**	-.139**	-.257**	-.024	.344**
2 Age squared		1	-.131**	-.249**	-.016	.351**
3 Education			1	-.106*	-.112*	-.218**
4 CESD-10 1986				1	.492**	.048
5 Sleep 1986					1	.171**
6 CMC 1986						1
Black Female						
1 Age	1	.991**	-.350**	-.202**	-.058	.509**
2 Age squared		1	-.354**	-.195**	-.054	.511**
3 Education			1	-.080	-.035	-.397**

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	6	5	4	3	2	1
1						
.097	1					
.508**		1				
1			1			
4 CESD-10 1986	-.015					986
5 Sleep 1986						1986
6 CMC 1986						1986

Note CMC: Chronic Medical Conditions, CESD; Center for Epidemiological Studies-Depression

* p<0.05;
** p<0.01
*** p<0.001;

Table 4

Summary of the cross-lagged model by race and gender

		Estimate (SE)	Black Male	Estimate (SE)	White Female	Estimate (SE)	Black Female
Lagged		White Male					
Sleep 1986	→ CMC 2001	-.061(.091)	.178(.171)	-.015(.063)		-.142(.126)	
CESD-10 1986	→ CMC 2001	.120(.021)*	-.196(.032)	-.005(.012)		.034(.021)	
Sleep 2001	→ CMC 2011	.145(.085)**	.001(.150)	.171(.060)***		.135(.089)*	
CESD-10 2001	→ CMC 2011	-.053(.021)	-.003(.034)	.023(.013)		-.002(.017)	
Autoregressive paths							
Sleep 1986	→ Sleep 2001	.225(.060)***	.403(.119)***	.221(.046)***		.239(.077)**	
CMC 1986	→ CMC 2001	.384(.085)***	.383(.117)***	.477(.049)***		.397(.089)***	
CESD-10 1986	→ CESD-10 2001	.462(.052)***	.418(.095)***	.402(.038)***		.416(.063)***	
CMC 2001	→ CMC 2011	.457(.056)***	.442(.115)***	.449(.046)***		.504(.055)***	
Covariates							
Age	→ CMC 2011	.442(.036)	-.037(.070)	.639(.025)*		.594(.035)	
Age squared	→ CMC 2011	-.402(.000)	.045(.001)	-.583(.000)		-.643(.000)	
Education	→ CMC 2011	-.062(.020)	-.006(.036)	-.009(.016)		-.086(.023)	

* p<0.05;

** p<0.01

*** ; p<0.001;

CMC; Chronic Medical Conditions, CESD; Depression