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Mediating effects of body mass index, physical activity, and emotional distress on the relationship between short sleep and cardiovascular disease

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Abstract

The current study investigated the mediating effects of body mass index (BMI), physical activity, and emotional distress on the association between short sleep duration (<7 hours per 24-hour period) and cardiovascular disease (CVD) and risk factors.

We used data from the National Health Interview Survey, an ongoing nationally representative cross-sectional study of noninstitutionalized US adults (≥18 years) from 2004 to 2013 (N = 206,049). Participants provided information about anthropometric features (height and weight), sociodemographic factors, health behaviors (smoking and physical activity), emotional distress, and physician-diagnosed health conditions, including hypertension, coronary heart disease, diabetes, heart attack, stroke, kidney disease, and cancer. Structural equation modeling was used to assess the mediating effects of physical activity, BMI, and emotional distress on the relationship between short sleep and CVDs and risk factors (coronary heart disease, hypertension, diabetes, chronic kidney disease, heart attack, and stroke).

Of the sample, 54.7% were female, 60.1% identified as white, 17.7% as Hispanic, and 15.4% as black. The mean age of the respondents was 46.75 years (SE = 0.12), with a mean BMI of 27.11 kg/m^2 (SE = 0.02) and approximately 32.5% reported short sleep duration. The main relationship between short sleep and CVD and risk factors was significant (β = 0.08, $P < .001$), as was the mediated effect via BMI (indirect effect = 0.047, $P < .001$), emotional distress (indirect effect = 0.022, $P < .001$), and physical activity (indirect effect = -0.022, $P = .035$), as well as after adjustment for covariates, including age, race, sex, marital status, and income: short sleep and CVD (B = 0.15; SE = 0.01; $P < .001$), BMI (B = 0.05; SE = 0.00; $P < .001$), emotional distress (B = 0.02; SE = 0.00; $P < .001$), and physical activity (B = 0.01; SE = 0.00; $P < .001$).

Our findings indicate that short sleep is a risk factor for CVD and that the relationship between short sleep and CVD and risk factors may be mediated by emotional distress and obesity, and negatively mediated by physical activity.

Abbreviations: BMI = body mass index, CDC = Center for Disease Control and Prevention, CHD = coronary heart disease, CKD = chronic kidney disease, CVD = cardiovascular diseases and risk factors, HPA axis = hypothalamic pituitary adrenal axis, HTN = hypertension, NHIS = National Health Interview Survey, PA = physical activity.

Keywords: cardiovascular disease, emotional distress, obesity, physical activity, sleep

1. Introduction

There is clear and compelling evidence that pharmacotherapy and healthy behavioral and lifestyle practices are effective primary and adjunct strategies for reducing or preventing cardiovascular diseases (CVDs) risk, as well as managing the exacerbating progression of chronic CVD and its comorbidities, such as diabetes and chronic kidney disease (CKD).[1–3] However, limited access and poor adherence to medications

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are significant challenges in reducing CVD risk at the population level.[1,2] Approximately 90% of CVD risk factors and diseases, such as CKD, diabetes, obesity, hypertension (HTN), and heart disease, are preventable through healthy lifestyles and behaviors such as lower BMI, regular physical activity, low emotional stress, healthy sleep, and healthy diet.[4–6]

Although BMI, emotional distress/stress, and physical inactivity are well-established CVD risk factors, there is growing evidence that short sleep duration (<7 hours/24-hour period) is a novel CVD risk factor, as it is associated with HTN, obesity, diabetes,[7,8] myocardial infarctions,[9] and CVD-related mortality.[10] The association between short sleep and CVD might be explained by several direct and indirect biological and physiological mechanisms such as endothelial damage, carotid artery intima thickness, arterial calcification (markers of atherosclerosis), remodeling of the heart, cardiac load/cardiovascular stress, and hormonal dysregulation.[11–16] In general, chronic short sleep duration stresses the body's organ systems by overstimulating the sympathetic, nervous, and endocrine systems, which in turn has deleterious effects on cardiovascular health.[14] Further confirmatory evidence linking short sleep and CVDs and risk factors can be seen in recent research indicating that chronic short sleep duration may cause significant structural changes to the heart, as it is associated with plasma B-type natriuretic peptides, which are counter regulatory hormones linked to ventricular stretching and cardiovascular remodeling caused by increased blood volume.[16]

Conversely, chronic short sleep duration may also be indirectly linked to CVD through emotional distress, physical inactivity, and obesity.[17–25] The indirect effects of short sleep on CVD via emotional distress, obesity, and physical activity are buttressed by evidence indicating that weight loss[26,27]), increased physical activity,[28,29] and low levels of emotional distress[30–32] improve sleep and reduce CVD risk.

Despite the available evidence that habitual short sleep duration is directly and indirectly associated with CVD, to our knowledge, there are no studies that have examined the single and combined mediating roles of emotional distress, BMI, and physical activity on the relationship between short sleep and CVD risk factors and diseases. This study aimed to assess the mediating effects of emotional distress, BMI, and physical activity on the relationship between short sleep duration and CVD risk factors and diseases, using a large nationally representative dataset.

2. Methods

Data (N=911,773) were obtained from the National Health Interview Survey (NHIS) for years 2004 to 2013. For the purpose of our analyses, we included only participants who provided complete data for sleep variables and CVD (N=206,049). The NHIS is a publicly available dataset with de-identified information of noninstitutionalized US adults (> 18 years old) from all 50 states and the District of Columbia in the United States. An original informed consent was obtained by the Center for Disease Control and Prevention (CDC) from subjects, as the data are de-identified and publicly available additional consent was not necessary for the current study. Data can be accessed by visiting the IPUMS Health Survey website at nhis.ipums.org/nhis. The Ethics Review Board of the CDC and the National Center for Health Statistics provided the appropriate guidelines for this survey and is in accordance with the Declaration of Helsinki. The annual response rate for all states was approximately 80%. Participants provided information using computer-assisted personal interviewing program (CAPI).[33,34]

2.1. Variables

2.1.1. Cardiovascular diseases and risk factors. The primary outcome of the current study is a latent construct of 6 self-reported physician diagnosed CVD risk factors and diseases, which include coronary heart disease (CHD), HTN, diabetes, CKD, heart attack, and stroke.

2.1.2. Short sleep duration. Sleep duration, the independent variable, was assessed in full hour increments.[34] Participants were asked "On average, how many hours of sleep do you get in a 24-hour period?" Short sleep duration was coded as total sleep time < 7 hours. Average sleep duration was coded for 7 to 8 hours of sleep in a 24-hour period.

2.1.3. Mediators. Our mediating variables were BMI, emotional distress, and physical activity. BMI (kg/m^2) was calculated on the basis of self-reported height and weight. Emotional distress was measured using the Kessler-6 (K-6) scaling system, which assesses general mood and anxiety symptoms within the last 30 days, with a score of ≥ 13 indicating significant emotional distress.[34,35] Physical activity was assessed using survey questions asking participants their self-reported minutes of moderate and vigorous physical activity per week. Physical activity was defined as a latent variable using tertiles of moderate minutes of physical activity and vigorous minutes of activity per week as indicators. Tertiles of physical activity were classified as none (0 minutes of activity per week), some (1–30 minutes of activity week), and high levels (>30 minutes of physical activity week). Vigorous physical activity was defined as activities causing "heavy sweating or large increases in breathing or heart rate." Moderate physical activity was defined as activities that "cause light sweating or a slight to moderate increase in breathing or heart rate."

2.1.4. Covariates. The covariates used for adjustment in our analyses included age, sex, race/ethnicity, income, education, and marital status. Age and sex were self-reported. Racial/ethnic group was divided into categories (non-Hispanic white, Hispanic, Black/African–American, Native American/Alaskan Native, and Asian/Pacific Islander). Family income was also coded dichotomously (less $35,000 per year vs ≥$35,000 per year). Education was coded using 6 levels (e.g., "never attended school," "grade 1–4," "grade 5–8," "grade 9–12," "1–4 years of college," and "5 or more years of schooling after high school"). Marital status was coded dichotomously (married vs unmarried).

2.1.5. Statistical analysis. Analyses were based on sampling weights provided by the NHIS (from 2004 to 2013) to ensure representativeness of the sample. According to the Center for Disease Control and Prevention, the multifaceted survey analysis technique accounted for the weights, strata, and clusters in this survey design. SPSS version 22 (IBM Corp. Released 2013. IBM SPSS Statistics for Windows, Version 22.0. Armonk, NY: IBM Corp.)[36] and R version 3.3.0 statistical software (Vienna Austria)[37] were used to perform descriptive and inferential analyses, such as the prevalence of variables of interest (age, BMI, short sleep duration, obesity, physical activity, emotional distress, medical conditions). The analysis package "survey"[38] was used to deal with the complex sample design of NHIS and to generate *P* values for Chi-square and Kruskal–Wallis comparisons of participant characteristics frequencies and means between individuals with short and average sleep, excluding individuals who reported long sleep duration (>8 hrs.).

CVD risk factors and diseases, short sleep, and emotional distress were coded as dichotomous variables and BMI was coded continuously with moderate levels of skewness (skewness

statistic = 0.827 in raw data and 0.835 in the clustered data). Physical activity was coded using 2 continuous variables (minutes of moderate physical activity and minutes of vigorous physical activity). These were not normally distributed but were both used as indicators of a latent physical activity variable for the mediation analysis. To remove possible bias caused by these skewed variables, moderate and physical activity were recoded by dividing the variable into tertiles (0, 1–30, and >30 minutes of activity per week).

A structural equation model (SEM) was constructed via MPLUS version 7.2 (Los Angeles, CA: Muthen & Muthen)[39] software to assess main effects of short sleep and mediating role of emotional distress, BMI, and physical activity on CVD. CVD and risk factors construct (hereon forward will be called CVD) was operationalized as a latent variable comprised of 6 conditions: CHD, HTN, diabetes, CKD, heart attack, and stroke. Fit indices for the measurement model of CVD were examined to assure that our latent CVD measure was properly specified. Next, the association between short sleep and latent CVD was examined in unadjusted, covariate-adjusted, and covariate and mediator-adjusted SEMs. We then assessed the direct effect of short sleep on CVD as well as the mediating role of short sleep via our proposed mediators (physical activity, BMI, and emotional distress). We adjusted effects of age, sex, race, family annual income, marital status, and education on all pathways of the SEM. Model was assessed using the following fit indices: Comparative Fit Index (CFI) and Root Mean Square Error of Approximation (RMSEA). Standardized root mean square of the residual (SRMR) was not used as a fit index for the current analyses, as this index is not calculated for a weighted, clustered, or stratified model. Model Chi-square (χ^2) was also reported for all SEM models but was not used to evaluate model fit, as Chi-squared statistics for model fit are easily biased by sample size.[40]

3. Results

3.1. Descriptive statistics

Of the total sample, 60.1% identified as white, 17.7% as Hispanic, and 15.4% as black, and 54.7% as female. The sample had a mean age of 46.8 years (SE = 0.12), a mean BMI of 27.11 kg/m^2 (SE = 0.02), and approximately 32.5% reported short sleep duration (See Table 1).

The prevalence of individual indicators of CVD risk factors and diseases (HTN, heart attack, CHD, stroke, diabetes, and kidney disease) were all significantly greater in Chi-squared analyses in the group with short sleep duration compared with

Table 1

Characteristics of average and short sleepers.

		Total sample (N = 206,049)	Short sleep (N = 66,945)	Average sleep (N = 139,104)	P
Participant characteristics					
Age, mean (SE)		46.75 (0.12)	46.44 (0.13)	46.89 (0.13)	<.001
Male		93,437 (45.3%)	30,061 (44.9%)	63,376 (45.6%)	.990
Race	White	123,629 (60.1%)	19,012 (28.4%)	87,218 (62.7%)	<.001
	Hispanic	36,470 (17.7%)	10,912 (16.3%)	89,304 (64.2%)	
	Black	31,731 (15.4%)	13,121 (19.6%)	19,057 (13.7%)	
	Asian	11,332 (5.5%)	3615 (5.4%)	7789 (5.6%)	
	Native American/Pacific Islander	2266 (1.1%)	803 (1.2%)	1391 (1.0%)	
Family income	<$35,000/y	87,721 (42.6%)	30,574 (45.7%)	57,147 (41.1%)	<.001
	≥$35,000/y	118,328 (57.4%)	36,371 (54.3%)	81,957 (58.9%)	
Education	No schooling	816 (0.4%)	269 (0.4%)	547 (0.4%)	<.001
	Grade 1–4	2349 (1.1%)	752 (1.1%)	1597 (1.1%)	
	Grade 5–8	9888 (4.8%)	3054 (4.6%)	6834 (4.9%)	
	Grade 9–12	71,999 (35.0%)	24,787 (37.0%)	47,212 (33.9%)	
	1–4 y of college	99,326 (48.3%)	32,394 (48.4%)	66,932 (48.1%)	
	5+ y of college or grad school	21,1134 (10.3%)	5525 (8.3%)	15,609 (11.2%)	
Married		94,572 (45.9%)	28,005 (41.8%)	66,567 (47.9%)	<.001
CVD outcomes					
Diabetes		17,654 (8.6%)	6734 (10.1%)	10,920 (7.9%)	<.001
Hypertension		59,478 (28.9%)	21,842 (32.6%)	37,636 (27.1%)	<.001
Kidney disease		3507 (1.7%)	1607 (2.4%)	1900 (1.4%)	<.001
Heart attack		6520 (3.2%)	2541 (3.8%)	3979 (2.9%)	<.001
Coronary heart disease		9034 (4.4%)	3381 (5.1%)	5653 (4.1%)	<.001
Stroke		5158 (2.5%)	2046 (3.1%)	3112 (2.2%)	<.001
Mediators					
BMI, mean (SE)		27.11 (0.02)	27.71 (0.03)	26.83 (0.02)	<.001
Significant emotional distress (≥13)		6699 (3.3%)	4177 (6.2%)	2522 (1.8%)	<.001
Moderate physical activity		27.13 (46.53)	26.93 (48.88)	27.23 (45.36)	.064
Vigorous physical activity		22.73 (42.07)	22.57 (43.65)	22.81 (41.29)	.111
Moderate physical activity	None	90,279 (44.0%)	30,904 (46.3%)	59,375 (42.9%)	<.001
	Some (1–30)	67,699 (33.0%)	20,901 (31.3%)	46,798 (33.8%)	
	High (>30)	47,235 (23.0%)	14,879 (22.3%)	32,356 (23.4%)	
Vigorous physical activity	None	123,871 (60.2%)	41,349 (61.9%)	82,522 (59.4%)	<.001
	Some (1–30)	32,481 (15.8%)	9992 (15.0%)	22,489 (16.2%)	
	High (>30)	49,327 (24.0%)	15,475 (23.2%)	33,852 (24.4%)	

P values represent differences in characteristics between short sleep and average sleep participants based on weighted, clustered, and stratified Chi-squared and Kruskal–Wallis tests. BMI = body mass index, CVD = cardiovascular disease and risk factors.

the group with normal/average sleep duration ($P < .001$; Table 1). Similarly, short sleep was positively associated with higher BMI and levels of emotional distress ($P < .001$). Physical activity levels did not significantly differ between short (<7 hours) and average (7–8 hours) sleep groups in descriptive analyses (See Table 1).

3.2. Inferential statistics

Using SEM analysis, we determined that all 6 CVDs and risk factors loaded well onto the latent construct of CVD (Fig. 1; CFI = 0.967; RMSEA = 0.042). First, we examined the association between short sleep and latent CVD (Fig. 2). Short sleep was found to be significantly and positively associated with latent CVD in unadjusted, covariate-adjusted, and covariate and mediator-adjusted models (Table 2). Second, we investigated the direct and indirect effects of BMI, emotional distress, and physical activity on the relationship between short sleep and CVD (Fig. 2). The indices of model fit were within normally accepted limits (Chi-square estimate = 8217.85, $P < .001$; CFI = 0.934; RMSEA = 0.029). Short sleep duration was directly associated with CVD (B = 0.08; SE = 0.02; $P < .001$). Mediation analyses showed that the individual indirect effects between short sleep and CVD via BMI (B = 0.04; SE = 0.00; $P < .001$), emotional distress (B = 0.02; SE = 0.00; $P < .001$), and physical activity (B = 0.02; SE = 0.00; $P < .001$) were all statistically significant. The total indirect relationship between short sleep via all 3 mediators on CVD was also found to be significant (B = 0.07; SE = 0.002; $P < .001$). After covariate adjustment for age, sex, race, income, education, and marital status on all pathways of our mediational model, indices of fit remained acceptable (Chi-square estimate = 14,710.98, $P < .001$; CFI = 0.874; RMSEA = 0.028). In the adjusted model, short sleep retained a significant direct relationship with latent CVD (B = 0.15; SE = 0.01; $P < .001$). In addition, the specific indirect relationships between short sleep and CVD mediated via BMI (B = 0.05; SE = 0.00; $P < .001$), emotional distress (B = 0.02; SE = 0.00; $P < .001$), and physical activity (B = 0.01; SE = 0.00; $P < .001$) were all statistically significant. The total indirect relationship between short sleep and latent CVD also remained significant after covariate adjustment (B = 0.07; SE = 0.00; $P < .001$).

4. Discussion

We found that compared with average sleepers, short sleepers had a higher prevalence of all CVDs and risk factors, such as diabetes, HTN, kidney disease, heart attack, CHD, and stroke. Short sleepers also had higher BMI, reduced levels of physical activity, and reported higher levels of emotional distress. Emotional distress, BMI, and physical activity all significantly mediated the relationship between short sleep duration and CVDs and risk factors (Fig. 2). However, as the relationship between short sleep duration and CVD remained significant after covariate adjustment, we infer that emotional distress, BMI, and physical activity only partially mediated the short sleep–CVD

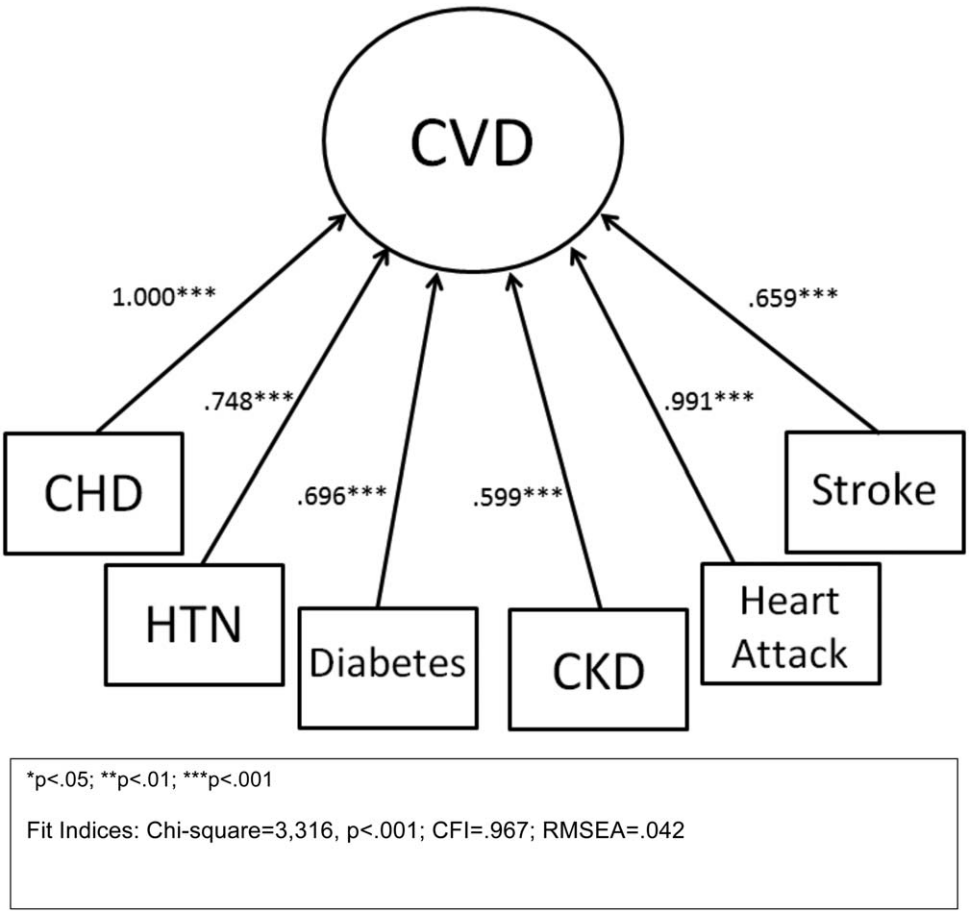

Figure 1. Factor loading of cardiovascular diseases and risk factors latent variable. The fit indices range from 0.599 to 1.00. CVD = cardiovascular diseases and risk factors, CHD = coronary heart disease, CKD = chronic kidney disease, HTN = hypertension.

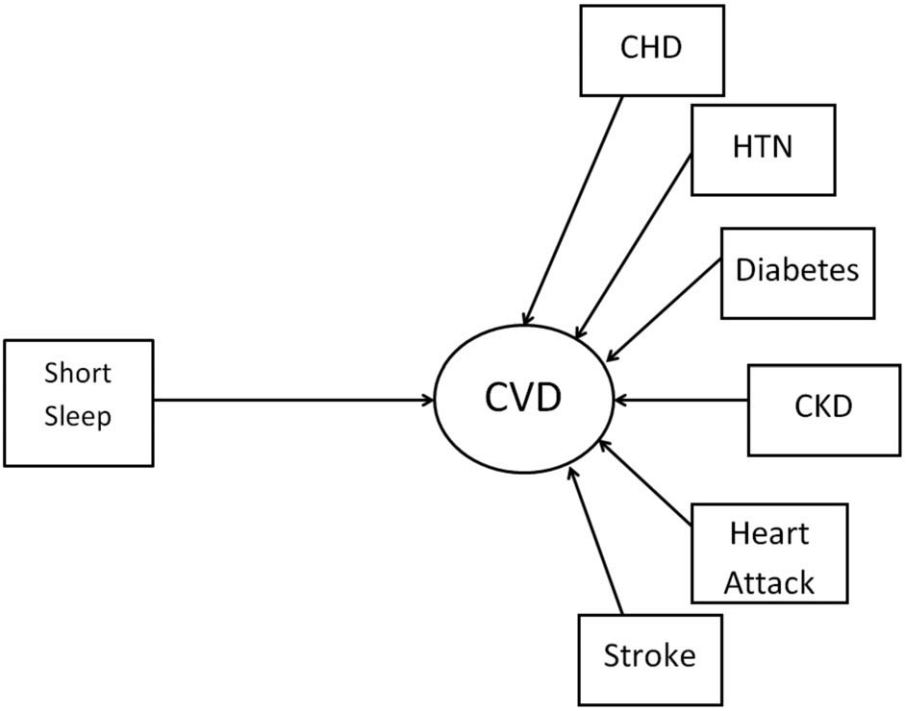

Figure 2. Structural equation model conceptual framework of the relationship between self-reported short sleep duration (≤6 h/24-h period) and latent variable of cardiovascular disease and risk factors (coronary heart disease, hypertension, diabetes, chronic kidney disease, heart attack, and stroke). Cardiovascular diseases and risk factors were coded dichotomously as whether the individual had a physician diagnosis of the following conditions.

Table 2

Associations between short sleep and latent CVD: model fit indices and path coefficients.

	Model fit indices				Path coefficients	
	Chi-squared	*P*	CFI	RMSEA	B (SE)	*P*
Unadjusted model						
Short sleep	3414.06	<.001	.968	.029	0.15 (0.01)	<.001
Covariate-adjusted model*						
Short sleep	9852.51	<.001	.892	.029	0.23 (0.01)	<.001
Covariate-adjusted model with mediators as predictors of CVD*						
Short sleep	13,194.30	<.001	.911	.026	0.15 (0.01)	<.001
BMI					0.29 (0.00)	<.001
Kessler Distress					0.59 (0.02)	<.001
Physical activity					−0.22 (0.04)	<.001

Variables: BMI = body mass index, CVD = cardiovascular disease and risk factors.
CFI = Comparative Fit Index, RMSEA = Root Mean Square Error of Approximation.
* Covariates: age, sex, race, income, education, and marital status.

relationship, and other mediating variables remain to be identified. Overall, our findings suggest that higher BMI and emotional distress as well as lower physical activity levels are associated with an increased likelihood of cardiovascular risk factors and diseases among short sleepers (Fig. 3).

4.1. Main effects

Our findings corroborate previous work indicating that short sleepers are at a significant risk of developing CVD and corollary adverse health conditions (e.g., HTN, diabetes, obesity, CHD, stroke, and kidney disease). In our study, compared with average sleepers (7–8 hours), short sleepers had a higher prevalence of medical comorbidities (Table 1).[11–16] Our findings are supported by previous work that show short sleep disrupts endocrine and cardiometabolic functions by reducing leptin (a hormone that curbs appetite)[41,42] and decreasing glucose metabolism, which increases the risk of glucose intolerance and diabetes.[17] Spiegel et al[14] highlight that short sleepers may not receive sufficient rapid eye movement activity during sleep, which is linked to restorative sleep and is needed for healthy metabolic and endocrine functioning. Short sleepers are more likely to have elevated sympathetic and pituitary adrenal axis activity resulting in decreased cerebral glucose metabolism and elevation in growth hormone, all of which can precipitate glucose intolerance, insulin insensitivity, and subsequent development of diabetes.[14]

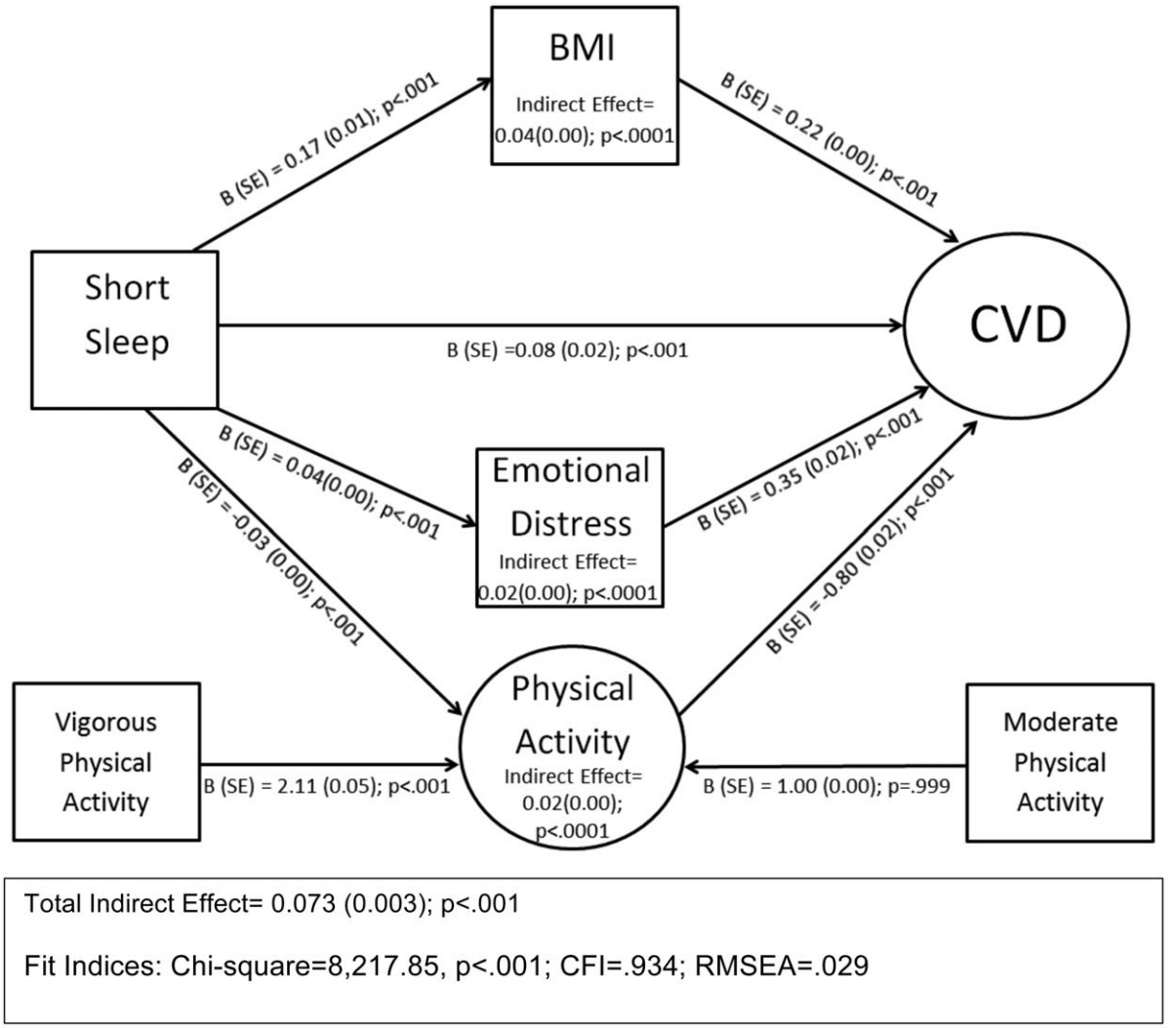

Figure 3. Structural equation model of the mediating effects of body mass index, emotional distress, and physical activity on the relationship between short sleep duration and cardiovascular diseases and risk factors. Physical activity was a latent variable that consisted of moderate and vigorous physical activity.

Another possible pathophysiological explanation for the short sleep–CVD relationship is rooted in the idea that short sleep may cause carotid intima disruption, which increases the risk of HTN and carotid plaque formation, all proximal predictors of CVD.[13]

4.2. Mediating effects

In spite of epidemiological and biological evidence that habitual short sleep duration increases CVD risk, our study found that behavioral and lifestyle factors such as BMI, physical activity, and emotional distress may mediate the relationship between short sleep on CVD and its associated risk factors.

4.2.1. Negative mediating effects. In the current analyses, we found that short sleep was associated with reduced physical activity, which was associated with greater levels of CVD and risk factors (Fig. 2). Our findings are consistent with previous studies that have highlighted the health benefits of physical activity, which includes enhancing mental health well-being, reducing CVD events and other chronic diseases, and decreasing mortality risk.[43–45] Physical activity may improve cardiovascular health by buffering the adverse effects of stress on cardiovascular health.[46–48] In a randomized trial, Blumenthal et al[48] demonstrated that medically stable CHD patients who engaged in consistent physical activity and stress management had less emotional distress and less CVD risk markers compared with patients who received medical treatment alone. In addition, some studies have even suggested that consistent physical activity may reverse the negative cardiovascular effects of sleep deprivation.[23,49–53]

4.2.2. Positive mediating effects. Both BMI and emotional distress positively mediated the relationship between short sleep and CVDs and risk factors. Of the 3 mediators we considered, BMI had the strongest mediation effect on the short sleep CVD relationship.[25,54–56] However, given the cross-sectional nature of the dataset, we were unable to determine a precise causal function of BMI. It is possible that habitual short sleep increased risk of elevated BMI, which in turn increases risk for CVD.[25,55] Laboratory studies indicate that short sleep is associated with an increase in the appetite stimulating hormone called ghrelin and a

decrease in the satiety hormone called leptin resulting in increased hunger, especially increased rich foods.[57] This pathophysiological argument might explain the increased BMI of short sleepers. However, the above causal chain is debunked in light of evidence indicating that not all individuals with elevated BMI are short sleepers.[58] Nevertheless, findings by Jean-Louis et al[59] and Donat et al[60] have shown that the obesity epidemic coincided with the rise in short sleep duration in the US. In addition, several prospective studies have shown that short sleep is associated with incident weight gain and/or obesity.[61–63] Despite the limitation of being unable to make causal claims, our study nevertheless makes a significant and unique contribution to the field based on our use of structural equation modeling a more robust analytic technique, relative to traditional regression analyses. The use of SEM techniques to model a latent construct for CVD risk factors and diseases reduces the problem of the correlations between BMI and any one comorbidity and allows us to test and quantify direct and indirect relationships among short sleep, CVD, and BMI, while adjusting for effects of confounders.

We also found that emotional distress positively mediated the relationship between short sleep and CVD. Emotional distress was more common among short sleepers and was positively associated with CVD. This observation was expected given that short sleep may induce emotional distress, irritability, and mood disturbances.[18,19] DRIVE, a robust Australian prospective study, concluded that self-reported short sleep duration was associated with psychological/emotional distress.[64] Similarly, in a 9-year clinical trial, Denollet and Brutsaert[32] examined the impact of treating emotional distress among male patients with CHD. The results showed that reduction in negative affect and emotional distress through a rigorous mental health rehabilitation program reduced likelihood of mortality among CHD patients. The direct link between short sleep and emotional distress is not well established in the literature; however, much can be inferred indirectly. Previous research shows that short sleep negatively affects the hypothalamic pituitary adrenal axis (HPA axis), which regulates stress in the body.[14,17,65] Activity of HPA axis is reduced during sleep onset and early stages of sleep, while it is activated during latter stages of sleep, such as rapid eye movement stage. If an individual experiences chronic short sleep, their HPA axis and cortisol levels will remain high and activated leaving the individual in a state of heightened alertness and stress. This heightened state of alertness can compromise the immune system and increase stress-related inflammation, which can lead to poor cardiovascular health. The HPA axis also plays a vital role in emotional and mental wellbeing and mediates the relationship between short sleep duration and emotional distress.[66] Evidence of this is observed in the negative effects of insomnia-related short sleep on emotional distress and depressed mood.[67–69] Prolonged sleep-related stress increases the risk of CVD and risk factors such as obesity, diabetes, and HTN[65,70] via HPA axis and other endocrine disruption.[71,72]

4.3. Limitations

Our study has several methodological and conceptual limitations. First, the cross-sectional design of the current study prevents us from inferring any casual relationships. For example, it is not clear whether a physical activity intervention could mitigate CVD risk factors and diseases associated with short sleep. Second, sleep duration was not measured objectively or prospectively, reducing precision of estimates of sleep duration. Third, BMI, physical activity, and medical conditions were not objectively determined and may be over- or underestimated. Fourth, as for the assessment of emotional distress, we were unable to confirm diagnosable mental illness. In addition, the Kessler scale is limited in assessing chronicity of emotional distress, which could affect our findings. It should be noted that the analyses in the present study did not take sleep apnea or insomnia into account, which overlap with short sleep duration[73] and are well-characterized risk factors for CVD risk factors and diseases, as well as depression, anxiety disorders, and suicide.[74–76] It is possible that the mediating effect of emotional distress on the relationship between short sleep duration and CVD risk factors and diseases may be confounded by sleep apnea or insomnia, which were not measured in the current study.

We recognize that our interpretation lacks nuance in capturing the confounding effects severity and chronicity of medical conditions may have on physical activity and sleep behaviors in this population.[55,77] Another limitation in our analyses was our inability to capture time-lagged and cumulative effect of short sleep on physical activity, which may provide greater insights on the long-term benefits of physical activity on cardiovascular health.

Another limitation is our inability to make significant generalizable inferences about all racial and ethnic groups, specifically Hispanics, as Hispanics were grouped into white race category. Current analyses did not adjust racial categories based on Hispanic ethnicity such as Hispanic and nonwhite Hispanic. Therefore, our race categories combined heterogeneous groups, such as Hispanics, which may have failed to capture actual racial differences in the covariate adjusted models. In addition, there is a lack of information on the number of dependents/children for the participants of this study. Having dependents/young children could have possibly been a confounder of short sleep status.

Additional questions remain for future work. First, the present study suggests that it is plausible that the cardioprotective benefits of physical activity may partially mitigate the detrimental effects of short sleep. Although the cross-sectional results of this study support this hypothesis, future intervention studies are needed to determine if this is the case. Second, this study cannot ascertain whether different types, levels, or categories of physical activity, emotional distress, and BMI may differentially affect the relationship between short sleep and health outcomes. More careful assessment of types of physical activity may address this issue. Third, certain factors may moderate relationships observed in our analyses; for example, individuals of differing age, sex, and race/ethnicity groups may differentially experience the cardioprotective benefits of exercise, as it relates to risks associated with sleep.

5. Conclusion

Despite these limitations, our findings provide evidence that BMI, emotional distress, and physical activity may affect the short sleep-CVD relationship. Future studies should take a personalized behavioral medicine approach to examine the nature of mediational models demonstrated in our analyses. Specifically, future studies should investigate whether specific sleep durations (short = less than 7 hours, average = 7–8 hours, or long = greater than 8 hours) when combined with BMI, emotional distress, and physical activity or sedentary behavior lead to lower and higher CVD risk. We propose that future studies should also investigate whether all short sleepers benefit equally from the mitigating effects of lower BMI, lower emotional distress, and increased physical activity.

We believe the current study adds significantly to the literature by providing a nuanced understanding of the complex interrelationships among short sleep, CVD, and behavioral and lifestyle mediators. It quantifies the direct and indirect effects of behavioral and lifestyle factors on the relationship between short sleep duration and CVD. Such insights are likely to lead to multipronged behavioral strategies to reduce the inimical effects short sleep has on CVD.

Author contributions

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