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Bereavement, self-reported sleep disturbances and inflammation: Results from Project HEART

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Abstract

Objective: Spousal bereavement is linked to increased mortality and morbidity from inflammatory conditions. It also has a significant impact on sleep disturbances. Evidence from experimental studies indicates that chronic stress may prime individuals to have an exaggerated inflammatory response to acute stress. In this study, we examined the association between self-reported sleep disturbances and inflammation after adjusting for depressive symptoms, and determined whether this association varies by bereavement status (bereaved individuals vs. controls).

Methods: Participants included 54 bereaved individuals and 47 controls with a mean age of 67.12 (SD=12.11). Inflammation was measured using C-reactive protein (CRP). Self-reported sleep disturbances were measured using the Pittsburgh Sleep Quality Index (PSQI). Depressive symptoms were measured using the Center for Epidemiologic Studies Depression Scale (CES-D).

Results: Sleep disturbances were not associated with elevated levels of CRP in the overall group (B=0.030, standardized β=0.122, 95% Confidence Intervals [CI]=−0.027 – 0.087, p=0.299) after adjusting for depressive symptoms. Results indicated, however, that bereavement moderated the association between inflammation and sleep disturbances (B=0.104, β=0.517, 95% CI=0.009 – 0.198, p=0.032). Stratified analyses demonstrated that these associations differed across groups. Associations were significant among bereaved individuals (B=0.104, β=0.406, 95% CI=0.013 – 0.196, p=0.026) and not controls (B=−0.016, β=−0.066, 95% CI=−0.096 – 0.065, p=0.690).

Conclusions: These findings provide preliminary evidence that bereavement moderates the association between self-reported sleep disturbances and inflammation. Future studies should

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examine the course of sleep disturbances following bereavement and establish whether objective sleep has differential associations with inflammation among bereaved adults.

Keywords

Sleep disturbances; sleep duration; sleep quality; insomnia; inflammation; bereavement

INTRODUCTION

Sleep disturbances, such as insomnia complaints, extremes of sleep duration and sleep fragmentation, adversely influence various aspects of health [1, 2], including elevated levels of inflammation markers [3, 4]. Observational studies, primarily those among middle-aged adults, consistently show that sleep disturbances are associated with inflammation [3, 5]. In fact, a recent meta-analysis which included results from over 50,000 people, demonstrated that sleep disturbances, particularly when measured by a well-validated instrument, were associated with increased levels of interleukin-6 (IL-6) and C-reactive protein (CRP). Shorter sleep duration has been associated with increased levels of CRP in observational studies. While the findings are still equivocal, other sleep disturbances such as poor self-reported sleep quality are also associated with increases in inflammation markers [6–8]. Fewer studies have examined this link in older adulthood, and a better understanding of these associations among older individuals is imperative, particularly given the high prevalence of both sleep disturbances [9] and inflammatory chronic conditions [10] in this age group.

The prevalence of stressful life events (e.g. personal medical illness, death/illness of a family member) is also consistently high among older persons [11]. Spousal bereavement is regarded as one of the most stressful life events of older adulthood [12]. This event may have a significant impact on sleep, leading to poor sleep quality and overall sleep disturbances [13]. Spousal bereavement is also significantly associated with an increased risk of mortality [14, 15] and elevated rates of chronic inflammatory conditions, such as cardiovascular disease (CVD) [16, 17]. An increased production of inflammatory cytokines is central to all stages of CVD, from initial lesion to end-stage thrombotic complications [18]. An exaggerated immunological response to stress, following the stressful life event of bereavement may contribute to the increased risk for morbidity and mortality in this population.

Evidence from experimental studies indicates that chronic stress may prime individuals to have an exaggerated inflammatory response to acute stress [19]. For example, in a laboratory study, clinically depressed women who underwent a “mock job interview” task showed greater increases in circulating CRP when compared to controls [20]. In another study, male patients who were diagnosed with major depression and experienced considerable early life stress exhibited enhanced inflammatory responsiveness (IL-6) to the Trier Social Stress Test [21] in comparison to those who did not experience early life stress and were not diagnosed with major depression. Similar responses have been reported in other chronic stressors, such as perceived discrimination [22], early life low socioeconomic status [23], and loneliness [24].Bereavement is usually conceptualized as both a chronic and acute stressor. The Dual

Process Model of coping with bereavement [25, 26] delineates two categories of stressors associated with bereavement: *loss*-versus *restoration-oriented*. *Loss-orientation* refers to the bereaved individual's concentration on, appraising and processing of some aspect of the loss experience itself. This stressor is chronic as it involves a painful dwelling on the loss. Restoration-orientation stress, on the other hand, is more "acute" and refers to the secondary stressors that may arise as a consequence of bereavement. These may include learning how to do chores that the deceased spouse did before, handling finances, getting used to sleeping alone and dealing with newly developed sleep disturbances. If the individual fails to adjust to life without the deceased, these stressors may also become chronic.

In line with the previous experimental studies, we propose that bereavement may prime individuals to have an exaggerated immunological response to sleep disturbances. Thus, individuals who are going through the bereavement process may be more susceptible to the effects of sleep disturbances on inflammation. No study to date has examined the impact of bereavement in the association between sleep and inflammation. In light of this gap in the literature, the primary aims of this study were to (1) examine the association between self-reported sleep disturbances and the inflammatory marker, CRP, in a sample of older adults after adjusting for important confounding factors, and to (2) determine the moderating role of spousal bereavement in the association between self-reported sleep disturbances and CRP.

METHODS

Study Sample

Our sample was comprised of individuals participating in Project Heart. The primary aim of this study was to determine the mechanisms that underlie the increased cardiovascular disease (CVD) risk among bereaved adults. Individuals who recently experienced the loss of their spouse (M= 89.6 days, SD = 15.9 days) were contacted and recruited from obituaries, support groups, flyer distribution, online postings, and community events. Control participants were also recruited through flyers, community events, and online advertisements. We excluded those with evidence of kidney failure, significant visual, auditory or cognitive impairment, those pregnant or nursing (women), those with autoimmune and inflammatory diseases (e.g. rheumatoid arthritis, ulcerative colitis, drug abuse). Individuals in the control group were also excluded if they had experienced bereavement due to loss of a spouse, another loved one or divorce within the past 5 years. All participants were English-speakers to ensure proper understanding of the questionnaires.

Research assistants administered assessments at the participants' home or in the Bioscience Research Collaborative's Community Research Center in the Houston medical center. During these visits, participants completed a questionnaire packet, which included self-report demographic questionnaires and clinical questionnaires. Furthermore, anthropometric measurements, including weight, height, and waist circumference and non-fasting blood samples were collected during the early hours of the morning. Participants were asked if they were experiencing any illness symptoms (e.g., fever, congestion, sore throat, or acute infections due to injury), and to avoid strenuous physical activity 48 hours before all visits. Participants were rescheduled if they were ill or did not follow exercise restrictions. All participants provided informed consent and procedures were approved by the Rice

University Institutional Review Board (IRB). Data for the present analyses were collected between December, 2015 and June, 2017. Our analytical sample included a total of 101 participants (54 bereaved, 47 control). A total of 9 participants were excluded from analysis due to CRP levels ≥10 mg/L, as these values are likely a sign of infection [27].

Measures

Sleep Disturbances.—The Pittsburgh Sleep Quality Index (PSQI) was used as a self-reported measure of sleep disturbance. The PSQI is a widely used instrument for the evaluation of sleep disturbances which consists of seven component scores (Subjective Sleep Quality, Sleep Latency, Sleep Duration, Sleep Efficiency, Sleep Disturbances, Use of Sleep Medication and Daytime Dysfunction) that are aggregated in a global score with a range of 0–21 [28]. Higher scores on the PSQI global score are indicative of greater sleep disturbance. A recent paper [29] examined the factor structure of the PSQI and found a clear three-factor solution that distinguished 3 domains of sleep disturbance: Sleep Efficiency (comprised of the Sleep Duration and Habitual Sleep Efficiency subscales), Perceived Sleep Quality (Subjective Sleep Quality, Sleep Latency, and Sleeping Medication Use), and Daily Disturbances (Sleep Disturbances and Daytime Dysfunction). Results indicate that subscale scores load robustly into one of the three factors. Accordingly, we computed scores for each domain (factor) by summing their respective subscale scores. As with the PSQI global score, higher scores on each domain indicate greater sleep disturbance.

Inflammation.—Laboratory analyses were performed on blood samples to evaluate high-sensitivity CRP as a marker of systemic information. Levels of high-sensitivity CRP were determined using Roche (immunoturbidomatric) assays per manufacturer specifications in a clinical core laboratory at Baylor College of Medicine. Quality control was performed at a minimum of twice per day. All assessments were performed blindly by laboratory technicians with no knowledge of the participant's bereavement status.

Depression.—The Center for Epidemiologic Studies Depression Scale (CES-D) was used as a measured of depression, and included in regression models as a control variable due to its close association with inflammation [30]. The CES-D is a widely utilized measure of depression and has been validated across populations [31]. Higher scores on this scale indicate greater depressive symptomatology. The CES-D item measuring sleep disturbances was excluded when computing CES-D total scores.

Covariates.—Demographic factors, health behaviors, comorbid conditions and body mass index (BMI) were included in models as covariates. Demographic information (age, gender, race/ethnicity and education) was collected via self-report questionnaires. Health behaviors included caffeine consumption (number of cups consumed per day). BMI was computed as weight in kilograms divided by height in meters squared. The variable assessing presence of comorbid chronic conditions was computed using information from the medical history performed during the clinical visit. Participants were categorized as having a chronic condition (1=Presence of Chronic Condition) if they reported having any of the following conditions: CVD, dementia, chronic pulmonary disease, connective tissue disease, peptic ulcer disease, liver disease, diabetes, hemiplegia or paraplegia or renal disease. Categorical

variables including gender (1=Female), and race/ethnicity (1=Minority Status) were dichotomized for regression analysis.

Statistical Analysis

Preliminary statistical analysis included descriptive statistics and assessment of normality of distributions. Non-normal variables including CRP values were log-transformed. Data for continuous variables are presented as means and standard deviations and were compared between groups with the independent *t* test or the Mann-Whitney-U test as needed. Proportions are presented as percentages and were compared with the χ^2 test.

Multiple linear regression analyses were used to test the association between sleep disturbances and inflammation. Demographic variables including age, gender, race/ethnicity, education, caffeine consumption, obesity, and comorbid conditions were included in multiple regression models as covariates due to their well-established impact on inflammation markers [32]. Separate regression models were fitted for each PSQI domain as well as the PSQI Global Score. The moderating effect of bereavement was tested by creating several group X PSQI domain/Global Score interaction terms and following a 3-step approach. Covariates were entered into the model (Step 1) before entering each PSQI domain (centered) and group (bereaved group coded as 1) main effects (Step 2), and each interaction term (Step 3). Once significant interaction terms were identified, stratified analyses by bereavement status (bereaved vs. control) were conducted. All tests were two-sided and $\alpha<0.05$ was considered to be statistically significant.

RESULTS

Descriptive Characteristics of the Study Sample

The study sample was comprised of 101 participants, 54 bereaved individuals and 47 controls. A total of 70.30% of the sample was female. Mean age was 67.12 (SD=12.11). Bereaved participants had significantly lower BMI when compared to control participants ($M_{bereaved}$=26.24, $M_{control}$=29.27, p=0.007). Similarly, they had higher depressive symptoms ($M_{bereaved}$=16.91, $M_{control}$=8.76, p<0.001), higher scores on the PSQI Global Scale ($M_{bereaved}$=7.94, $M_{control}$=6.17, p=0.025), the Sleep Efficiency domain ($M_{bereaved}$=1.85 $M_{control}$=1.09, p=0.030) and the Daily Disturbances domain ($M_{bereaved}$=2.40, $M_{control}$=1.96, p=0.022). Both groups were comparable in terms of age, gender, education, race/ethnicity, education, caffeine use, comorbid conditions, medication use, CRP, and in their scores on other PSQI domains. Other important characteristics of the study sample are presented in Table 1.

Sleep Disturbances and Inflammation

The association between various sleep disturbances and inflammation were examined in separate multiple regression models. Models controlled for age, gender, education, race/ethnicity, caffeine use, BMI, depressive symptoms, comorbid conditions, and group (control vs. bereaved). Main effects for the association between sleep disturbances and inflammation were not significant for the PSQI Global Score or any of the PSQI domains. Main effects for all scales are described in Table 2, Column 2.

The Moderating Role of Bereavement

In order to elucidate the role of bereavement as a moderator in the associations between self-reported sleep disturbances and inflammation, a group by PSQI score interaction was created for the Global Score as well as each domain, and included in separate multivariate regression models (Table 2, Model 3). We adjusted for age, gender, education, race/ethnicity, caffeine use, BMI, depressive symptoms, and comorbid conditions. Results showed that there was a significant moderating effect of bereavement on the association between the PSQI Global Score (B=0.104, 95% CI= 0.009 – 0.198, standardized β=0.517, p=0.032), and the PSQI Sleep Efficiency domain (B=0.216, 95% CI= 0.010 – 0.422, β=0.307, p=0.040) and inflammation. It is worth noting that there was a trend for the Sleep Quality domain (p=0.061). Further adjustment for antidepressant and statin use did not alter these associations. Table 3 presents stratified analyses by bereavement status (bereaved vs. control group). As shown in Table 3, the associations between the PSQI Global Score (B=0.104, 95% CI= 0.013 – 0.196, β=0.406, p=0.026) and the PSQI Sleep Efficiency Domain (B=0.202, 95% CI= 0.053 – 0.351 β=0.397, p=0.009) with inflammation were only significant among the bereaved group.

DISCUSSION

In this study, we examined the association between self-reported sleep disturbances and inflammation in a sample of bereaved individuals and controls. We found no significant associations between self-reported sleep and inflammation in the overall sample. However, we provide novel data on the moderating role of bereavement in these associations. We demonstrated that after adjusting for confounding factors, including demographic characteristics, caffeine use, depressive symptoms, BMI and comorbid conditions, the association between global self-reported sleep and inflammation was significant among those who recently lost a spouse, but not for those who did not experience this stressful life event. Assessment of key PSQI domains (sleep efficiency, perceived sleep quality and daily disturbances) indicated that the association is primarily driven by self-reported sleep efficiency.

The differential association between self-reported sleep disturbances and inflammatory levels by bereavement status is in line with results of studies demonstrating that chronic stress promotes an exaggerated immune response to acute stressors in the laboratory [20–24]. The etiology of bereavement-related sleep disturbances may be important in understanding how sleep may lead to greater increases in inflammation markers among bereaved individuals. Sleep disturbances, for example, may develop as a part of, or following, a major depressive episode. Many widows and widowers meet formal criteria for major depressive disorder [33, 34]. In this case, depression may interact with sleep disturbances to increase inflammation. While our study adjusted for the presence of depressive symptoms, our study was not sufficiently powered to assess three way interactions (depression X sleep disturbances X bereavement). Future studies should determine whether depression interacts with self-reported sleep disturbances and bereavement to increase inflammation. Similarly, three-way interactions with other factors

impacting sleep disturbances, inflammation and bereavement, such as obesity, should be explored.

Another mechanism by which sleep might be disrupted in bereaved spouses is through the disruption of their circadian rhythms. Spouses often keep an individual's circadian rhythm properly entrained, and the absence of a spouse may promote irregular sleep patterns and meal times, which may have a detrimental impact on metabolism. Furthermore, the absence of a partner may promote social isolation and obesity-promoting behaviors such as lack of physical activity and consumption of high-fat diets [35–37]. Future studies should comprehensively examine circadian rhythms following bereavement. Further, the role of weight gain should be explored as a potential mediator in the association between sleep disturbances and inflammation.

Our results indicated that greater disruption in the sleep efficiency domain, in particular, was associated with higher levels of inflammation among bereaved individuals. The sleep efficiency domain was calculated by integrating the scores of the PSQI sleep duration and sleep efficiency subscales. It is important to note that we adjusted for relevant covariates, including levels of depressive symptoms measured by the CESD. In line with these findings, previous reports showed a significant link between sleep efficiency and inflammation [38, 39]. For example, results from the Midlife in the United States (MIDUS) Study showed that sleep efficiency was associated with greater inflammation, particularly among men with low social engagement (low scores in a positive relations measures) [39]. Similarly, in a sample of older caregivers and controls, sleep efficiency was associated with increased IL-6 levels [38]. Significant links between short sleep duration and inflammation have also been reported in the literature. In fact, a recent meta-analysis reviewed a total of 72 studies ($n>50,000$) and concluded that shorter sleep duration was associated with higher levels of CRP [40]. No significant associations were reported between self-reported sleep disturbances or sleep domains and inflammation among individuals in the control group. Lower overall variability and range in self-reported sleep-disturbances and sleep domains among individuals in the control group may have reduced power to detect associations in this particular group. These factors, in addition to the relatively small sample size, indicate that these findings (particularly the lack of sleep-inflammation association among non-stressed elders) require further confirmation.

Important strengths of our study include a rigorous methodology as well as the assessment of a well-validated marker of inflammation. The present study is limited by its relatively small sample size and cross-sectional design. Therefore, causality in the association between sleep domains and inflammation cannot be determined. While our measure of sleep disturbances, the PSQI, is a valuable tool for assessing sleep disturbances, it heavily relies on individuals' self-report. Other measures such as the assessment of depressive symptoms were also self-reported by participants. It is possible that the distress experienced by bereaved individuals may have resulted in negative cognitive biases. Thus, individuals in the bereaved group may have rated their sleep worse than it actually was.

Future studies should examine the link between objective measures of sleep, such as actigraphy or polysomnography, and inflammation among bereaved individuals to determine

the degree of sleep disturbances in this population. Research efforts should also be aimed at examining the link between sleep domains, sleep efficiency in particular, and inflammation using longitudinal designs to examine the trajectory of sleep symptoms following bereavement. Examining which aspects of sleep disturbances are more detrimental to bereaved adults is also an important priority. Determining whether disruptions in sleep duration vs. circadian rhythms are more prominent in this sample is an important step toward designing tailored interventions aimed at reducing sleep disturbances in this population. Finally, future reports should determine whether sleep disturbances in bereavement have an impact on physical health outcomes, including CVD morbidity and mortality.

CONCLUSIONS

In sum, our study demonstrated that the association between self-reported sleep disturbances and inflammation varies by bereavement status. We showed that the association between higher levels of inflammation and self-reported sleep disturbances, in particular lower sleep efficiency, is present among individuals who recently underwent the loss of a spouse. While these findings are still preliminary and require further confirmation, they highlight a potential role of sleep disturbance as a pathway leading to increased inflammation among bereaved individuals. *If* sleep disturbance is an important mechanism underlying increased inflammation and cardiovascular risk in spousal bereavement, designing both psychosocial and pharmaceutical interventions aimed at reducing sleep disturbance may be an important step towards reducing cardiovascular risk in this population. Future studies should focus on prospectively examining the association between sleep disturbances, measured subjectively as well as objectively, and inflammation among bereaved individuals. Further, specifying the nature of sleep disturbances (e.g. insufficient sleep duration vs. disruption in circadian rhythms) in this population is an important priority. A better understanding of the nature of sleep disturbances among bereaved individuals will inform the design of appropriately tailored interventions aimed at improving sleep in this population.

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List of Abbreviations:

CRP	C-reactive protein
IL-6	Interleukin-6
TNF-α	Tumor necrosis factor-alpha
LPS	Lipopolysaccharide
PBMCs	Stimulated peripheral blood mononuclear cells
IRB	Institutional Review Board
PSQI	Pittsburgh Sleep Quality Index

CES-D	Center for Epidemiologic Studies Depression Scale
BMI	Body mass index
CI	Confidence Intervals

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Table 1.

Descriptive characteristics of the study sample

	All (n=101) M (SD)/ Median (IQR)	Control (n=47) M (SD)/ Median (IQR)	Bereaved (n=54) M (SD)/ Median (IQR)	*p*-value
Demographic				
Age, years	67.12 (12.11)	64.81 (12.65)	69.13 (11.31)	0.073
Gender, % female	70.30	66.00	74.10	0.373
Race/Ethnicity, % Minority Status	35.60	44.70	27.80	0.077
Education, years	16.23 (2.67)	16.21 (2.71)	16.24 (2.66)	0.958
Lifestyle/Risk Factors				
Caffeine use, cups per day	1.46 (1.31)	1.27 (1.25)	1.63 (1.35)	0.175
Body Mass Index, kg/m^2	27.65 (5.67)	29.27 (6.35)	26.24 (4.60)	**0.007**
Comorbid Conditions, % yes	12.90	17.00	9.30	0.245
Medications				
Statins, %yes	30.70	23.40	37.00	0.138
Anti-depressants, %yes	17.80	14.90	20.40	0.473
Psychosocial Scales				
Depression, CES-D Score	13.16 (10.58)	8.76 (8.80)	16.91 (11.12)	<**0.001**
PSQI, Global Score	7.11 (3.97)	6.17 (3.88)	7.94 (3.89)	**0.025**
PSQI, Sleep Efficiency Domain	1.50 (1.78)	1.09 (1.59)	1.85 (1.87)	**0.030**
PSQI, Perceived Sleep Quality Domain	3.44 (2.31)	3.13 (2.32)	3.70 (2.30)	0.213
PSQI, Daily Disturbances Domain	2.19 (0.96)	1.96 (0.88)	2.40 (0.99)	**0.022**
Inflammation				
C-reactive protein, mg/L	1.40 (0.70–3.60)	1.60 (0.80 – 4.40)	1.30 (0.60 – 2.90)	0.340

M=mean, SD=standard deviation, IQR=interquartile ranges.

Table 2.

Regression analysis of covariates, main effects and interactions models in the association between sleep disturbances on inflammation

	B	95% CI	β	*p*-value
Model 1 (Covariates)				
Age	0.007	−0.008 to 0.023	0.095	0.360
Gender	0.139	−0.249 to 0.527	0.069	0.481
Education	0.024	−0.045 to 0.093	0.070	0.475
Race/Ethnicity	0.221	−0.193 to 0.634	0.114	0.293
Caffeine Consumption	0.015	−0.118 to 0.149	0.022	0.821
Body Mass Index	0.081	0.049 to 0.114	0.503	**<0.001**
CES-D Total Score	0.007	−0.01 to 0.024	0.078	0.406
Comorbid Conditions	−0.452	−0.959 to 0.056	−0.167	***0.079***
Main Effects Models[£]				
PSQI Global Score[2]	0.030	−0.027 to 0.087	0.122	0.299
PSQI Sleep Efficiency Domain[3]	0.097	−0.017 to 0.210	0.182	0.094
PSQI Perceived Sleep Quality Domain[4]	0.002	−0.087 to 0.090	0.004	0.970
PSQI Daily Disturbances Domain[5]	0.086	−0.124 to 0.295	0.087	0.419
Interaction Model				
PSQI Global Score[6]	0.104	0.009 to 0.198	0.517	**0.032**
PSQI Sleep Efficiency Domain[7]	0.216	0.010 to 0.422	0.307	**0.040**
PSQI Perceived Sleep Quality Domain[8]	0.150	−0.007 to 0.307	0.254	***0.061***
PSQI Daily Disturbances Domain[9]	−0.180	−0.562 to 0.202	−0.137	0.351

n=101, CI=confidence intervals, CES-D=Center for Epidemiologic Studies Depression Scale, PSQI=Pittsburgh Sleep Quality Index.

£all Main Effects and Interaction Models controlled for variables in Model 1.

Table 3.

Stratified regression analyses of significant interactions by bereavement status

	Bereaved				Control			
	B	**95% CI**	**β**	***p*-value**	**B**	**95% CI**	**β**	***p*-value**
Model 1 (Covariates)								
Age	0.014	−0.012 to 0.040	0.167	0.296	0.006	−0.018 to 0.030	0.080	0.611
Gender	0.253	−0.358 to 0.863	0.123	0.408	−0.039	−0.624 to 0.547	−0.020	0.895
Education	−0.036	−0.132 to 0.060	−0.099	0.453	0.087	−0.038 to 0.213	0.247	0.168
Race/Ethnicity	0.455	−0.149 to 1.059	0.221	0.136	0.271	−0.519 to 1.061	0.144	0.491
Caffeine Consumption	0.031	−0.159 to 0.221	0.046	0.743	−0.006	−0.228 to 0.215	−0.008	0.954
Body Mass Index	0.111	0.052 to 0.171	0.550	**<0.001**	0.076	0.027 to 0.126	0.520	**0.003**
CES-D Total Score	0.013	−0.015 to 0.042	0.140	0.348	0.004	−0.033 to 0.041	0.034	0.821
Comorbid Conditions	−0.495	−1.354 to 0.364	−0.161	0.251	−0.472	−1.204 to 0.259	−0.192	0.199
Sleep Domain Models[£]								
PSQI Global Score[2]	0.104	0.013 to 0.196	0.406	**0.026**	−0.016	−0.096 to 0.065	−0.066	0.690
PSQI Sleep Efficiency Domain[3]	0.202	0.053 to 0.351	0.397	**0.009**	−0.027	−0.227 to 0.173	−0.045	0.787

n=101, CI=confidence intervals, PSQI=Pittsburgh Sleep Quality Index,

[£]all Sleep Domain Models controlled for variables in Model 1.