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The relationship between Pittsburgh Sleep Quality Index subscales and diabetes control

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Abstract

Objectives: Data suggest that poor sleep quality as measured by the Pittsburgh Sleep Quality Index (PSQI) contributes to suboptimal diabetes control. How the subscales comprising the PSQI individually relate to diabetes control is poorly understood.

Methods: In order to explore how PSQI subscales relate to diabetes control, we analyzed baseline data from a trial of a telemedicine intervention for diabetes. We used multivariable modeling to examine: (1) the relationship between the global PSQI and hemoglobin A1c (HbA1c); (2) the relationships between the 7 PSQI subscales and HbA1c; and (3) medication nonadherence as a possible mediating factor.

Results: Global PSQI was not associated with HbA1c ($n = 279$). Only one PSQI subscale, sleep disturbances, was associated with HbA1c after covariate adjustment; HbA1c increased by 0.4 points for each additional sleep disturbances subscale point (95%CI 0.1 to 0.8). Although the sleep disturbances subscale was associated with medication nonadherence (OR 2.04, 95%CI 1.27 to 3.30), a mediation analysis indicated nonadherence does not mediate the sleep disturbances-HbA1c relationship.

Discussion: The sleep disturbances subscale may drive the previously observed relationship between PSQI and HbA1c. The mechanism for the relationship between sleep disturbances and HbA1c remains unclear, as does the impact on HbA1c of addressing sleep disturbances.

Keywords

Sleep quality; Pittsburgh Sleep Quality Index; sleep disturbance; diabetes; hemoglobin A1c

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Introduction

The incidence of type 2 diabetes is increasing, and poor diabetes control drives the devastating complications and costs associated with this disease.¹ A diverse array of factors appear to impact glycemic control, including medication adherence, treatment regimen complexity, and psychosocial factors.² Recognizing and modifying these barriers to improved glycemic control is critical to reducing diabetes-related complications and costs.

One under-recognized factor that may impact diabetes control is sleep quality. Assessment of overall sleep quality often relies on the Pittsburgh Sleep Quality Index (PSQI).³⁻⁶ Based on two recent reviews, poor global sleep quality as measured by PSQI has often been associated with higher hemoglobin A1c (HbA1c).^{7,8} Of note, PSQI is a general measure, and includes subscales that assess total sleep time, sleep onset latency, degree of fragmentation, sleep efficiency, sleep disturbances, sleep medication use, daytime alertness, and total awake time.⁹ Currently, little is known about how the distinct constructs that comprise the PSQI each individually relate to diabetes control. Examining how each PSQI subscale relates to HbA1c could enhance our understanding of the link between poor overall sleep quality and diabetes control, and potentially point toward sleep-focused strategies for lowering HbA1c.

In order to explore the relationship between self-reported sleep quality and diabetes control, we used data from a randomized trial to examine relationships between the global PSQI measure and HbA1c, and then the seven PSQI subscales and HbA1c. Because medication non-adherence negatively affects diabetes control and may in turn be exacerbated by poor sleep,^{2,10,11} we also evaluated medication non-adherence as a potential mediator of relationships between sleep quality and HbA1c.

Methods

We analyzed baseline clinical and survey data from participants enrolled in the Simultaneous risk factor control using Telehealth to sLOw Progression of Diabetic Kidney Disease (STOP-DKD) trial. STOP-DKD ([Clinicaltrials.gov NCT01829256](https://clinicaltrials.gov/ct2/show/study/NCT01829256)) is a randomized, controlled trial examining the effect of a telemedicinebased medication management and self-management educational intervention on diabetic kidney disease (DKD) progression. The study is approved by the Duke University Institutional Review Board.

Patient population

STOP-DKD study participants were identified from the administrative and clinical database of Duke University Health System (DUHS). Eligible patients had type 2 diabetes (ICD-9 codes 250.x0, 250.x2), uncontrolled hypertension (blood pressure (BP) 140/90 mm Hg or two recent elevated values), and most recent estimated glomerular filtration rate greater than 45 mL/min/1.73 m². Other eligibility criteria were age 18 to 75 years, management in a participating DUHS primary care clinic, prescriptions for at least one medication for diabetes and hypertension, ability to complete a baseline survey, and evidence of diabetic nephropathy (as indicated by presence of albuminuria, history of albuminuria prior to

angiotensin-converting enzyme inhibitor or angiotensin receptor blocker therapy and/or previous documentation of diabetic retinopathy and/or laser therapy).

Eligible patients were mailed an invitation to participate in the study, which was followed by a telephone screening to arrange an in-person baseline study visit. A survey, body mass index (BMI), BP, and laboratory tests (including HbA1c) were completed at study baseline; all clinical and survey data used in the present analysis derived from this baseline study assessment.

Measure of sleep quality

To evaluate sleep quality, the PSQI measure, which assesses overall sleep quality over the prior month,¹² was administered at study baseline by a trained research assistant. The instrument comprises 19 items that are grouped into seven subscales measuring the following constructs: self-reported sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication and daytime dysfunction. Each subscale is assigned a score between 0 and 3. The sum of these components provides the global PSQI score, which may range from 0 to 21, with higher scores indicating poorer sleep quality.¹²

For the purpose of describing our population, we dichotomized patients as poor sleepers (PSQI >5) or good sleepers (PSQI ≤ 5). A global score of PSQI >5 has high sensitivity in differentiating poor sleep quality from good sleep quality.¹² For subsequent analyses, we analyzed PSQI as a continuous measure, and also conducted sensitivity analyses examining PSQI as a dichotomous variable using cutoffs of both >5 and >8 to define poor sleep quality. Previous studies have examined both of these cutoffs.⁷

Additional measures

We examined relevant covariates from the STOP-DKD baseline survey, organized in three categories: demographic, medical, and psychosocial. Demographic covariates included age, sex, race (dichotomized as Black/African American and non-Black/non-African American), and annual income (categorized into three groups <\$30,000, \$30,000, and refused/don't know).

Medical covariates included diabetes treatment regimen (dichotomized as insulin-based and noninsulin based), BP, BMI (dichotomized as <30 and ≥ 30 kg/m²), and low-density lipoprotein (LDL) cholesterol levels.

We considered multiple psychosocial covariates as potential confounders. We examined medication adherence using an 8-item measure that consists of seven yes/no questions and one question with a 5-point Likert response (“never” to “all the time”).¹³ Using a validated approach, we summed responses to yield a score ranging from 0 to 8.¹⁴ Because we reverse-scored questions (such that a high score indicated nonadherence), medication nonadherence was defined by a total score ≥ 3.¹⁴ We assessed depression using the Patient Health Questionnaire-2 (PHQ-2), which evaluates frequency of depressed mood and anhedonia over the past two weeks. The PHQ-2 consists of two questions with scores ranging from 0–6;¹⁵ the score was analyzed as a continuous variable. The Confusion, Hubbub, and Order Scale

(CHAOS) examines confusion and disorganization in the home environment.¹⁶ We used a 4-item scale adapted from the previously validated 6-item measure (Cronbach's α calculated for the 4-item scale using our analytic dataset was 0.62 (95% CI 0.54, 0.69)), which represents an acceptable value given the limited number of included items.¹⁷ The sum of 4 of the items ("my life is organized," "my daily activities from week to week are unpredictable," "keeping a schedule is difficult for me," and "I don't like to make appointments too far in advance because I don't know what might come up") yields a score ranging from 0 to 16 where higher scores indicate a more chaotic lifestyle; the score was analyzed as a continuous variable.

Outcome

Our primary outcome was diabetes control as measured by HbA1c. A blood sample was collected at each patient's baseline study assessment and HbA1c testing was performed using a high-performance liquid chromatography assay.

Statistical analysis

We first examined demographic and clinical characteristics of poor sleepers (PSQI >5) and good sleepers (PSQI \leq 5). For descriptive purposes, groups were compared using the Wilcoxon–Mann–Whitney test for continuous variables and Fisher's exact test for categorical variables, with no adjustment for multiple testing. We used multivariable linear regression to assess the association between continuous HbA1c and global sleep quality as measured by PSQI, adjusted for age, sex, race, income, BMI, depression, and life chaos. As above, we analyzed PSQI as a continuous variable, but also conducted sensitivity analyses examining PSQI as a dichotomous variable. Multivariable logistic regression was also used to examine the association between medication nonadherence and global sleep quality, adjusted for the same covariates. We performed mediation analyses to determine if the association between HbA1c and sleep quality was mediated by nonadherence.¹⁸ Nonadherence was regressed on PSQI using a logit model and adjusted for potential confounders; HbA1c was then regressed on PSQI and nonadherence using a linear model, adjusted for the same confounders. These models provided the information needed to estimate the mediating effect, and hypothesis testing was performed via bootstrapping. Using a similar approach, we then examined the association between each of the seven PSQI subscales, HbA1c, and non-adherence.

All statistical tests were two sided with no correction for multiple testing. Analyses were performed using R 3.3.0 (R Core Team, Vienna, Austria) and SAS 9.4 (SAS institute, Cary, NC).

Results

Population characteristics

Table 1 summarizes the 281 STOP-DKD study participants' baseline demographic, medical, and psychosocial characteristics. Compared to good sleepers (PSQI \leq 5), poor sleepers (PSQI >5) were more often female (61% vs. 34%, $p < 0.01$), more likely to report medication nonadherence (44% vs. 24%, $p < 0.01$), more likely to screen positive for depression (16%

vs. 4%, $p < 0.01$), and had higher life chaos scores (7.4 vs. 6.1, $p = 0.01$). The mean PSQI score among poor sleepers was 9.18 ± 3.24 , while the mean score among good sleepers was 2.97 ± 1.51 .

Sleep and HbA1c

On multivariable linear ($n = 279$, two individuals excluded from all models due to missing income data), global PSQI (continuous variable) was not associated with HbA1c at baseline (β 0.09, 95% CI (-0.54, 0.36)). Because our sensitivity analyses examining PSQI as a dichotomous variable (using cutoffs of >5 and >8) likewise showed no association with HbA1c, we proceeded with subsequent analyses using the continuous PSQI. We examined each individual component of the PSQI in relation to HbA1c (Table 2) to explore how specific components of sleep relate to diabetes control. Of the seven PSQI components, only the sleep disturbance subscale was significantly associated with HbA1c after multivariable adjustment, with each additional point reflecting an model-estimated mean increase in HbA1c of 0.4 points ($p = 0.02$) (Table 2). Stated differently, participants with a sleep disturbance score of zero had a model-estimated mean HbA1c of 7.4%, while those with a sleep disturbance score of 3 had HbA1c of 8.8% (model-estimated mean difference 1.4%).

Examination of adherence as a possible mediator

On multivariable logistic regression ($n = 279$, two individuals excluded from all models due to missing income data), we observed a significant association between the global PSQI score and medication non-adherence; a one point increase in the global score resulted in a 9% increase in the odds of nonadherence ($p = 0.03$). When we examined the individual sleep components, we found that sleep disturbances (OR 2.04, 95% CI 1.27–3.30) and daytime dysfunction (OR 2.05, 95% CI 1.31–3.22) were each significantly associated with medication nonadherence (Table 3). Because the sleep disturbances subscale was associated with both HbA1c and medication nonadherence, we conducted a mediation analysis. We saw no evidence for a mediating effect for nonadherence for sleep disturbance ($p = 0.25$).

Discussion

In order to explore the relationship between self-reported sleep quality and diabetes control, we examined how each individual PSQI subscale related to HbA1c in a population with type 2 diabetes. One PSQI subscale, sleep disturbances, had a statistically significant relationship with HbA1c that persisted after adjustment for age, gender, **BMI** and other factors such as life chaos, depression and income. The association between sleep disturbances and HbA1c was clinically significant; participants with the highest sleep disturbances score (score of 3) had an HbA1c that was, on average, 1.4 points higher than those with the lowest score (score of 0). We also conducted an analysis that suggested medication nonadherence does not mediate the relationship between sleep disturbances and HbA1c.

Based on a meta-analysis of nine studies, a recent systematic review by Lee et al.⁷ reported that poor overall sleep quality is associated with higher HbA1c levels (weighted mean difference 0.35 points, 95% CI 0.12–0.58). The investigators noted moderate heterogeneity on this meta-analysis ($Q = 22.84$, $I^2 = 65\%$, $p < 0.001$), reflecting the fact that included

studies varied in their estimates of the relationship between the global PSQI scale and HbA1c. One possible explanation for this heterogeneity is that included studies used different cutoffs to define poor sleep quality (>5 to >8). However, our analyses showed no association between global PSQI and HbA1c regardless of whether PSQI was defined continuously or using cutoffs of >5 or >8, which may argue against PSQI definition as an important source of heterogeneity. Other potential causes for variability in the reported relationship between the global PSQI and HbA1c include study population demographics and degree of glycemic control.

Our analysis specifically sought to determine whether individual PSQI subscales have varying associations with HbA1c. We found sleep disturbances to be the sole PSQI component that had a significant association with HbA1c. Merging the different PSQI subscales may have in effect diluted the association between sleep disturbances and HbA1c in our study, explaining the lack of association between the global PSQI measure and HbA1c. Of note, we identified one prior analysis that examined PSQI subscales in relation to HbA1c.¹⁹ Sleep disturbances were not significantly associated with HbA1c in this small study ($n = 46$ Taiwanese patients), but a different PSQI component, sleep efficiency, was. As with the global PSQI, it is therefore possible that relationships between PSQI components and HbA1c may be influenced by population factors. Further examination of the relationship between PSQI subscales and HbA1c is warranted to confirm our findings and to explore how population differences affect the relationship between sleep quality and diabetes control.

Our findings suggest that self-reported medication adherence does not mediate the observed relationship between the sleep disturbances subscale and HbA1c. Given that the sleep disturbances subscale probes problems pertaining to interruption of sleep, including nocturnal awakening, waking overnight to use the bathroom, breathing issues, and others, alternative mediators could include physiologic and behavioral factors. In physiologic studies, sleep onset has an inhibitory effect on cortisol release, while awakening stimulates cortisol secretion.²⁰ Sleep disturbances may therefore increase cortisol levels, which could negatively affect diabetes control by exacerbating insulin resistance and contributing to obesity. These issues may represent additional barriers to diabetes self-management and participation in weight loss activities. Inflammatory markers like TNF and IL-6, which are elevated in the presence of poor sleep, may also contribute to diabetes progression and poor control.²¹ Behavioral factors that could mediate the relationship between sleep disturbances and HbA1c include diabetes-related distress, diabetes burden, and other factors that were not assessed in our survey. Future studies should explore alternative physiologic behavioral factors that might mediate the relationship between sleep disturbances and HbA1c.

In light of the observed relationship between the PSQI sleep disturbances subscale and HbA1c in this analysis, disturbances like nocturnal awakening, overnight bathroom usage, and breathing issues may be deleterious to glycemic control. Such disturbances may be associated with a variety of conditions that commonly coexist with diabetes, including medication effects (e.g. insulin-related hypoglycemia), hyperglycemia-associated polyuria, and obstructive sleep apnea. Because these conditions are treatable using available approaches—continuous positive airway pressure for obstructive sleep apnea and medication adjustment for polyuria and hypoglycemia^{22,23}—sleep disturbances may represent an

important treatment target in diabetes. While the current cross-sectional analysis was not designed to ascertain whether treating sleep disturbances translates to better diabetes outcomes, it is notable that individuals with the highest sleep disturbances score had HbA1c values that were on average 1.4 points above those with the lowest score. If eliminating sleep disturbances could translate to HbA1c lowering of this degree, the effectiveness of this approach would equal or surpass that expected with most diabetes medications. The impact on diabetes control of therapeutic approaches targeting sleep disturbances should be explored prospectively.

Limitations

Along with those discussed above, this analysis has additional limitations. Several factors may affect the generalizability of our findings, including our study population's demographics (majority Black/African-American, income >\$30,000) and the STOP-DKD study's inclusion criteria (e.g. ability to complete self-reported instruments). Further, because our cohort had reasonably good diabetes control overall (mean HbA1c 8.0%), our findings may not apply to more poorly-controlled patients. Our population, while uniquely well-characterized with survey instruments and other data collected as part of a randomized trial, may not have provided adequate power to detect all relevant associations between PSQI subscales and HbA1c. However, insufficient power would not invalidate the observed relationship between sleep disturbances and HbA1c.

Despite the use of validated instruments to measure sleep quality, medication adherence, depression, and other psychosocial constructs, these self-reported measures may be susceptible to social desirability, recall, and other biases. Our use of a self-reported medication adherence measure may have limited our mediation analysis, because self-reported measures correlate imperfectly with objective adherence measures like pill count or refill frequency.¹³ Unfortunately, such objective measures are not utilized clinically to assess insulin adherence, so self-reported measures may represent the best available option, despite their limitations.²⁴

Importantly, this study used cross-sectional data, so while the observed association between sleep disturbances and HbA1c is noteworthy, establishing causation or directionality is beyond the scope of this analysis.

Conclusions

Our study demonstrated that the PSQI sleep disturbances subscale was associated with higher HbA1c among patients with diabetes, while other subscales were not associated with HbA1c. Although long-term prospective studies are needed to confirm these results, our findings suggest that sleep disturbances may be an important target for intervention to enhance diabetes control and reduce the continued accrual of complications and costs.

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DKD trial patient baseline characteristics demographic and clinical characteristics by PSQI score.*

Table 1.

Variable	Overall (n = 281)	PSQI 5 (n = 132)	PSQI > 5 (n = 149)	p-Value ^a
Demographics/patient characteristics				
Mean age ± SD	61.9 ± 8.8	63.0 ± 8.3	60.9 ± 9.2	0.06
Sex				<0.01
Female	136 (48.4)	45 (34.1)	91 (61.1)	
Male	145 (51.6)	87 (65.9)	58 (38.9)	
Race				0.41
White or Caucasian	116 (41.3)	60 (45.5)	56 (37.6)	
Black or African American	156 (55.5)	68 (51.5)	88 (59.1)	
Other	9 (3.2)	4 (3.0)	5 (3.4)	
Income				0.16
<\$30,000	82 (29.2)	33 (25.0)	49 (32.9)	
\$30,000	189 (67.3)	95 (72.0)	94 (63.1)	
Don't know/refused	8 (2.9)	2(1.5)	6 (4.0)	
Missing	2 (0.7)	2(1.5)	0 (0)	
Medical comorbidities				
Diabetes treatment regimen				
Insulin-based	124 (44.1)	62 (47.0)	62 (41.6)	0.34
Noninsulin-based	155 (55.2)	68 (51.5)	87 (58.4)	
Missing	2 (0.7)	2(1.5)	0 (0)	
BP (mmHg)				
Mean systolic BP ± SD	134.3 ± 19.5	134.7 ± 17.9	134.0 ± 20.8	0.45
Mean diastolic BP (SD)	76.3 ± 13.5	75.1 ± 12.5	77.3 ± 14.3	0.19
Mean HbA1c (SD)	8.0 ± 1.8	7.9 ± 1.7	8.0 ± 1.9	1
Mean LDL cholesterol (SD)	89.8 ± 39.1	85.2 ± 39.6	93.8 ± 38.4	0.04
BMI				
BMI <25	11 (3.9)	5 (3.79)	6 (4.0)	0.33
BMI 25–29.9	58 (20.6)	32 (24.2)	26 (17.5)	
BMI >30	210 (74.7)	93 (70.5)	117 (78.5)	

Variable	Overall (n = 281)	PSQI 5 (n = 132)	PSQI > 5 (n = 149)	p-Value ^a
Missing	2 (0.7)	2 (1.5)	0 (0)	
Psychosocial factors				
8-item medication adherence Measure				<0.01
Nonadherent (<6)	97 (34.5)	32 (24.2)	65 (43.6)	
Adherent (6–8)	182 (64.8)	98 (74.2)	84 (56.4)	
Missing	2 (0.7)	2 (1.5)	0 (0)	
Depression (PHQ-2)				<0.01
PHQ-2 < 3	250 (89.0)	125 (94.7)	125 (83.9)	
PHQ-2 ≥ 3	29 (10.3)	5 (3.8)	24 (16.1)	
Missing	2 (0.7)	2 (1.5)	0 (0)	
Mean life chaos score	6.81 ± 4.0	6.11 ± 3.8	7.42 ± 4.0	0.01
PSQI	6.26 ± 4.0	2.97 ± 1.5	9.18 ± 3.2	NA

BMI: body mass index; BP: blood pressure; HbA1c: hemoglobin A1c; LDL: low-density lipoprotein; NA: not applicable; PHQ-2: Personal Health Questionnaire-2; PSQI: Pittsburgh Sleep Quality Index; SD: standard deviation.

* Two patients had missing information and are shown in the table.

^a p-Values not adjusted for multiple comparisons, included as descriptive measures of imbalance between groups.

Table 2.

PSQI components and association with HbA1c ($n = 279$).^a

PSQI Subscale ^b	Estimate ^c	95% CI	p-Value
Global PSQI score	0.01	-0.05 0.07	0.71
PSQI subscales			
Subjective sleep quality	-0.05	-0.32 0.22	0.72
Sleep latency	0.07	-0.14 0.29	0.52
Sleep duration	0.03	-0.19 0.24	0.81
Habitual sleep efficiency	0.08	-0.12 0.28	0.44
Sleep disturbances	0.41	0.06 0.76	0.02
Use of sleep medication	-0.13	-0.32 0.07	0.20
Daytime dysfunction	0.14	-0.20 0.48	0.41

CI: confidence interval; PSQI: Pittsburgh Sleep Quality Index.

^aTwo patients excluded from model for missing income data.

^bIndividual model fit for each PSQI subscale.

^cEstimate reflects model-estimated mean change in HbA1C for each 1-point increase in score, adjusted for age, sex, race, income, BMI, depression, and life chaos.

Table 3.

PSQI components and association with medication nonadherence ($n = 279$).^a

PSQI Subscale	OR ^b	95% CI	P-Value	
Global PSQI score	1.09	1.01	1.17	0.027
PSQI subscales				
Subjective sleep quality	1.29	0.91	1.83	0.148
Sleep latency	1.02	0.78	1.35	0.862
Sleep duration	1.17	0.89	1.54	0.265
Habitual sleep efficiency	1.26	0.97	1.63	0.085
Sleep disturbances	2.04	1.27	3.30	0.003
Use of sleep medication	1.05	0.82	1.34	0.713
Daytime dysfunction	2.05	1.31	3.22	0.002

CI: confidence interval; OR: odds ratio; PSQI: Pittsburgh Sleep Quality Index.

^aTwo patients excluded from model for missing income data.

^bAdjusted for age, sex, race, income, BMI, depression, and life chaos.