



The prevalence of poor sleep quality and its associated factors in patients with interstitial lung disease: a cross-sectional analysis

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ABSTRACT

Background: Many patients with interstitial lung disease (ILD) experience poor sleep quality, which may contribute to decreased quality of life. Sleep disordered breathing is commonly associated with ILD but there is less information on other factors that may contribute to poor sleep quality.

Methods: We conducted a cross-sectional analysis of 101 patients with a diagnosis of ILD at a pulmonary rehabilitation assessment clinic. We assessed the prevalence of poor sleep quality using the Pittsburgh Sleep Quality Index (PSQI) and performed multivariable logistic regression analysis to determine factors independently associated with poor sleep quality.

Results: Median forced expiratory volume in 1 s was 64% predicted (interquartile range (IQR) 50–77%) and vital capacity was 62% predicted (IQR 48–78%). 67 (66%) out of 101 patients reported poor sleep quality. The median PSQI was 8 units (IQR 4–11 units). There were no significant differences in physical or physiological parameters including age, sex distribution, body mass index or spirometry values between subjects with good sleep quality and those with poor sleep quality (all $p > 0.1$). Multivariable logistic regression showed that depression ($p = 0.003$) and Epworth Sleepiness Scale ($p = 0.03$) were independently associated with poor sleep quality.

Conclusion: Poor sleep quality is common in patients with ILD and is independently associated with increasing symptoms of depression and sleepiness. Routine assessment of sleep quality should be undertaken and interventions targeting depression and coexisting sleep disorders may be required in symptomatic patients to determine if sleep quality and ultimately, health-related quality of life improves as a result.



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Introduction

Interstitial lung diseases (ILDs) are a heterogeneous group with diffuse parenchymal lung involvement and considerable variation in clinical courses and prognoses [1]. As ILD progresses over time it is often associated with significant disease burden, and as a result, progressive dyspnoea and poorer quality of life may occur [2, 3]. Many of the ILDs deteriorate over time despite treatment of the underlying disease process; therefore, symptom management and improving quality of life is an essential target for treatment [4–7]. Symptom management in ILD has mainly focused on dyspnoea and cough. However, sleep quality is also an important consideration in this population, as poor sleep quality can contribute to decreased quality of life [8–10].

Most studies of sleep in patients with ILD have focused on polysomnographic abnormalities and nocturnal desaturation [11–16], but a greater understanding of psychological and patient-reported outcomes is required to identify factors associated with poor sleep quality to help guide future treatment and management. Furthermore, apart from a recent study looking at the impact of sleep disordered breathing on quality of life in idiopathic pulmonary fibrosis (IPF) patients [17], previous studies on sleep quality in ILD patients have only reported mean or median Pittsburgh Sleep Quality Index (PSQI) values and there are scant data regarding the prevalence of poor sleep quality. Therefore, we wanted to assess the prevalence of poor sleep quality in a diverse population of patients with ILD, find independently associated factors using a multivariable logistic regression model to identify possible intervention targets, and examine individual PSQI components to understand the different symptomatology between good sleepers and poor sleepers.

Methods

Study design and setting

We performed a retrospective, cross-sectional study of 101 community-based patients with ILD referred to an outpatient hospital-based pulmonary rehabilitation programme between June 2014 and June 2018. This study was approved by the Western Sydney Local Health District Human Research Ethics Committee (HREC2017/7/6.9(5228) QA).

Participants

Subjects were referred by respiratory physicians for suitability for pulmonary rehabilitation and were assessed at either of two university teaching hospitals (Westmead Hospital and Blacktown Hospital in Sydney, Australia). Eligibility criteria for study entry included a respiratory physician diagnosis of ILD and age over 18 years.

We assessed sleep quality with the PSQI questionnaire [8], which is a self-administered, validated 19-item questionnaire assessing sleep disturbance and usual sleep habits in the past month. It consists of seven components which include sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbance, use of sleeping medications, and daytime dysfunction with each item being scored from 0 to 3 on a Likert scale. The sum of the seven components results in a global PSQI score ranging between 0 and 21, where higher scores represent worse sleep quality. A score of >5 had 89.6% sensitivity and 86.5% specificity in distinguishing good sleepers from poor sleepers. In addition to the Likert scale items, the PSQI contains questions regarding bedtime, subjective sleep latency, wake time and subjective sleep duration.

All subjects underwent a comprehensive multidisciplinary assessment prior to commencement of the rehabilitation programme. We collected anthropometric data including age, sex, body mass index (BMI), neck circumference, smoking status and lung function as measured by spirometry (MicroLab 36-ML3500 MK8-STK, CareFusion). We assessed health-related quality of life with the St George's Respiratory Questionnaire (SGRQ) [18], anxiety and depression with the Hospital Anxiety and Depression Scale (HADS) [19] and breathlessness with the modified Medical Research Council (mMRC) dyspnoea scale [20]. We assessed daytime subjective sleepiness with the Epworth Sleepiness Scale (ESS) [21] and screened for obstructive sleep apnoea (OSA) using the STOP-Bang questionnaire [22], which is an eight item "yes–no" questionnaire that determines the risk of OSA; "yes" to less than three items inferred a low risk of OSA, while "yes" to three or more items inferred an intermediate to high risk of OSA. We recorded all medications that patients were on at the time of assessment. All data were recorded and available for review *via* electronic medical records. Available data were transferred onto an Excel spreadsheet and de-identified prior to data analysis.

Data analysis

Our primary outcome variable was sleep quality using the PSQI questionnaire. Subjects were categorised as having poor sleep quality (PSQI >5) or good sleep quality (PSQI ≤ 5) [8] to estimate poor sleep quality

prevalence. We compared subject data using the Mann–Whitney test for non-parametric continuous variables or an unpaired t-test for parametric continuous variables. Fisher’s exact test was used for categorical variables. We expressed parametric data as mean±SD and non-parametric data as median (interquartile range (IQR)). We performed univariable logistic regression to screen for factors associated with poor sleep quality. Our dependent variable was PSQI expressed as a binary outcome (*i.e.* poor sleep quality *versus* good sleep quality). STOP-Bang data were categorised as a binary variable with low risk *versus* intermediate/high risk of OSA [22]. For smoking status, we created a “never smoker” group and combined ex-smokers and smokers as there was only one current smoker in the study. BMI was expressed as a tertile. Age, HADS, ESS and SGRQ total score were expressed as quartiles. Independent variables with $p < 0.25$ in the univariable analysis were included in a base model and a backward elimination method was used to select variables with $p < 0.05$ in our multivariable model. We used pairwise deletion analysis to deal with missing data. We assessed the multivariable model goodness of fit with the Hosmer–Lemeshow test and the strength of fit with the c-statistic. We examined regression diagnostic plots for any unusual or influential points. We used SAS Studio version 3.71 (SAS Institute, Cary, NC, USA) for statistical analyses. A two-sided p-value of < 0.05 was considered statistically significant.

Results

101 patients with a known diagnosis of ILD were assessed for enrolment into pulmonary rehabilitation and included in the analysis. 38 (38%) out of 101 subjects had a diagnosis of IPF. A further 16 had connective tissue disease-associated ILD, 10 had nonspecific interstitial pneumonitis, 14 had combined pulmonary fibrosis and emphysema, four had asbestosis and four had sarcoidosis. The remainder ($n=15$) were reported as having pulmonary fibrosis with no specific details from their referring physician. We had very few missing data overall; we did not have spirometry data for three subjects, SGRQ for two subjects, and HADS and STOP-Bang data for one subject each (table S1).

Median forced expiratory volume in 1 s (FEV₁) was reduced at 64% predicted (IQR 50–77%), as was vital capacity (VC) at 62% predicted (IQR 48–78%). A majority of patients (67 (66%) out of 101) reported poor sleep quality based on a PSQI score of more than 5 units (figure 1). The median PSQI was 8 units (IQR 4–11 units). There were no significant differences in physical or physiological parameters, including age, sex distribution, BMI or neck circumference, between subjects with good sleep quality and those with poor sleep quality (all $p > 0.1$; table 1). In particular, there was no significant difference in FEV₁ ($p=0.4$) or forced vital capacity (FVC) ($p=0.54$). Although there was no difference between the two groups in the risk of OSA based on the STOP-Bang questionnaire, subjects with poor sleep quality had increased subjective sleepiness with a higher ESS than those with good sleep quality (7 *versus* 4 arbitrary units (au), respectively; $p=0.003$). The poor sleep quality group had a higher total SGRQ score (58.0±16.0 *versus* 46.1±16.8 au, respectively; $p=0.001$), and higher HADS scores for both anxiety (HADS-A 7.1±4.6 *versus* 4.4±3.4 au, respectively; $p=0.004$) and depression domains (HADS-D 6.8±3.8 *versus* 4.4±3.9 au, respectively; $p=0.004$). The overall prevalence of clinically significant depression symptoms (*i.e.* HADS-D > 7 au) was 32% and was higher in those with poor sleep quality (43%) compared with the good sleep quality group (15%). Clinically significant anxiety (*i.e.* HADS-A > 7 au) was present in 34% of patients overall and was higher in the group with poor sleep quality at 43% compared with 18% in the good sleep quality group. There was no significant difference in mMRC dyspnoea scale between the poor and good

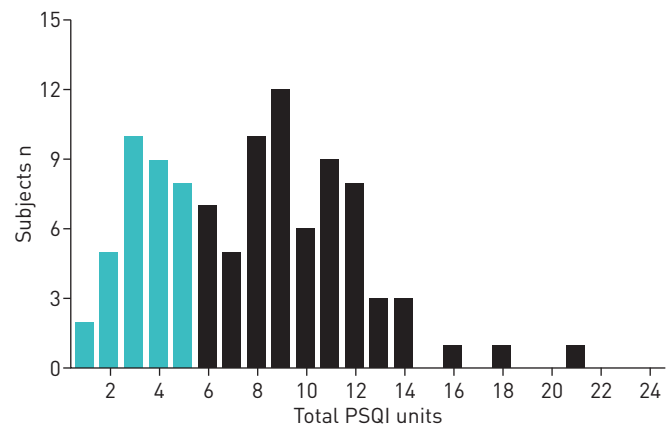


FIGURE 1 Frequency histogram of the distribution of Pittsburgh Sleep Quality Index (PSQI) for 101 patients. Green shaded columns represent normal PSQI (*i.e.* ≤ 5 units), black columns represent increased PSQI (> 5 units).

TABLE 1 Baseline characteristics of patients with interstitial lung disease

Variable	Poor sleep quality (PSQI>5)	Good sleep quality (PSQI≤5)	p-value
Subjects n	67	34	
Age years	68.3±10.4	71.6±10.3	0.13
Males n (%)	35 (54%)	21 (62%)	0.52
BMI kg·m⁻²	28.1 [25.2–32.9]	27.9 [23.8–32.5]	0.57
FEV₁ % pred	65.1±19.2	68.7±20.4	0.40
FVC % pred	63.3±18.8	65.9±20.5	0.54
STOP-Bang au	4.0±1.6	3.6±1.4	0.19
ESS au	7 [4–8]	4 [2–7]	0.003
Neck circumference cm	39.7±4.8	39.3±3.7	0.75
mMRC au	2.3±1.1	1.9±1.0	0.08
SGRQ-Symptoms	57.2±20.7	39.4±19.2	<0.0001
SGRQ-Activities	75.8±19.4	66.7±19.0	0.03
SGRQ-Impacts	48.0±20.2	35.7±19.7	0.005
SGRQ-Total au	58.0±16.0	46.1±16.8	0.001
HADS-Anxiety au	7.1±4.6	4.4±3.4	0.004
0–7 n/N (%)	37/65 (57%)	28/34 (82%)	
8–21 n/N (%)	28/65 (43%)	6/34 (18%)	
HADS-Depression au	6.8±3.8	4.4±3.9	0.004
0–7 n/N (%)	37/65 (57%)	29/34 (85%)	
8–21 n/N (%)	28/65 (43%)	5/34 (15%)	

Data are presented as mean±SD or median (interquartile range), unless stated otherwise. PSQI: Pittsburgh Sleep Quality Index; BMI: body mass index; FEV₁: forced expiratory volume in 1 s; FVC: forced vital capacity; ESS: Epworth Sleepiness Scale; mMRC: modified Medical Research Council Dyspnoea Scale; SGRQ: St George's Respiratory Questionnaire; HADS: Hospital Anxiety and Depression Scale (0–7=normal, 8–10=borderline abnormal, 11–21=abnormal); au: arbitrary units.

sleep quality groups (2.3±1.1 *versus* 1.9±1.0 au, respectively; *p*=0.08). More patients with poor sleep quality (20 (29.9%) out of 67 patients) were on medications that could affect sleep compared with only three (8.8%) out of 34 patients with good sleep quality (*p*=0.014; see table S2 for list of medications).

PSQI questionnaire data showed the subjective median sleep latency was longer in patients with poor sleep quality compared with good sleep quality (30 min (IQR 15–45) *versus* 10 min (IQR 5–15); *p*<0.0001), with a shorter subjective sleep duration (6 h (IQR 5–7) *versus* 8 h (IQR 7–9); *p*<0.0001; table 2). All seven components making up the PSQI global score were higher in the group with poor sleep quality (all *p*≤0.004; table 2). Of note, the group with poor sleep quality had more daytime dysfunction (*p*=0.001) and greater use of sleep medications (*p*=0.004). Review of individual components of PSQI component 5 (sleep disturbance) showed that patients with poor sleep were more troubled by a variety of disruptions

TABLE 2 Sleep latency, sleep duration and Pittsburgh Sleep Quality Index (PSQI) component score data obtained from PSQI questionnaire

Variable	Poor sleep quality (PSQI>5)	Good sleep quality (PSQI≤5)	p-value
Sleep latency min	30 [15–45]	10 [5–15]	<0.0001
Sleep duration h	6 [5–7]	8 [7–9]	<0.0001
PSQI global score au	9.8±2.9	3.5±1.2	
PSQI component scores[#]			
Sleep quality	1.43±0.66	0.68±0.64	<0.0001
Sleep latency	1.77±0.90	0.44±0.61	<0.0001
Sleep duration	1.25±1.05	0.12±0.41	<0.0001
Habitual sleep efficiency	1.68±1.09	0.15±0.36	<0.0001
Sleep disturbance	1.97±0.69	1.38±0.55	<0.0001
Use of sleep medications	0.62±1.14	0.03±0.17	0.004
Daytime dysfunction	1.25±0.79	0.71±0.72	0.001

Data are presented as mean±SD or median (interquartile range), unless stated otherwise. au: arbitrary units. [#]: Each component score ranges from 0 (no difficulty) to 3 (severe difficulty) on a Likert scale.

TABLE 3 Individual questions for component 5 (sleep disturbance) in the Pittsburgh Sleep Quality Index (PSQI) questionnaire

Variable	Poor sleep quality (PSQI>5)	Good sleep quality (PSQI≤5)	p-value
Cannot get to sleep within 30 min	2 [1–3]	0 [0–1]	<0.0001
Wake up in the middle of the night or early morning	3 [2–3]	2 [0–3]	0.002
Have to get up to use the bathroom	3 [2–3]	3 [2–3]	0.45
Cannot breathe comfortably	2 [0–3]	0 [0–2]	0.02
Cough or snore loudly	3 [1–3]	1 [0–3]	0.07
Feel too cold	1 [0–3]	1 [0–1.25]	0.02
Feel too hot	1 [0–2.5]	0 [0–0.25]	0.0004
Have bad dreams	1 [0–3]	0 [0–0]	0.14
Have pain	1 [0–3]	0 [0–2]	0.006
Other reasons	1 [0–3]	0 [0–1]	0.001

Data presented as median (interquartile range), unless stated otherwise. Scores are as follows. 0: not during the past month; 1: less than once a week; 2: one or twice a week; 3: three or more times a week.

to their sleep including “cannot breathe comfortably” and “cough or snore loudly” compared with patients with good sleep (all $p < 0.05$; table 3); the exceptions being “have to get up to use the bathroom” which troubled both groups ($p = 0.45$), and “having bad dreams” ($p = 0.14$).

Univariable logistic regression demonstrated that poor sleep quality was associated with SGRQ total score ($\chi^2 = 14.1$, 3 DF, $p = 0.003$), HADS-A ($\chi^2 = 9.37$, 3 DF, $p = 0.025$), HADS-D ($\chi^2 = 13.54$, 3 DF, $p = 0.004$) and ESS ($\chi^2 = 7.71$, 3 DF, $p = 0.05$) (see online supplementary material). Age and mMRC dyspnoea scale were not significantly associated with poor sleep quality but were included in the multivariable base model (all $p < 0.25$). All other physical and physiological variables were not associated with poor sleep quality (all $p > 0.5$), including FEV₁ % predicted ($\chi^2 = 1.49$, 3 DF, $p = 0.68$) and VC % predicted ($\chi^2 = 0.62$, 3 DF, $p = 0.89$). An increased risk of OSA (*i.e.* STOP-Bang score ≥ 3) was also not associated with poor sleep quality ($\chi^2 = 0.27$, 1 DF, $p = 0.60$).

Multivariable logistic regression showed that HADS-D and ESS were independently associated with poor sleep quality (table 4). There is strong evidence that HADS-D was associated with poor sleep quality ($\chi^2 = 13.78$, 3 DF, $p = 0.003$) after adjusting for subjective sleepiness. The odds of poor sleep quality were 10 times higher (95% CI 2.5–40.0) in patients with HADS-D scores between 5 and 8 compared with patients with HADS-D scores between 0 and 2 after adjusting for the effect of sleepiness. There was also evidence of an association between increased sleepiness and poor sleep quality ($\chi^2 = 8.90$, 3 DF, $p = 0.03$) after adjusting for the effects of depression symptoms. Patients with ESS between 6 and 8 had an increased odds of poor sleep quality of 5.6 (95% CI 1.2–26.5) compared with patients with ESS of 0 to 2, after

TABLE 4 Final model from multivariable logistic regression

	Adjusted odds ratio [#] (95% CI)	Wald χ^2	DF	p-value
HADS-Depression score				
0 to 2	1.0 [¶]	13.78	3	0.003
3 to 4	2.59 [0.69–9.75]			
5 to 8	10.05 [2.5–40.0]			
≥ 9	8.91 [2.1–37.8]			
ESS score				
0 to 2	1.0 [¶]	8.90	3	0.03
3 to 5	1.19 [0.33–4.25]			
6 to 8	5.64 [1.2–26.5]			
≥ 8	4.47 [1.2–16.5]			

HADS: Hospital Anxiety and Depression Scale; ESS: Epworth Sleepiness Scale; DF: degrees of freedom.
[#]: adjusted for HADS-D and ESS; [¶]: reference category.

adjusting for HADS-D. Our final multivariable model also showed that there was no longer a significant association of SGRQ total score with poor sleep quality after adjusting for HADS-D and ESS.

The Hosmer–Lemeshow test demonstrated that our model fitted the data well ($\chi^2=3.13$, 7 DF, $p=0.87$) and the *c*-statistic was 0.78, indicating that our fitted regression had acceptable discrimination between subjects with good sleep *versus* poor sleep quality. Review of regression diagnostic plots did not indicate that there were unusual datapoints which had an undue influence on the results.

Discussion

To our knowledge, we present the largest study looking at sleep quality among patients with ILD. A recent study of 34 patients with IPF demonstrated the prevalence of poor sleep quality to be 47% [17]. We similarly demonstrate a high prevalence with two-thirds (66%) of our patients with ILD having poor sleep quality and this was not associated with any anthropomorphic or physiological factors, but was independently associated with more depression symptoms and increasing sleepiness. In particular, there were no independent associations with age, lung function or breathlessness. Our subjective sleep apnoea risk assessment using the Stop-BANG questionnaire was also not associated with poor sleep quality; however, a recent study found an association between poor sleep quality, sleep arousals and sleep disordered breathing [17], suggesting that these factors may also be important contributors to poor sleep quality in IPF.

Prior studies evaluating sleep quality in patients with ILD using the global PSQI score have shown elevated mean PSQI values. Our elevated median PSQI of 8 units is consistent with published data, including a cross-sectional study of 41 patients demonstrating a mean \pm SD PSQI of 6.3 \pm 3.7 [10], 15 patients with a mean PSQI of 9.8 \pm 2.3 [23] and 52 patients with a mean PSQI of 7.0 \pm 3.9 [24]. In a recent study of 34 IPF patients, median PSQI was 5 units but a subgroup with OSA and sleep-related hypoxaemia had an elevated median PSQI at 8.5 units [17]. Poor sleep quality was not associated with BMI, age, sex or lung function, which was consistent with our findings, but was significantly associated with decreased quality of life as assessed by SF-36 [10] and SGRQ [17], clinically meaningful depression [24], and the number of awakenings and apnoea index on polysomnography (PSG) [17]. Although we did not find an independent association of SGRQ with PSQI in our multivariable regression analysis as recently shown by Bosi *et al.* [17], this may be due to different measured characteristics of the two populations in these studies, or the use of different independent variables in our statistical analyses. However, our univariable analysis did show an association between these variables ($p=0.003$; see table S1), but an independent association with SGRQ was not supported by our multivariable regression model.

Our findings differ from previous studies in that we have not only looked at the total PSQI score, but also evaluated the individual domains making up the PSQI as well as the subjective sleep duration and sleep latency times, allowing for a better understanding of the poor sleep quality. Patients with poor sleep quality were more troubled by sleep initiation insomnia symptoms, reported waking up in the night frequently, had reduced self-reported sleep duration, and felt they were not able to breathe comfortably (table 3). It is possible that patients with ILD and poor sleep quality have a heightened sensitivity to factors that disturb sleep, as their sleep was also more affected by temperature changes and pain, albeit to a lesser degree. The potential impact of insomnia in ILD cannot be understated as there is a strong association between insomnia and major depression in general [25, 26], and insomnia is independently associated with worse health-related quality of life even after adjusting for the effects of depression, anxiety and comorbidities in patients with chronic illness [27]. As our patients with poor sleep quality subjectively took longer to fall asleep and had a shorter total sleep time in the setting of higher depression scores, targeting psychological distress through interventions such as mindfulness or cognitive behavioural therapy might be beneficial in improving sleep quality [28, 29] and subsequent quality of life. Medications for mood disorders and pain known to affect sleep may also be associated with poor sleep quality as 30% of patients with poor sleep quality were on these medications compared with <9% with good sleep quality.

There is a high prevalence of clinically meaningful depression symptoms in patients with ILD, which has been documented in up to 25% of patients [24, 30, 31]. We have demonstrated a prevalence of HADS-D >7 units in 43% of patients with poor sleep quality compared with only 15% of patients with good sleep quality, consistent with established strong links between poor sleep quality and psychological factors such as depression [32]. Furthermore, our data show that clinically significant depression scores are not necessarily required to have the association with poor sleep quality, as even patients with HADS-D values above 4 units had a nine to 10-fold odds of poor sleep quality, after adjusting for the effect of sleepiness.

Overall, our patients did not report excessive sleepiness. Despite a higher median ESS of seven in the group with poor sleep quality indicating more sleepiness than the group with good sleep quality, these ESS values fall within the normal range of sleepiness (*i.e.* ESS \leq 10) in reference to a normal population [21].

This finding has also been demonstrated in other studies of ILD patients [23, 33] and also in patients with chronic obstructive pulmonary disease [34] where overall ESS was found to be within the normal range. We have shown that an ESS above only 5 units was associated with over 4.5 times the odds of having poor sleep quality after adjusting for the effects of depression. Therefore, in ILD patients with poor sleep quality and an ESS >5 units, causes of poor sleep quality other than depression should be considered, including the possibility of comorbid symptomatic OSA, in view of the high prevalence of OSA in patients with ILD at between 59% and 90% [11–14, 35]. However, a full assessment of sleep quality cannot be obtained by PSG alone as the PSQI is a subjective global assessment over the previous month and has not been shown to correlate with single night polysomnographic variables in adult community populations [8, 36] or even with actigraphy over 14 days in older adults [37]. Therefore, obtaining both subjective and objective measures of sleep quality is required in patients with ILD who complain of sleep problems to cover the spectrum of causes for their poor sleep quality [37].

There are several limitations of our study. Although we have studied a mixed group of patients with ILDs, including IPF, connective tissue-related interstitial lung diseases, asbestosis and nonspecific interstitial pneumonitis, we are unable to determine if differences in sleep quality exist for different diagnoses of ILD. As this study occurred in the setting of assessment for pulmonary rehabilitation, we relied on a respiratory physician diagnosis of ILD and did not have access to radiology or histopathology to differentiate and confirm types of fibrotic lung disease. We did not perform overnight PSG on our patients to look for sleep disordered breathing or nocturnal hypoxaemia; correlates with objective nocturnal parameters would have been useful in confirming pathophysiological associations with poor sleep quality [17]. Finally, it is not possible to determine causation of poor sleep quality due to the cross-sectional design of this study. Nevertheless, our findings raise important hypotheses regarding possible causes of poor sleep quality and future targets for interventional studies.

Conclusion

There is a high prevalence of poor sleep quality in patients with ILD referred for pulmonary rehabilitation and this is independently associated with increasing symptoms of depression and sleepiness. Insomnia symptoms are also highly prevalent in these patients with poor sleep quality. Studies looking at comparison of subjective sleep assessment with objective measurements of sleep in ILD patients with poor sleep quality are required to target other potential areas of sleep to improve overall quality of life in this population. Intervention studies targeting depression and coexisting sleep disorders such as OSA are required to determine if sleep quality and ultimately, health-related quality of life improve as a result.

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Conflict of interest: None declared.

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