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Insomnia in Patients with COPD

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Study Objectives: Chronic obstructive pulmonary disease (COPD) is a leading cause of morbidity and mortality and may frequently be associated with sleep disturbances. However, the correlates of insomnia in COPD patients have not been well characterized. The aim of the current study was to describe the prevalence of insomnia disorder in COPD and to elucidate the demographic and clinical characteristics of COPD patients that are associated with insomnia.

Design: Cross-sectional study.

Setting: Clinic-based sample from an academic hospital.

Participants: Patients with stable COPD.

Measurements: An interviewer-conducted survey was administered to 183 participants with COPD. Seventy-two of these participants (30 with and 42 without insomnia) maintained a sleep diary and underwent actigraphy for 7 days.

Results: Insomnia (chronic sleep disturbance associated with impaired daytime functioning) was present in 27.3% of participants. Current tobacco users (odds ratio (OR), 2.13) and those with frequent sadness/anxiety (OR, 3.57) had higher odds, but oxygen use was associated with lower odds (OR, 0.35) of insomnia. Patients with insomnia had worse quality of life and a higher prevalence of daytime sleepiness. Actigraphy revealed shorter sleep duration and lower sleep efficiency, and a sleep diary revealed worse self-reported sleep quality in participants with insomnia.

Conclusion: Insomnia disorder is highly prevalent in patients with COPD; current tobacco use and sadness/anxiety are associated with a higher prevalence, and oxygen use with a lower prevalence of insomnia; patients with insomnia have poorer quality of life and increased daytime sleepiness; and insomnia is associated with worse objective sleep quality.

Keywords: Sleep, COPD, insomnia, smoking, depression, oxygen

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INTRODUCTION

Insomnia is a common sleep complaint and is present chronically in approximately 10% of the general population.[1] Primary insomnia is a major public health problem and leads to worse health-related quality of life (QOL) and decreased productivity.[2] However, relatively little attention has been paid to insomnia in medical disorders, including chronic obstructive pulmonary disease (COPD).

COPD affects approximately 24 million people in the United States and is a major cause of mortality and disability.[3] This constitutes a major burden on the public health system.[4] Klink and Quan[5] reported a high prevalence of insomnia symptoms in subjects with COPD. A later study by the same investigators correlated the presence of respiratory symptoms with insomnia complaints.[6] More recent studies have provided further evidence for association between COPD and insomnia.[7] However, the prevalence estimates for insomnia in these studies have varied, in part due to differences in definitions of insomnia, age ranges, and methodology. Furthermore, the correlates of insomnia in this population are yet to be clearly elucidated. Finally, QOL is reduced in COPD patients.[8] Similarly, reduced QOL is an established consequence of insomnia.[2] Whether QOL in those with COPD and concomitant insomnia is worse than that in patients with COPD but no insomnia is not known.

In contrast with the subjective self-report, which has been used in most previous studies, objective measures may provide additional information about sleep in patients with COPD. Actigraphy has been used to systematically evaluate sleep in medical conditions such as cirrhosis and postcoronary artery bypass grafting but not in COPD.[9] It has the advantage of providing data in the home environment over the duration of several days.

The aim of the current study was to describe the prevalence of insomnia disorder in patients with physician-diagnosed COPD, and to elucidate the demographic and clinical characteristics associated with insomnia in patients with COPD. We defined insomnia as combined sleep-specific complaints with associated daytime symptoms, in accordance with the American Academy of Sleep Medicine-suggested Research Diagnostic Criteria (RDC).[10] We also compared QOL in patients with COPD and insomnia and those without insomnia. Finally, we determined the relationship between objective sleep findings in patients with COPD and subjective sleep complaints by using actigraphy and sleep diaries.

METHODS

Study Design

We conducted the study in 2 stages. For the 1st stage, an interviewer-conducted survey was administered to 183 consecutive consenting patients with COPD seen in the pulmonary clinic of the Southern Arizona Veterans Affairs Health Care System (SAVAHCS). COPD was defined as the presence of a compatible history, forced expiratory volume in 1 sec/forced vital capacity (FEV1/FVC) < 0.70 on spirometry and no

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known history of asthma or cystic fibrosis. Exclusion criteria: all patients were assessed by board-certified pulmonologists, and those with an acute exacerbation within the prior 3 mo or on steroid taper were excluded. Other exclusion criteria included medical conditions that could result in disturbed sleep, such as significant chronic pain, symptomatic heart failure, restrictive lung disease, and nocturia (average > 4 times/night); history of depression; shift work; inability to obtain informed consent; mental retardation; and inability to speak or understand English. Furthermore, anyone with a history consistent with a sleep disorder other than insomnia was excluded. All participants were asked whether they would be willing to participate in the 2nd stage of the study, with a positive response from almost all respondents. One third of these respondents (n = 60) were randomly selected to participate in this part of the study. The sample was enriched with patients who reported having insomnia to achieve at least 30 subjects in each group. Eventually, from the 183 participants, 30 with insomnia and 42 without insomnia were recruited to maintain a sleep diary and undergo actigraphy for 7 days. The study was approved by the University of Arizona Human Subjects Protection Program and the SAVAHCS R&D Committee.

Assessment of COPD

For all prospective subjects, medical records were reviewed to determine whether spirometry had been performed in the preceding 12 mo. For those whose spirometry results were not available during this time frame (approximately 10%), spirometry was performed using a handheld Koko spirometer (Ferraris Respiratory, Louisville, CO). The Global Initiative for Chronic Obstructive Lung Disease (GOLD, http://www.goldcopd.com) criteria[11] were used to categorize severity. Dyspnea severity was assessed using the Medical Research Council dyspnea scale.[12]

Sociodemographic and Clinical Data

Age, sex, race, and body mass index were recorded in the interviewer-administered survey. Participants were shown different inhalers available through VA and were asked which ones they used regularly. They were also asked whether they used the inhalers within 2 hr before falling asleep. The inhalers used and the frequency of use were collated with the electronic patient health records.

Assessment of Sleep Disorders and QOL

The interviewer-administered survey included the Global Sleep Assessment Questionnaire (GSAQ) to screen for the presence of sleep disorders.[13] This questionnaire has a sensitivity of 93% for both sleep apnea and periodic limb movement, 96% for restless legs syndrome, and 100% for parasomnias.[13] The survey also included questions on sleep, daytime symptoms, excessive daytime sleepiness (Epworth Sleepiness Scale score, ESS),[14] sleep habits, presence of depression, alcohol and tobacco use, and shift work as well as the medications. Patients who performed shift work or with circadian rhythm sleep disorders were excluded. Insomnia was defined as history of frequent difficulty in initiating or maintaining sleep and significant disruption of daytime functioning for at least 1 mo.[10] After the initial interviewer-administered survey, all participants completed the Short Form-36 questionnaire (SF-36)[15] to determine QOL.

Actigraphic Evaluation and Sleep Diary

The 72 recruited participants were asked to wear a motion logger (Actiwatch, Ambulatory Monitoring Inc, Ardsley, NY) for 7 days. The participants were also required to maintain a logbook and document bedtime, rise time, estimated sleep latency, and total sleep time during the same period as actigraphy. The 7-day sleep logs maintained by the 72 participants were analyzed to evaluate daily sleep hours and subjective sleep quality (on a scale of 1–10, 10 being the best sleep quality). The subjects were also asked to rate how they felt every day on a scale of 1–3: 1 = tired, 2 = somewhat refreshed, 3 = refreshed. Logbook information was also used to set appropriate windows of analysis in actigraphic recordings.

Statistical Analyses

We compared sociodemographic factors, symptom scores, and severity of COPD between those with and those without insomnia using the Student *t*-test or analysis of variance. Comparisons of categorical variables were performed using the chi-square test. Multivariate regression model using variables significant at level $P < 0.1$ were constructed to determine factors independently associated with insomnia. The results were considered statistically significant at level $P < 0.05$. We also investigated the relationship between history of insomnia and QOL and ESS scores. Finally, actigraphic sleep measures were compared between subjects with and without insomnia. Data are expressed as mean ± standard deviation unless otherwise stated. For all tests, 2-tailed statistical significance was set at $P < 0.05$. Statistical analyses were conducted using SPSS 10.0 for Windows (SSPS Inc, Chicago, IL).

RESULTS

Baseline Characteristics

The characteristics of the 183 participants as well as the subgroup that maintained sleep logs (n = 72) are shown in Table 1. Of all participants, 57% had severe COPD (FEV1 < 50%). As expected from a VA population, most participants were male and elderly.

Sleep Complaints

Seventy-two percent of all subjects had at least 1 sleep complaint, and 50% had 1 or more sleep complaints more than 3 times a week. Insomnia (chronic sleep disturbance associated with impaired daytime functioning) was present in 27.3% (50 of 183) of participants. Current smokers had a significantly higher prevalence of insomnia than former smokers (41% versus 22%, P = 0.01). In contrast, the prevalence of insomnia was lower in participants who were receiving oxygen either intermittently/nocturnally or continuously than those not receiving supplemental oxygen (14.3% versus 34.2%, P = 0.03). A higher percentage of patients with insomnia compared with those without insomnia (24.5% versus 8.2%, P = 0.01) reported frequent feelings of sadness or anxiety. The prevalence of daytime sleepiness (ESS > 10) was also higher in patients with insomnia (36.4% versus 14.6%, P = 0.004).

Insomnia Disorder and COPD Severity

The FEV1 was not different among participants with insomnia (48.9% predicted) or those without insomnia (44.7% pre-

dicted, P = 0.17). The prevalence of insomnia was not significantly different in different GOLD stages of COPD. Table 2 shows the prevalence of insomnia, oxygen use, smoking, and inhaler use in different GOLD stages.

There was a significant inverse correlation between Medical Research Council (MRC) dyspnea scale score and FEV_1 (R = −0.34, P < 0.001). However, we did not find a correlation between MRC dyspnea scale score and insomnia (R = 0.03, P = 0.62).

Table 1—Baseline characteristics of the study participants

Variable	All participants (n = 183)	Participants who maintained sleep diary (n = 72)
Mean age (SD), yr	69.8 (9.2)	69.5 (9.7)
Male, n (%)	175 (95.6)	68 (94.4)
Mean body mass index (SD), kg/m^2	28.2 (5.7)	28.5 (5.9)
% predicted postbronchodilator FEV (SD), %	45.9 (18.6)	52.5 (17.6)
FEV1/FVC ratio (SD)	49.6 (12.5)	54.7 (11.2)
Oxygen users, n (%)	63 (34.4)	14 (19.4)
Current smoker, n (%)	56 (30.6)	22 (30.6)
ß-agonist inhaler users, n (%)	119 (65.0)	37 (51.4)
Anticholinergic inhaler users (ipratropium or tiotropium), n (%)	101 (55.2)	27 (37.6)
Feeling sad usually or always, n (%)	23 (12.6)	13 (18.1)

FEV1/FVC, forced expiratory volume in 1 sec/forced vital capacity; SD, standard deviation.

Table 2—Characteristics of participants according to the Global Initiative for Chronic Obstructive Lung Disease stages

Variable	Stage 1 (n = 6)	Stage 2 (n = 72)	Stage 3 (n = 53)	Stage 4 (n = 52)	P
Prevalence of insomnia (%)	33.3	30.6	35.8	13.5	0.059
Oxygen users (%)	0	13.9	34.0	67.3	< 0.001
Current smokers (%)	0	33.8	36.5	29.8	0.322
ß-agonist inhaler users (%)	50.0	38.9	73.6	94.2	< 0.001
Anticholinergic inhaler users (%)	16.7	29.2	62.3	88.5	< 0.001
Tiotropium users (%)	0	16.7	47.2	59.6	< 0.001
Formoterol	16.7	23.6	62.3	67.3	< 0.001
Feeling sad (%)	16.7	15.3	7.5	13.5	0.609

Table 3—Comparison of prevalence of insomnia among the participants who were or were not using specific inhalers regularly

Inhaler (number of patients using)	Insomnia among users (%)	Insomnia among non-users (%)	P
Ipratropium (33)	33.3	26.7	0.67
Combivent (22)	36.4	26.1	0.22
Tiotropium (68)	20.6	31.3	0.13
Albuterol (88)	22.7	31.6	0.19
Formoterol (86)	20.9	33.0	0.09
Steroid inhaler: mometasone/flunisolide/fluticasone (86)	22.1	32.0	0.18
β-agonist; albuterol or formoterol (119)	21.8	37.5	0.03
Anticholinergic inhaler; ipratropium or tiotropium (101)	24.0	30.8	0.37
Long-acting inhalers; tiotropium or formoterol (105)	21.0	35.9	0.03

Inhalers and Sleep

Different models were created (one with tiotropium as an anticholinergic agent and another with tiotropium as a long-acting inhaler) to assess whether anticholinergic inhalers or long-acting inhalers, respectively, affect sleep. Similarly, formoterol was assessed as a ß-agonist and a long-acting inhaler in different models.

Table 3 shows prevalence of insomnia among those who were or were not using specific inhalers regularly. There was a lower prevalence of insomnia in the participants using ß-agonist inhalers or either (or both) of the two long-acting inhalers, formoterol or tiotropium. Analyses revealed no difference in the number of hours before bedtime that the patients with insomnia or no insomnia last used their inhalers (2.5 ± 3.0 hr versus 2.4 ± 2.7 hr, P = 0.77). There was a nonsignificant trend toward lower prevalence of insomnia in those using ß-agonist inhalers (albuterol or formoterol) within 2 hr of bedtime (21.3% versus 33.8%, P = 0.07). None of the inhalers, when used within 2 hr of bedtime, was associated with a higher prevalence of insomnia.

Insomnia and QOL

The mean SF-36 scores for all participants are shown in Table 4. The scores were lower in insomniacs than noninsomniacs in several domains.

Multivariable Analysis

Multiple regression analyses were performed with presence of insomnia as the dependent factor. The analyses revealed that absence of oxygen use, smoking, and presence of sadness/anxiety were the only significant independent predictors of insomnia (Table 5).

Sleep Diary and Actigraphy

These analyses included the participants who maintained a sleep diary and underwent actigraphy. There was a significant correlation between sleep times reported in a sleep diary and that found on actigraphy (r = 0.56, P < 0.001). Overall, sleep

Table 4—The quality of life assessed by SF-36 scores in insomniac and non-insomniac participants with chronic obstructive pulmonary disease[a]

SF-36 dimension	Insomnia (mean ± SD)	No insomnia (mean ± SD)	P
Physical functioning (PF)	36.5 ± 28.2	39.0 ± 24.3	0.55
Role physical (RP)	26.5 ± 37.6	35.7 ± 40.3	0.16
Bodily pain (BP)	51.6 ± 22.6	67.3 ± 24.6	< 0.001
General health (GH)	43.9 ± 19.2	47.1 ± 21.7	0.35
Vitality (VT)	33.8 ± 19.0	48.8 ± 20.1	< 0.001
Social functioning (SF)	62.0 ± 28.1	73.9 ± 22.3	0.003
Role-emotional (RE)	63.2 ± 43.7	76.3 ± 38.6	0.05
Mental health (MH)	68.6 ± 20.6	76.4 ± 17.1	0.03

[a]The 'scores in COPD' refer to those seen in an earlier study of more than 300 male COPD participants.[18] The last column provides scores seen in participants with severe insomnia in another study for comparison.[17] COPD, chronic obstructive pulmonary disease; SD, standard deviation.

Table 5—Multiple logistic regression results for prediction of insomnia among participants with chronic obstructive pulmonary disease

Variable[a]	OR	95% CI	P
Oxygen use	0.35	0.14 – 0.90	0.029
Smoking	2.13	1.02 – 4.44	0.044
ß-agonist use	0.83	0.27 – 2.43	0.69
ß-agonist within 2 hr of bedtime	0.91	0.34 – 2.45	0.85
Tiotropium/formoterol use	0.75	0.28 – 1.98	0.55
Sadness/anxiety	3.57	1.31 – 9.62	0.013

[a]Oxygen use was associated with lower odds of insomnia, whereas smoking and frequently feeling sad or anxious were associated with higher odds of insomnia. CI, confidence interval; OR, odds ratio.

Table 6—Actigraphically determined sleep variables in participants with chronic obstructive pulmonary disease with and without insomnia[a]

	Insomnia	No insomnia	P
Mean sleep time (minutes, mean ± SD)	379.5 ± 65.3	448.8 ± 75.6	< 0.001
Sleep efficiency (%, mean ± SD)	75.2 ± 9.4%	80.6 ± 11.1%	0.03
Wakefulness after sleep onset (min, mean ± SD)	116.7 ± 51.7	96.9 ± 56.0	0.02

[a]Insomniacs had lower sleep time and sleep efficiency and higher wakefulness after sleep onset compared with those without insomnia. SD, standard deviation.

time reported in a sleep diary was not significantly different from the actigraphic sleep time (426.1 ± 86.6 min versus 412.5 ± 80.6 min, P = 0.2). The persons with insomnia had shorter self-reported sleep time on sleep diary than those without insomnia (403.3 ± 88.8 min versus 444.5 ± 91.0 min, P = 0.04). Actigraphy confirmed lower total sleep time, sleep efficiency, and higher wakefulness after sleep onset in insomniacs (Table 6). There was a trend toward shorter sleep latency in patients with insomnia in comparison with those without insomnia (8.5 ± 33.8 min in those with insomnia versus 11.2 ± 28.9 min in those without insomnia, P = 0.05). The participants with insomnia had lower average self-reported sleep quality (5.8 ± 1.4 versus 7.8 ± 1.6, P < 0.001; 10 being the best sleep quality and 1 being the worst) than those without insomnia. The mean score of the question "how do you feel today (1 = tired, 2 = somewhat refreshed, 3 = refreshed)" was also lower in those with insomnia than those without insomnia (1.6 ± 0.5 versus 2.2 ± 0.5, P < 0.001).

DISCUSSION

The current study used face-to-face interviews and found an approximately 3-fold higher prevalence of insomnia disorder in patients with COPD compared to what has been reported in the general population.[16] Current tobacco use and presence of sadness/anxiety were associated with a higher prevalence of insomnia, but oxygen use was associated with lower prevalence of insomnia. Presence of insomnia, in turn, was associated with increased daytime sleepiness, poorer QOL, shorter sleep duration, and worse self-reported sleep quality.

The high prevalence of insomnia in patients with COPD confirms earlier reports suggesting disturbed sleep in COPD.[5,17] The prevalence of insomnia would probably have been even higher if we had included patients with depression, a condition that can frequently occur in patients with chronic medical disorders. The strength of the current study is that it used the rigorous American Academy of Sleep Medicine-suggested diagnostic criteria[10] to assess the prevalence of insomnia symptoms accompanied by daytime consequences in this understudied population. Subjects with insomnia and daytime consequences typically have higher health care utilization than those reporting sleep complaints only.[18] The study also found that presence of insomnia portends a further reduction of the already reduced QOL in patients with COPD. The scores in patients with COPD and no insomnia were generally similar to those shown in more than 300 male patients with COPD in an earlier study.[19] Considering the current limited efficacy of medical therapy in significantly improving QOL in COPD, our data suggest that approaches to improve sleep will have a favorable impact on lifestyle in patients with COPD and insomnia.

Another finding of this study was the significantly lower prevalence of insomnia in patients on oxygen. Supplemental oxygen has several salutary effects in patients with COPD[20]: it may decrease minute ventilation,[21] alleviate nocturnal hypoxemia-associated arousals,[22] lower pulmonary artery pressures,[23-25] and lessen anxiety and depression,[26] all of which can potentially enhance sleep. Unfortunately, the role of oxygen in effecting sleep improvement and the effect of nocturnal versus daytime oxygen supplementation cannot be determined from this study. Prior studies have provided conflicting results on the effect of oxygen on sleep in patients with COPD. One study

revealed no effect of supplemental oxygen therapy on arousal frequency in patients with COPD.[27] Another study found no difference in sleep quality in a comparison of consecutive nights of compressed air or oxygen supplementation in patients with severe COPD.[28] In contrast, 2 other studies found a beneficial effect of oxygen on sleep.[29,30] The studies usually had a small number of subjects and assessed the effect of short-term rather than longer-term oxygen supplementation. Given the conflicting results of the effect of oxygen on sleep in patients with COPD from various studies, including the current study, a randomized clinical trial is needed.

The current study provides further evidence for a relationship between cigarette smoking and sleep disturbances.[31-34] Nicotine may disrupt sleep by stimulating the nicotine-acetylcholine receptors in the brain.[35] Nicotine withdrawal may have an adverse effect on sleep. Although assessment of the relationship between the interval between last cigarette smoked and bedtime and insomnia may provide some evidence for this hypothesis, time of last cigarette smoked was not collected in this study. Finally, although it is likely that the smoking may result in sleep disturbances, it is also possible that the poor sleep itself may contribute to smoking.

Prior studies have shown mixed effects of inhaled bronchodilator medications on sleep quality.[36-38] Our study did not show an adverse influence of any inhalers on sleep. Univariate analyses suggested lower insomnia prevalence in patients on ß-agonist inhalers, on long-acting inhalers (muscarinic or ß-agonist), and those using ß-agonists within 2 hr of sleep, although the effects were not statistically significant in multivariate analyses. Thus, our results challenge conventional wisdom that ß-agonist inhaler use in proximity to sleep adversely affects sleep quality.

The study did not demonstrate a relation between COPD severity and sleep quality. This observation might be explained by the presence of several other factors that might affect overall sleep quality. Hypoxemia, cigarette smoking, and psychiatric comorbidities, as shown in this study, may be some of these factors. Other factors not assessed in this study may include abnormal ventilatory mechanics and nocturnal respiratory symptoms such as cough and sputum production.[6] In addition, we did not find a correlation between daytime dyspnea and sleep complaints. Several physiologic alterations may contribute to worse dyspnea during sleep.[39] Functional residual capacity decreases in patients in the supine position. Hypoventilation, especially during rapid eye movement sleep, may contribute to hypoxemia and hypercapnia. Furthermore, the supine position may worsen ventilation/perfusion mismatch, cause nocturnal desaturations and hypercapnia, and may lead to disruption of sleep even in relatively compensated patients.[40] It may be important in future studies to differentiate nocturnal from diurnal dyspnea.

The study also demonstrates that patients with COPD and insomnia disorder have significant levels of sleepiness. This is in contrast to two small studies that did not find daytime sleepiness associated with poor sleep in patients with COPD.[41,42] One of these studies enrolled 14 patients with COPD[41] and the other assessed sleepiness in 15 nondiabetic postmenopausal women with moderate to severe COPD.[42] The current study is significantly larger and included daytime sleepiness as one of the criteria constituting daytime impairment; this may also partly explain the different results. Nevertheless, presence as well as consequences of sleepiness, including traffic accidents and decreased productivity, should be explored further.

We used actigraphy and sleep records to assess sleep in this study. Although not as accurate as polysomnography (PSG) for evaluating sleep on a single night, the ability to record sleep for longer periods allows actigraphy to provide an objective insight into the nightly sleep variability often seen in patients with insomnia, making it more practical than PSG.[9] Both objective and subjective reports of sleep obtained from actigraphy and a sleep diary, respectively, indicated poor sleep in patients with insomnia. Although sleep latency appeared to be shorter in patients with COPD and insomnia, which may reflect increased sleepiness and primarily sleep-maintenance insomnia, we suggest caution in deriving conclusions from this finding because actigraphy has been suggested to be less accurate in determination of this sleep variable.[43] The fact that the time from when the sleep diary was used to infer bedtime further decreases the reliability of this measure. The sleep latency in patients with COPD in the current study was slightly lower than that seen in a recent polysomnographic study.[44] Notably, a recent study demonstrated that actigraphy underestimated sleep latency when compared with PSG.[45] The current study did not find differences in actigraphy or sleep diary reports in those with depression symptoms or a history of oxygen use or smoking in comparison with those without these respective traits, which may be due to the smaller numbers in this part of the study. Larger trials need to be carried out to assess the effect of these specific variables on objective sleep quality.

The study has several limitations. First, the study collected cross-sectional rather than prospective longitudinal data, precluding determination of causality. Second, although we excluded patients with current or prior diagnosis of or on therapy for depression, it is possible that the presence of depression or anxiety symptoms may be primary contributors to sleep problems in the study participants reporting insomnia. However, the associations of cigarette smoking and oxygen use with insomnia were independent of feelings of sadness or anxiety. Third, actigraphy, unlike PSG, does not provide information about sleep-disordered breathing and periodic leg movement syndrome. We excluded the patients with diagnosed sleep apnea and those with suggestive symptoms such as loud snoring or witnessed apneas, and used GSAQ to help with detecting these disorders, albeit subjectively. However, although GSAQ helps exclude several sleep disorders, it has not been specifically validated in people with COPD. Fourth, the study lacked an age-matched control group free of respiratory disease; however, in comparison with historically reported figures in the general population, the prevalence of insomnia in our cohort was very high. Fifth, although we found a lower prevalence of insomnia in patients receiving supplemental oxygen, we do not have information about the number of hours every day that the therapy was used. Future studies using objective adherence data will help further clarify the effect of oxygen on diverse outcomes such as insomnia in COPD. Sixth, other factors such as socioeconomic status and caregiver status may influence sleep quality but were not assessed in the study. Finally, most study participants were men. Notably, studies have suggested that women are more likely to report insomnia than men.[18] It is, therefore, likely that a study of primarily female patients with COPD may find an even higher prevalence of insomnia than the current study.

In conclusion, we demonstrated that insomnia is highly prevalent in patients with COPD, and is independently associated with smoking and depression or anxiety symptoms and not being on supplemental oxygen therapy. Presence of insomnia in patients with COPD is also associated with increased daytime sleepiness and worse QOL. Considering the disease burden of COPD and insomnia, these results have important clinical and public health implications. Prospective studies need to be designed to ascertain the causality of factors associated with insomnia in patients with COPD. Furthermore, studies assessing effect of various therapies such as cognitive behavioral therapy, supplemental oxygen, or inhalers, administered singularly or in combination, on insomnia prevalence will help improve clinical decision making in patients with COPD, with a goal of improving daytime functioning and QOL in these patients.

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