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RESEARCH ARTICLE

Association between lung function and sleep disorder symptoms in a community-based multi-site case-finding study

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Summary

Obstructive airway disease is associated with sleep disturbances. We aimed to assess the relationship between lung function and sleep disorder symptoms using cross-sectionally collected data between March 2017 and August 2021 from the Undiagnosed Chronic Obstructive Pulmonary Disease and Asthma Population study, a prospective community-based multi-site case-finding study. Undiagnosed Chronic Obstructive Pulmonary Disease and Asthma Population study participants with respiratory symptoms but without diagnosed lung disease who completed spirometry and

the Global Sleep Assessment Questionnaire were included. We conducted multivariate linear regression models for forced expiratory volume in 1 s, forced vital capacity and forced expiratory volume in 1 s/forced vital capacity by Global Sleep Assessment Questionnaire responses adjusted for confounders. The same models were employed to examine respiratory symptoms, as reported on the St George's Respiratory Questionnaire and Chronic Obstructive Pulmonary Disease Assessment Test, by Global Sleep Assessment Questionnaire responses. Logistic regression models were used to assess the association of undiagnosed obstructive airway disease with sleep symptoms. Amongst 2093 adults included in the study, 48.3% were female and the median age was 63 years (interquartile range 53–72). Two-hundred and five (9.79%) subjects met spirometry criteria for undiagnosed chronic obstructive pulmonary disease, and 191 (9.13%) for undiagnosed asthma. There were no significant associations between spirometry measures and sleep symptoms ($p > 0.5$), controlling for age, sex, body mass index, smoking and comorbidities. Those with undiagnosed asthma were more likely to report insomnia "at least sometimes" versus "never" (odds ratio 2.58, 95% confidence interval: 1.27–6.19, $p = 0.02$). Respiratory symptoms were associated with sleep symptoms, with significant ($p < 0.05$) increases in St George's Respiratory Questionnaire and Chronic Obstructive Pulmonary Disease Assessment Test scores in those reporting most sleep symptoms. Overall, we found an association between undiagnosed asthma and insomnia, and between respiratory and sleep disorder symptoms.

KEYWORDS
obstructive airway disease, respiratory symptoms, sleep disorders, spirometry

1 | INTRODUCTION

Chronic obstructive pulmonary disease (COPD) and asthma are the most common forms of obstructive airway disease (OAD), both with high morbidity and mortality globally (Agustí et al., 2023; Venkatesan, 2023). Sleep is a complex physiological and behavioural process (Troynikov et al., 2018) associated with altered respiratory physiology compared with the wakeful state (Appelberg et al., 2007; Newton et al., 2014). Sleep disorders and sleep disorder symptoms are important comorbidities in OAD, and the body of literature on their association has grown substantially over the last decade (Alanazi et al., 2021; Belachew et al., 2022; Braido et al., 2021; Brumpton et al., 2017; Campos et al., 2017; Jonassen et al., 2018; Kim et al., 2021; Kleisiaris et al., 2014; Lal et al., 2020; Li et al., 2022; Luyster et al., 2016; Luyster et al., 2020; Nobeschi et al., 2020; Prasad et al., 2020; Shorofsky et al., 2019; Silva Júnior et al., 2017; Sundbom et al., 2020; Theorell-Haglöw et al., 2016; Vukoja et al., 2018; Wang et al., 2023).

Existing evidence suggests a possible link between OAD and sleep symptoms; however, the nature of this relationship is not well understood. Several studies note a higher prevalence of insomnia amongst those with COPD than in the general population (Agusti et al., 2011; McNicholas et al., 2013), and in particular increased sleep-onset latency in COPD compared with healthy individuals (McSharry et al., 2012; Theorell-Haglöw et al., 2016; Vukoja et al., 2018). While a strong association has also been identified between obstructive sleep apnea (OSA) and asthma, especially in uncontrolled asthma (Alanazi et al., 2021; Jonassen et al., 2018; Julien et al., 2009; Prasad et al., 2020), the association with COPD is less clear (Agusti et al., 2011; Kleisiaris et al., 2014; McNicholas et al., 2013; Sanders et al., 2003). OSA and COPD may coexist by chance rather than a pathophysiological association, but do share risk factors (such as increased body mass index [BMI] and smoking), and demonstrate an interplay of respiratory mechanics and ventilatory control (Verbraecken & McNicholas, 2013). Sleep quality is a self-perceived measure of great interest in the study of sleep disorders, and is typically measured via questionnaires such as the Pittsburgh Sleep Quality Index (PSQI; Alanazi et al., 2021; Braido et al., 2021; Campos et al., 2017; Nobeschi et al., 2020; Shorofsky et al., 2019; Soler et al., 2013; Vukoja et al., 2018; Zohal et al., 2013). Poor sleep quality has been shown to be an independent predictor of poor asthma control and is also associated with COPD (Agusti et al., 2011; McNicholas et al., 2013; McSharry et al., 2012; Nobeschi et al., 2020; Soler et al., 2013; Theorell-Haglöw et al., 2016; Vukoja et al., 2018; Zohal et al., 2013), with some studies showing lower sleep quality is associated with COPD exacerbations (Omachi et al., 2012; Shorofsky

et al., 2019), emergency health care utilization and mortality (Omachi et al., 2012).

There is a lack of consensus on the association between symptoms of sleep disorders and objective measures of lung function. While some studies demonstrate reduced lung function is associated with greater subjective sleep disturbances (Jonassen et al., 2018; Theorell-Haglöw et al., 2016; Zohal et al., 2013), others show an association with fewer objective indicators of sleep disorders (McSharry et al., 2012; Sanders et al., 2003). Adding to this contradiction, some studies have found no association between lung function and sleep disturbances, measured subjectively (Lal et al., 2020; Nobeschi et al., 2020; Vukoja et al., 2018) or objectively (Silva Júnior et al., 2017). Most data have also been gathered from clinically based samples as opposed to community-based studies (Brumpton et al., 2017; Jonassen et al., 2018; Lal et al., 2020; Luyster et al., 2020; Sanders et al., 2003; Shorofsky et al., 2019), and are therefore subject to referral bias.

To address this knowledge gap, our community-based study aimed to examine the association between decreased lung function, as measured by spirometry, and self-reported frequency of sleep disorder symptoms. We conducted this analysis in patients with respiratory symptoms but no prior diagnosis of lung disease, thus assessing the impact of undiagnosed OAD on sleep disorder symptoms. We hypothesized there would be a positive association between increased frequency of sleep disorder symptoms and reduced lung function.

2 | METHODS

2.1 | Study population

We conducted secondary data analyses using data cross-sectionally collected between March 2017 and August 2021 from the Undiagnosed COPD and Asthma Population (UCAP) study, a prospective community-based multi-site case-finding study. The details of the UCAP study design and protocol have been published previously (Huynh et al., 2022). Briefly, the UCAP study used random-digit dialling to recruit adults from the 17 most populous metropolitan areas in Canada. Participants were enrolled if they were ≥ 18 years old, experienced one or more respiratory symptom (i.e., shortness of breath, wheezing, increased mucus or sputum, or prolonged cough) in the past 6 months, and had no prior physician diagnosis of asthma, COPD or any other lung diseases. All potential participants, regardless of age, completed the Asthma Screening Questionnaire (ASQ; Shin et al., 2010). Participants ≥ 60 years old, and those < 60 years old with a score of < 6 on the ASQ, also completed the COPD-Diagnostic Questionnaire (COPD-DQ; Kotz et al., 2008). Participants scoring ≥ 20 points on the COPD-DQ or ≥ 6 on the ASQ, thresholds conveying high risk for COPD or asthma, respectively, were invited to the local study site for pre- and post-bronchodilator spirometry to confirm, or rule out, OAD. Amongst 1,182,406 individuals who received automated phone calls, 236,301 (20.0%) engaged with the call and 38,353 (3.2%) were interviewed to determine trial eligibility. Ultimately, following eligibility assessment, 2857 (0.2%) individuals completed pre- and post-bronchodilator spirometry (Aaron et al., 2024). Participants were then assessed for sleep disorder symptoms by completing the Global Sleep Assessment Questionnaire (GSAQ; Roth et al., 2002). We included all individuals from the UCAP study who completed both spirometry and the GSAQ.

2.2 | Diagnosis of OAD

Spirometry was performed following American Thoracic Society guidelines (Miller et al., 2005), and individuals with spirometry data that could not be interpreted were excluded ($n = 54$). Spirometry interpretation was performed by a UCAP study respirologist, who had access to the participants' smoking history to aid in OAD categorization. Those with forced expiratory volume in 1 s (FEV_1) improved by ≥ 12% and ≥ 200 ml following bronchodilator administration with 400 μg of salbutamol were categorized as "asthma." Participants whose post-bronchodilator FEV1/forced vital capacity (FVC) ratio was below the lower 95% confidence limit for a healthy individual, adjusted for sex, age and height, were categorized as "COPD." Participants meeting neither criterion were classified as "no OAD."

2.3 | Global Sleep Assessment Questionnaire

The GSAQ was administered to assess sleep disorder symptoms. The GSAQ is an externally validated (Vaidya et al., 2017; Vaidya et al., 2020), self-administered, 11-item questionnaire that screens for and distinguishes between seven sleep disorders: insomnia; insomnia associated with a mental disorder; OSA; restless legs syndrome (RLS); periodic limb movement disorder (PLM); parasomnias; and shiftwork sleep disorder (Roth et al., 2002). Participants responded to each questionnaire item with "never", "sometimes", "usually" or "always." The GSAQ's discriminative ability to identify sleep disorders as measured by the area under the curve (AUC) was 72% for insomnia, 78% for insomnia-mental, 88% for OSA, 84% for RLS and PLM, 95% for parasomnias, and 88% for shiftwork. Test–retest reliability of the GSAQ, as measured by the intra-class correlation coefficient, was also acceptable for insomnia (0.86), insomnia–mental (0.72), OSA (0.88), RLS (0.77), PLM (0.80), shiftwork (0.92), no-disorder (0.77), and multiple sleep disorder (0.75; Roth et al., 2002). In a recent prospective case–control study, for which sleep disturbances were confirmed by polysomnography (PSG) or disorder-based questionnaire, the overall sensitivity and specificity for sleep disorders in individuals with COPD were 90.9% and 70.58%, respectively. For insomnia, OSA and RLS, the sensitivity and specificity were 87% and 75%, 77% and 67%, and 90% and 80%, respectively (Vaidya et al., 2020).

For our analysis, responses to GSAQ item 4 ("*Did work or other activities prevent you from getting enough sleep?*") were excluded, as disturbance of sleep due to work was not relevant to our objective. Responses to item 10 ("*Did the following things disturb your sleep: pain,*

other physical problems, worries, medications, other?") and item 11 (*"Did you feel sad or anxious?"*) were also excluded from the analysis as the questions were not specific to sleep disorder symptoms.

Details of the GSAQ items can be found in Supplemental Table A.1 and in the data supplement.

2.4 | St George's Respiratory Questionnaire (SGRQ) and COPD Assessment Test (CAT)

The SGRQ (Jones et al., 1991) and CAT (Jones et al., 2009) are two self-administered questionnaires assessing respiratory disease-specific health and quality of life, with higher scores indicating a lower health status. The SGRQ measures health impairments in patients with asthma or COPD, with the questionnaire divided into three distinct components, comprising the Symptom score, Activity score, and Impacts score. Each component carries a score of 0–100, and all three are combined in a Total score of 0–100, with higher scores connoting greater burden of symptoms (Jones et al., 1991). The CAT questionnaire, scored 0–40, provides a reliable measure of COPD-related health status. See Text A.1 for questionnaire validation metrics. While the CAT was designed and validated for patients with COPD, asthma and COPD share symptomatology, and so this questionnaire was considered relevant in answering our question for the OAD population as a whole. In the present analysis, we used the SGRQ Symptom and Total scores as well as the CAT score. Of the 2093 individuals included in our study, 2059 (98.4%) completed the SGRQ and 2091 (99.9%) the CAT.

2.5 | Risk of OSA

Information on any prior diagnosis of OSA was not collected for UCAP participants. In our study, we therefore estimated the risk of OSA for all participants using the STOP-Bag (Waseem et al., 2022) risk score, which assigns points for snoring, tiredness, observed apneas, blood pressure, BMI, age and male gender. STOP-Bag has performed well in the diagnosis of moderate to severe OSA, with no improvement of diagnostic capacity after inclusion of neck circumference (DeLong's test; $p = 0.67$ and $p = 0.33$, respectively; Waseem et al., 2022). We combined demographic data and answers to GSAQ items 2 (daytime sleepiness), 3 (bothersome daytime sleepiness), 5 (snoring) and 6 (apneas) to calculate the STOP-Bag score. Neck circumference was not available for our participants. We analysed the score as a continuous variable, then dichotomized scores, with a score of ≥ 3 conferring high risk and < 3 conferring low risk for OSA (Waseem et al., 2022).

2.6 | Confounders and risk factors

The potential confounders and risk factors considered in our analysis were age, sex, BMI, smoking status and comorbidities. Smoking was separated by tobacco and marijuana products, and participants were categorized as tobacco smokers or marijuana smokers (Yes/No) if they currently smoked or had ever smoked any quantity of the respective substance. The following comorbidities were counted in our analysis if patients reported any history of a diagnosis: anaemia, cancer (excluding skin cancer), congestive heart failure, coronary artery disease (angina, heart attack), depression/anxiety, diabetes mellitus, gastro-esophageal reflux disease, systemic hypertension, liver disease, renal disease and stroke.

2.7 | Statistical analysis

We used descriptive statistics to characterize the study population as a whole and by OAD of interest. We used univariate (Model 1) and multivariate (Models 2–4) linear regression models to assess the relationship between post-bronchodilator FEV_1, FVC or FEV_1/FVC (continuous variables) and responses to individual GSAQ items. Responses of "sometimes", "usually" and "always" were compared with "never." We then dichotomized responses as "never" and "at least sometimes", the latter category consisting of the cumulative responses of "sometimes", "usually" plus "always", given bias associated with differentiating amongst affirmative answers. Confounders were entered in the statistical models sequentially: univariate analysis (Model 1), additionally controlled for age and sex (Model 2), then additionally controlled for BMI (Model 3) and, finally, additionally controlled for smoking status and comorbidities (Model 4).

We used chi-square tests to examine the association between undiagnosed COPD or asthma (Yes/No) with reported sleep disorder symptoms. Logistic regression models were then employed to adjust for confounders. As with the aforementioned linear regression models, confounders were included in this analysis sequentially in Models 1–4.

To better understand the association between OAD and sleep disturbances, we further explored the relationship between respiratory-related symptoms, measured by SGRQ and CAT total scores, and GSAQ responses. The SGRQ Symptom and Total scores as well as CAT scores were used as continuous variables to assess the burden of respiratory symptoms. As with spirometry measures, we used multivariable linear regression models to examine the effect of respiratory symptoms on sleep disorder symptoms, with each questionnaire score considered separately in the statistical model: first in the univariate analysis (Model 1), then adjusting sequentially for confounders (Models 2–4).

Finally, we examined the association between previously undiagnosed COPD or asthma and the risk of OSA based on STOP-Bag score. We first conducted a linear regression analysis using STOP-Bag scores as continuous variables, and then a logistic regression analysis using dichotomous STOP-Bag scores (low versus high risk of OSA). The same modelling approach as described above was applied. All statistical analyses were performed using R Statistical Software (version 1.4.1717; R Core Team, 2021).

3 | RESULTS

3.1 | Participants

Of 2420 individuals with a positive respiratory symptom screen, 2093 were included in this study after completing both valid spirometry and the GSAQ (Figure 1). The baseline characteristics of the study population as a whole and by OAD of interest are detailed in Table 1. Participants were 48.3% female, with a median age of 63 years (interquartile range [IQR] 53–72 years) and a mean BMI of 30.9 ± 7.2 kg m^{-2}. The mean STOP-Bag score was 3.8 ± 1.4, with 372 (17.8%) subjects identified at high risk for OSA. For post-bronchodilator measures, the mean FEV_1 percent predicted was 96.3% ± 17.9%, FVC percent predicted was 96.4% ± 15.8%, and FEV_1/FVC was 75.9 ± 8.98. Spirometry interpretation revealed 191 (9.1%) subjects meeting criteria for asthma and 205 (9.8%) for COPD. The median SGRQ Symptom score was 47.8 (IQR 32.7–62.8), and the mean SGRQ Total score was 36.6 ± 17.3, while the mean CAT score was 16.7 ± 6.9.

3.2 | Sleep disorder symptoms and spirometry measures

Suffering from bothersome daytime sleepiness "at least sometimes" (versus "never") was associated with increased FEV_1 in the univariate analysis; however, this finding became non-significant after controlling for confounders (Supplemental Table A.2). There was no significant association with FVC (Supplemental Table A.3). Experiencing insomnia symptoms and bothersome daytime sleepiness "at least sometimes" (versus "never") was associated with an increased FEV_1/FVC in the univariate analysis, but again these associations became non-significant controlling for confounders (Supplemental Table A.4). Reporting apneas corresponded with an increase in mean FEV_1/FVC in the univariate analysis, and this finding remained significant after adjusting for age and sex; nonetheless, further adjusting for BMI, smoking and comorbidities rendered the association non-significant (Supplemental Table A.4). Overall, there were no significant associations between any spirometry measure and sleep disorder symptoms reported on the GSAQ following adjustment for confounders (Figure 2).

3.3 | Sleep disorder symptoms and OAD

Responses to each GSAQ item are shown in Supplemental Table A.1 for all study participants, as well as stratified by OAD category. Individuals with undiagnosed asthma (versus without asthma) had higher odds of reporting insomnia symptoms "at least sometimes" versus "never" after adjusting for confounders: odds ratio (OR) 2.58, 95% confidence interval (CI): 1.27–6.19 ($p = 0.02$; Table 2). There was also a significant univariate association between reporting insomnia or apneas and not having undiagnosed COPD; however, this became non-significant after controlling for confounders (Table 3). Adjusted analyses revealed no other significant associations between sleep disorder symptoms and asthma or COPD diagnosis.

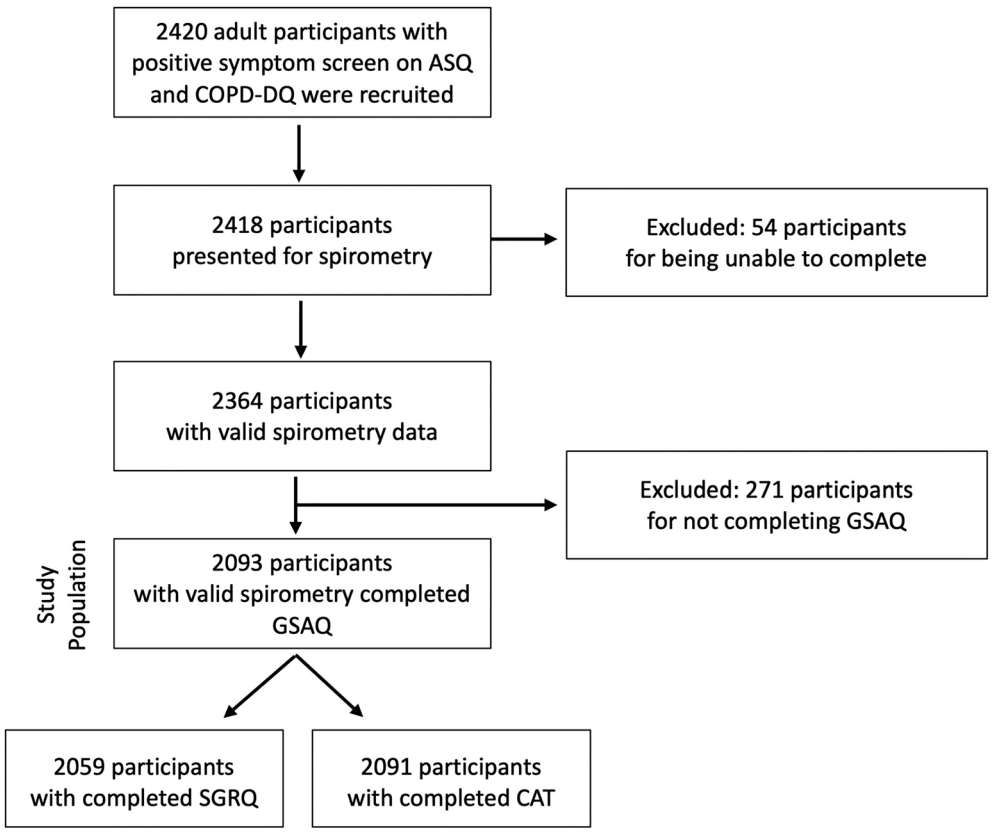

FIGURE 1 Study population flow chart. ASQ, Asthma Screening Questionnaire; CAT, COPD Assessment Test; COPD-DQ, COPD-Diagnostic Questionnaire; GSAQ, Global Sleep Assessment Questionnaire; SGRQ, St George's Respiratory Questionnaire.

TABLE 1 Baseline characteristics of the study population ($N = 2093$)

Variables	All participants[a]	OAD[a]		
		Asthma	COPD	No OAD
Demographics[b]				
Age, years – median (IQR)	63 (53–72)	62 (53–71)	68 (61–73)	63 (51–71)
Female gender	1011 (48.3)	85 (44.5)	77 (37.6)	849 (50.0)
BMI, kg m^{-2} – mean ± SD	30.9 ± 7.19	30.7 ± 7.34	28.8 ± 5.93	31.2 ± 7.31
Tobacco smokers[c]	1237 (59.1)	119 (62.3)	169 (82.4)	949 (55.9)
Marijuana smokers[c]	967 (46.3)	101 (52.9)	101 (49.3)	765 (45.1)
Number of comorbidities[d] – median (IQR)	2 (1–3)	2 (1–3)	1 (0–2)	2 (1–3)
Spirometry				
Pre-bronchodilator – mean ± SD				
FEV_1 (L)	2.68 ± 0.85	2.47 ± 0.77	2.06 ± 0.71	2.81 ± 0.82
FEV_1 (% predicted)	92.4 ± 18.3	82.7 ± 14.1	72.7 ± 16.3	96.9 ± 15.8
FVC (L)	3.66 ± 1.06	3.62 ± 1.11	3.51 ± 1.07	3.69 ± 1.05
FVC (% predicted)	96.0 ± 25.3	91.7 ± 15.6	92.0 ± 17.3	97.4 ± 27.0
FEV_1/FVC	73.2 ± 8.96	68.5 ± 7.50	58.6 ± 8.25	76.1 ± 5.97
Post-bronchodilator – mean ± SD				
FEV_1 (L)	2.79 ± 0.87	2.82 ± 0.85	2.16 ± 0.72	2.89 ± 0.85
FEV_1 (% predicted)	96.3 ± 17.9	94.6 ± 14.6	76.5 ± 16.3	99.7 ± 16.2
FVC (L)	3.69 ± 1.06	3.84 ± 1.09	3.61 ± 1.08	3.68 ± 1.05
FVC (% predicted)	96.4 ± 15.8	97.6 ± 14.9	94.6 ± 18.1	96.5 ± 15.6
FEV_1/FVC	75.9 ± 8.98	73.8 ± 7.60	60.3 ± 8.51	78.6 ± 5.88
Spirometry interpretation				
Asthma	191 (9.13)			
COPD	205 (9.79)			
Degree of obstruction[e]				
Mild	78 (38.4)			
Moderate	107 (52.7)			
Severe	18 (8.9)			
No OAD	1697 (81.1)			
Questionnaire scores				
STOP-Bag – mean ± SD	3.8 ± 1.4	4.0 ± 1.5	3.9 ± 1.3	3.8 ± 1.5
Low risk for OSA (< 3)	372 (17.8)	32 (16.8)	32 (15.6)	304 (18.4)
High risk for OSA (≥ 3)	1721 (82.2)	159 (83.2)	173 (84.4)	1348 (81.6)
CAT – mean ± SD	16.7 ± 6.9	17.7 ± 6.6	16.8 ± 6.5	16.5 ± 6.9
SGRQ				
Symptom score – median (IQR)	47.8 (32.7–62.8)	51.9 (37.3–67.5)	49.9 (32.73–63.11)	47.0 (32.2–61.9)
Total score – mean ± SD	36.6 ± 17.3	40.1 ± 17.4	36.0 ± 16.1	36.0 ± 17.3

[a]Unless otherwise specified, values represent number (%) of participants.
[b]Averages are represented as means ± SD for normally distributed variables and medians (IQR) for non-normally distributed variables.
[c]Current or previous smoking.
[d]Comorbidities: anaemia, cancer, congestive heart failure, coronary artery disease, depression/anxiety, diabetes mellitus, gastroesophageal reflux disease, systemic hypertension, liver disease, renal disease, stroke.
[e]COPD severity according to GOLD criteria.
BMI, body mass index; CAT, COPD Assessment Test; COPD, chronic obstructive pulmonary disease; FEV_1, forced expiratory volume in 1 s; FVC, forced vital capacity; IQR, interquartile range; OAD, obstructive airway disease; OSA, obstructive sleep apnea; SGRQ, St George's Respiratory Questionnaire.

3.4 | Respiratory symptom burden and sleep disorders

Respiratory symptoms reported on the SGRQ and CAT were significantly associated with sleep disorder symptoms, as represented by GSAQ responses (Supplemental Tables A.5–A.7). Reporting any sleep disorder symptoms "at least sometimes" (versus "never") corresponded with a significant increase in SGRQ Symptom scores, SGRQ Total scores and CAT scores (Figure 3) in the univariate analysis. These findings remained significant after adjusting for

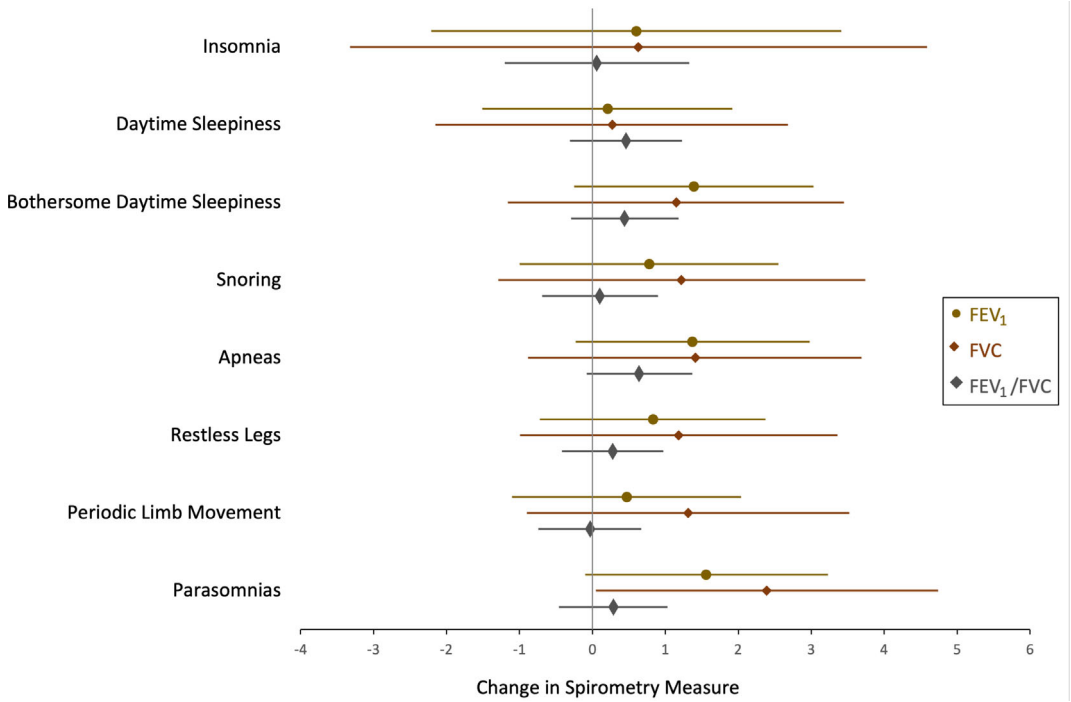

FIGURE 2 Change in spirometry measures, with 95% confidence intervals (CI), with reporting sleep disorder symptoms "at least sometimes" versus "never" on the Global Sleep Assessment Questionnaire (GSAQ), as determined using linear regression analysis accounting for confounders (age, sex, BMI, smoking and comorbidities). BMI, body mass index.

TABLE 2 The odds of sleep disorder symptoms occurring "at least sometimes" versus "never" (GSAQ responses) with undiagnosed asthma in the univariate analysis (Model 1) and multivariable logistic regressions (Model 2-4)[a]

GSAQ item	Model 1 OR (95% CI), *p*-value	Model 2	Model 3	Model 4
Item 1: Insomnia	2.56 (1.28–6.10), 0.02*	2.60 (1.29–6.21), 0.02*	2.61 (1.29–6.24), 0.02*	2.58 (1.27–6.19), 0.02*
Item 2: Daytime sleepiness	0.99 (0.72–1.39), 0.96	0.99 (0.71–1.38), 0.93	0.99 (0.71–1.39), 0.96	0.98 (0.71–1.38), 0.91
Item 3: Bothersome daytime sleepiness	0.97 (0.72–1.32), 0.85	0.97 (0.71–1.32), 0.83	0.97 (0.71–1.32), 0.83	0.95 (0.69–1.31), 0.74
Item 5: Snoring	1.15 (0.82–1.64), 0.44	1.14 (0.81–1.63), 0.47	1.14 (0.81–1.64), 0.46	1.13 (0.80–1.62), 0.50
Item 6: Apneas	1.17 (0.87–1.59), 0.29	1.15 (0.85–1.56), 0.36	1.16 (0.85–1.57), 0.34	1.15 (0.84–1.56), 0.39
Item 7: RLS	1.05 (0.78–1.41), 0.77	1.06 (0.79–1.43), 0.70	1.06 (0.79–1.43), 0.69	1.06 (0.78–1.43), 0.71
Item 8: PLM	1.19 (0.88–1.61), 0.25	1.17 (0.86–1.58), 0.31	1.17 (0.86–1.58), 0.31	1.16 (0.85–1.57), 0.35
Item 9: Parasomnias	1.13 (0.82–1.55), 0.43	1.11 (0.80–1.51), 0.53	1.11 (0.80–1.51), 0.54	1.08 (0.78–1.48), 0.64

Estimates are presented as OR and 95% CI.

[a]The univariate analysis is represented in Model 1, with confounders added sequentially: age and sex (Model 2), BMI (Model 3), smoking and comorbidities (Model 4).

*Indicates statistically significant values.

CI, confidence interval; GSAQ, Global Sleep Assessment Questionnaire; OR, odds ratio; PLM, periodic limb movement disorder; RLS, restless legs syndrome.

confounders, with one exception–snoring. Insomnia, daytime sleepiness, bothersome daytime sleepiness, apneas, RLS, PLM and parasomnias were all associated with respiratory symptoms. The largest effect was noted for bothersome daytime sleepiness (GSAQ Item 3), with reporting this symptom "at least sometimes" conferring an increase in the SGRQ Symptom Score of 7.61 points (5.81–9.41,

TABLE 3 The odds of sleep disorder symptoms occurring "at least sometimes" versus "never" (GSAQ responses) with undiagnosed COPD in the univariate analysis (Model 1) and multivariable logistic regressions (Model 2–4)[a]

GSAQ item	Model 1 OR (95% CI), *p*-value	Model 2	Model 3	Model 4
Item 1: Insomnia	0.57 (0.37–0.90), 0.01*	0.73 (0.48–1.17), 0.18	0.77 (0.49–1.23), 0.25	0.78 (0.50–1.26), 0.29
Item 2: Daytime sleepiness	0.99 (0.72–1.37), 0.96	0.98 (0.71–1.37), 0.92	1.07 (0.77–1.49), 0.70	1.06 (0.77–1.50), 0.71
Item 3: Bothersome daytime sleepiness	0.85 (0.64–1.15), 0.29	1.02 (0.75–1.38), 0.91	1.04 (0.77–1.41), 0.80	1.09 (0.80–1.50), 0.57
Item 5: Snoring	0.93 (0.67–1.30), 0.65	0.92 (0.66–1.29), 0.61	1.03 (0.74–1.46), 0.86	1.01 (0.72–1.44), 0.94
Item 6: Apneas	0.71 (0.52–0.97), 0.03*	0.71 (0.52–0.97), 0.04*	0.78 (0.56–1.07), 0.12	0.76 (0.55–1.05), 0.10
Item 7: RLS	1.08 (0.81–1.44), 0.62	1.11 (0.83–1.49), 0.48	1.14 (0.85–1.54), 0.37	1.12 (0.83–1.51), 0.48
Item 8: PLM	1.01 (0.75–1.35), 0.95	1.12 (0.83–1.51), 0.46	1.16 (0.86–1.56), 0.33	1.13 (0.83–1.53), 0.43
Item 9: Parasomnias	0.72 (0.52–1.00), 0.05	0.81 (0.58–1.11), 0.20	0.84 (0.60–1.16), 0.29	0.81 (0.57–1.12), 0.21

Estimates are presented as OR and 95% CI.

[a]The univariate analysis is represented in Model 1, with confounders added sequentially: age and sex (Model 2), BMI (Model 3), smoking and comorbidities (Model 4).

*Indicates statistically significant values.

CI, confidence interval; GSAQ, Global Sleep Assessment Questionnaire; OR, odds ratio; PLM, periodic limb movement disorder; RLS, restless legs syndrome.

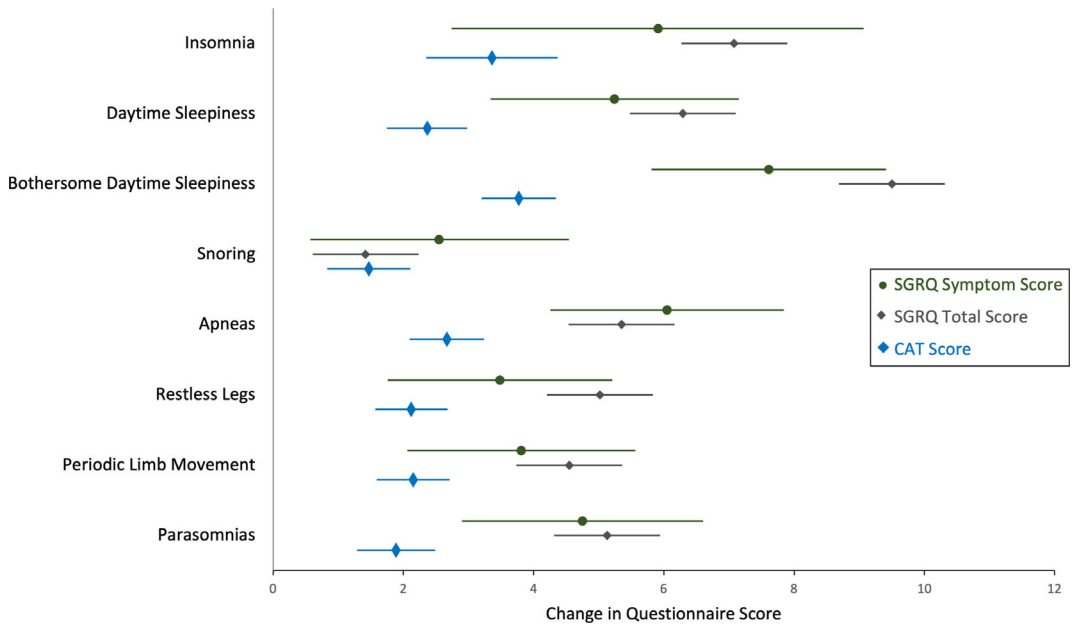

FIGURE 3 Change in questionnaire scores, with 95% confidence intervals (CI), with reporting sleep disorder symptoms "at least sometimes" versus "never" on the Global Sleep Assessment Questionnaire (GSAQ), as determined using linear regression analysis accounting for confounders (age, sex, BMI, smoking and comorbidities). BMI, body mass index; CAT, COPD Assessment Test; SGRQ, St George's Respiratory Questionnaire.

$p < 0.0001$; Table A.5). Similarly, a 9.50 point increase (8.08–10.9; $p < 0.0001$) was seen for the SGRQ Total Score (Table A.6) and a 3.77 point increase (3.20–4.34; $p < 0.0001$) for the CAT Score (Table A.7).

3.5 | OSA and OAD

The risk of OSA, represented by the STOP-Bag score, was not significantly associated with either undiagnosed asthma or COPD (Table 4).

TABLE 4 The relationship between undiagnosed OAD (asthma or COPD) and risk of OSA, as measured by the STOP-Bag score considered as a continuous variable (STOP-Bag score 0–7) and dichotomous variable (high-risk OSA [STOP-Bag ≥ 3] versus low-risk OSA [STOP-Bag < 3])[a]

OAD	STOP-Bag	Model 1 OR (95% CI), *p*-value	Model 2	Model 3	Model 4
Asthma	Continuous score, per one point increase	1.06 (0.96–1.18), 0.28	1.07 (0.95–1.21), 0.24	1.11 (0.97–1.28), 0.14	1.11 (0.96–1.29), 0.15
	High- versus low-risk for OSA	1.08 (0.74–1.63), 0.70	1.09 (0.72–1.70), 0.69	1.11 (0.72–1.76), 0.64	1.10 (0.71–1.75), 0.67
COPD	Continuous score, per one point increase	1.03 (0.94–1.14), 0.51	0.86 (0.76–0.96), 0.01	0.96 (0.84–1.11), 0.59	0.97 (0.84–1.12), 0.66
	High- versus low-risk for OSA	1.19 (0.81–1.79), 0.39	0.71 (0.46–1.10), 0.11	0.88 (0.57–1.39), 0.58	0.91 (0.58–1.45), 0.68

Estimates are presented as OR and 95% CI.

[a]The univariate analysis is represented in Model 1, with confounders added sequentially: age and sex (Model 2), BMI (Model 3), smoking and comorbidities (Model 4).

CI, confidence interval; COPD, chronic obstructive pulmonary disease; OAD, obstructive airway disease; OR, odds ratio; OSA, obstructive sleep apnea.

4 | DISCUSSION

In this community-based study, amongst adults without a prior respiratory diagnosis but with a positive respiratory symptom screen, indicating high risk for asthma and/or COPD, we found no significant association between spirometry measures (post-bronchodilator FEV_1, FVC, FEV_1/FVC) and subjective measures of sleep disorders. However, undiagnosed asthma was significantly associated with increased frequency of insomnia symptoms. No other relationship emerged between objectively-diagnosed OAD and sleep disorder symptoms. Importantly, we did nonetheless find significant associations between self-reported high respiratory symptom burden and sleep disorder symptoms, after controlling for confounders.

There is limited literature with disparate findings on the association between lung function and sleep disorder symptoms. The lack of relationship between spirometry measures and sleep disorder symptoms in our study is consistent with a number of previous studies (Lal et al., 2020; Nobeschi et al., 2020; Silva Júnior et al., 2017; Vukoja et al., 2018): spirometry data have not been associated with sleep disorder symptoms subjectively reported on several questionnaires such as the PSQI and Epworth Sleepiness Scale (ESS; Lal et al., 2020; Nobeschi et al., 2020; Vukoja et al., 2018), or with an objectively measured apnea–hypopnea index on PSG (Silva Júnior et al., 2017). More broadly, spirometry also has not correlated with respiratory symptoms in individuals without airway disease, despite increased respiratory morbidity and mortality amongst those with chronic respiratory symptoms (Çolak et al., 2019). Antithetically, amongst the authors who have reported significant associations between spirometry and sleep disorder symptoms, there is disagreement as to whether reduced lung function is positively or negatively associated with sleep disorders. Some studies show an inverse relationship between severity of sleep disorder measures or symptoms and airflow obstruction. For instance, FEV_1 has been seen as an independent positive predictor of arousal index, and fewer apnea–hypopnea events have been seen with worsening lung function, as measured by FEV_1/FVC (Sanders et al. 2003). However, other studies show a direct association between sleep disorders and airflow obstruction. Amongst male patients with COPD, for example, lower FVC was associated with greater sleep disturbances (Theorell-Haglöw et al., 2016). In analyses of subjective sleep symptoms and lung function, reduced sleep quality (higher PSQI scores) has been seen in patients with COPD with lower FEV_1 (Zohal et al., 2013), and increased OSA symptoms have been associated with both lower FVC and women with FEV_1/FVC < 0.7 (Jonassen et al., 2018). One explanation for some of these discrepant findings may be the heterogeneity of COPD, with subgroups of emphysema and chronic bronchitis as well as varying BMI having differential impacts on sleep. For example, those with emphysema-predominant COPD and severe airflow obstruction may be protected against OSA due to reduced collapsibility of the upper airway with hyperinflation (Suri & Suri, 2021). Given all of this, with most prior studies focusing mainly on COPD and COPD subgroups, but not COPD phenotypes, (McSharry et al., 2012; Nobeschi et al., 2020; Silva Júnior et al., 2017; Theorell-Haglöw et al., 2016; Vukoja et al., 2018; Zohal et al., 2013) as well as limited data from community-based studies (Jonassen et al., 2018; Lal et al., 2020), further studies are needed to delineate whether the relationship between lung function and sleep disorder symptoms is a function of COPD phenotypes.

Our data showed a significant association between asthma and insomnia. This is consistent with other studies demonstrating a high prevalence of insomnia in patients with asthma (Belachew et al., 2022; Brumpton et al., 2017; Lal et al., 2020; Luyster et al., 2016; Luyster et al., 2020; Sundbom et al., 2020), with more insomnia symptoms amongst those with worse asthma control (Alanazi et al., 2021; Belachew et al., 2022; Sundbom et al., 2020). Asthma symptom burden, particularly nocturnal symptoms (Lal et al., 2020), and immune modulation with activation of shared inflammatory pathways (Brumpton et al., 2017; Kim et al., 2021; Li et al., 2022; Prasad et al., 2020) have been posited as reasons for this association. Interestingly, two recent genetic epidemiology studies showed unidirectional causality between asthma and insomnia, with genetically predicted insomnia as a risk factor for developing asthma (Kim et al., 2021; Li et al., 2022). This was also seen clinically in the HUNT study, a large prospective study in which the odds of incident asthma were three times greater in those with insomnia symptoms (Brumpton et al., 2017).

While the literature has identified further links between sleep disorder symptoms apart from insomnia and both asthma and COPD (Braido et al., 2021; Campos et al., 2017; Jonassen et al., 2018; Li et al., 2022; McNicholas et al., 2013; McSharry et al., 2012; Nobeschi et al., 2020; Shorofsky et al., 2019; Silva Júnior et al., 2017; Soler et al., 2013; Theorell-Haglöw et al., 2016; Vukoja et al., 2018; Zohal et al., 2013), these were not found in our study. In particular, we did not identify any association between OAD and risk of OSA. A higher risk for OSA or OSA symptoms is associated with asthma (Alanazi et al., 2021; Jonassen et al., 2018; Julien et al., 2009; Prasad et al., 2020), evidenced by a recent systematic review and meta-analysis (Wang et al., 2023). Many prior analyses though have shown OSA occurs with a similar prevalence in COPD and the general population (Agusti et al., 2011; Kleisiaris et al., 2014; McNicholas et al., 2013; Sanders et al., 2003).

The discrepancy between our findings and prior studies may be due to a number of factors. The number of individuals with COPD or asthma in our study was relatively small, therefore limiting statistical power to detect any association between OAD and sleep disorder symptoms, including risk of OSA. Furthermore, our community-based study population included individuals at high risk of OAD but without prior respiratory diagnosis. Thus, it is likely the participants who met spirometry criteria for undiagnosed asthma or COPD represent a group with lower severity OAD and better respiratory symptom control or lower perception of respiratory symptoms. The limited severity of OAD in our participants is reflected in their spirometry, with few individuals with OAD having severe airflow obstruction by FEV_1. We expect those with worse symptom control and more abnormal spirometry would be more inclined to seek out healthcare, and consequently more likely to obtain respiratory diagnoses and treatment, thereby excluding them from our study. Because sleep disorder symptoms have been correlated with worse OAD control (Agusti et al., 2011; Alanazi et al., 2021; Belachew et al., 2022; Braido et al., 2021; Campos et al., 2017; Julien et al., 2009; Luyster et al., 2016; McNicholas et al., 2013; Prasad et al., 2020; Sundbom et al., 2020; Vukoja et al., 2018; Wang et al., 2023), the selection of individuals with previously undiagnosed OAD in our study may explain why we did not see relationships between spirometry measures and sleep disorders. Another possible explanation for the discrepancy in our findings and the literature is that FEV_1/FVC and FEV_1 may not fully capture COPD severity (Ni et al., 2021). It has been posited that diffusing capacity of carbon monoxide (DLCO) may be a more comprehensive measure of COPD severity (Choi et al., 2021; Ni et al., 2021). Additionally, OSA is increasingly being recognized as a heterogeneous condition, with clinical, pathophysiological, cellular and molecular characteristics (Zinchuk et al., 2017). As we were only able to identify individuals with clinical features of OSA, and in the absence of assessing DLCO, we may have missed certain associations between sleep disorder symptoms and OAD severity.

It is interesting, though not surprising, that subjective respiratory symptom burden—but neither lung function nor to a great extent OAD diagnosis—was significantly associated with sleep disorder symptoms in our study. Amongst those with OAD, there is extensive evidence that respiratory symptoms, measured by the Asthma Control Test or CAT questionnaires, correspond with reduced sleep quality by PSQI (Alanazi et al., 2021; Belachew et al., 2022; Braido et al., 2021; Campos et al., 2017; Luyster et al., 2016; Vukoja et al., 2018) and other sleep disorder symptoms (Lal et al., 2020). This is true even amongst populations where lung function parameters were not associated with sleep disorder symptoms (Lal et al., 2020; Vukoja et al., 2018). We expand on the existing literature by showing the relationship between respiratory and sleep symptoms holds true in community dwellers with respiratory symptoms even in the absence of OAD. There may be a need to screen for co-existing sleep disorder symptoms in community members with respiratory symptoms, given the known ramifications of sleep disorders on respiratory disease control and quality of life (Belachew et al., 2022; Luyster et al., 2016; Soler et al., 2013; Wang et al., 2023).

Our study has some important limitations. While there is strength in the community-based nature of our study, random population-based sampling likely also generated a selection bias amongst participants. Enrolment in UCAP required a registered land-line telephone or cell-phone number, and older adults were more likely to participate in the study (Huynh et al., 2022). This could explain the older median age of our asthma cohort, which appeared similar to our COPD cohort. Our findings may thus be limited in their generalizability amongst adults with asthma, who tend to be younger (Venkatesan, 2023). We employed spirometry to objectively diagnose COPD and asthma, but were unable to perform any analysis on participants with both undiagnosed asthma and COPD (asthma–COPD overlap) due to the small number of participants meeting spirometry criteria for OAD. Our small sample size also prevented us from categorizing asthma severity or control, though this may be an important predictor of sleep disorder symptoms. We assessed the role of mental health disorders in our analyses by including depression/anxiety in our comorbidity covariate, but excluded the insomnia-mental components of the GSAQ as this was not the focus of our study. As mental health may influence sleep disorder symptoms in OAD (Aldabayan, 2023), future larger studies are required to test the effect modification of mental health on the relationship between sleep disorders and spirometry measures or OAD diagnosis. Given the nature of our study as a secondary data analysis, some important covariates were not available. For instance, our analysis of OSA risk in relation to undiagnosed OAD is limited by lack of information on participants' prior diagnosis, phenotype and treatment of OSA. Participants with known OSA on positive airway pressure therapy may have been misclassified as low risk for OSA, resulting in unmeasured confounding bias. The cross-sectional and observational design furthermore precludes inferences regarding the causality of our findings. The GSAQ allowed us to assess subjective sleep disorder symptoms, but the questionnaire has not been widely adopted, so our comparisons to prior studies are limited. Symptoms reported on the GSAQ are also limited by recall and detection bias, and we did not measure sleep disturbances objectively via PSG.

5 | CONCLUSIONS

In a sample of community-dwelling adults with no prior respiratory diagnoses but symptoms suggestive of OAD, we identified a significant association between undiagnosed asthma and subjective insomnia symptoms, as well as between subjective respiratory and sleep disorder symptoms. Future larger-scale studies are needed to further delineate the relationship and mechanism between OAD and sleep disorder symptoms. These studies should focus on stratifying OAD by severity, including spirometry values and diffusing capacity, as well as COPD phenotype such as emphysema, chronic bronchitis and BMI. Sleep disorder symptoms identified through questionnaires should also be confirmed by objective measures.

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CONFLICT OF INTEREST STATEMENT

None of the authors has any conflicts of interest to disclose.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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