Allergologia et immunopathologia
Sociedad Española de Inmunología Clínica, Alergología y Asma Pediátrica
www.all-imm.com

ORIGINAL ARTICLE

OPEN ACCESS

Which sleep-related factors matter most in asthma? A prospective study of sleep duration, sleep quality, and nocturia

Müge Erbay[a,*], **Yılmaz Bülbül**[b]

[a]*Section of Immunology and Allergy Diseases, Department of Chest Diseases, Medical Faculty, Karadeniz Technical University, Trabzon, Turkey*
[b]*Department of Chest Diseases, Medical Faculty, Karadeniz Technical University, Trabzon, Turkey*

Received 12 January 2026; Accepted 30 April 2026
Available online 1 July 2026

KEYWORDS
asthma control;
chronic sinusitis;
nocturia;
sleep duration;
sleep quality

Abstract
Background: Sleep disturbances are highly prevalent in asthma, but the relative importance of sleep duration, sleep quality, and nocturnal symptoms for asthma outcomes remains unclear.
Objective: We aimed to investigate the association between sleep duration and sleep quality with asthma control and subsequent clinical outcomes in adults with asthma.
Material and Methods: We conducted a prospective cohort study of 53 adults with physician-diagnosed asthma who completed a 7-day sleep diary and the Pittsburgh Sleep Quality Index (PSQI) and were followed for 6 months. Short sleep duration was defined as <7 hours per night and poor sleep quality as PSQI ≥5. Asthma control was assessed using the Asthma Control Test (ACT), and asthma worsening during follow-up was defined as deterioration in ACT, increased exacerbations or hospitalizations, escalation of inhaled corticosteroids, or initiation of biologic therapy.
Results: Of the participants, 42 (79.2%) were female and the median age was 42.0 years (range 18-71). Short sleep duration was common (41.5%) but was not significantly associated with asthma control or future worsening. In contrast, patients with uncontrolled asthma (ACT <20) had significantly higher PSQI scores, more nocturia, and greater sleep fragmentation. In multivariable analysis, nocturia was the only independent sleep-related predictor of uncontrolled asthma (OR 6.70, 95% CI 1.09-41.27). During follow-up, 47.2% of patients experienced asthma worsening, and chronic sinusitis was the only independent predictor of clinical deterioration (OR 6.54, 95% CI 1.23-34.75), whereas sleep parameters were not associated with future worsening.
Conclusion: These findings suggest that in adults with asthma, qualitative aspects of sleep, particularly nocturia, are more closely associated with asthma control than sleep duration, while chronic sinusitis appears to be the dominant factor associated with subsequent asthma worsening.
© 2026 Codon Publications. Published by Codon Publications.

*Corresponding author: Müge Erbay, Section of Immunology and Allergy Diseases, Department of Chest Diseases, Medical Faculty, Karadeniz Technical University, Trabzon, Turkey. *Email address*: mugeerbay@ktu.edu.tr

https://doi.org/10.15586/aei.v54i4.1694
Copyright: Erbay M and Bülbül Y
License: This open access article is licensed under Creative Commons Attribution 4.0 International (CC BY 4.0). http://creativecommons.org/

Introduction

Asthma is a chronic inflammatory airway disease affecting more than 300 million people globally and is increasingly recognized as a heterogeneous disorder with multiple phenotypes and endotypes arising from distinct pathophysiological mechanisms. In patients with asthma, comorbid conditions, such as obesity, anxiety and depression, chronic rhinosinusitis, gastroesophageal reflux disease, and obstructive sleep apnea, substantially contribute to symptom burden, impaired quality of life, and poor asthma control.[1] Compared to the general population, asthma patients experience a disproportionately high burden of sleep disturbances. Between 22% and 47% of patients report difficulty initiating or maintaining sleep, and more than 75% report poor sleep quality.[2] Moreover, the prevalence of short sleep duration (≤6 h per night) in case of asthma patients ranges from 45% to 50.8%, substantially exceeding that observed in the general population.[3] Current consensus guidelines recommend that adults should have at least 7 h of sleep per night to maintain optimal health, and both short and long sleep duration in the general population is associated with adverse health outcomes, including cardiometabolic diseases and increased mortality.[4-6] Data from the Severe Asthma Research Program demonstrated that insomnia and poor sleep quality are highly prevalent in asthma patients and are strongly associated with increased symptom burden, impaired quality of life, and greater healthcare utilization.[2] Several epidemiologic and clinical studies have linked short sleep duration to increased asthma symptoms, impaired lung function, and higher proportions of asthma-related exacerbations and emergency department visits.[7,8] However, findings across studies remain inconsistent, partly because of differences in definition of sleep duration (e.g., <5, <6, or <7 h) and variability in patient populations, disease severity, and outcome measures.

Asthma management aims to achieve and maintain optimal symptom control and to minimize future risk, including exacerbations and treatment burden. Both the Global Initiative for Asthma (GINA) symptom score and the Asthma Control Test (ACT) explicitly include nocturnal symptoms and nighttime awakenings as key components of asthma control.[1] Therefore, a fundamental goal of asthma treatment is to enable patients to sleep through night without clinical manifestations. However, it remains uncertain whether reduced sleep duration *per se* is a major determinant of poor asthma control, or whether qualitative aspects of sleep, such as nocturnal awakenings, sleep fragmentation, and nocturia, are more relevant clinically.

Therefore, in this study, we aimed to investigate the association between sleep duration and sleep quality with asthma control and subsequent clinical outcomes in asthmatic adults. Specifically, we examined whether short sleep duration and poor sleep quality are associated with worse asthma control, and clinical worsening during prospective follow-up. By simultaneously evaluating sleep quantity, sleep quality, and nocturnal disturbances, we sought to clarify which sleep-related factors are most closely linked to asthma control in real-world clinical practice.

Materials and Methods

Study design and participants

This was a prospective observational cohort study conducted at Faculty of Medicine, Karadeniz Technical University, Trabzon, Turkey, between December 2023 and January 2025. Adult patients (aged ≥18 years) with physician-diagnosed asthma who attended the Pulmonology and Allergy–Immunology outpatient clinics were consecutively recruited. Patients with a diagnosis of obstructive sleep apnea, and those receiving antipsychotic or antidepressant medications were excluded from the study. Standardized screening tools for obstructive sleep apnea (e.g., STOP-Bang or Berlin questionnaire) were not applied. All eligible patients were consecutively invited to participate and were provided with a 7-day sleep diary at baseline. Of the 85 patients who initially consented and received the diary, 32 did not return a complete diary after 1 week and were, therefore, excluded from the final analysis. In all, 53 patients with complete sleep diary data were included in the study. A comparison between included and excluded patients was performed to assess potential selection bias (Table S1). This study was approved by the Faculty of Medicine Scientific Research Ethics Committee, Karadeniz Technical University, Trabzon, Turkey (No.: 2023-191, dated:October 09, 2023). Written informed consent was obtained from all participants.

Demographic characteristics (age, gender, marital status, height, weight, smoking, and alcohol use) and comorbidities (diabetes mellitus, hypertension, thyroid disease, chronic rhinosinusitis, and allergic rhinitis) were recorded. Asthma-related variables included disease duration, current asthma medications, and asthma-related emergency department visits and hospitalization in the previous year. Laboratory data (total serum immunoglobulin E [IgE] and peripheral blood eosinophil count) and spirometric indices (forced expiratory volume in 1 s [FEV1]/forced vital capacity [FVC], and measure of air in smaller airways [FEF25–75]) were also collected.

Sleep assessment

Patients who had been free from asthma exacerbations for at least 1 month and had not experienced any acute illness affecting sleep (e.g., upper or lower respiratory tracts infection, urinary tract infection, gastroenteritis, or menstruation) within the previous week were provided with a sleep diary. Participants completed the diary for 7 consecutive days.

The diary included daily records of bedtime, duration of smartphone use before sleep, estimated sleep onset latency, number of nocturnal awakenings, reasons for awakenings (e.g., nocturia, asthma symptoms, and environmental factors), timing and duration of awakenings, wake-up time, whether awakening occurred at a planned time, and subjective difficulty in waking up. Mean night sleep duration was calculated across the 7-day period. For each participant, the mean “rested upon awakening” score was calculated as the average of seven daily ratings.

Short sleep duration (SSD) was defined as an average sleep duration of <7 h per night, while ≥7 h of sleep was classified as adequate sleep duration, in accordance with commonly used epidemiologic cut-offs.[9]

Sleep quality was assessed after completion of sleep diary using the Pittsburgh Sleep Quality Index (PSQI), which has been validated in Turkish populations.[10] Global PSQI scores range from 0 to 21, with higher scores indicating worse sleep quality. A global score of ≥5 was used to define poor sleep quality.

Nocturia was defined as self-reported awakenings to void during the night as recorded in the sleep diary. In patients who did not complete the sleep diary, nocturia was assessed at baseline using a structured self-report question.

Asthma assessment

Asthma control was evaluated before the sleep diary period using ACT, a validated 5-item questionnaire assessing symptoms, reliever use, and activity limitation over the preceding 4 weeks. ACT scores of 5–15 indicated uncontrolled asthma, 16–19 partially controlled asthma, and 20–25 well-controlled asthma. For dichotomous analyses, ACT ≥20 was defined as controlled asthma and ACT <20 as uncontrolled asthma.

After completion of sleep diary, all patients were prospectively followed for 6 months, and their asthma status was reassessed either during routine outpatient visits or, for those who did not attend the clinic, through structured telephone interviews. During follow-up, data were collected on emergency department visits and hospitalization because of asthma, Asthma Control Test score, inhaled corticosteroid (ICS) dose, and biologic therapy status (initiated, continued, or discontinued). Patients were classified as having asthma worsening if at least one of the following occurred during the 6-month follow-up: initiation of biologic therapy, a decrease of ≥3 points in ACT score, an increase in asthma exacerbations, an increase in asthma-related hospitalizations, or escalation of ICS dose. Patients who did not meet any of these criteria were classified as having stable or lessened asthma.

Psychological assessment

Depressive symptoms were assessed using the Beck Depression Inventory (BDI), with scores <10 considered normal, 10–16 mild, 17–29 moderate, and >29 severe depression.[11] Anxiety symptoms were evaluated using the Beck Anxiety Inventory (BAI), with scores <8 considered normal, 8–15 mild, 16–25 moderate, and >26 severe anxiety.[12]

Statistical analysis

Data were analyzed using IBM SPSS v23. Continuous variables were assessed for normality using the Shapiro–Wilk test and visual inspection of histograms. Normally distributed variables are presented as mean ± standard deviation (SD), whereas non-normally distributed variables are presented as median with minimum and maximum values. Categorical variables are presented as numbers and percentages. Comparisons between two groups (short sleep duration vs. adequate sleep duration; worsening asthma vs. stable asthma; ACT <20 vs. ACT ≥20) were performed using the Student's *t*-test for normally distributed continuous variables and the Mann–Whitney *U* test for non-normally distributed variables. Categorical variables were compared using the Chi-square test or Fisher's exact test, as appropriate.

Univariate logistic regression analyses were first performed to identify variables associated with uncontrolled asthma and asthma worsening. Given the relatively small sample size and limited number of outcome events, a parsimonious multivariable modeling strategy was used to reduce the risk of overfitting. Therefore, variables with $P < 0.05$ in univariate analysis were inserted into the primary multivariable logistic regression model. In addition, to address potential confounding, a sensitivity analysis was performed, including clinically relevant variables, such as age, gender, body mass index (BMI), lung function (FEV1), and smoking status. Because nocturia and poor sleep quality (PSQI ≥ 5) represent overlapping aspects of sleep disturbance, PSQI was excluded from multivariable models because nocturia is one of its core components and showed strong correlation with the PSQI global score, leading to multicollinearity.Odds ratios (ORs) with 95% confidence interval (CIs) were calculated. Model fit was assessed using the Hosmer–Lemeshow goodness-of-fit test and classification accuracy.

An *a priori* sample size calculation was conducted based on the secondary outcome (uncontrolled asthma; ACT < 20), which reflects the most direct clinical correlate of sleep-related impairment. Assuming a two-sided alpha of 0.05, 80% power, and a large effect size for the association between sleep impairment (short sleep duration <7 h/night or poor sleep quality defined as PSQI ≥ 5) and uncontrolled asthma (corresponding to an OR of approximately ≥3.0), the minimum required sample size was estimated at approximately 50 participants. The significance level was set at $P < 0.05$.

Results

Baseline characteristics

The final study population comprised 53 adults with physician-diagnosed asthma, of whom 42 (79.2%) were females. The median age was 42.0 years (min–max: 18–71), and the mean BMI was 28.7± 6.6 kg/m^2.

Demographic and clinical characteristics are summarized in Table 1. Of the total participants, 54% were married, 45.3% had a university degree, and 34% were housewives. The most common comorbidity was allergic rhinitis in 39 (73.6%), followed by chronic sinusitis in 16 (30.2%), and hypertension in 10 (18.9%) participants. Normal anxiety score (<8) was observed in 17 patients (32.1%), and normal depression score (<10) in 31 patients (58.5%).

The median duration of asthma was 7 years (min–max: 1–50). The median ACT score was 20 (min–max: 6–25).

According to ACT categories, 27 (50.9%) patients had well-controlled asthma. The median number of asthma-related emergency department visits was 0 (min-max: 0-12), and the median number of asthma-related hospitalization was 0 (min-max: 0-6).

Baseline characteristics of included and excluded patients are presented in Table S1. These findings suggest that the analyzed cohort does not represent a substantially different asthma severity profile.

Sleep characteristics

Based on a 7-day sleep diary, mean sleep duration was 7.3 ± 1.6 h. Short sleep duration (<7 h/night) was present in 22 patients (41.5%), while 31 (58.5%) had adequate sleep duration (≥7 h). The median PSQI score was 6 (min-max: 1-18), and 35 patients (66.0%) had poor sleep quality (PSQI ≥ 5).

Nocturia was reported by 39 patients (73.6%), with a median of 1 (min-max: 0-4) episode per night. Among patients with nocturia (n = 39), 30 (76.9%) did not have hypertension, while 9 (23.1%) had hypertension, and this difference was not statistically significant (P = 0.19).

Patients with short sleep duration were older than those with adequate sleep duration (median 47.5 vs. 34 years, P = 0.05) (Table 1). Asthma-related emergency visits and hospitalizations in the previous year did not differ between sleep-duration groups (Table 1).

Patients with poor sleep quality were older (median 44 vs. 27.5 years, P = 0.03), had a higher prevalence of nocturia (88.6% vs. 44.4%, P = 0.001), and higher nocturia frequency (median 1 vs. 0 episodes/night, P = 0.007). Anxiety (median 17.5 vs. 5.5, P < 0.001) and depression (median 9 vs 5.5, P = 0.02) scores were higher among patients with poor sleep.

The use of upper airway treatments, such as oral antihistamines, intranasal corticosteroids, intranasal antihistamines, and montelukast, was not significantly associated with nocturia or sleep quality (all P > 0.05).

Factors associated with uncontrolled asthma

Figure 1 shows that patients with ACT < 20 had higher PSQI scores (P = 0.001), more nocturia (P = 0.04), and greater sleep fragmentation (P = 0.02), whereas sleep duration did not differ between ACT groups (P = 0.52). Although patients with ACT < 20 reported feeling less rested upon waking, this difference was not statistically significant (P = 0.16).

In univariate logistic regression analysis, chronic sinusitis, nocturia, poor sleep quality (PSQI ≥ 5), and anxiety were significantly associated with uncontrolled asthma. Patients in the poor sleep quality group (PSQI ≥ 5) had a significantly higher likelihood of having uncontrolled asthma, compared to those with good sleep quality (OR = 5.92; 95% CI: 1.61-21.86; P = 0.008). In multivariable logistic regression analysis, including chronic sinusitis, nocturia, and anxiety, nocturia emerged as the only independent predictor of uncontrolled asthma (OR 6.70; 95% CI 1.09-41.27; P = 0.04) (Table S2).

Although patients with short sleep had lower ACT scores compared to patients having adequate sleep (median 16.5 vs. 21), this difference was not statistically significant (P = 0.06). Similarly, uncontrolled asthma was more frequent among patients with short sleep duration, compared with those with adequate sleep (45.5% vs. 16.1%, P = 0.06) (Table 1).

Asthma control test scores were significantly lower in patients with poor sleep quality (median 18 vs. 22, P = 0.005). Uncontrolled asthma was present in 42.9% of patients with poor sleep quality, whereas no patient with good sleep quality had uncontrolled asthma (P = 0.003). Asthma-related emergency visits in the previous year were more frequent in the poor sleep quality group (P = 0.03).

Factors associated with asthma worsening

During 6-month follow-up, 25 patients (47.2%) experienced asthma worsening, whereas 28 patients (52.8%) remained stable or showed clinical lessening (Table S3).

Chronic sinusitis was more frequent in patients with asthma worsening compared to those with stable disease (48% vs. 14.3%, P = 0.02). In parallel, patients with asthma worsening exhibited a greater psychological burden, with higher anxiety scores (median 12 vs. 8, P = 0.03) and a higher prevalence of depression (54.2% vs. 23.1%, P = 0.02) than patients who remained stable (Table S3).

Asthma control tended to be poorer in patients with asthma worsening, with a lower median ACT score (18 vs. 21, P = 0.07). Sleep-related parameters, such as nocturia, sleep duration, and poor sleep quality (PSQI ≥ 5), did not differ significantly between patients with asthma worsening and those with stable asthma (Table S3).

Table 2 summarizes the univariate and multivariable logistic regression analyses of variables that were clinically relevant and/or associated with asthma worsening in the initial screening analysis. In multivariate logistic regression analysis, chronic sinusitis was the only independent factor associated with worsening of asthma (OR 6.54; 95% CI 1.23-34.75; P = 0.03) (Table 2).

In the sensitivity analysis, including core clinical confounders (age, gender, BMI, lung function, and smoking status), the association between chronic sinusitis and asthma worsening was attenuated and reached borderline statistical significance, while the direction and magnitude of the effect remained similar (Table S4).

Discussion

This prospective study provides three clinically relevant insights into the complex relationship between sleep and asthma. First, although short sleep duration was common among adults with asthma, it was not independently associated with asthma control or clinical worsening. Second, poor sleep quality and nocturnal symptoms were linked to poor asthma control. Third, nocturia emerged as independent sleep-related predictor of uncontrolled asthma, while chronic sinusitis was the only independent determinant of asthma worsening during follow-up.

Short sleep duration (<7 h/night) showed only nonsignificant trends toward poorer asthma control and should not be considered a confirmed risk factor in this cohort.

Table 1 Demographic and clinical characteristics of patients with asthma, grouped by sleep duration.

	Total (n = 53)	Short sleep duration (<7 h) (n = 22)	Adequate sleep duration (≥7 h) (n = 31)	P-value
Age, years, median (min-max)	41 (18-71)	47.5 (18-71)	34 (18-65)	0.05
Gender, Female, n (%)	42 (79.2)	18 (81.8)	24 (77.4)	0.75
BMI (kg/m^2), mean ± SD	28.7 ± 6.6	29 ± 7.9	28.5 ± 5.7	0.79
Smoking status, n (%)				0.21
Never smoker	39 (73.6)	16 (72.7)	23 (74.2)	
Ex-smoker	12 (22.6)	4 (18.2)	8 (25.8)	
Current smoker	2 (3.8)	2 (9.1)	0	
Married, n (%)	29 (54.7)	13 (59.1)	16 (51.6)	0.59
Educational level, n (%)				0.23
Postgraduate	3 (5.7)	1 (4.5)	2 (6.5)	
University	24 (45.3)	9 (40.9)	15 (48.4)	
High School	12 (22.6)	5 (22.7)	7 (22.6)	
Middle School	4 (7.5)	4 (18.2)	0	
Primary School	8 (15.1)	2 (9.1)	6 (19.4)	
Illiterate	2 (3.8)	1 (4.5)	1 (3.2)	
Occupational status, n (%)				0.17
Housewives	18 (34)	7 (31.8)	11 (35.5)	
Students	12 (22.6)	4 (18.2)	8 (25.8)	
Retirees	6 (11.3)	5 (22.7)	1 (3.2)	
Others	17 (32.1)	6 (27.3)	11 (35.5)	
Comorbidities, n (%)				
Allergic rhinitis	39 (73.6)	14 (63.6)	25 (80.6)	0.17
Chronic sinusitis	16 (30.2)	7 (31.8)	9 (29)	0.83
Hypertension	10 (18.9)	7 (31.8)	3 (9.7)	0.07
Diabetes mellitus	8 (15.1)	3 (13.6)	5 (16.1)	1.00
Medications, n (%)				
LTRAs	45 (84.9)	17 (77.3)	28 (90.3)	0.25
LAMA	11 (20.8)	6 (27.3)	5 (16.1)	0.32
Oral corticosteroid	1 (1.9)	0	1 (3.2)	1.00
Immunotherapy	3 (5.7)	1 (4.5)	2 (6.5)	1.00
Biologics	12 (22.6)	7 (31.8)	5 (16.1)	0.18
ICS dose category, n (%)				0.09
As-needed	4 (7.5)	0	4 (12.9)	
Low dose	6 (11.3)	1 (4.5)	5 (16.1)	
Medium dose	15 (28.3)	9 (40.9)	6 (19.4)	
High dose	28 (52.8)	12 (54.5)	16 (51.6)	
Spirometry, mean ± SD				
FEV1/FVC, %	82 ± 9.2	82.7 ± 7.5	80.2 ±10.1	0.32
FEV1, L	2.80 ± 0.8	2.61 ± 0.7	2.89 ±0.8	0.21
FEV1, %	99 ± 19.6	101 ±19	96.8 ± 18.4	0.43
FVC, L	3.35 ± 0.8	3.14 ± 0.8	3.52 ± 0.8	0.09
FVC, %	95.9 ± 17.6	95.2 ±18.4	96.6 ± 15.4	0.76
FEF25-75, L	3.00 ± 1.4	2.91 ± 1.4	2.90 ± 1.4	0.97
FEF25-75, %	91.5 ± 34.5	95.7 ± 32.1	84.1 ± 35	0.23
Laboratory findings, median (min-max)				
Total IgE, kU/L	234.7 (11.7-2630)	246.6 (14-2374.6)	224.9 (11.7-2630)	0.83
Eosinophil (10^9/L)	145 (0-2590)	135 (30-1230)	200 (0-2590)	0.58
Nocturia, n (%)	39 (73.6)	19 (86.4)	20 (64.5)	0.12
Nocturia episodes/night, median (min-max)	1 (0-4)	1 (0-4)	1 (0-3)	0.19
PSQI score, median (min-max)	6 (1-18)	7.5 (1-14)	5 (1-18)	0.02
Poor sleep quality (PSQI ≥ 5), n (%)	35 (66)	17 (77.3)	18 (58.1)	0.15
Asthma duration, years, median (min-max)	7.5 (1-50)	8.5 (1-50)	7 (1-30)	0.45

(*continues*)

Table 1 Continued.

	Total (n = 53)	Short sleep duration (<7 h) (n = 22)	Adequate sleep duration (≥7 h) (n = 31)	P-value
ACT score, median (min-max)	19 (6-25)	16.5 (9-25)	21 (6-25)	0.06
Asthma control category, n (%)				0.06
Uncontrolled	15 (28.3)	10 (45.5)	5 (16.1)	
Partially controlled	11 (20.8)	3 (13.6)	8 (25.8)	
Well controlled	27 (50.9)	9 (40.9)	18 (58.1)	
Emergency visits in previous year, median (min-max)	0 (0-12)	0 (0-4)	0 (0-12)	0.46
Hospitalizations in previous year, median (min-max)	0 (0-6)	0 (0-1)	0 (0-6)	0.21
Asthma worsening, n (%)	25 (47.2)	10 (45.5)	15 (48.4)	0.83
Beck anxiety total score, median (min-max)	10 (0-39)	11 (0-39)	8 (0-28)	0.19
Beck Depression total score, median (min-max)	8 (0-34)	8 (0-34)	8 (0-25)	0.34

Notes: BMI: body mass index; LTRAs: Leukotriene receptor antagonists; LAMA: long-acting antimuscarinic; ICS: inhaled corticosteroid; SD: standard deviation; ACT: asthma control test; PSQI: Pittsburgh Sleep Quality İndex.

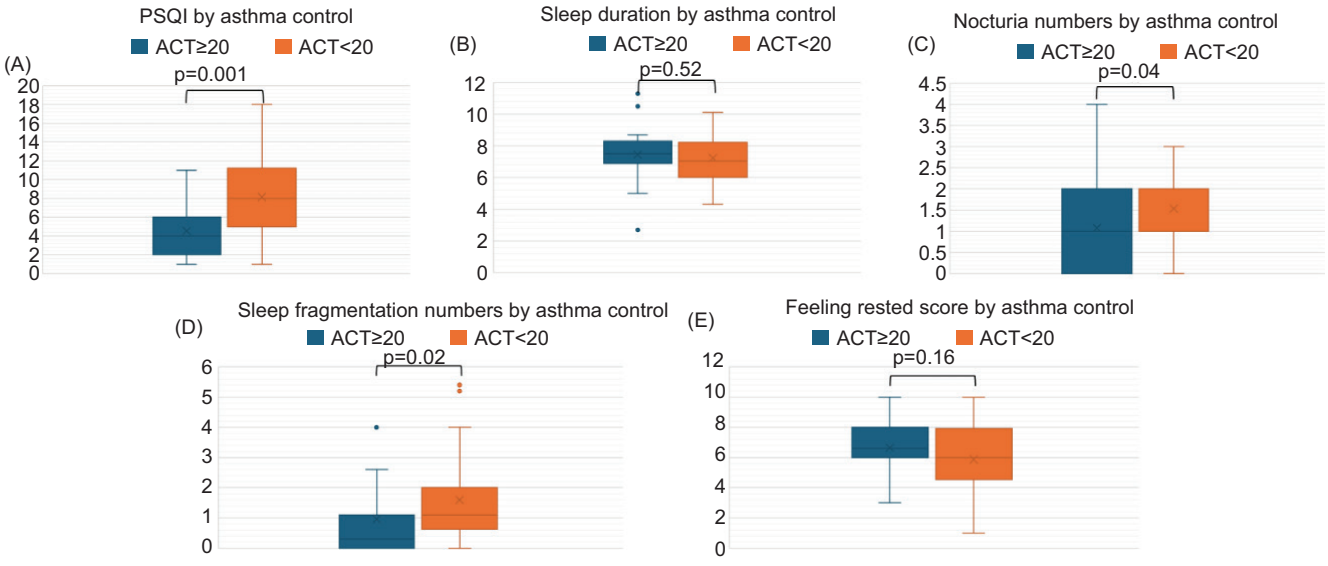


Figure 1 Sleep characteristics according to asthma control (ACT ≥ 20 vs. ACT < 20). PSQI: Pittsburgh Sleep Quality Index; ACT: asthma control test.

Table 2 Factors associated with asthma worsening in logistic regression analysis.

Variables	Univariate analysis		Multivariate analysis	
	OR (95% CI)	P-value	OR (95% CI)	P-value
Chronic sinusitis	4.93 (1.33-18.31)	0.02	6.54 (1.23-34.75)	0.03
Anxiety score	1.07 (1.002-1.131)	0.04	1.05 (0.97-1.14)	0.24
Depression	5.09 (1.45-17.92)	0.01	1.15 (0.18-7.23)	0.88

Notes: ORs > 1 indicate increased risk of asthma worsening.
Anxiety score was entered as a continuous variable.
Variables with P < 0.05 in univariate analysis were included in the multivariate logistic regression model.
ACT: asthma control test; OR: odds ratio; CI: confidence interval.

In contrast, in a prospective cohort of more than 500 adults, Wang et al. showed that short sleep duration (<6 h) was associated with a nearly threefold higher risk of poor asthma control and a twofold increase in exacerbations, together with increased airway inflammation.[13] Heterogeneity in the definition of short sleep duration across studies (<5 h, <6 h, or <7 h per night) probably captures populations with differing degrees of sleep deprivation and biological vulnerability, contributing to inconsistent findings. Moreover, our cohort consisted of clinically stable patients receiving controller therapy, which may have further attenuated the impact of reduced sleep duration on asthma outcomes.

In contrast, sleep quality was strongly associated with asthma control. Patients with ACT < 20 had significantly worse PSQI scores and nearly sixfold higher odds of poor sleep quality than those with ACT ≥ 20. In the Italian multicenter cohort reported by Braido et al., PSQI scores differed significantly across asthma control categories, and uncontrolled asthma was associated with a 3.3-fold higher risk of poor sleep quality.[14] Similarly, Vukoja et al. reported substantially lower PSQI scores in well-controlled asthma, compared to uncontrolled asthma (3.2 ± 0.6 vs. 6.7 ± 3.7, P < 0.001).[15] This relationship was also confirmed in other populations, including females with asthma and community-based cohorts from Ethiopia, where very poorly controlled asthma was associated with a fourfold higher likelihood of poor sleep quality.[16] Beyond epidemiological consistency, biological plausibility further supports this association: experimental models show that sleep deprivation aggravates airway inflammation through circadian and oxidative stress pathways, an effect that can be attenuated by melatonin.[17] Together, these data support a bidirectional and self-reinforcing relationship between asthma and sleep disturbance, in which nocturnal airway inflammation and clinical manifestations disrupt sleep, while chronic sleep impairment further amplifies airway inflammation and worsens asthma control—an interaction clearly reflected in our real-world cohort.

We did not observe significant differences in sleep duration according to asthma treatments or ICS dose. This may suggest that sleep duration, as a quantitative parameter, is less sensitive to treatment-related differences, compared to qualitative aspects of sleep. Similarly, ICS dose did not show a significant relationship with sleep duration, which may reflect that sleep duration is influenced more by nocturnal symptoms, sleep fragmentation, and individual behavioral or environmental factors, rather than controller treatment intensity alone.

Among all sleep-related variables, nocturia emerged sleep-related marker associated with uncontrolled asthma. Nocturnal asthma manifestations, autonomic imbalance, and repeated awakenings can increase the likelihood of voiding during the night, making nocturia a consequence of poor asthma control. Conversely, nocturia itself contributes to sleep fragmentation, which can further impair asthma control through increased airway inflammation, immune dysregulation, reduced adherence to controller therapy, and heightened perception of manifestations. This bidirectional relationship was demonstrated by Sato et al., who showed that nocturnal voiding and sleep disruption are closely linked to worse asthma control.[18] Nocturia is also a recognized manifestation of obstructive sleep apnea (OSA), related to sleep fragmentation and atrial natriuretic peptide release during apneic events.[19,20] Therefore, its presence may identify a subgroup of patients with possible occult sleep-disordered breathing contributing to asthma instability. Although patients with known OSA were excluded, undiagnosed sleep-disordered breathing cannot be completely ruled out, as standardized screening tools, such as STOP-Bang or the Berlin questionnaire, were not applied. However, these tools primarily identify individuals at higher risk, particularly those with obesity (BMI ≥ 30 kg/m^2). In our cohort, the mean BMI was 28.7 kg/m^2 and the population was not predominantly obese, suggesting that the overall burden of undiagnosed OSA may be relatively limited. Taken together, these findings suggest that nocturia in our study may reflect both asthma-related nocturnal pathophysiology—such as recurrent bronchoconstriction, intrathoracic pressure changes, and autonomic dysregulation—and, to a lesser extent, potential unrecognized sleep-disordered breathing. Clinically, asking about nocturia represents a simple, low-cost strategy to identify patients at higher risk of nocturnal asthma activity and impaired sleep quality. Moreover, patients reporting prominent nocturia may benefit from targeted evaluation for sleep-disordered breathing, and selected patients may be appropriate candidates for comprehensive sleep assessment with polysomnography, and lessening of nocturia may serve as a pragmatic treatment-response marker reflecting restoration of sleep continuity and control of underlying operators.

In a 6-month follow-up analysis, sleep-related variables were not independently associated with asthma worsening, whereas chronic sinusitis emerged as the sole predictor of future clinical deterioration. This finding is highly consistent with the data from the Severe Asthma Research Program-3, which demonstrated that chronic rhinosinusitis was the only upper airway comorbidity associated with a 1.7-fold increased proportion of of severe asthma exacerbations (95% CI 1.4–2.1), independent of asthma severity, lung function, and treatment.[21] In line with this, GINA 2025 identifies chronic sinusitis as a major "treatable trait" that increases exacerbation risk independent of symptom burden, together with low lung function and type 2 airway inflammation.[1] Our data provide prospective real-world support for this framework by demonstrating that chronic sinusitis identifies patients at risk for future deterioration.

This study has several limitations. Although the sample size met the *a priori* target, the study may still be underpowered to detect small-to-moderate associations between sleep duration/quality and asthma worsening, which is a multifactorial outcome. Therefore, nonsignificant findings—particularly for sleep duration—should be interpreted as absence of a large effect rather than definitive evidence of no effect. Sleep parameters were assessed using self-reported tools rather than objective measurements, and undiagnosed sleep-disordered breathing could not be excluded; however, the use of a 7-day sleep diary provides more detailed and reliable information than the single-question sleep duration measures commonly used in epidemiologic studies. Asthma worsening was defined using a composite outcome that integrated symptom deterioration, healthcare utilization, and treatment escalation, which increases clinical relevance but may have diluted

associations with individual predictors because of the heterogeneity of its components. Another limitation is that diuretic use and urologic conditions, which may contribute to nocturia, were not assessed systematically. Therefore, we cannot fully exclude the influence of non-respiratory factors on nocturnal voiding. However, in our cohort, the majority of patients with nocturia did not have hypertension, suggesting that nocturia was unlikely to be primarily driven by diuretic use. Although some baseline differences were observed between included and excluded patients, these differences were mainly related to sociodemographic characteristics and medication patterns, rather than core asthma severity or nocturnal symptom variables. Therefore, while selection bias cannot be fully excluded, its impact on the main study findings is likely limited. Despite these limitations, the prospective design, detailed sleep characterization, and systematic follow-up provide valuable real-world insight into the relative importance of sleep-related factors in asthma control.

Conclusion

This prospective study highlights that in adults with asthma, the clinical relevance of sleep lies not in how long patients sleep, but in how well they sleep and how often their sleep is disrupted. Our findings suggest that nocturia may serve as a clinically useful marker of poor asthma control, and that chronic sinusitis identifies patients at increased risk of subsequent worsening. Future studies should investigate whether targeted interventions aimed at lessening of nocturnal symptoms, sleep continuity, and upper airway inflammation can translate into better long-term asthma outcomes and reduced disease progression.

Mandatory AI Disclosure

During the preparation of this manuscript, the authors used ChatGPT (OpenAI) solely to improve the English language, grammar, and clarity of the text. No artificial intelligence tool was used for data collection, data analysis, interpretation of results, or generation of scientific content. The authors reviewed and edited all outputs and take full responsibility for the final content of the manuscript.

Author Contributions

Both authors contributed equally to this article.

Conflicts of Interest

The authors declared no potential conflicts of interest with respect to research, authorship, and/or publication of this article.

Funding

None.

References

1. Global Initiative for Asthma. Global strategy for asthma management and prevention, 2025. [Internet]. [Updated 2025 Nov 15]. Available from: www.ginasthma.org.
2. Luyster FS, Strollo PJ Jr, Holguin F, Castro M, Dunican EM, Fahy J, et al. Association between insomnia and asthma burden in the Severe Asthma Research Program (SARP) III. Chest. 2016 Dec;150(6):1242-50. https://doi.org/10.1016/j.chest.2016.09.020
3. Goral A, Lipsitz JD, Muhsen K, Gross R. Depressive symptoms, risk factors and sleep in asthma: Results from a national Israeli health survey. Gen Hosp Psychiatry. 2012 Jan-Feb;34(1):17-23. https://doi.org/10.1016/j.genhosppsych.2011.09.007
4. Watson NF, Badr MS, Belenky G, Bliwise DL, Buxton OM, Buysse D, et al. Recommended amount of sleep for a healthy adult: A joint consensus statement of the American Academy of Sleep Medicine and Sleep Research Society. Sleep. 2015 Jun 1;38(6):843-4. https://doi.org/10.5665/sleep.4716
5. Itani O, Jike M, Watanabe N, Kaneita Y. Short sleep duration and health outcomes: A systematic review, meta-analysis, and meta-regression. Sleep Med. 2017 Apr;32:246-56. https://doi.org/10.1016/j.sleep.2016.08.006
6. Jike M, Itani O, Watanabe N, Buysse DJ, Kaneita Y. Long sleep duration and health outcomes: A systematic review, meta-analysis and meta-regression. Sleep Med Rev. 2018 Jun;39:25-36. https://doi.org/10.1016/j.smrv.2017.06.011
7. Hu Z, Tian Y, Zeng F, Song X. The association between sleep duration, asthma-related episodes/attacks and emergency department visits. Nature Sci Sleep. 2020 Apr 29;12:253-62. https://doi.org/10.2147/NSS.S243882
8. Hu Z, Zhang H, Hu K, Song X. Associations between sleep duration, lung function, FeNO and blood eosinophils among current asthmatics (NHANES 2007-12). J Breath Res. 2021 Jan 23;15(2):026008. https://doi.org/10.1088/1752-7163/abd192
9. Abreu GA, Silva TLN, Teixeira LR, Bloch KV. Quality analysis of self-reported information on sleep duration in school children in the Study of Cardiovascular Risk Factors in Adolescents (ERICA). Cad Saude Publica. 2019 Oct 21;35(10):e00152918.
10. Agargun MY, Kara H, Anlar O. The validity and reliability of the Pittsburgh Sleep Quality Index.Turk J Psychiatry. 1996;7(2):107-15. (in Turkish)
11. Hisli N. Validity and reliability of the Beck Depression Inventory for a Turkish sample. Turk Psikol Derg. 1988;6:118-22. (in Turkish)
12. Ulusoy M, Sahin NH, Erkmen H. Turkish version of the Beck Anxiety Inventory: Psychometric properties. J Cogn Psychother. 1998;12:163-72. (in Turkish)
13. Wang CY, Wang J, Zhang L, Zhang SW, Wang L, Zhao SZ, et al. Self-reported ınsufficient sleep ıs associated with clinical and ınflammatory features of asthma: A prospective cohort study. J Allergy Clin Immunol Pract. 2023 Apr;11(4):1200-10. https://doi.org/10.1016/j.jaip.2022.12.011
14. Braido F, Baiardini I, Ferrando M, Scichilone N, Santus P, Petrone A, et al. The prevalence of sleep impairments and predictors of sleep quality among patients with asthma. J Asthma. 2021 Apr;58(4):481-7. https://doi.org/10.1080/02770903.2019.1711391
15. Vukoja M, Kopitovic I, Milicic D, Maksimovic O, Pavlovic-Popovic Z, Ilic M. Sleep quality and daytime sleepiness in patients with COPD and asthma. Clin Respir J. 2018 Feb;12(2):398-403. https://doi.org/10.1111/crj.12528
16. Gete M, Bitew MS, Wondie T, Bewket B, Amiha H, Mulugeta H, et al. Sleep quality and associated factors among people with asthma at public hospitals in east gojjam zone, North West Ethiopia, 2022. PLoS One. 2025 May 21;20(5):e0323284. https://doi.org/10.1371/journal.pone.0323284

17. Kim JY, Lee YD, Kim BJ, Kim SP, Kim DH, Jo KJ, et al. Melatonin improves inflammatory cytokine profiles in lung inflammation associated with sleep deprivation. Mol Med Rep. 2012 May;5(5):1281–4.
18. Sato S, Saito J, Fukuhara A, Uematsu M, Suzuki Y, Rikimaru M, et al. Association between sleep characteristics and asthma control in middle-aged and older adults: A prospective cohort study. J Asthma Allergy. 2021 Apr 6;14:325–34. https://doi.org/10.2147/JAA.S301444
19. Dos Santos GM, Coelho PS, Marques JG, Serranho S, Santos S, Brito A, et al. Obstructive sleep apnea: A risk for uncontrolled and more severe asthma in adults that we should keep an eye on. Eur Ann Allergy Clin Immunol. 2026 Jan;58(1):24–31. https://doi.org/10.23822/EurAnnACI.1764-1489.324
20. Di Bello F, Napolitano L, Abate M, Colla Ruvolo C, Morra S, Califano G, et al. Nocturia and obstructive sleep apnea syndrome: A systematic review. Sleep Med Rev. 2023 Jun;69:101787. https://doi.org/10.1016/j.smrv.2023.101787
21. Denlinger LC, Philips BR, Ramratnam S, Ross K, Bhakta NR, Carlos Cardet J, et al. Inflammatory and comorbid features of patients with severe asthma and frequent exacerbations. Am J Respir Crit Care Med. 2017 Feb 1;195(3):302–13.

Supplementary

Table S1 Comparison of baseline characteristics between included patients and excluded patients with incomplete sleep diary data.

	Included patients (n=53)	Excluded patients (n=32)	P value
Age, years, median (min-max)	42 (18-71)	42 (18-74)	0.74
Gender, Female, n (%)	42 (79.2)	28 (87.5)	0.39
BMI (kg/m^2), mean ± SD	28.7 ± 6.6	30.2 ± 5.7	0.30
Smoking status, n (%)			0.03
Never smoker	39 (73.6)	23 (71.9)	
Ex-smoker	12 (22.6)	3 (9.4)	
Current-smoker	2 (3.8)	6 (18.8)	
Married, n (%)	29 (54.7)	25 (78.1)	0.03
Educational level, n (%)			0.12
Postgraduate	3 (5.7)	3 (9.4)	
University	24 (45.3)	5 (15.6)	
High School	12 (22.6)	10 (31.3)	
Middle School	4 (7.5)	6 (18.8)	
Primary School	8 (15.1)	7 (21.9)	
Illiterate	2 (3.8)	1 (3.1)	
Occupational Status, n (%)			0.01
Housewives	18 (34)	16 (50)	
Students	12 (22.6)	4 (12.5)	
Retirees	6 (11.3)	3 (9.4)	
Others	17 (32.1)	9 (28.1)	
Comorbidities, n (%)			
Allergic rhinitis	39 (73.6)	19 (59.4)	0.17
Chronic sinusitis	16 (30.2)	10 (31.3)	0.92
Hypertension	10 (18.9)	10 (31.3)	0.19
Diabetes mellitus	8 (15.1)	3 (9.4)	0.52
Medications, n (%)			
LRTAs	45 (84.9)	19 (59.4)	0.01
LAMA	11 (20.8)	8 (25)	0.65
Oral Antihistamines	44 (83)	17 (53.1)	0.003
Oral corticosteroid	1 (1.9)	0	1.00
Immunotherapy	3 (5.7)	0	0.29
Biologics	12 (22.6)	8 (25)	0.80
ICS dose category, n (%)			0.24
As-needed	4 (7.5)	6 (18.8)	
Low dose	6 (11.3)	3 (9.4)	
Medium dose	15 (28.3)	12 (37.5)	
High dose	28 (52.8)	11 (34.4)	
Spirometry, mean ± SD			
FEV1/FVC, %	81.2 ± 9.1	83.8 ± 8.3	0.19
FEV1, L	2.77 ± 0.8	2.81 ± 0.7	0.82
FEV1, %	98.6 ± 18.6	105.4 ± 15.8	0.09
FVC, L	3.36 ± 0.8	3.35 ± 0.7	0.96
FVC, %	96 ± 16.5	99.5 ± 15.8	0.34
FEF25-75, L	2.90 ± 1.4	3.17 ± 1.2	0.37
FEF25-75, %	88.9 ± 34	102.2 ± 33.3	0.08
Laboratory findings, median (min-max)			
Total IgE, kU/L	228.1 (11.7-2630)	114.8 (14-1571.2)	0.05
Eosinophil (10^9/L)	160 (0-2590)	150 (20-1260)	0.96
Nocturia, n (%)	39 (73.6)	19 (59.4)	0.297
Nocturia episodes/night, median (min-max)	1 (0-4)	1 (0-8)	0.19
Asthma duration, years, median (min-max)	7 (1-50)	5.5 (1-25)	0.31
ACT score, median (min-max)	20 (6-25)	21 (7-25)	0.85
Controlled asthma, n (%)	27 (50.9)	20 (62.5)	0.299
Emergency visits in previous year, median (min-max)	0 (0-12)	0 (0-1)	0.03
Hospitalizations in previous year, median (min-max)	0 (0-6)	0 (0)	0.05

Included patients refer to those who completed the 7-day sleep diary and were included in the final analysis. Excluded patients refer to those who did not return a complete sleep diary and were therefore excluded from the analysis.
BMI, body mass index; LTRAs, Leukotriene receptor antagonists; LAMA, long-acting antimuscarinic; ICS, inhaled corticosteroid; SD, standard deviation; ACT, asthma control test.

Table S2 Multivariable logistic regression analysis for uncontrolled asthma.

	OR	95% CI	p-value
Chronic sinusitis	5.110	0.986-26.490	0.05
Nocturia	6.698	1.087-41.266	0.04
Anxiety	4.157	0.903-19.136	0.07

Notes: Variables were selected based on univariate P < 0.05. PSQI was excluded due to collinearity.
OR, odds ratio; CI, confidence interval.

Table S3 Demographic and clinical characteristics of patients with asthma, grouped by asthma follow-up.

	Asthma worsening (n=25)	Asthma stable (n=28)	P value
Age, years, median (min-max)	34 (18-66)	42 (18-71)	0.96
Gender, Female, n(%)	20 (80)	22 (78.6)	0.90
BMI (kg/m^2), mean ± SD	28.8 ± 6.5	28.3 ± 5.9	0.96
Smoking status, n (%)			0.30
Never smoker	16 (64)	23 (82.1)	
Ex-smoker	8 (32)	4 (14.3)	
Current-smoker	1 (4)	1 (3.6)	
Comorbidities			
Allergic rhinitis	17 (68)	22 (78.6)	0.38
Chronic sinusitis	12 (48)	4 (14.3)	0.02
Hypertension	4 (16)	6 (21.4)	0.73
Diabetes mellitus	3 (12)	5 (17.9)	0.71
ICS dose category, n (%)		0.14	
As-needed	1 (4)	3 (10.7)	
Low dose	4 (16)	2 (7.1)	
Medium dose	10 (40)	5 (17.9)	
High dose	10 (35.7)	18 (64.3)	
Spirometry, mean ± SD			
FEV1/FVC, %	79.6 ± 10.3	84.1 ± 7.8	0.09
FEV1, L	2.65 ± 0.8	2.92 ± 0.8	0.36
FEV1, %	94.3 ± 18.9	102.9 ± 19.6	0.08
FVC, L	3.30 ± 0.8	3.40 ± 0.8	0.86
FVC, %	94.5 ± 15.9	97 ± 19.1	0.48
FEF25-75, L	2.72 ± 1.5	3.23 ± 1.3	0.24
FEF25-75, %	83.7 ± 37.3	98.1 ± 31.2	0.12
Laboratory findings, median (min-max)			
Total IgE, kU/L	224.9 (14-2374.6)	448.3 (11.7-2630)	0.99
Eosinophil (109/L)	190 (0-2590)	140 (0-930)	0.14
Asthma duration, years, median (minmax)	10 (1-35)	7 (1-50)	0.74
ACT score, median (min-max)	18 (6-23)	21 (8-25)	0.07
Asthma control category, n (%)			0.18
Uncontrolled	10 (40)	5 (17.9)	
Partially controlled	5 (20)	6 (21.4)	
Well controlled	10 (40)	17 (60.7)	
Emergency visits in previous year, median (min-max)	0 (0-4)	0 (0-12)	0.14
Hospitalizations in previous year, median (min-max)	0 (0-6)	0 (0-1)	0.30
Nocturia, n (%)	20 (80)	19 (67.9)	0.32
Poor sleep quality (PSQI ≥5)	16 (64)	19 (67.9)	0.77
Sleep duration, median (min-max)	7.4 (4.3-10.5)	7.5 (2.7-11.3)	0.91
Anxiety score, median (min-max)	12 (4-39)	8 (0-32)	0.03
Depression (BECK depression score ≥10), n (%)	13 (54.2)	6 (23.1)	0.02

BMI, body mass index; ICS, inhaled corticosteroid; SD, standard deviation; ACT, asthma control test; PSQI, Pittsburgh sleep quality index.

Table S4 Factors associated with asthma worsening in logistic regression analysis

Variables	Univariate analysis		Multivariate analysis	
	OR (95%CI)	p-value	OR (95%CI)	p-value
Chronic sinusitis	4.93 (1.33-18.31)	0.02	6.99 (0.99-49.19)	0.051
Anxiety score	1.07 (1.002-1.131)	0.04	1.05 (0.97-1.14)	0.23
Depression	5.09 (1.45-17.92)	0.01	1.05 (0.14-8.05)	0.96
Age	0.995 (0.96-1.03)	0.79	0.99 (0.94-1.04)	0.65
Sex (female)	1.20 (0.32-4.55)	0.79	0.74 (0.12-4.69)	0.75
BMI	0.97 (0.89-1.06)	0.50	1.01 (0.88-1.16)	0.91
FEV1 (%)	0.97 (0.94-1.01)	0.09	0.98 (0.94-1.01)	0.23
Smoking status	2.00 (0.098-41.003)	0.65	0.76 (0.02-29.21)	0.88

Odds ratios >1 indicate increased risk of asthma worsening. Anxiety score was entered as a continuous variable. Smoking status was included in the model, with never smokers as the reference category; results are presented for current smokers. ACT, asthma control test; OR, odds ratio; CI, confidence interval; BMI, body mass index.