BMC Pulmonary Medicine

RESEARCH

Open Access

Associations between nocturnal bedtime and asthma among adults in the United States

Huawei Zhuang[1†], Xin Huang[1†], Hui Huang[2] and Lizhong Guo[1*]

Abstract

Background Sleep disorders have a significant impact on asthma. The aim of this study was to explore the association between nocturnal bedtime and asthma among adults in the United States.

Methods This study was a cross-sectional analysis involving 11,475 participants from the National Health and Nutrition Examination Survey (NHANES) during the period of 2015–2018. Nocturnal bedtime was categorized into three distinct groups: 2100 h or earlier, between 2100 h and 2300 h, and 2300 h or later. The association between night bedtime and asthma was detected using multivariable logistic regression analyses. Additionally, subgroup analyses were conducted to assess the impact of subgroups.

Results After adjustment for confounders, a positive association was revealed between later bedtime (after 2300 h) and the prevalence of asthma (OR = 1.20, 95%CI: 1.01–1.43). In the subgroup analysis, the following factors were associated with increased risk: 18–39 years (OR = 1.23, 95%CI: 1.02–1.48); female sex (OR = 1.30, 95%CI: 1.01–1.68); Hispanic patients (OR = 1.66, 95%CI: 1.17–2.37); heavy drinkers (OR = 1.52, 95%CI: 1.17–1.96); Body Mass Index (BMI) (< 25 kg/m^2) (OR = 1.45, 95%CI: 1.13–1.87); vigorous physical activity (OR = 1.32, 95%CI: 1.05–1.65);Significant interactions were found between nocturnal bedtime and asthma based on age, sex, eosinophils (EOS) percent and depression ($P_{Interaction} < 0.05$).

Conclusion Our results confirmed a moderately increased risk of asthma attributed to later bedtime, especially in 18–39 years, women and patients of Hispanic ethnicity. Future studies should investigate the underlying mechanisms of this association and explore the clinical implications for asthma management.

Keywords Sleep disorders, Circadian rhythm, Asthma, National health and nutrition examination survey (NHANES), A cross-sectional survey

†Huawei Zhuang and Xin Huang contributed equally to this work.

*Correspondence:
Lizhong Guo
lzg1073@sina.com
[1]The First Clinical Medical College, Nanjing University of Chinese Medicine, 138 Xianlin Road, Nanjing 210023, Jiangsu, China
[2]Department of Respiratory, Kunshan Hospital of Traditional Chinese Medicine, Kunshan, Jiangsu, China

Introduction

Asthma is a prevalent and chronic respiratory disease affecting approximately 339 million people with at least 250,000 deaths per year, which results in substantial morbidity and increased annual healthcare burden [1–3]. The prevalence of asthma is 10–18% in different countries, with a dramatic rise over the past decade [4, 5]. It is necessary to take appropriate measures to prevent and treat asthma. Previous studies found that obesity, metabolic syndrome, diabetes, cigarette smoking, and allergic rhinitis are associated with an increased risk of asthma [6–9]. Sleep disorders such as chronic insomnia symptoms [10] and insufficient sleep [11] have been found to have a significant impact on the development of asthma [12].

Bedtime at night is a behavioral habit affected by biological and sociological factors [13]. The influence of bedtime on circadian rhythms has been proposed, with any disturbance to these rhythms potentially increasing the susceptibility to health-related complications [14]. Numerous observational studies have demonstrated a significant association between bedtime and various health outcomes including obesity [15] and depression [16] in children and adolescents, and non-alcoholic fatty liver disease [17], angina pectoris [18], and type 2 diabetes [19] in adults. Only two studies [20, 21] have been conducted to identify the associations of late bedtime with asthma, but these studies are limited to children under the age of 12 years.

The relationship between night bedtime and asthma has been rarely reported among adult individuals. To address this research gap, we aimed to explore the correlation between nocturnal bedtime and asthma by utilizing a considerably extensive sample cohort of adults in the United States.

Methods

Study population

NHANES [22] is a cross-sectional survey that aims to acquire nationally representative estimates of the health and nutritional status of the non-institutionalized civilian population.

A total of 19,225 participants completed all surveys from 2015 to 2018, which included questions on demographics, body measures, daily alcohol drinking, physical activity, medical conditions, sleep disorders, smoking, high-sensitivity C-reactive protein, complete blood count, depression, health insurance and urine pregnancy test data. Finally, 11,475 participants were included for the final analysis after excluding 7,750 individuals. The exclusion criteria were as follows: (a) age < 18 years (n=7,377); (b) pregnant women (n=120); (c) missing bedtime data or bedtime between 0800 h and 1800 h (n=243); and (d) incomplete asthma survey (n=10) (Fig. 1).

All related data came from the open NHANES database, and no asthmatics were required to be recruited. NHANES study was approved by the IRB the of the National Center for Health Statistics of the U.S. Centers for Disease Control and Prevention. The Institutional Review Board at Nanjing University of Chinese Medicine exempted the study from review.

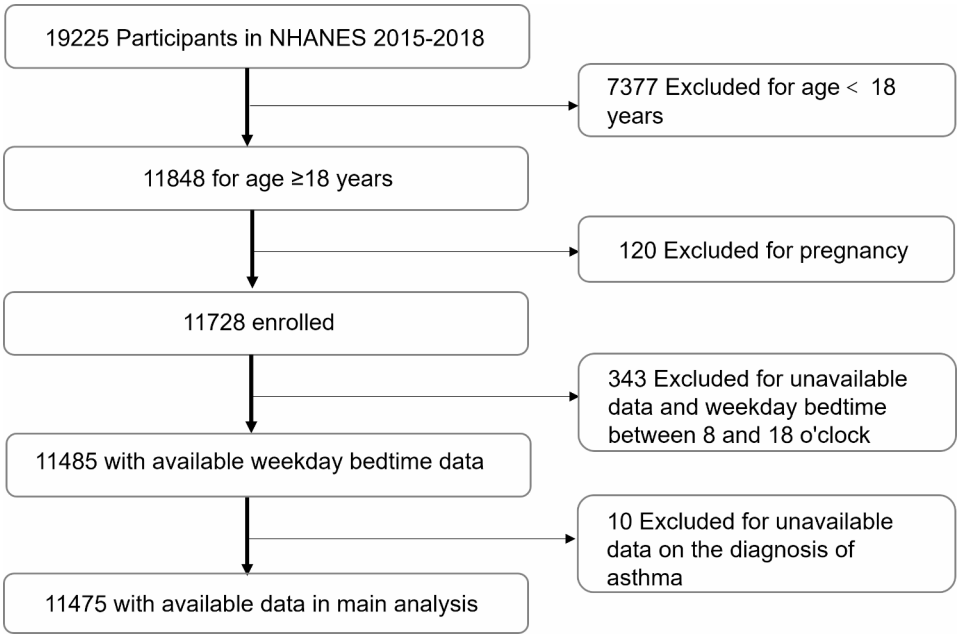

Fig. 1 Flow chart of included population

Study variables

Primary exposure

The determination of nocturnal bedtimes was based on the following question (NHANES questionnaire variables SLQ300): "Usual sleep time on weekdays or workdays" and were categorized into three distinct groups: 2100 h or earlier, between 2100 h and 2300 h, and 2300 h or later [23].

Outcomes

Bronchial asthma is a heterogeneous disease characterized by chronic inflammation of the airways, which is associated with airway hyperresponsiveness. It typically presents with widespread and variable reversible expiratory airflow restriction, leading to recurrent symptoms such as wheezing, shortness of breath, chest tightness, and/or cough, with varying intensity over time. The diagnosis of asthma typically requires a comprehensive assessment of various clinical features and test results, but there is no single "gold standard" for a definitive diagnosis. To determine asthma, Self-reported asthma was determined using the NHANES questionnaire variables MCQ010 "Ever been told you have asthma: yes/no" or MCQ035 "Still have asthma: yes/no."

Covariates

After conducting a covariate check and screening using Empowerstats software, covariates were selected based on the criterion that their inclusion or exclusion in the complete model resulted in a change of the regression coefficient of X by more than 10%. We also collected sociodemographic characteristics and other covariates as potential confounding factors; age, sex, and race were the first factors to be considered. The current study classified age into three groups, namely 18–39, 40–59, and ≥60 years. Race/ethnicity was classified as Hispanic, Non-Hispanic White, Non-Hispanic Black, Non-Hispanic Asian, and other races (American Indian or Alaska Native, Native Hawaiian or other Pacific Islander, and multiracial persons). BMI (kg/m^2) was classified as <25.0 kg/m^2, 25.0–30.0 kg/m^2, 30.0–35.0 kg/m^2, and ≥35.0 kg/m^2.

In the current study, covariates were ascertained based on known confounders from previously described methods and clinical practice. Smoking was classified as never, former (quit smoking), or current (smoking now and smoked at least 100 cigarettes per year). Based on the criteria of the National Institute on Alcohol Abuse and Alcoholism (NIAAA), alcohol consumption was categorized as moderate (1–2 drinks/day for men or 1 drink/day for women), or heavy (≥3 drinks/day for men or ≥2 drinks/day for women). Furthermore, in accordance with the Physical Activity Guidelines recommendation, physical activity was categorized into three distinct levels: active (≥75 min/week of vigorous or ≥150 min/week of moderate physical activity), less active (<75 min/week of vigorous or <150 min/week of moderate physical activity), and inactive (no physical activity). Moreover, the percentage of eosinophils (EOS) was classified into three categories: <4%, 4–15%, and 15–50%. The Patient Health Questionnaire-9 (PHQ-9) [24] was utilized as a screening tool for identifying depressive symptoms. Comprising nine items aligned with the diagnostic criteria for depressive disorders, the PHQ-9 is recognized for its reliability and validity in assessing the severity of depression. Scores falling within the range of 0 to 4 typically suggest the absence of a depressive disorder. Participants were classified into two groups: those without depression (no depressive spectrum disorder; PHQ-9 score 0–4) and those with depression (indicating a high likelihood of a depressive spectrum disorder diagnosis; PHQ-9 score≥5). Participants reported their health insurance coverage ("yes" or "no") from any source (e.g., private individual insurance, employer provided, Medicare, Medicaid, and Veteran's Administration). Include health insurance obtained through employment or purchased directly as well as government programs like Medicare and Medicaid that provide medical care or help pay medical bills.

Statistical analysis

The data were classified as either categorical or continuous variables. The variables were represented as weighted mean ± SD or weighted % (95% confidence interval). To assess differences in clinical characteristics among various groups, the weighted chi-square test was employed for categorical variables, while the weighted linear regression model was utilized for continuous variables.

Univariable and multivariable logistic regression models were employed to calculate the odds ratio (OR) and 95% confidence interval (CI) to elucidate the risk factors associated with the occurrence of asthma. In accordance with the guidelines outlined in the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement [25], an independent association was conducted by constructing three logical regression models: (1) unadjusted; (2) adjusted for age, sex, and race; and (3) adjusted for age, sex, race, alcohol, body mass index, physical activity, smoking, sleep duration, HSCRP, EOS percent, depression and health insurance. Additionally, subgroup analysis was performed to further explore the relationship between bedtime and asthma. Interaction analyses were also conducted between nocturnal bedtime and asthma risk factors (including age, sex, race, alcohol, body mass index, smoking, physical activity, EOS percent, depression and health insurance.) in the final multivariable logistic regression model.

All statistical analyses were conducted using R version 3.4.3, Empowerstats software, and STATA 17.0. The complex survey design was accounted for by applying appropriate examination weights, with a p-value of <0.05 indicating statistically significant.

Results

Study population characteristics

A cumulative count of 11,475 individuals were deemed suitable in the ultimate analysis, of whom 48.5% were male and 51.5% were female, with an average age of 47.5 years. Among these eligible participants, 1,729 (15.4%) individuals from the general population reported a history of asthma.

Table 1 provides a comprehensive summary of the sociodemographic characteristics and descriptive clinical baseline characteristics within the three groups. Significant variations were observed in many outcomes across quartiles of nocturnal bedtime among all participants, with the exception of EOS percent ($p>0.05$). Besides, individuals with later bedtime (2300 h or later) tended to be younger (18–39 years), male, Non-Hispanic Asian, had the lowest BMI (<25 kg/m^2), engaged in less physical activity, and had shorter sleep duration (7.1 ± 1.6). In contrast, participants with earlier bedtime (2100 h or earlier) were middle-aged and older (>40 years), education level less than 9th grade, overweight status (BMI between 30 and 35 kg/m^2), former smoker, and longer sleep duration (8.5 ± 1.5) (Table 1).

Association of nocturnal bedtime and asthma

Over the study period, univariable logistic regression showed that having nocturnal bedtimes>2300 h (OR: 1.26; 95%CI: 1.06–1.50; $P=0.011$) exhibited a positive association with the prevalence of asthma compared to the reference group (2100–2300 h). After adjusting for age, sex, and race, individuals with a bedtime of 2300 h or later were found to have a 24% higher risk of asthma (OR: 1.24, 95%CI: 1.04–1.49, $P=0.021$). Additionally, after adjusting for age, sex, race, poverty index, alcohol consumption, smoking, physical activity, body mass index, diabetes, sleep duration (total sleeping time), high-sensitivity C-reactive protein, and EOS percent, individuals with nocturnal bedtimes after 2300 h still had a 20% increased risk of asthma (OR: 1.20; 95%CI: 1.02–1.42) (Table 2).

Subgroup analysis

We further investigated the impact of covariates using subgroup analyses and interaction analyses (Fig. 2). Subgroup analyses stratified by age, sex, race, poverty index, alcohol consumption, smoking, physical activity, body mass index, diabetes, and EOS percent were conducted to address the potential heterogeneous population. Compared to the reference group (between 2100 h and 2300 h), stratified analysis indicated that the correlation of a later bedtime (2300 or later) and asthma remained significant in 18–39 years (OR=1.23, 95%CI: 1.02–1.48); woman (OR=1.30, 95%CI: 1.01–1.68); Hispanic (OR=1.66, 95%CI: 1.17–2.37); heavy drinkers (OR=1.52, 95%CI: 1.17–1.96); BMI<25 kg/m^2 (OR=1.45, 95%CI: 1.13–1.87); vigorous physical activity (OR=1.32, 95%CI: 1.05–1.65); Statistically significant interactions were observed between nocturnal bedtime and asthma in relation to age, sex, eosinophils (EOS) percent and depression ($P_{\text{Interaction}}<0.05$).

Discussion

Our findings showed a statistically significant relationship between bedtime at night and the prevalence of asthma. Notably, our research findings indicate a significant association between a bedtime of 2300 h or later and asthma, as determined through the construction of multivariable logistic models. Importantly, this association remained significant even after controlling for all potential confounding factors. Later sleep may affect the circadian rhythm and cause a decrease in melatonin secretion [26]. This reduction in melatonin secretion has been shown to effectively inhibit the chronic inflammatory response associated with asthma [27]. In addition, a delayed bedtime was observed, along with an atypical elevation in bronchial eosinophil levels, and the release of their associated mediators, including histamine. This phenomenon subsequently may lead to nocturnal airway hyperreactivity, thereby contributing to the development or exacerbation of asthma [28]. Moreover, pulmonary function exhibits a diurnal rhythm even in healthy adults, with a small circadian variation in FEV1; FEV1/FEVC; and PEF (forced expiratory volume in the first second [FEV1], forced vital capacity [FVC], FEV1/FVC, forced expiratory flow rate, and mid-exhalation [FEF]). Disordered circadian rhythms can correspondingly affect changes in lung function, such as significantly lower FEV1, FEV1%, PEF, and PEF% [29], which are commonly used to confirm the diagnosis of asthma [30].

Thus far, only two studies have focused on the association between nocturnal bedtime and asthma [20, 21], which is limited to subjects<12 years old. Contrary to our study, a study on 4,876 preschool children in China [20] and 2,529 children in The Netherlands [21] suggested no significant associations between late bedtime and asthma. The observed disparities among the existing studies could potentially be attributed to dissimilarities in the variables and sample sizes employed in this investigation compared to those utilized in previous studies. Furthermore, the questionnaire content could also be a contributing factor.

Table 1 General characteristics of participants ($n = 11475$) stratified by weekdays bedtime in the NHANES 2015–2018

Characters	Total	Total(%)	before 9 p.m.	9–11 p.m.	After 11 p.m.	P value
	11475		(n = 592)	(n = 7244)	(n = 3639)	
Age(years)		47.5 ± 17.7	53.4 ± 16.1	48.6 ± 17.3	44.4 ± 18.3	< 0.001
Age groups						
18-39years	3997	37.2(35.3–39.3)	19.7(14.6–26)	34.1(32.2–36)	46.6(43.3–50)	
40-59years	3501	34.5(32.7–36.4)	43.9(37.4–50.8)	36.7(34.5–38.9)	28.4(26.1–30.8)	
60-80years	3977	28.2(26-30.6)	36.4(31.5–41.6)	29.2(26.9–31.7)	24.9(22.4–27.7)	
Gender						< 0.001
Men	5584	48.5(47.3–49.7)	43.8(39.1–48.6)	46.9(45.3–48.5)	52.6(50.2–55)	
Women)	5891	51.5(50.3–52.7)	56.2(51.4–60.9)	53.1(51.5–54.7)	47.4(45–49.8)	
Race/ethnicity						< 0.001
Hispanic	3124	15.9(12.8–19.6)	17.1(11.6–24.6)	16.3(13–20.4)	14.7(11.8–18)	
Non-Hispanic White	3841	62.9(58.1–67.5)	60.6(51.3–69.2)	65(59.7–70)	58.7(54.8–62.5)	
Non-Hispanic Black	2476	11.1(8.7–14.1)	14.9(10.9–20.1)	9.7(7.4–12.6)	13.6(11–16.7)	
Non-Hispanic Asian)	1531	5.8(4.4–7.7)	2.7(1.6–4.6)	5.2(3.7–7.1)	7.8(6.1–9.9)	
Other races[a]	503	4.2(3.6–5)	4.6(2.7–7.8)	3.8(3–4.7)	5.2(4.2–6.5)	
Daily alcohol drinking status						< 0.001
Moderate-drinkers	3363	34.5(32.5–36.5)	23.2(19.3–27.8)	35.7(33.6–37.9)	33.4(30–36.9)	
Heavy-drinkers	3726	38.6(36.6–40.6)	42(36–48.3)	37.6(35.6–39.7)	40.1(36.9–43.3)	
Not recorded	4386	27(25.4–28.6)	34.7(29.9–39.9)	26.7(24.7–28.7)	26.5(23.6–29.7)	
BMI group						< 0.001
<25	3013	27.8(25.7–30)	20.5(16.2–25.6)	27.6(25.2–30.1)	29.2(27–31.6)	
25–30	3414	31(29.7–32.4)	31.4(25–38.5)	32.1(30.5–33.9)	28.5(26.2–30.9)	
30–35	2285	21.5(20–23)	22.2(17.6–27.6)	22.1(20.4–23.9)	20(18.1–22.1)	
≥35	2031	18.5(17–20.2)	23.6(17.3–31.3)	17(15.5–18.7)	21(18.2–24.1)	
Not recorded	732	1.2(1–1.5)	2.4(1.4–4)	1.1(0.8–1.6)	1.2(0.8–1.8)	
Eosinophils percent groups						0.147
Not recorded	1123	4.4(3.7–5.3)	6(3.6–9.7)	4.2(3.4–5.1)	4.7(3.7–6)	
<4%	8274	77.1(76.1–78.1)	79(74.4–83)	77.2(75.8–78.5)	76.6(74.7–78.4)	
4%-15%	2049	18.3(17.3–19.3)	14.8(11.3–19.2)	18.5(17.1–19.9)	18.4(16.9–20.1)	
15%-50%	29	0.2(0.1–0.3)	0.2(0–0.9)	0.1(0.1–0.3)	0.2(0.1–0.5)	
Physical activity level						< 0.001
Inactive	3737	25.1(23.4–26.9)	32.8(28.4–37.5)	24.2(22.3–26.2)	25.9(23.1–28.9)	
Less active	1381	12.6(11.5–13.6)	9.5(7.2–12.4)	12.1(10.9–13.3)	14(12–16.2)	
Active	6326	62.2(60.6–63.7)	57.4(52.3–62.3)	63.5(61.7–65.4)	59.9(57–62.8)	
Not recorded	31	0.2(0.1–0.3)	0.3(0.1–1)	0.2(0.1–0.3)	0.2(0.1–0.5)	
Smoking status, %						< 0.001
Never smoker	6835	58.1(56.1–60.0)	51.7(46.2–57.1)	58.6(56.6–60.6)	57.8(54.1–61.5)	
Former smoker	2594	24.5(23.1–26)	25.7(21.1–31)	25.5(23.7–27.4)	22.2(19.9–24.5)	
Current smoker	2036	17.4(15.9–19)	22.6(17.6–28.5)	15.8(14.2–17.6)	20(17.7–22.5)	
Not recorded	10	0.1(0.0–0.2)	0.1(0–0.4)	0.1(0–0.2)	0(0–0.1)	
HSCRP(mg/L)		3.8 ± 7.1	5.1 ± 11.7	3.6 ± 6.0	4.1 ± 8.3	< 0.001
Sleep duration(h)		7.7 ± 1.4	8.7 ± 1.6	7.9 ± 1.3	7.1 ± 1.6	< 0.001
Asthma						< 0.001
NO	9746	84.6(83.7–85.5)	83.3(78.6–87.1)	85.6(84.4–86.7)	82.5(80.6–84.3)	
YES	1729	15.4(14.5–16.3)	16.7(12.9–21.4)	14.4(13.3–15.6)	17.5(15.7–19.4)	
Depression						< 0.001
NO	7404	70.5(68.8–72.1)	65.2(58.3–71.4)	73.8(71.7–75.8)	64.1(62.1–66.1)	
YES	2539	22.4(21.1–23.8)	25.2(19.9–31.4)	19.4(18-20.9)	28.6(26.5–30.8)	
Not recorded	1532	7.1(5.9–8.4)	9.6(7.2–12.7)	6.8(5.6–8.4)	7.3(5.6–9.5)	
Health Insurance						< 0.001
NO	1886	13.6(11.4–16.1)	12.6(10-15.7)	12.4(10.2–15.1)	16.1(13.4–19.4)	

Table 1 (continued)

Characters	Total	Total(%)	before 9 p.m.	9–11 p.m.	After 11 p.m.	P value
	11475		(n = 592)	(n = 7244)	(n = 3639)	
YES	9555	86.2(83.7–88.4)	87.2(84–89.8)	87.3(84.7–89.6)	83.8(80.5–86.5)	
Not recorded	34	0.2(0.1–0.3)	0.2(0.1–0.7)	0.2(0.1–0.4)	0.1(0–0.3)	

NOTE: Values are weighted mean ± SD or weighted % (95% confidence interval). *P*-values are weighted. Other races[a] include American Indian or Alaska Native, Native Hawaiian or other Pacific Islander, and multiracial persons. % (Mean+/-SD) for: Age, Gender, Race, Education, Daily alcohol drinking status, BMI, EOS percent, Physical activity level, Smoking status, Depression, Health Insurance, Asthma. *P* value was calculated by weighted chi-square test. Mean+/-SD for: Age(years), HSCRP(mg/L), Sleep duration(h). *P* value was calculated by weighted linear regression model

Abbreviations: BMI: body mass index; HSCRP: High Sensitivity C-Reactive Protein (mg/L); EOS: Eosinophils; NHANES: National Health and Nutrition Examination Survey;

Table 2 Associations between weekdays bedtime and asthma (*n* = 11475), NHANES 2015–2018

Weekdays bedtime	*N* (*n* = 11475)	Model1 OR (95% CI), *P*	Model2 OR (95% CI), *P*	Model3 OR (95% CI), *P*
Before 9 p.m.	592	1.2(0.89,1.6)0.224	1.21(0.9,1.63)0.194	1.15(0.85,1.54)0.355
9–11 p.m.	7244	Reference	Reference	Reference
After 11 p.m.	3639	1.26(1.06,1.5)0.01	1.24(1.04,1.48)0.02	1.20(1.01,1.43)0.042

Model 1: non-adjusted

Model 2: adjusted for age, sex, race

Model 3: adjusted for Age, Gender, Race, Daily alcohol drinking status, BMI, EOS percent, Physical activity level, Smoking status, HSCRP, Sleep duration, Depression, Health Insurance

Subsequently, we performed a stratified analysis of the effects of bedtime on asthma in several categories, and the findings revealed that the effects of nocturnal bedtime on asthma differed across groups. In the later bedtime cohort, 18-39years, womam, BMI < 25 kg/m^2, Hispanic ethnicity, heavy alcohol drinkers, those engaged in vigorous physical activities were more prone to developing asthma than others.

First, Asthma is more common in males during childhood, but becomes more prevalent in females during adolescence and young adulthood due to the impact of female hormones on the immune response, including the modulation of inflammatory response by estrogens and the activity of various cells and proteins [31]. Another study revealed that having a persistently low BMI at an early age was associated with higher risk of asthma [32]. Moreover, this may involve a plethora of factors, including well-established anatomical, hormonal, or smoking-related distinctions within the respiratory system [33]. Second, in comparison to other racial groups, Hispanics demonstrated a higher prevalence of asthma. Epigenetic mechanisms such as distinct patterns of DNA methylation (DNAm) in whole blood, have been found to validate associations between asthma DNAm and Hispanic groups [34]. This finding is consistent with previous reports [35]. Third, heavy drinkers were more likely to develop asthma than other groups, which is consistent with the UK Biobank cohort study [36]. One potential explanation is the heightened presence of acetaldehyde in the bloodstream, which triggers the degranulation of mast cells or basophils, subsequently leading to the release of histamine and potentially exacerbating asthma symptoms [37].

Therefore, we conducted interaction analyses and found that age, sex, EOS percent and depression modified the association between nocturnal bedtime and asthma risk. Recent studies suggested that blood EOS count was a potential candidate biomarker for both asthma and nocturnal bedtime. There is increasing evidence that EOS levels are linked to disease outcomes and treatment response. In asthma, exacerbations are more frequent in patients with high counts (>400 cells·μL-1) than those with counts below this threshold [38]. Depression is a common comorbidity in asthmatic outpatients and is linked to uncontrolled asthma and lower ACT scores [39]. Major depression can cause dysregulation of the hypothalamic-pituitary-adrenal axis and autonomic nervous system, leading to chronic secretion of cortisol and catecholamines, decreased expression of glucocorticoid and β2-adrenergic receptors, and ultimately aberrant immune responses, heightened airway inflammation, and diminished efficacy of short-acting β2-agonists in individuals with asthma [40].

Limitations

Our study has some limitations, especially because we used the NHANES data. First, certain variables such as asthma, sleep factors and lifestyle factors were assessed through questionnaire surveys, which may have introduced a recall bias. Second, the data obtained for this study exclusively focused on the bedtimes during weekdays; no information regarding variables associated with weekend bedtimes was gathered. In addition, people have different thresholds for consulting a general practitioner, which in turn influences the probability of getting the diagnosis of both insomnia disorder and asthma. We

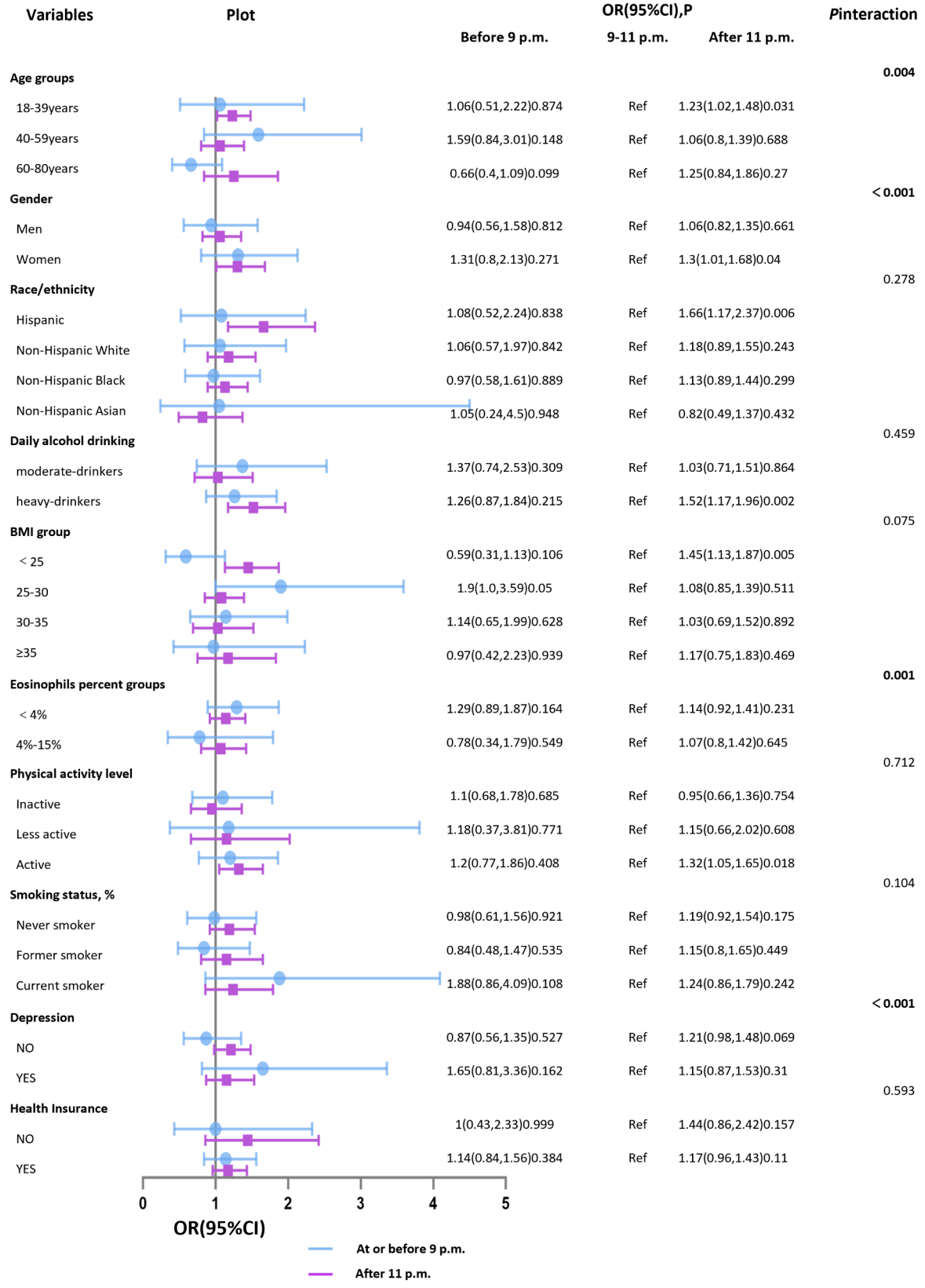

Fig. 2 Subgroup and interaction analyses for the impact of covariates on the association of nocturnal bedtime and Asthma. adjusted for Age, Gender, Race, Daily alcohol drinking status, BMI, EOS percent, Physical activity level, Smoking status, HSCRP, Sleep duration, Depression, Health Insurance. BMI: body mass index; HSCRP: High Sensitivity C-Reactive Protein (mg/L); EOS: Eosinophils; NHANES: National Health and Nutrition Examination Survey

cannot rule out the possibility that such information bias influences our results.

Conclusion

This study evaluated the impact of nocturnal bedtime on the occurrence of asthma, revealing a modestly heightened risk of asthma associated with later bedtime, particularly among women and individuals of Hispanic ethnicity. This finding contributes to the expansion and reinforcement of the consensus and endorsement surrounding asthma. Subsequent research should delve into the underlying mechanisms of this correlation and explore the potential clinical ramifications for the management of asthma.

Abbreviations

BMI	Body Mass Index
CI	Confidence interval
NHANES	National Health and Nutrition Examination Survey
NIAAA	National Institute on Alcohol Abuse and Alcoholism
OR	Odds ratio
PIR	Poverty-Income Ratio
HSCRP	High Sensitivity C-Reactive Protein (mg/L)
EOS	Eosinophil

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12890-024-03245-w.

Supplementary Material 1

Acknowledgements

We express our gratitude for the invaluable support and insightful discussions provided by our colleagues in the respiratory department.

Author contributions

H-WZ, XH, HH and L-ZG contributed to the conception and design, acquisition, analysis, interpretation of the data, drafting of the article, and critical revision for important intellectual content. H-WZ wrote the first draft of the manuscript. All authors contributed to the study conception and design and commented on the previous versions of the manuscript and read and approved the final manuscript.

Funding

This work was supported by the National Natural Science Foundation of China (82004271) and Natural Science Foundation of Jiangsu Province (BK20200260).

Data availability

The datasets analyzed during the current study are available in the Dataverse repository: https://www.cdc.gov/nchs/nhanes/index.htm. These datasets were derived from the following public domain resources: https://wwwn.cdc.gov/Nchs/Nhanes/2015-2016/DEMO_I.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2015-2016/BMX_I.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2015-2016/CBC_I.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2015-2016/HSCRP_I.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2015-2016/UCPREG_I.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2015-2016/ALQ_I.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2015-2016/INQ_I.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2015-2016/MCQ_I.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2015-2016/PAQ_I.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2015-2016/SLQ_I.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2015-2016/SMQ_I.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2015-2016/DPQ_I.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2015-2016/HIQ_I.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2017-2018/DEMO_J.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2017-2018/BMX_J.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2017-2018/CBC_J.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2017-2018/HSCRP_J.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2017-2018/UCPREG_J.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2017-2018/ALQ_J.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2017-2018/DIQ_J.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2017-2018/INQ_J.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2017-2018/MCQ_J.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2017-2018/PAQ_J.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2017-2018/SLQ_J.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2017-2018/SMQ_J.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2017-2018/DPQ_J.htm. https://wwwn.cdc.gov/Nchs/Nhanes/2017-2018/HIQ_J.htm.

Declarations

Ethics approval and consent to participate

This article does not include the use of animal or human data or tissue, therefore this section is deemed "Not applicable."

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

Received: 21 March 2024 / Accepted: 23 August 2024
Published online: 28 August 2024

References

1. Naghavi M, Abajobir AA, Abbafati C, Abbas KM, Abd-Allah F, Abera SF, et al. Global, regional, and national age-sex specific mortality for 264 causes of death, 1980–2016: a systematic analysis for the global burden of Disease Study 2016. Lancet. 2017;390:1151–210.
2. Singh S, Salvi S, Mangal DK, Singh M, Awasthi S, Mahesh PA, et al. Prevalence, time trends and treatment practices of asthma in India: the Global Asthma Network study. ERJ Open Res. 2022;8:00528–2021.
3. Dharmage SC, Perret JL, Custovic A. Epidemiology of asthma in children and adults. Front Pediatr. 2019;7:246.
4. Palomares O, Untersmayr E, Gutermuth J, Agache I, Ajeganova S, Bavbek S et al. Biologicals in allergic diseases and asthma: Toward personalized medicine and precision health: Highlights of the 3rd EAACI Master Class on Biologicals, San Lorenzo de El Escorial, Madrid, 2019. Allergy. 2020;75:936–40.
5. Schneider A, Brunn B, Hapfelmeier A, Schultz K, Kellerer C, Jörres RA. Diagnostic accuracy of FeNO in asthma and predictive value for inhaled corticosteroid responsiveness: a prospective, multicentre study. eClinicalMedicine. 2022;50:101533.
6. Roshan Lal T, Cechinel LR, Freishtat R, Rastogi D. Metabolic contributions to Pathobiology of Asthma. Metabolites. 2023;13:212.
7. He X, Cheng G, He L, Liao B, Du Y, Xie X, et al. Adults with current asthma but not former asthma have higher all-cause and cardiovascular mortality: a population-based prospective cohort study. Sci Rep. 2021;11:1329.
8. Showalter MR, Nonnecke EB, Linderholm AL, Cajka T, Sa MR, Lönnerdal B, et al. Obesogenic diets alter metabolism in mice. PLoS ONE. 2018;13:e0190632.
9. Huang K, Yang T, Xu J, Yang L, Zhao J, Zhang X, et al. Prevalence, risk factors, and management of asthma in China: a national cross-sectional study. Lancet. 2019;394:407–18.
10. Liu X, Hong C, Liu Z, Fan L, Yin M, Chen Y, et al. Association of sleep disorders with asthma: a meta-analysis. BMJ Open Respir Res. 2023;10:e001661.
11. Long-term outcomes. and predictors of chronic insomnia: a prospective study in Hong Kong Chinese adults - PubMed. https://pubmed.ncbi.nlm.nih.gov/22425578/. Accessed 28 Dec 2023.
12. Cy W, Sw JWLZ, Sz ZLW et al. Z,. Self-Reported Insufficient Sleep Is Associated With Clinical and Inflammatory Features of Asthma: A Prospective Cohort Study. The journal of allergy and clinical immunology In practice. 2023;11.
13. Rüger M, Scheer FAJL. Effects of circadian disruption on the cardiometabolic system. Rev Endocr Metab Disord. 2009;10:245–60.

14. Mr D, Jk W, Ag H, Sleep. Circadian Rhythms, and Risk Across Health Domains in Adolescents With an Evening Circadian Preference. Journal of clinical child and adolescent psychology: the official journal for the Society of Clinical Child and Adolescent Psychology, American Psychological Association, Division 53. 2019;48.
15. Anderson SE, Andridge R, Whitaker RC. Bedtime in Preschool-aged children and risk for adolescent obesity. J Pediatr. 2016;176:17–22.
16. Sakamoto N, Nanri A, Kochi T, Tsuruoka H, Pham NM, Kabe I, et al. Bedtime and Sleep Duration in relation to depressive symptoms among Japanese workers. J Occup Health. 2013;55:479–86.
17. Association between bedtime at. Night and nonalcoholic fatty liver disease diagnosed by liver ultrasound transient elastography. Diabetes Res Clin Pract. 2022;184:109195.
18. Li B, Fan Y, Zhao B, Yang L, Yang J, Ma X, et al. Later bedtime is associated with angina pectoris in middle-aged and older adults: results from the Sleep Heart Health Study. Sleep Med. 2021;79:1–5.
19. Seo JA, Lee DY, Yu JH, Cho H, Lee SK, Suh S, et al. Habitual late sleep initiation is associated with increased incidence of type 2 diabetes mellitus in Korean adults: the Korean Genome and Epidemiology Study. Sleep. 2019;42:zsz090.
20. Ma Y, Tang J, Wen Y, Hu Y, Liang J, Jiang L, et al. Associations of sleep problems with asthma and allergic rhinitis among Chinese preschoolers. Sci Rep. 2022;12:8102.
21. van Maanen A, Wijga AH, Gehring U, Postma DS, Smit HA, Oort FJ, et al. Sleep in children with asthma: results of the PIAMA study. Eur Respir J. 2013;41:832–7.
22. Tc C, Mk JC, Lk R, Thi M. F. National Health and Nutrition Examination Survey, 2015–2018: Sample Design and Estimation Procedures. Vital and health statistics Series 2, Data evaluation and methods research. 2020.
23. Li X, Ding D, Zhao Q, Wu W, Xiao Z, Luo J, et al. Sleep timing and risk of Dementia among the Chinese Elderly in an Urban Community: the Shanghai Aging Study. Front Neurol. 2021;12:629507.
24. Patel PO, Patel MR, Baptist AP. Depression and Asthma outcomes in older adults: results from the National Health and Nutrition Examination Survey. J Allergy Clin Immunol Pract. 2017;5:1691–e16971.
25. Von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP. The strengthening the reporting of Observational studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. Lancet. 2007;370:1453–7.
26. Fonken LK, Nelson RJ. The effects of Light at Night on Circadian Clocks and Metabolism. Endocr Rev. 2014;35:648–70.
27. Shin I, Park J, Shin N, Jeon C, Kwon O, Lee M, et al. Melatonin inhibits MUC 5 AC production via suppression of MAPK signaling in human airway epithelial cells. J Pineal Res. 2014;56:398–407.
28. The Role of Histamine in the Pathophysiology of Asthma and the Clinical Efficacy of Antihistamines in Asthma Therapy - PubMed. https://webvpn.njucm.edu.cn/https/webvpn014c6fc268675e690c733c4efdd571640acf6dab7c74feddc586348f9075568f/30965592/. Accessed 30 Oct 2023.
29. Spengler CM, Shea SA. Endogenous circadian rhythm of pulmonary function in healthy humans. Am J Respir Crit Care Med. 2000;162:1038–46.
30. Venkatesan P. 2023 GINA report for asthma. Lancet Respir Med. 2023;11:589.
31. C I, I ER, Gw MMC. C, G S. Sex in respiratory and skin allergies. Clin Rev Allergy Immunol. 2019;56.
32. Gb A, Ds B, Cj L, Nt W, Jl P, C S et al. Infant body mass index trajectories and asthma and lung function. J Allergy Clin Immunol. 2021;148.
33. Gellman MD, editor. Centers for Disease Control and Prevention. Encyclopedia of behavioral medicine. Cham: Springer International Publishing; 2020. pp. 409–409.
34. Herrera-Luis E, de la Rosa-Baez C, Huntsman S, Eng C, Beckman KB, Lenoir MA, et al. Novel insights into the whole blood DNA methylome of asthma in ethnically diverse children and youth. Eur Respir J. 2023:2300714.
35. Ph AZ, C RBC. B, S D, J B, Childhood asthma incidence, early and persistent wheeze, and Neighborhood socioeconomic factors in the ECHO/CREW Consortium. JAMA Pediatr. 2022;176.
36. Nyberg ST, Batty GD, Pentti J, Madsen IEH, Alfredsson L, Bjorner JB, et al. Association of alcohol use with years lived without major chronic diseases: a multicohort study from the IPD-Work consortium and UK Biobank. Lancet Reg Health - Europe. 2022;19:100417.
37. Cooper WM, Kaniecki RG, Taylor FR. Abstracts Citations Headache. 2015;55:871–81.
38. Benson VS, Hartl S, Barnes N, Galwey N, Van Dyke MK, Kwon N. Blood eosinophil counts in the general population and airways disease: a comprehensive review and meta-analysis. Eur Respir J. 2022;59:2004590.
39. Ciprandi G, Schiavetti I, Rindone E, Ricciardolo FLM. The impact of anxiety and depression on outpatients with asthma. Ann Allergy Asthma Immunol. 2015;115:408–14.
40. Rj VL, Gm JB. M. A review of candidate pathways underlying the association between asthma and major depressive disorder. Psychosom Med. 2009;71.

Publisher's note