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RESEARCH ARTICLE

Exposure to polycyclic aromatic hydrocarbons increases the risk of poor sleep pattern in US adults: results from the NHANES (2005–2010)

Qian Zhang[1] · Kun Peng[2] · Li-Hong Xin[1] · Jie Zhao[1] · Yu-Jie Li[1]

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Abstract

Recently, polycyclic aromatic hydrocarbons (PAHs) were found to be linked to various diseases. The current study's objective was to explore whether or not there was a relation between PAH exposure and poor sleep pattern. We evaluated nine urine PAH metabolites as exposures in our cross-sectional research based on the National Health and Nutrition Examination Survey (NHANES) from 2005 to 2010. Logistic regression, restricted cubic spline regression (RCS) model, weighted quantile sum (WQS) regression, subgroup analysis, and mediation analysis were used to assess the associations between PAH metabolism and poor sleep pattern risk. After controlling for all confounding variables, several primary PAH metabolites, namely 1-hydroxynapthalene (1-NAP, OR 1.32, 95% CI 1.04–1.68), 2-hydroxyfluorene (2-FLU, OR 1.34, 95% CI 1.05–1.71), 1-hydroxyphenanthrene (1-PHE, OR 1.30, 95% CI 1.03–1.64), 9-hydroxyfluorene (9-FLU, OR 1.38, 95% CI 1.09–1.74), and ∑PAHs (OR 1.33, 95% CI 1.05–1.69), compared to the bottom tertile, were associated with increased risk of poor sleep pattern. The WQS regression analysis showed that 9-FLU and 1-NAP comprised the two most important factors related to poor sleep pattern. Mediation analysis revealed that inflammation acted as a mediator between PAHs and the prevalence of poor sleep pattern. In conclusion, exposure to PAHs may be associated with poor sleep pattern. Inflammation is a mediator of the effects of PAH exposure on poor sleep pattern.

Keywords Polycyclic aromatic hydrocarbons · Poor sleep pattern · Trouble sleeping · Short sleep duration · Inflammation · NHANES

Introduction

The importance of sleep to human health is becoming more widely acknowledged. According to a World Health Organization assessment, almost one-third of the world's population suffers from sleep disorders (Lancet 2022). It is reported that insomnia constitutes a common health concern, with an estimated incidence ranging from 5 to 50% (Morin and Jarrin 2022), which is associated with a number of negative consequences such as hypertension (Jarrin et al. 2018), cardiac events (Zhong et al. 2023), mood/anxiety disorders (Palagini et al. 2022), and cancer (Blask 2009). In addition, our bodies require appropriate sleep duration to perform regular physiological, cognitive and psychological processes, and excessive or insufficient sleep length can be harmful to our health (Diekelmann and Born 2010). Previous research has indicated that insufficient sleep duration is linked to an elevated risk of obesity (Itani et al. 2017), type 2 diabetes (Lee et al. 2017), coronary heart disease (Lao et al. 2018), and hypertension (Guo et al. 2013). In the meantime, studies of epidemiology have revealed that sleeping for lengthy periods of time is closely connected with higher risk of cardiovascular disease (Krittanawong et al. 2019), obesity (Liu et al. 2019), diabetes (Shan et al. 2015), and stroke

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Qian Zhang and Kun Peng contributed equally to this work.

✉ Yu-Jie Li
18761436058@163.com

[1] Department of Respiratory and Critical Care Medicine, The Second Affiliated Hospital of Xuzhou Medical University, Xuzhou, Jiangsu, China

[2] Department of Respiratory and Critical Care Medicine, Second Affiliated Hospital of Anhui Medical University, Hefei, Anhui, China

(He et al. 2017). Sleep quality is affected by many characteristics, conditions, and stimuli such as age, sex, physical exercise, mental or physical health, and the environment (Billings et al. 2020). It has been shown that air pollution disrupts sleep and can cause a variety of issues. According to several studies, longer sleep duration is connected with worsening air pollution (An et al. 2019; An and Yu 2018). Xu et al. (2021a) found that excessive exposure to higher concentrations of PM2.5 or O_3 increased the incidence of trouble sleeping. In brief, exposure to air pollutants is a significant environmental risk factor that should be given enough attention due to its potential impact on sleep.

Polycyclic aromatic hydrocarbons (PAHs) comprise a type of air pollution that exerts a negative impact on human health (Marzooghi and Toro 2017). They are principally produced by the incomplete combustion of organic substances like coal, oil, natural gas, wood, waste, and tobacco—as well as by cooking, smoking, and grilling foods (Bostrom et al. 2002; Hoseini et al. 2018; Wang et al. 2016a, b). PAHs can enter the body via the mouth, nose, and epidermis, among other entry points (McClean et al. 2004; Veyrand et al. 2013; Zhang et al. 2014). After a few hours in the body, PAHs are broken down into different products that are primarily excreted in the urine, and the urinary monohydroxy-PAHs such as naphthalene, fluorene, phenanthrene, and pyrene are thought to be able to assess the body's level of PAH exposure (Dalton et al. 2002). Assessing 1-hydroxypyrene (1-PYR) is the most prevalent means used to assess PAH exposure, so it is often used to analyze how much PAH a person is exposed to (Shi et al. 2021). PAH pollution has been studied widely in recent years and has been linked to diseases such as chronic obstructive pulmonary disease (COPD) (Peng et al. 2023), hypertension (Wang et al. 2022a, b), diabetes (Stallings-Smith et al. 2018), rheumatoid arthritis (Sun et al. 2020), metabolic syndrome (Yang et al. 2022), and cancer (Rahman et al. 2022). Current research demonstrates that PAH is associated with trouble sleeping (Chen et al. 2023) as well as short sleep duration (Han and Wang 2023). However, their studies are limited to a single dimension of sleep, which does not provide a comprehensive assessment of a person's sleep. At the same time, earlier research has highlighted the importance of including a variety of sleep components in analyses (Hestetun et al. 2018).

In addition, considering that there are many opportunities for people to be exposed to PAHs and that there are fewer studies on the relationship between PAHs and sleep, further research on the relation between the two is imperative. As a result, the aim of this study was to inspect the exposure burden of PAHs and evaluate their effects on poor sleep pattern (a combined sleep behaviors) using the 2005–2010 National Health and Nutrition Examination Survey (NHANES) data. This study provides epidemiologic evidence of the effects of PAHs on public health, and we call for the development of prevention and control policies to reduce the effects of environmental pollutants on the sleep health of the public.

Materials and methods

Study population

In this study, we utilized data from four NHANES cycles (2005–2006, 2007–2008, and 2009–2010) that involved a total of 31,034 participants. We excluded individuals younger than 20 years or elder than 80 years ($n = 15{,}160$) and those with missing co-variables or outcome-variable data ($n = 12{,}234$), leaving 3640 adults (Fig. 1). There were 595 participants identified with poor sleep pattern and 3045 non-poor sleep pattern participants. The NHANES is a cross-sectional study of the US's population that provides a wealth of information to support the formulation of nutrition and health policies. Since the NHANES publishes open-access data, an institutional review board analysis was not necessary for our study.

Assessment of PAH metabolites

The researchers collected the urine samples from the subjects, refrigerated them, and then sent them to the testing center for pretreatment before analyzing the PAH metabolite concentrations in urine. A total of nine PAH metabolites were analyzed: 1-hydroxynapthalene (1-NAP), 2-hydroxynapthalene (2-NAP), 3-hydroxyfluorene (3-FLU), 2-hydroxyfluorene (2-FLU), 3-hydroxyphenanthrene (3-PHE), 1-hydroxyphenanthrene (1-PHE), 2-hydroxyphenanthrene (2-PHE), 1-hydroxypyrene (1-PYR), and 9-hydroxyfluorene (9-FLU). The levels of urinary PAH metabolites were adjusted in all analyses for the concentration of creatinine (ng/g creatinine). The adjusted PAH levels were equal to the PAH concentration multiplied by 100 and divided by the concentration of creatinine in the urine (Middleton et al. 2019). Given that the values of PAH metabolites in urine were skewed, a logarithmic change was performed before statistical analysis to ensure a normal distribution.

Assessment of poor sleep pattern

The inquiry "How much sleep do you usually get at night on weekdays or workdays?" was used to collect sleep duration data. The responses to the questions "Have you been asked by a doctor or other health professional if you have trouble sleeping?" and "Have you been asked by a doctor or other health professional if you have sleep disorder?" were used to determine whether someone had trouble sleeping or sleep disorder. A poor sleep pattern was defined as having at least two of the following sleep issues: (1) abnormal sleep

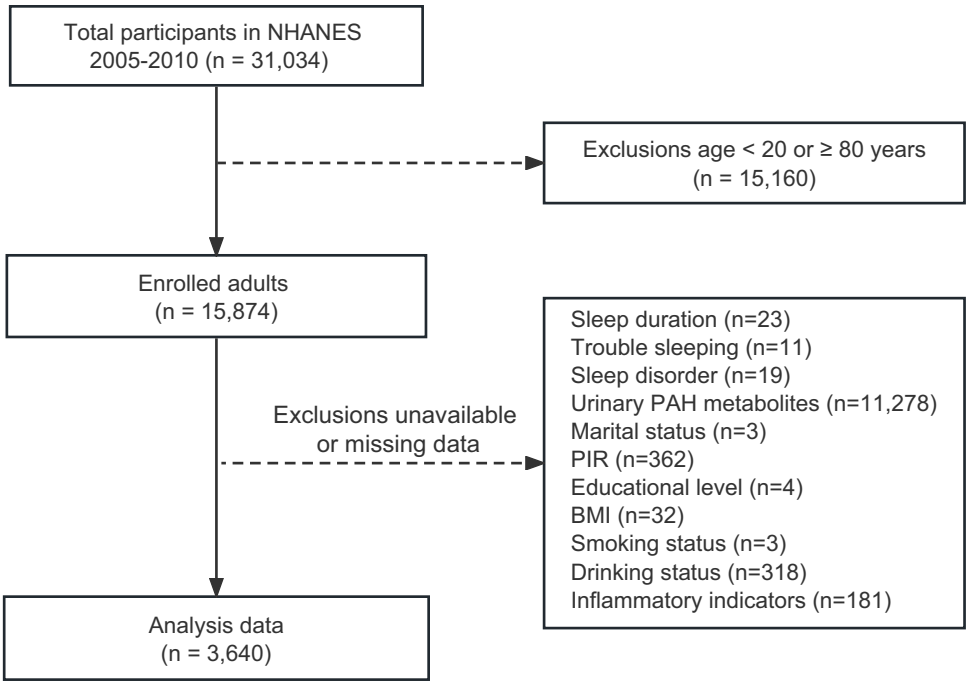

Fig. 1 Flowchart describing the selection of participants. NHANES, National Health and Nutrition Examination Survey; PAH, polycyclic aromatic hydrocarbons; PIR, poverty-to-income ratio; BMI, body mass index

duration, which was defined as < 7 h or > 9 h per night, (2) trouble sleeping, and (3) self-reported sleep disorder.

Covariates

Covariates included age, sex, race, educational level, marital status, poverty-to-income ratio (PIR), body mass index (BMI), smoking status, and alcohol intake. The categories were as follows: age (20–39, 40–59, 60–79), sex (male, female), race (Mexican American, non-Hispanic White, non-Hispanic Black, others), educational level (less than 9th grade, 9–11th grade, high school graduate or equivalent, some college or AA degree, college graduate or above), marital status (married/living, windowed/divorced/separated, never married), PIR (0–4.99, ≥ 5), BMI (< 25.0, 25.0–29.9, ≥ 30.0), smoking status (yes, no), and alcohol intake (< 12 drinks/year, ≥ 12 drinks/year).

Statistical analysis

For categorical data, counts (%) were used to describe the demographic characteristics. We used the chi-squared test to compare categorical variates between individuals with poor sleep pattern and their peers. The coefficients of association among PAH metabolites were calculated using Spearman correlation analysis. The relation between PAH and poor sleep pattern was evaluated employing multivariate logistic regression analysis. Individual PAHs were classified into tertiles, with the bottom tertile serving as the reference. We first explored the relation between each PAH metabolite and poor sleep pattern in model 1, and then adjusted the regression model by adding age, sex, and race (model 2). Finally, the link between PAH metabolites and poor sleep pattern was further explored in model 3 as adjusted for age, gender, race, educational level, marital status, PRI, BMI, smoking status, alcohol intake. In addition, we adopted restricted cubic spline (RCS) analysis to investigate the dose–response connection between PAH metabolites and poor sleep pattern. We employed weighted quantile sum (WQS) regression to elucidate the total effects and relative weights of each PAH metabolites on poor sleep pattern in order to shed further light on the relationship between PAH and sleep. Finally, mediated effect analysis was employed to determine if PAHs influence the occurrence of poor sleep pattern via inflammation. The average causal mediated effect (ACME) and average direct effect (ADE) are the two most important parameters in mediation analysis. We executed the statistical software R Studio to conduct all data analyses and to generate diagrams. In the current investigation, P values < 0.05 were considered statistically significant.

Results

Demographic information of participants, using data from NHANES, 2005–2010

As shown in Table 1, there were 595 (16.3%) subjects who exhibited poor sleep pattern, and these subjects were more likely to be older (30.9% vs. 26.8%, $P < 0.001$), non-Hispanic White (55.0% vs. 47.6%, $P < 0.001$), smokers (57.1% vs. 45.8%, $P < 0.001$), and to have had a greater BMI (49.4%

Table 1 The characteristics of the study participants

Characteristic	Total	Poor sleep pattern		*P* value
		No	Yes	
No. of participants, *n* (%)	3640 (100)	3045 (83.7)	595 (16.3)	-
Age, years, *n* (%)				< 0.001
20–39	1345 (37.0)	1199 (39.4)	246 (24.5)	
40–59	1294 (35.5)	1029 (33.8)	165 (44.5)	
60–79	1001 (27.5)	817 (26.8)	184 (30.9)	
Gender, *n* (%)				0.066
Female	1832 (50.3)	1512 (49.7)	320 (53.8)	
Male	1808 (49.7)	1533 (50.3)	275 (46.2)	
Race, *n* (%)				< 0.001
Mexican American	683 (18.8)	603 (19.8)	80 (13.4)	
Non-Hispanic black	727 (20.0)	600 (19.7)	127 (21.3)	
Non-Hispanic white	1777 (48.8)	1450 (47.6)	327 (55.0)	
Others	453 (12.4)	392 (12.9)	61 (10.3)	
Educational level, *n* (%)				0.043
Less than 9th grade	374 (10.3)	313 (10.3)	61 (10.3)	
9–11th grade	572 (15.7)	480 (15.8)	92 (15.5)	
High school graduate or equivalent	896 (24.6)	735 (24.1)	161 (27.1)	
Some college or AA degree	1023 (28.1)	842 (27.7)	181 (30.4)	
College graduate or above	775 (21.3)	675 (22.2)	100 (16.8)	
Marital status, *n* (%)				< 0.001
Married/living	2302 (63.2)	1934 (63.5)	368 (61.8)	
Never married	613 (16.8)	538 (17.7)	75 (12.6)	
Windowed/divorced/separated	725 (19.9)	573 (18.8)	152 (25.5)	
PIR, *n* (%)				0.541
0–4.99	2959 (81.3)	2470 (81.1)	489 (82.2)	
≥ 5	681 (18.7)	575 (18.9)	106 (17.8)	
BMI, *n* (%)				< 0.001
< 25.0	1023 (28.1)	890 (29.2)	133 (22.4)	
25.0–29.9	1225 (33.7)	1057 (34.7)	168 (28.2)	
≥ 30.0	1392 (38.2)	1098 (36.1)	294 (49.4)	
Smoking status, *n* (%)				< 0.001
No	1904 (52.3)	1649 (54.2)	255 (42.9)	
Yes	1736 (47.7)	1396 (45.8)	340 (57.1)	
Alcohol intake, *n* (%)				0.676
< 12 drinks/year	978 (26.9)	814 (26.7)	164 (27.6)	
≥ 12 drinks/year	2662 (73.1)	2231 (73.3)	431 (72.4)	

Abbreviation: *PIR* poverty-to-income ratio, *BMI* body mass index

vs. 36.1%, $P < 0.001$). However, we detected no differences in gender, PIR or alcohol consumption.

Distribution and correlation of PAH metabolites

The distribution with respect to PAH exposure among the study cases, adjusted for urine creatinine, is shown in Table 2. Participants were most exposed to 2-NAP, with geometric mean concentrations of 4155.7 ng/g and median concentrations of 3713.7 ng/g. The least PAH exposure was noticed for 2-PHE of 69.9 ng/g (median = 63.5 ng/g). When we analyzed the correlations among nine PAH metabolites, our results revealed that the Spearman's correlation coefficients ranged from 0.40 to 0.95, and that 2-FLU and 3-FLU exhibited the strongest association ($r = 0.95$, $P < 0.001$; Fig. 2).

Associations between PAH metabolites and poor sleep pattern

The tertile analyses for model 1 illustrated that the highest levels of 1-NAP (OR 1.58; 95% CI 1.27–1.96), 2-NAP

Table 2 Distribution of urinary metabolites of PAH in the NHANES, 2005–2010

PAH metabolites (ng/g creatinine)			Creatinine-corrected			
	Abbreviation	GM	GM	Minimum	Median	Maximum
1-hydroxynapthalene	1-NAP	2768.3	2720.5	30.0	2112.0	39,226,536.0
2-hydroxynapthalene	2-NAP	4228.7	4155.7	316.1	3713.7	272,079.8
3-hydroxyfluorene	3-FLU	119.9	117.9	7.25	801.5	8074.3
2-hydroxyfluorene	2-FLU	311.2	305.9	38.2	225.7	29,914.3
3-hydroxyphenanthrene	3-PHE	89.9	88.4	8.4	79.1	7533.6
1-hydroxyphenanthrene	1-PHE	145.1	142.6	18.1	86.3	8341.9
2-hydroxyphenanthrene	2-PHE	69.9	68.7	7.3	63.5	4552.3
1-hydroxypyrene	1-PYR	112.1	110.2	3.1	101.5	11,554.9
9-hydroxyfluorene	9-FLU	331.6	325.9	33.9	293.0	34,257.9

Abbreviation: *PAH* polycyclic aromatic hydrocarbon, *GM* geometric mean, *NHANES* National Health and Nutrition Examination Survey

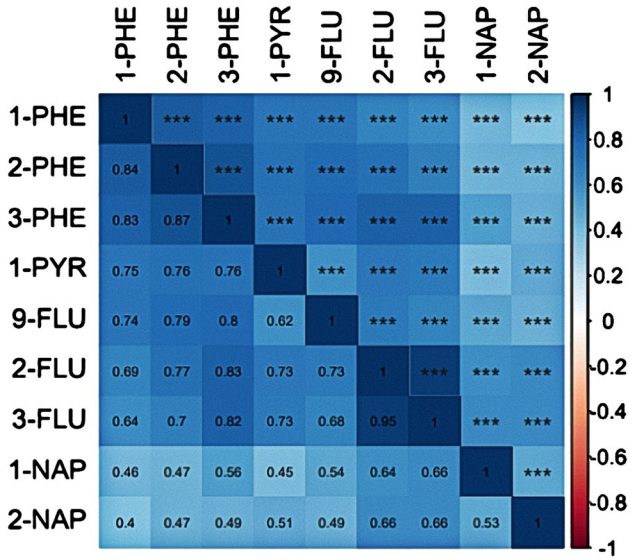

Fig. 2 Spearman correlation among 9 PAH metabolites. Abbreviation: 1-NAP, 1-hydroxynapthalene; 2-NAP, 2-hydroxynapthalene; 3-FLU, 3-hydroxyfluorene; 2-FLU, 2-hydroxyfluorene; 3-PHE, 3-hydroxyphenanthrene; 1-PHE, 1-hydroxyphenanthrene; 2-PHE, 2-hydroxyphenanthrene; 1-PYR, 1-hydroxypyrene; 9-FLU, 9-hydroxyfluorene. *$P<0.05$, **$P<0.01$, ***$P<0.001$

(OR 1.35; 95% CI 1.09–1.68), 3-FLU (OR 1.26; 95% CI 1.03–1.56), 2-FLU (OR 1.58; 95% CI 1.28–1.96), 3-PHE (OR 1.40; 95% CI 1.14–1.73), 1-PHE (OR 1.55; 95% CI 1.25–1.92), 2-PHE (OR 1.58; 95% CI 1.28–1.95), 9-FLU (OR 1.71; 95% CI 1.38–2.12), and ∑PAHs (OR 1.56; 95% CI 1.26–1.93) were positively correlated with the risk of poor sleep pattern. While in model 3 adjusted for all confounding factors, only highest concentrations of 1-NAP (OR 1.32; 95% CI 1.04–1.68), 2-FLU (OR 1.34; 95% CI 1.05–1.71), 1-PHE (OR 1.30; 95% CI 1.03–1.64), 2-PHE (OR 1.27; 95% CI 1.01–1.60), 9-FLU (OR 1.38; 95% CI 1.09–1.74), and ∑PAHs (OR 1.33; 95% CI 1.05–1.69) still showed considerable favorable associations with poor sleep pattern, compared to the lower concentrations (Table 3).

The dose–response relationships between PAH metabolites and the prevalence of poor sleep pattern

Figure 3 shows a definite connection between PAH metabolites and poor sleep pattern. After log-transformation and adjusting for age, sex, race, educational degree, marital status, PIR, BMI, smoking status, and alcohol consumption, RCS exhibited a linear relationship between 1-NAP, 3-PHE, 1-PHE, and 9-FLU and the risk of poor sleep pattern (P for non-linearity > 0.05). However, 2-NAP, 3-FLU, 2-FLU, 2-PHE, and 1-PYR were nonlinearly associated with poor sleep pattern (P for non-linearity < 0.05).

Associations between combined and individual PAH and poor sleep pattern

We then executed the WQS model to investigate the effects of mixed exposures and the proportional weight of each PAH metabolite with regard to poor sleep pattern (Fig. 4A and B). The WQS results showed that 9-FLU was the most heavily weighted component in both models (weighted 0.443 and 0.441, respectively). In the crude model, 2-PHE and 1-NAP were weighted heavily (weighted 0.187 and 0.168, respectively), while 1-NAP and 2-PHE were weighted heavily in the modified model (weighted 0.180 and 0.109, respectively).

Relations between PAH metabolites and poor sleep pattern in different subgroups

We observed heterogeneity in the correlations of specific PAH metabolites with poor sleep pattern across age, gender, and smoking status in subgroup analyses (Table 4).

Table 3 Multiple logistic regression analysis of the association between PAH metabolites and poor sleep pattern in NHANES, 2005–2010

PAH metabolites	Urinary PAH metabolites levels, OR (95% CI)			*P* for trend
	Tertile 1	Tertile 2	Tertile 3	
1-NAP				
Model 1	Ref	1.12 (0.89–1.41)	1.58 (1.27–1.96)***	< 0.001
Model 2	Ref	1.04 (0.83–1.31)	1.42 (1.14–1.77)**	0.002
Model 3	Ref	1.09 (0.86–1.38)	1.32 (1.04–1.68)*	0.025
2-NAP				
Model 1	Ref	1.07 (0.85–1.33)	1.35 (1.09–1.68)**	0.004
Model 2	Ref	1.10 (0.88–1.38)	1.39 (1.12–1.73)**	0.002
Model 3	Ref	1.02 (0.81–1.29)	1.17 (0.92–1.49)	0.200
3-FLU				
Model 1	Ref	0.83 (0.66–1.04)	1.26 (1.03–1.56)*	0.018
Model 2	Ref	0.80 (0.64–1.00)	1.22 (0.98–1.51)	0.051
Model 3	Ref	0.83 (0.66–1.04)	1.08 (0.85–1.38)	0.500
2-FLU				
Model 1	Ref	1.00 (0.80–1.26)	1.58 (1.28–1.96)***	< 0.001
Model 2	Ref	0.96 (0.76–1.21)	1.51 (1.22–1.88)***	< 0.001
Model 3	Ref	0.93 (0.73–1.17)	1.34 (1.05–1.71)*	0.031
3-PHE				
Model 1	Ref	0.88 (0.70–1,10)	1.40 (1.14–1.73)**	0.001
Model 2	Ref	0.83 (0.66–1.05)	1.26 (1.01–1.56)*	0.031
Model 3	Ref	0.86 (0.68–1.08)	1.21 (0.96–1.53)	0.120
1-PHE				
Model 1	Ref	1.01 (0.80–1.27)	1.55 (1.25–1.92)***	< 0.001
Model 2	Ref	0.95 (0.76–1.20)	1.37 (1.09–1.71)**	0.004
Model 3	Ref	0.95 (0.75–1.20)	1.30 (1.03–1.64)*	0.020
2-PHE				
Model 1	Ref	0.96 (0.76–1.21)	1.58 (1.28–1.95)***	< 0.001
Model 2	Ref	0.92 (0.73–1.16)	1.44 (1.16–1.80)***	< 0.001
Model 3	Ref	0.87 (0.69–1.10)	1.27 (1.01–1.60)*	0.028
1-PYR				
Model 1	Ref	0.82 (0.65–1.02)	1.22 (0.99–1.51)	0.039
Model 2	Ref	0.85 (0.68–1.07)	1.25 (1.01–1.56)	0.026
Model 3	Ref	0.83 (0.66–1.05)	1.14 (0.90–1.45)	0.200
9-FLU				
Model 1	Ref	1.11 (0.88–1.40)	1.71 (1.38–2.12)***	< 0.001
Model 2	Ref	1.07 (0.85–1.35)	1.55 (1.24–1.93)***	< 0.001
Model 3	Ref	1.01 (0.80–1.28)	1.38 (1.09–1.74)**	0.008
∑PAHs				
Model 1	Ref	1.04 (0.83–1.31)	1.56 (1.26–1.93)***	< 0.001
Model 2	Ref	1.04 (0.82–1.31)	1.49 (1.20–1.85)***	< 0.001
Model 3	Ref	1.01 (0.80–1.28)	1.33 (1.05–1.69)*	0.026

Model 1: crude; model 2: adjusted for age, gender, race; Model 3: Adjusted for covariates adjusted for in model 2 and educational level, marital status, PRI, BMI, smoking status, and alcohol intake

Abbreviation: *PAH* polycyclic aromatic hydrocarbon, *1-NAP* 1-hydroxynapthalene, *2-NAP* 2-hydroxynapthalene, *3-FLU* 3-hydroxyfluorene, *2-FLU* 2-hydroxyfluorene, *3-PHE* 3-hydroxyphenanthrene, *1-PHE* 1-hydroxyphenanthrene, *2-PHE* 2-hydroxyphenanthrene, *1-PYR* 1-hydroxypyrene, *9-FLU* 9-hydroxyfluorene, *OR* odds ratio, *CI* confidence interval, ∑*PAHs* total urinary PAH metabolites

*$P < 0.05$, **$P < 0.01$, ***$P < 0.001$

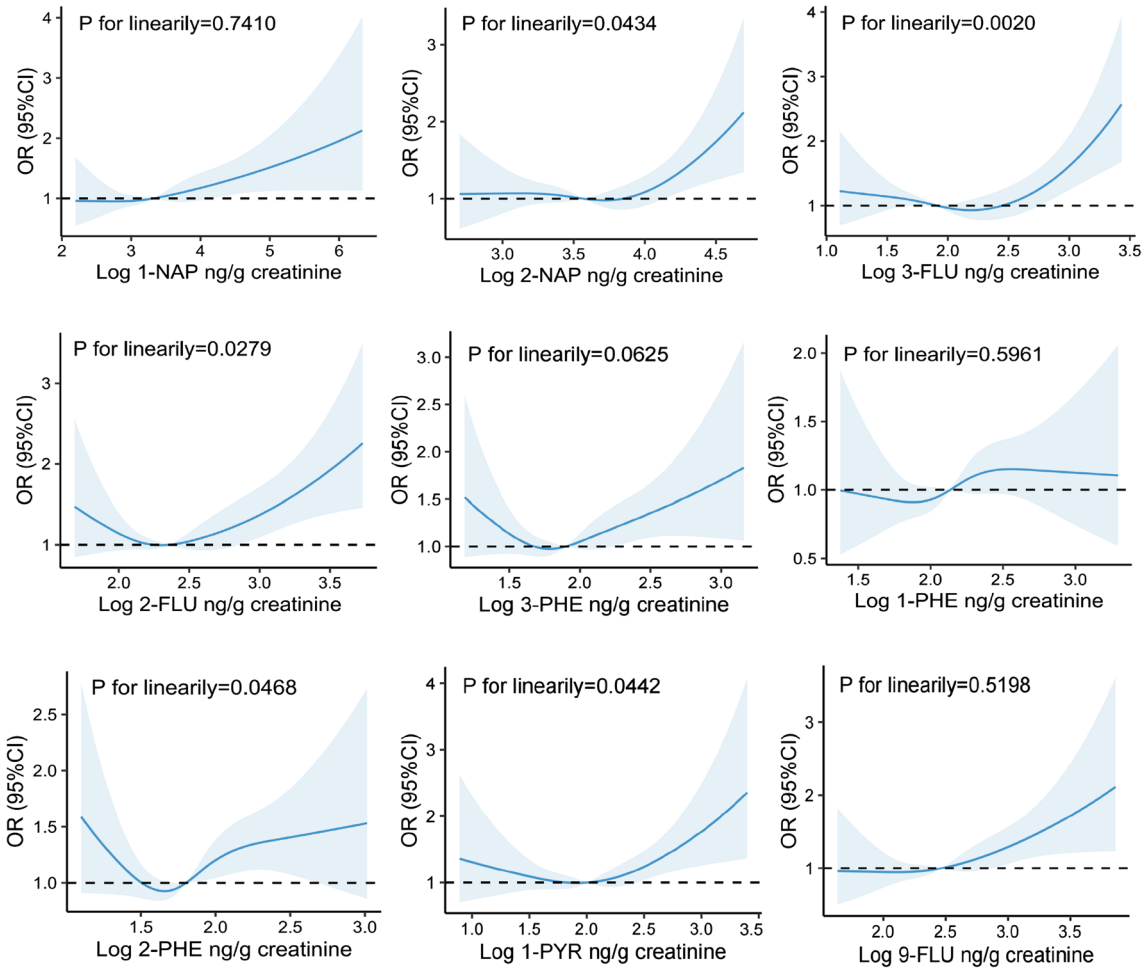

Fig. 3 Association between PAH metabolites and poor sleep pattern evaluated by restricted cubic splines (RCS). Adjusted for age, gender, race, educational level, marital status, PRI, BMI, smoking status, and alcohol intake. 1-NAP, 1-hydroxynapthalene; 2-NAP, 2-hydroxynapthalene; 3-FLU, 3-hydroxyfluorene; 2-FLU, 2-hydroxyfluorene; 3-PHE, 3-hydroxyphenanthrene; 1-PHE, 1-hydroxyphenanthrene; 2-PHE, 2-hydroxyphenanthrene; 1-PYR, 1-hydroxypyrene; 9-FLU, 9-hydroxyfluorene; OR, odds ratio; CI, confidence interval

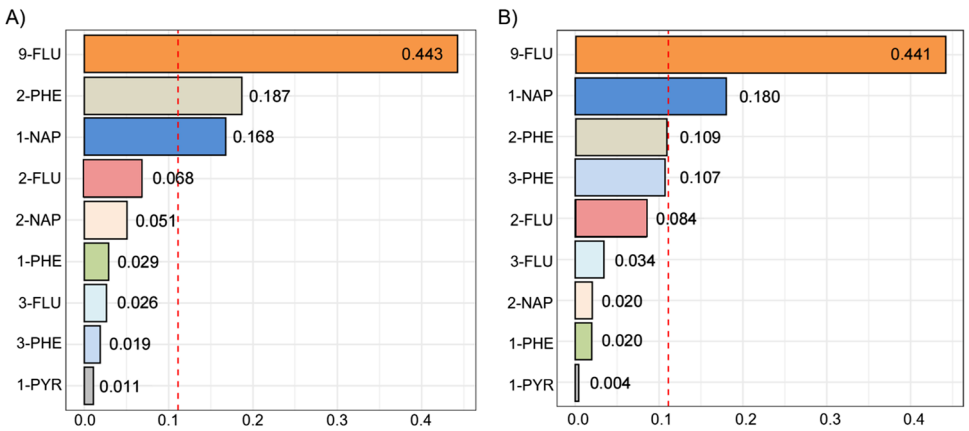

Fig. 4 Weights for weighted quantile sum (WQS) regression index for mixed PAH exposure in **A** crude model; **B** adjusted for age, gender, race, educational level, marital status, PRI, BMI, smoking status and alcohol intake in NHANES, 2005–2010. Abbreviation: 1-NAP, 1-hydroxynapthalene; 2-NAP, 2-hydroxynapthalene; 3-FLU, 3-hydroxyfluorene; 2-FLU, 2-hydroxyfluorene; 3-PHE, 3-hydroxyphenanthrene; 1-PHE, 1-hydroxyphenanthrene; 2-PHE, 2-hydroxyphenanthrene; 1-PYR, 1-hydroxypyrene; 9-FLU, 9-hydroxyfluorene

Table 4 Subgroup analyses of urinary PAH exposure and poor sleep pattern in NHANES, 2005–2010

	Age			Gender		Smoking status	
	20–39	40–59	69–79	Male	Female	No	Yes
1-NAP	1.28 (0.94–1.73)	**1.29 (1.05–1.58)**	1.04 (0.80–1.34)	1.15 (0.92–1.43)	**1.30 (1.08–1.55)**	1.05 (0.84–1.32)	**1.33 (1.11–1.60)**
p value	0.110	**0.020**	0.800	0.200	**0.004**	0.600	**0.002**
2-NAP	**1.88 (1.17–3.02)**	1.16 (0.80–1.68)	0.96 (0.63–1.45)	1.03 (0.73–1.44)	**1.50 (1.08–2.07)**	1.01 (0.67–1.51)	**1.40 (1.04–1.89)**
p value	**0.010**	0.400	0.800	0.900	**0.015**	0.900	**0.029**
2-FLU	**3.00 (1.85–4.88)**	1.20 (0.86–1.69)	0.91 (0.61–1.35)	121 (0.87–1.67)	**1.59 (1.18–2.14)**	1.26 (0.81–1.93)	**1.40 (1.07–1.82)**
p value	**< 0.001**	0.300	0.600	0.200	**0.003**	0.300	**0.013**
3-FLU	**2.29 (1.51–3.50)**	1.18 (0.88–1.59)	0.85 (0.59–1.21)	1.11 (0.84–1.48)	**1.46 (1.12–1.90)**	1.17 (0.79–1.72)	**1.27 (1.01–1.59)**
p value	**< 0.001**	0.300	0.400	0.500	**0.005**	0.400	**0.043**
9-FLU	**2.65 (1.13–4.29)**	1.42 (0.97–2.09)	0.90 (0.57–1.40)	**1.46 (1.04–2.05)**	**1.47 (1.04–2.07)**	1.33 (0.89–1.97)	**1.53 (1.12–2.10)**
p value	**< 0.001**	0.070	0.600	**0.029**	**0.028**	0.200	**0.007**
1-PYR	**2.56 (1.55–4.22)**	0.94 (0.65–1.35)	1.13 (0.73–1.75)	1.04 (0.74–1.45)	**1.63 (1.16–2.30)**	1.09 (0.73–1.60)	**1.43 (1.04–1.95)**
p value	**< 0.001**	0.700	0.600	0.800	**0.005**	0.700	**0.026**
1-PHE	**1.89 (1.04–3.41)**	1.18 (0.73–1.89)	0.93 (0.54–1.56)	1.11 (0.72–1.71)	1.44 (0.95–2.19)	1.06 (0.67–1.66)	1.40 (0.93–2.10)
p value	**0.040**	0.500	0.800	0.600	0.084	0.800	0.110
2-PHE	**2.79 (1.57–4.95)**	1.09 (0.69–1.71)	0.88 (0.52–1.46)	1.29 (0.85–1.93)	1.37 (0.92–2.04)	0.98 (0.62–1.53)	**1.61 (1.10–2.35)**
p value	**< 0.001**	0.700	0.600	0.200	0.120	0.900	**0.014**
3-PHE	**2.26 (1.30–3.94)**	1.21 (0.80–1.82)	0.79 (0.48–1.27)	1.12 (0.76–1.62)	1.46 (1.00–2.11)	1.06 (0.68–1.63)	1.34 (0.95–1.88)
p value	**0.004**	0.400	0.300	0.600	0.050	0.800	0.095
∑PAHs	1.50 (0.99–2.25)	1.31 (0.99–1.73)	1.09 (0.77–1.52)	1.16 (0.86–1.54)	**1.42 (1.11–1.82)**	1.06 (0.76–1.45)	**1.43 (1.13–1.82)**
p value	0.054	0.060	0.600	0.300	**0.005**	0.700	**0.003**

The models were adjusted for age, gender, race, educational level, marital status, PRI, BMI, smoking status, and alcohol intake. Numbers in bold represent statistical significance

Abbreviation: *1-NAP* 1-hydroxynapthalene, *2-NAP* 2-hydroxynapthalene, *3-FLU* 3-hydroxyfluorene, *2-FLU* 2-hydroxyfluorene, *3-PHE* 3-hydroxyphenanthrene, *1-PHE* 1-hydroxyphenanthrene, *2-PHE* 2-hydroxyphenanthrene, *1-PYR* 1-hydroxypyrene, *9-FLU* 9-hydroxyfluorene, *∑PAHs* total urinary PAH metabolites

Participants aged 20–39 years showed statistically significant associations of all PAH metabolites except for 1-NAP (OR 1.28; 95% CI 0.94–1.73), with poor sleep pattern. However, there is a borderline link between ∑PAHs (OR 1.50, 95% CI 0.99–2.25) and poor sleep pattern. Among the female participants, we observed a statistically significant association of 1-NAP (OR 1.30; 95% CI 1.08–1.55), 2-NAP (OR 1.50; 95% CI 1.08–2.07), 2-FLU (OR 1.59; 95% CI 1.18–2.14), 3-FLU (OR 1.46; 95% CI 1.12–1.90), 9-FLU (OR 1.47; 95% CI 1.04–2.07), 1-PYR (OR 1.63; 95% CI 1.16–2.30) and ∑PAHs (OR 1.42; 95% CI 1.11–1.82) with poor sleep pattern. In addition, subjects who were smokers manifested a greater association between PAH metabolites and poor sleep pattern, especially for 9-FLU (OR 1.53; 95% CI 1.12–2.10) and ∑PAHs (OR 1.43; 95% CI 1.13–1.82).

Associations between PAH metabolites and poor sleep pattern and its components

Supplementary Table S1 shows the associations between PAH metabolites and poor sleep pattern and its several components in adults. The logistic regression analyses showed significant associations of ∑PAHs with poor sleep pattern (OR 1.29; 95% CI 1.07–1.56), and trouble sleeping (OR 1.33; 95% CI 1.13–1.58) after adjusting for all potential confounders. In addition to being associated with poor sleep pattern, 1-NAP, 2-NAP, 2-FLU, 3-FLU, and 9-FLU had positive correlations with at least one of the components.

Analysis of inflammation as a mediator between PAH exposure and poor sleep pattern

In the present investigation, we conducted a mediation analysis to determine if inflammation acted as a mediator between PAHs and the prevalence of poor sleep pattern. PAHs have been demonstrated to contribute to the development of poor sleep pattern by altering white blood cell (WBC) and neutrophil count (NC) levels (Fig. 5). In detail, WBC-mediated efficacy accounted for 6.67% of the relationship between ∑PAHs and poor sleep pattern prevalence (ACME = 1.17e − 03, 95% CI 8.51e − 05, 0.01; ADE = 1.63e − 02, 95% CI 4.33e − 03, 0.02; Fig. 5A), while NC mediated efficacy accounted for 6.44% (ACME = 1.12e − 03, 95% CI 4.02e − 05, 0.01;ADE = 1.63e − 02, 95% CI 4.54e − 03, 0.02; Fig. 5B).

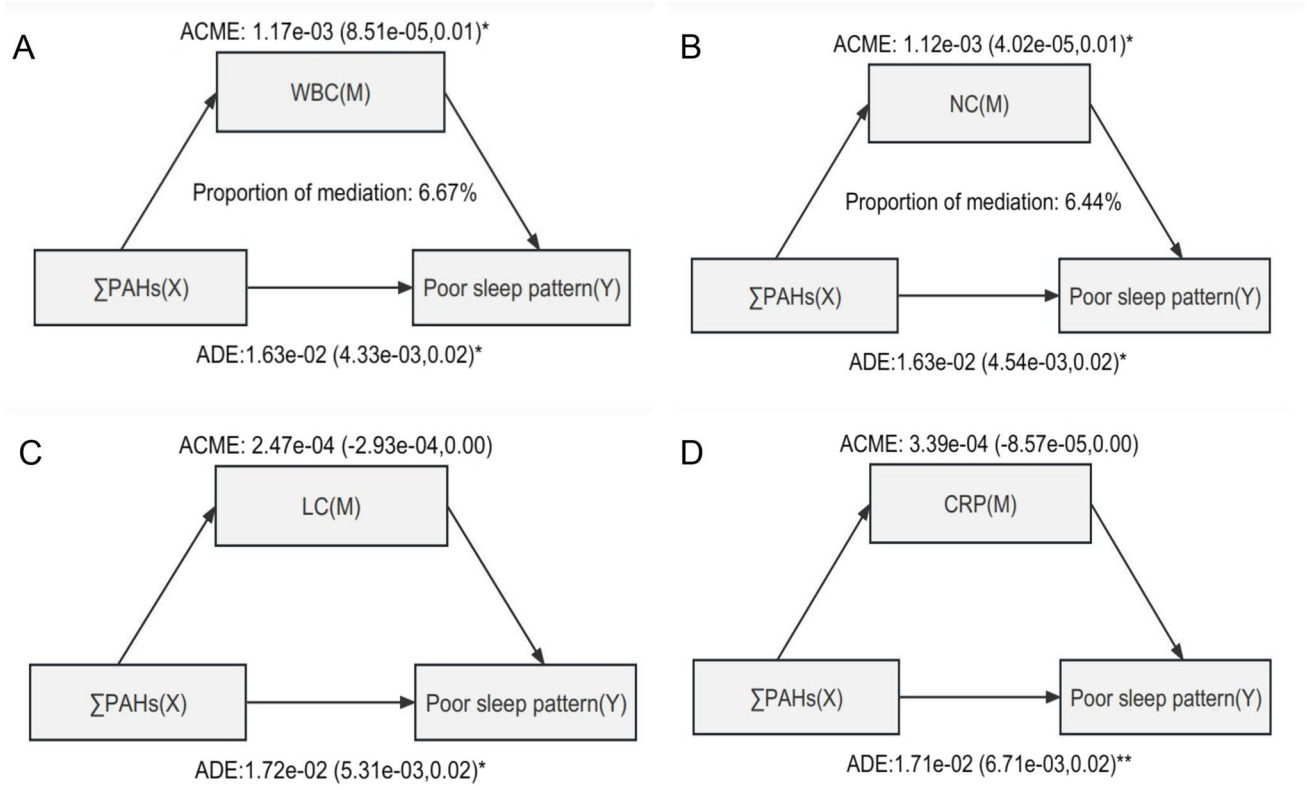

Fig. 5 Mediation analysis of inflammation indicators **A** WBC, **B** NC, **C** LC, and **D** CRP on the interaction between PAHs and prevalence of poor sleep pattern. WBC, white blood cell; NC, neutrophil; LC, lymphocyte; CRP, C-reactive protein. The models were adjusted for age, gender, race, educational level, marital status, PRI, BMI, smoking status, and alcohol intake

Discussion

The goal of our study was to examine PAH exposure burden and its link to the risk of poor sleep pattern using the NHANES database, 2005–2010. Our key results were as follows: (1) The geometric mean of urine 2-NAP concentrations was 4155.7 ng/g creatinine, designating it as the most abundant of all the PAH metabolites we evaluated; (2) higher levels of 1-NAP, 2-FLU, 1-PHE, 2-PHE, 9-FLU, and ∑PAHs were positive correlated with poor sleep pattern; (3) the most prevalent PAH metabolites in the WQS model for poor sleep pattern were 9-FLU and 1-NAP; (4) the levels of various PAH metabolites were linked to higher rates of poor sleep pattern in individuals who were women, smoker, and between 20 and 39 years of age; and (4) inflammation is a mediator between PAHs and the incidence of poor sleep pattern.

To our knowledge, this is the first-ever comprehensive study undertaken on the relationship between PAHs and poor sleep pattern. We herein ascertained that there were variations in age, race, smoking status, and BMI between the groups showing poor sleep pattern and those without poor sleep pattern. According to a survey about sleep issues among US adults, non-Hispanic Whites reflected the highest incidence of sleep issues (Wang et al. 2023), which was consistent with our results. Moreover, previous research showed that non-Hispanic Whites were more prone to use sleep medications than non-Hispanic Blacks or Mexican Americans (Ford et al. 2014; Vozoris 2019). Currently, it is believed that differences in healthcare availability, healthcare-seeking behaviors, and cultural norms may partially account for racial disparities in the prevalence of sleep issues (Flack et al. 1995; Gornick et al. 1996; Jean-Louis et al. 2001). We acknowledge that numerous physiological alterations accompany aging (including alterations in sleep and circadian rhythms), elevating the susceptibility of middle-aged and elderly people to sleep disorders (Patel et al. 2018); similarly, our findings revealed that subjects who had trouble sleeping were also elder (≥ 40 years of age). It has been shown that smoking, a prevalent lifestyle risk factor, is associated with poorer sleep quality (Woo et al. 2022), which is consistent with the data in the baseline of this study. A meta-analysis of relations between obesity and insomnia showed a significant weak positive correlation between insomnia and BMI (Chan et al. 2018). In the present study, while we discerned that subjects with poor sleep pattern had a greater BMI (49.4% vs. 36.1%, $P < 0.001$), a recent study from India illustrated that insomnia symptoms were inversely related to

obesity/overweight when compared to older individuals with a normal BMI (Muhammad et al. 2022). This contradicts our findings and might be attributed to the disparity in the study populations. As a result, research is sorely needed in the future to explicate the association between BMI, overweight, or obesity and sleep problems in order to provide a clinical reference.

There is less evidence of a link between air pollution and the likelihood of insomnia or other sleep-related symptoms. According to a Boston longitudinal survey, increased air pollution caused by vehicular traffic was significantly correlated with a diminution in sleep duration (Fang et al. 2015). Xu et al. (2021b) demonstrated that exposure to higher levels of PM2.5 and O_3 raised the incidence of insomnia. One cross-sectional study conducted by Wang et al. (2022a, b) on patients with obstructive sleep apnea (OSA) discovered a relationship between sleep-disordered breathing parameters and air pollutants, with PM10 and O_3 the most significant effectors. It was previously generally hypothesized that air pollutants impacted the quality of sleep via the central nervous system and/or due to changes in the physiology of the respiratory system (Park et al. 2019). In addition, a UK bank–based study has further confirmed that long-term exposure to air pollution can have an adverse impact on sleep health. (Li et al. 2022). We observed similar results in the current investigation. In a multivariate regression model adjusted for all confounding variates, we found the highest levels of 1-NAP, 2-FLU, 1-PHE, 2-PHE, and 9-FLU still reflected significant positive associations with poor sleep pattern in the general US population, revealing that air-pollutant PAHs may have exerted an adverse effect on sleep. However, additional research is necessitated to further elaborate on the mechanism(s) by which PAH exposure leads to trouble sleeping.

To elucidate the association between PAH and poor sleep pattern, we employed WQS regression to analyze the multiple effects and relative weights of each PAH on poor sleep pattern. The results illustrated that exposure to PAH was associated with an increased prevalence of poor sleep pattern, specifically, 9-FLU and 1-NAP. It is currently believed that 1-NAP and 2-NAP are the two isomers of naphthol, and 1-NAP (as naphthalene's primary metabolite) is found in cigarette smoke, pesticides, and herbicides containing carbamates, and in the beta-receptor blocker propranolol (Berman et al. 2016; Chen et al. 2020; Zhu et al. 2021). In addition, exposure to high concentrations of PAHs in the diet, cigarette smoke, and in automobile exhaust generate fluorene metabolites such as 2-FLU, 3-FLU, and 9-FLU (Cao et al. 2020; Lotz et al. 2016; Wang et al. 2016a, b). A recent study, based on the NHANES data, examined the relationship between PAHs and COPD, emphasizing the primary sources of each PAH metabolite for the first time (Peng et al. 2023). Therefore, the identification of the primary pollutants and the primary sources of those pollutants can enhance methods for the prevention and management of air pollution and, to a certain extent, provide substantial health benefits for individuals who suffer from sleep issues.

In subgroup analysis, participants aged 20 to 39 years were found to be more likely to suffer from poor sleep pattern due to PAH exposure. This might be because such people have a greater likelihood to be exposed to PAH due to factors such as work requirements, food habits, and smoking status. The sex-specific subgroup analysis revealed statistically significant associations between PAH metabolites and poor sleep pattern in the female population. According to an earlier study, this may have been attributable to the women's dietary habits or their different rates of PAH metabolism (Jain 2015). In addition, women are more vulnerable to chromosomal damage and oxidative stress due to PAH exposure than men (Guo et al. 2014). In addition, tobacco smoke is one of the major indoor sources of PAHs (Kim et al. 2013), and studies have shown that smokers have significantly higher concentrations of PAH metabolites in their urine than non-smokers (Cao et al. 2020). Exposure to tobacco leads to inflammation and oxidative stress in the body (Kuang et al. 2013; Lietz et al. 2013), which may be a possible pathway for the development of poor sleep pattern. The subgroup analysis results indicated that poor sleep pattern caused by PAH exposure was more prevalent in female, 20–39 years old, and smokers, providing a foundation for initiatives targeting reductions in air pollution.

Despite the fact that the mechanisms underlying the association between PAH exposure and poor sleep pattern have not been completely elucidated, there are a few lines of evidence to support the association. First, it was shown that exposure to PAHs is a factor in the onset of oxidative stress and systematic inflammation (Alshaarawy et al. 2013; Andersen et al. 2018; Bortey-Sam et al. 2017; Liu et al. 2018) that can affect the sleep–wake cycle (Pan et al. 2013). Second, numerous studies have shown that PAHs can regulate blood lipids and cause atherosclerosis (Holme et al. 2019; Ranjbar et al. 2015; Xu et al. 2021a, b, c; Yang et al. 2019). In a prior investigation, insomnia was linked to increased vascular stiffness and carotid atherosclerosis, and as the length of sleeplessness increased, the link became more significant (Pan et al. 2022). Third, Xing et al. applied data from NHANES 2011–2012 and found that individuals who were exposed to PAH had higher levels of thyroid hormones and thyroid autoantibodies (Xing et al. 2023), and Sridhar et al. (Sridhar et al. 2011) found an association between high levels of thyroid hormone and numerous aspects of sleep disorders such as increased sleep latency and trouble maintaining sleep. Furthermore, thyroid-stimulating hormone (TSH) levels, as well as T3 and T4, were found to be related to the severity of insomnia in a study by Xia et al. (Xia et al. 2013). To verify whether inflammation

mediated the relationship between PAH and sleep patterns, we performed mediation analyses. The results revealed that WBC and NC partly mediated the association between PAH exposure and poor sleep pattern. The results show that PAH exposure is associated with the development of systemic inflammation, which in turn disrupts the normal sleep cycle and increases the incidence of sleep problems. However, other possible mechanisms could not be further investigated due to incomplete information in the NHANES database, and additional clinical studies and animal experiments are needed in the future to demonstrate the precise mechanism by which PAH exposure causes sleep issues.

The strengths of the study were as follows: First, this study entailed a large sample size from the NHANES database, controls for numerous confounding variables to verify the validity of the findings, and offers new proof that exposure to PAH is a risk factor for poor sleep pattern. Second, to determine the contribution of each pollutant in the mixture and to identify special groups, WQS regression and subgroup analysis were utilized. Third, mediation analysis was employed in this study to elucidate inflammation acted as a mediator between PAHs and the prevalence of poor sleep pattern.

There were also some limitations to our study. First, because this was a cross-sectional study, no causal relationship could be drawn. However, with this study, we only explored the link between PAH exposure and poor sleep pattern in order to offer suggestions for future research. Second, although we controlled for several confounding factors, there are numerous factors that influence sleep, and therefore, additional research is needed in the future to confirm the link between PAH and sleep. Third, concentrations of PAH metabolites in urine were used to represent PAH exposure in humans in this study. Nevertheless, each individual's renal metabolism of PAHs may influence the assessment of PAH exposure in humans. It is thus imperative to develop novel techniques and methods for the detection of blood PAH markers. Lastly, this study's participants were Americans with distinctive cultural and behavioral norms. Therefore, care must be taken when extrapolating this study's findings to other nations.

Conclusion

According to the results of this investigation, 2-NAP was the predominant among the PAH metabolites in urine. Several PAH metabolites were found to reflect positive correlations with the risk of poor sleep pattern in US adults, with 9-FLU and 1-NAP achieving the greatest weight. Finally, we found that inflammation acted as a mediator between PAHs and the prevalence of poor sleep pattern. More research, including longitudinal studies, is required to confirm the precise relationship between PAH and sleep and to explore the potential mechanisms underlying this relationship. Our findings suggest that efforts should be undertaken to reduce PAH emissions in order to diminish the risk of poor sleep pattern due to environmental pollution.

Abbreviations *ACME:* Average causal mediated effect; *ADE:* Average direct effect; 1-*NAP:* 1-Hydroxynapthalene; 2-NAP: 2-Hydroxynapthalene; 3-*FLU*: 3-Hydroxyfluorene; 2-*FLU:* 2-Hydroxyfluorene; 3-*PHE:* 3-Hydroxyphenanthrene; 1-*PHE:* 1-Hydroxyphenanthrene; 2-*PHE:* 2-Hydroxyphenanthrene; 1-*PYR:* 1-Hydroxypyrene; 9-*FLU:* 9-Hydroxyfluorene; *PAH:* Polycyclic aromatic hydrocarbon; *NHANES:* National Health and Nutrition Examination Survey; COPD: Chronic obstructive pulmonary disease; *PIR:* Poverty-to-income ratio; *BMI:* Body mass index; *RCS:* Restricted cubic spline; *WQS:* Weighted quantile sum; *OR:* Odds ratio; *CI:* Confidence interval; *WBC:* White blood cell; *NC:* Neutrophil; *LC:* Lymphocyte; *CRP:* C-reactive protein; *TSH*: Thyroid-stimulating hormone

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Data availability The corresponding author will provide the data used and analyzed during the present investigation upon reasonable request.

Declarations

Ethics approval and consent to participate The NHANES publishes open-access data; an institutional review board analysis was not necessary for our study.

Consent for publication Consent was given by all authors.

Conflict of interest The authors declare no competing interests.

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