Tian *et al. BMC Public Health* (2025) 25:1679
https://doi.org/10.1186/s12889-025-22887-x

BMC Public Health

RESEARCH Open Access

# Correlation between nighttime sleep noise pollution and the risk of acute exacerbation of chronic obstructive pulmonary disease

Huijun Tian[1*], Yanwei Cheng[1], Lijie Qin[1], Peirong Zhang[1], Yun Li[1] and Bingwei Liang[1]

## Abstract

**Background** The role of noise pollution in chronic obstructive pulmonary disease (COPD) remains unclear. The aim of this study was to investigate the correlation between nighttime sleep noise levels and the risk of acute exacerbations of this lung disease.

**Methods** A total of 518 stable COPD patients were enrolled in the study. A portable sound level meter was used to measure the nighttime sleep noise levels. Subsequently, based on a threshold of 40 dB, the patients were divided into a high-noise group ($n=210$) and a low-noise group ($n=308$). The Pittsburgh Sleep Quality Index was employed to assess the sleep quality of the patients. During the 12-month follow-up, the events of acute exacerbation (i.e., Escalated therapy, Urgent hospitalization, and Admitted to intensive care unit) in these COPD patients were recorded. Multivariate logistic regression was utilized for data analysis.

**Results** When most potential confounding factors (i.e., demographic variable, chronic disease, risk factor of COPD, status of COPD, and inflammatory indicator) were adjusted, a higher nighttime sleep noise level was found to be associated with acute exacerbations of COPD during the follow-up period. After adjusting the sleep quality score, although the *P*-value of the above-mentioned correlation was still statistically significant, its level increased significantly, that is, the level of the *P*-value became closer to the threshold at which it would not be statistically significant.

**Conclusion** There is a significant correlation between relatively high nighttime sleep noise level and acute exacerbations of COPD. Sleep disorders might potentially contribute in some way to the above-mentioned correlation. The exact role of nighttime sleep noise in acute exacerbations of this lung disease and the underlying mechanisms still need further research.

**Keywords** Acute exacerbation, Chronic obstructive pulmonary disease, Noise pollution, Prognosis, Sleep disorder

*Correspondence:
Huijun Tian
tianhuijun0807@outlook.com
[1]Department of Emergency Medicine, People's Hospital of Henan Province, People's Hospital of Zhengzhou University, No. 7 Weiwu Road, Jinshui District, Zhengzhou City 450003, Henan Province, China

## Introduction

Currently, there are more than 384 million patients with chronic obstructive pulmonary disease (COPD) globally [1]. The main characteristic of this disease is persistent airflow limitation that is not fully reversible [2]. COPD can also undergo intermittent acute exacerbations, leading to a rapid deterioration of patients' respiratory function, and a notably elevated risk of mortality [3]. According to statistics, the number of patients who die from COPD globally each year reaches 3.23 million [1]. So, strengthening the prevention of acute exacerbations of COPD is of great significance for improving the overall prognosis of patients.

Existing studies have confirmed that long-term exposure to air pollution, occupational dusts and chemical substances, as well as smoking, may damage the airways and lungs, increasing the risk of acute exacerbations of COPD [4]. Elderly patients with COPD are more prone to acute exacerbations due to the decline in physical functions and immunity [5]. If patients with COPD have comorbid cardiovascular diseases and diabetes, the risk of their acute exacerbations will increase [6]. Respiratory infections often trigger acute exacerbations of COPD [7]. However, many other risk factors for acute exacerbations of COPD remain unclear, which is not conducive to the effective control of this disease.

As is well known, COPD is often accompanied by sleep disorders, and sleep disorders such as insomnia may be further associated with the acute exacerbation of COPD [8, 9]. In addition, some studies have indicated that there is a correlation between occupational noise exposure and the risk of developing COPD [10]. This seemingly implies that noise exposure may increase the risk of acute exacerbation of COPD, because noise exposure, especially nighttime noise exposure, is clearly linked to sleep disorders. More importantly, the harm of noise to health is multifaceted. Noise from various sources can not only affect sleep but also have an impact on the mental state and work ability of those affected [11–14]. Long-term and continuous exposure to high-decibel noise environments may even lead to the occurrence of certain cancers [15]. So, it is of great significance to directly study the correlation between noise exposure and the acute exacerbation of COPD.

Therefore, this cohort study aimed to enroll several hundred patients with COPD, and explored the correlation between the nighttime sleep noise levels and the risk of acute exacerbations of COPD during the follow-up period. The findings may contribute to a comprehensive understanding of the risk factors for this disease.

## Materials and methods

### Ethical requirements

This study was approved by the Medical Ethics Committee of Henan Provincial People's Hospital (ID: 2018-57). This study was conducted in accordance with the requirements of the World Medical Association Declaration of Helsinki [16]. All patients gave their consent to participate in this study.

### Participants

From January 1, 2019, to June 30, 2023, the screening of COPD patients was conducted across 10 large neighborhoods in Zhengzhou, and these localities were located in different parts of the city. Initially, the local Centers for Disease Control and Prevention established communication with the community physicians in these areas. These physicians had the health records of residents within their respective neighborhoods. Subsequently, the physicians made sequential telephone calls to the patients with COPD in accordance with the filing sequence. During these calls, they elaborated on the objectives and contents of this research and inquired about the patients' willingness to participate. After obtaining informed consent, they accompanied the researchers during home visits. Residents who met the predefined inclusion criteria were selected, while those who did not were excluded.

The inclusion criteria were as follows: (1) There were complete records in the medical records indicating that these patients had been diagnosed with COPD. The medical experts of our team reviewed these records and confirmed that they met the diagnostic criteria of the Global Initiative for COPD [17]. (2) Based on the results of the most recent pulmonary function test, current symptoms, and medication use, their conditions were determined to be in a stable stage. (3) Through communication with the patients, it was determined that they had basic cognitive abilities and communication skills, had no deafness or severe hearing impairment, and were able to cooperate with this study. (4) These patients had fixed and independent bedrooms and did not need to work night shifts. The bedrooms were neither adjacent to the street, nor to the boiler room, nor to the factory. In other words, the sleep environments of these patients were relatively stable, and there were no unstable noise sources. (5) These patients had no severe snoring and had never been diagnosed with sleep apnea syndrome, so as to exclude the influence of such conditions on the study results [18]. (6) The patients had no other serious diseases, such as malignant tumors. Common chronic diseases, such as type 2 diabetes, were in a stable state.

During the above-mentioned screening period, for the localities where the screening had been completed, if there were newly registered COPD patients, or for those patients who were in the acute exacerbation stage during

**Table 1** Basic characteristics of patients with chronic obstructive pulmonary disease at baseline

| Variable | High noise (*n* = 210) | Low noise (*n* = 308) | t / $\chi^2$ value | *P* value |
|---|---|---|---|---|
| Age (years) | 67.4 ± 7.2 | 68.1 ± 7.4 | 0.993 | 0.321 |
| Gender (n) | | | | |
| Male | 140 (66.7) | 191 (62.0) | 1.172 | 0.279 |
| Female | 70 (33.3) | 117 (38.0) | | |
| Ethnic group (n) | | | | |
| Han people | 186 (88.6) | 286 (92.9) | 2.834 | 0.092 |
| Other | 24 (11.4) | 22 (7.1) | | |
| Education (n) | | | | |
| >12 years | 77 (36.7) | 98 (31.8) | 1.312 | 0.252 |
| ≤12 years | 133 (63.3) | 210 (68.2) | | |
| Monthly income (n) | | | | |
| >5000 yuan | 71 (33.8) | 92 (29.9) | 0.899 | 0.343 |
| ≤5000 yuan | 139 (66.2) | 216 (70.1) | | |
| Chronic disease (n) | | | | |
| HTN | 115 (54.8) | 122 (39.6) | 11.549 | 0.001 |
| CHD | 75 (35.7) | 82 (26.6) | 4.885 | 0.027 |
| CVD | 31 (14.8) | 30 (9.7) | 3.031 | 0.082 |
| T2DM | 69 (32.9) | 78 (25.3) | 3.486 | 0.062 |
| Obesity | 55 (26.2) | 62 (20.1) | 2.623 | 0.105 |
| HLP | 94 (44.8) | 117 (38.0) | 2.374 | 0.123 |

Note: HTN = Hypertension, CHD = Coronary Heart Disease, CVD = Cardiovascular Disease, T2DM = Type 2 Diabetes Mellitus, HLP = Hyperlipidemia. Continuous variables are expressed as mean ± standard deviation, and categorical variables are expressed as frequencies and constituent ratios. A *P*-value less than 0.05 indicates statistical significance

**Table 2** Sleep status and average nighttime sleep noise level of chronic obstructive pulmonary disease patients at baseline

| Variable | High noise (*n* = 210) | Low noise (*n* = 308) | t / $\chi^2$ value | *P* value |
|---|---|---|---|---|
| PSQI score | 12.30 ± 2.30 | 9.95 ± 1.33 | 14.746 | <0.001 |
| Sleep quality | 1.76 ± 0.73 | 1.48 ± 0.50 | 5.223 | <0.001 |
| Sleep latency | 1.80 ± 0.74 | 1.54 ± 0.50 | 4.934 | <0.001 |
| Sleep duration | 1.84 ± 0.73 | 1.48 ± 0.50 | 6.695 | <0.001 |
| Sleep efficiency | 1.84 ± 0.71 | 1.52 ± 0.50 | 6.033 | <0.001 |
| Sleep disturbance | 1.77 ± 0.73 | 1.46 ± 0.50 | 5.595 | <0.001 |
| Sleeping medication | 1.49 ± 0.96 | 0.95 ± 0.72 | 7.287 | <0.001 |
| Daytime dysfunction | 1.80 ± 0.75 | 1.52 ± 0.50 | 5.106 | <0.001 |
| AIS score | 6.45 ± 0.92 | 5.49 ± 0.95 | 11.539 | <0.001 |
| SDRS score | 14.01 ± 1.22 | 11.98 ± 1.28 | 18.081 | <0.001 |
| Environmental noise (dB) | | | | |
| Noise on Day 1 | 44.97 ± 2.90 | 31.98 ± 3.99 | 40.474 | <0.001 |
| Noise on Day 3 | 44.98 ± 3.10 | 31.91 ± 4.20 | 38.534 | <0.001 |
| Noise on Day 5 | 45.01 ± 3.08 | 31.98 ± 4.13 | 38.934 | <0.001 |
| Average noise | 44.99 ± 2.98 | 31.96 ± 4.03 | 39.975 | <0.001 |

Note: HTN = Hypertension, CHD = Coronary Heart Disease, CVD = Cardiovascular Disease, T2DM = Type 2 Diabetes Mellitus, HLP = Hyperlipidemia, PSQI = Pittsburgh Sleep Quality Index, AIS = Athens Insomnia Scale, SDRS = Sleep Dysfunction Rating Scale. Continuous variables are expressed as mean ± standard deviation, and categorical variables are expressed as frequencies and constituent ratios. A *P*-value less than 0.05 indicates statistical significance

**Table 3** Risk factors for chronic obstructive pulmonary disease and its acute exacerbation

| Variable | High noise (*n* = 210) | Low noise (*n* = 308) | t / $\chi^2$ value | *P* value |
|---|---|---|---|---|
| High-risk occupations (n) | 33 (15.7) | 42 (13.6) | 0.435 | 0.509 |
| Cooking fume exposure (n) | 27 (12.9) | 51 (16.6) | 1.337 | 0.247 |
| Vehicle exhaust exposure (n) | 85 (40.5) | 107 (34.7) | 1.761 | 0.184 |
| Cigarette smoking (n) | 148 (70.5) | 203 (65.9) | 1.192 | 0.275 |
| Passive smoke (n) | 11 (5.2) | 27 (8.8) | 2.286 | 0.131 |
| Alcohol drinking (n) | 41 (19.5) | 46 (14.9) | 1.881 | 0.170 |
| Allergic asthma (n) | 64 (30.5) | 111 (36.0) | 1.727 | 0.189 |
| Family history of COPD (n) | 41 (19.5) | 78 (25.3) | 2.374 | 0.123 |
| Lack of exercise (n) | 88 (41.9) | 110 (35.7) | 2.026 | 0.155 |
| Malnutrition (n) | 76 (36.2) | 93 (30.2) | 2.042 | 0.153 |

Note: COPD = Chronic obstructive pulmonary disease. Continuous variables are expressed as mean ± standard deviation, and categorical variables are expressed as frequencies and constituent ratios. A *P*-value less than 0.05 indicates statistical significance

the first screening but whose conditions had been stable for more than four weeks, supplementary surveys would be conducted to ensure that all eligible patients could be included in this study.

### Baseline data collection

The following baseline data of the patients were collected through medical record review and face-to-face interviews: (1) Basic characteristics of the patients. (2) Risk factors for COPD and its acute exacerbation. (3) Status of COPD at baseline. (4) Cardiac and inflammatory indicators measured (within 3 months).

Specific categories are presented in Tables 1, 2, 3, 4 and 5. Patients with incomplete baseline data as mentioned above were excluded from this study. The definitions or descriptions of the above data are shown in Supplemental Table 1.

### Baseline sleep assessment

The patients' baseline sleep status was evaluated by researchers using the Pittsburgh Sleep Quality Index (PSQI), the Athens Insomnia Scale (AIS), and the Sleep Dysfunction Rating Scale (SDRS) [19–21].

The PSQI is composed of 18 questions, covering seven dimensions: sleep quality, sleep latency, sleep duration, sleep efficiency, sleep disturbance, sleeping medication, and daytime dysfunction. The score for each dimension ranges from 0 to 3 points, and the total score, which ranges from 0 to 21 points, is the sum of the scores of all dimensions. The AIS is used to evaluate the sleep status in the past month. It consists of 8 questions, involving aspects such as sleep latency, the number of awakenings at night, and early morning awakening. The total score ranges from 0 to 24 points. The SDRS involves aspects like difficulty falling asleep, light sleep, frequent dreaming, etc. It is composed of 10 items, and the total score

**Table 4** Status of chronic obstructive pulmonary disease at baseline and occurrence of acute exacerbations during the follow-up period

| Variable | High noise (*n* = 210) | Low noise (*n* = 308) | t / $\chi^2$ value | *P* value |
|---|---|---|---|---|
| Baseline GOLD classification | | | | |
| Grade 1 (n) | 35 (16.7) | 68 (22.1) | 2.295 | 0.130 |
| Grade 2 (n) | 80 (38.1) | 129 (41.9) | 0.744 | 0.388 |
| Grade 3 (n) | 68 (32.4) | 89 (28.9) | 0.718 | 0.397 |
| Grade 4 (n) | 27 (12.9) | 22 (7.1) | 4.761 | 0.029 |
| Baseline pulmonary function | | | | |
| $FEV_1$% pred (%) | 55.9 ± 22.8 | 59.9 ± 21.7 | 2.039 | 0.042 |
| DLCO% (%) | 61.5 ± 20.9 | 65.6 ± 19.3 | 2.302 | 0.022 |
| PaO2 (mmHg) | 76.1 ± 11.8 | 78.0 ± 11.3 | 1.841 | 0.066 |
| PaCO2 (mmHg) | 47.8 ± 7.6 | 45.7 ± 7.1 | 3.187 | 0.002 |
| Daily treatment | | | | |
| β-receptor agonists (n) | 183 (87.1) | 258 (83.8) | 1.125 | 0.289 |
| Anticholinergic drugs (n) | 141 (67.1) | 223 (72.4) | 1.654 | 0.198 |
| Glucocorticoids (n) | 112 (53.3) | 147 (47.7) | 1.570 | 0.210 |
| Oxygen inhalation (n) | 99 (47.1) | 125 (40.6) | 2.188 | 0.139 |
| Acute exacerbation | | | | |
| Escalated therapy (n) | 74 (35.2) | 66 (21.4) | 12.074 | 0.001 |
| Urgent hospitalization (n) | 56 (26.7) | 43 (14.0) | 13.039 | <0.001 |
| Admission to ICU (n) | 16 (7.6) | 8 (2.6) | 7.126 | 0.008 |

Note: GOLD = Global Initiative for Chronic Obstructive Lung Disease, $FEV_1$% pred = Forced Expiratory Volume in the First Second Percentage of Predicted Value, DLCO% = Diffusing Capacity of the Lung for Carbon Monoxide Percentage of Predicted Value, PaO2 = Partial Pressure of Oxygen in Arterial Blood, PaCO2 = Partial Pressure of Carbon Dioxide in Arterial Blood, ICU = Intensive Care Unit. Continuous variables are expressed as mean ± standard deviation, and categorical variables are expressed as frequencies and constituent ratios. A *P*-value less than 0.05 indicates statistical significance

**Table 5** Other physiological and psychological indicators of chronic obstructive pulmonary disease patients at baseline

| Variable | High noise (*n* = 210) | Low noise (*n* = 308) | t / $\chi^2$ value | *P* value |
|---|---|---|---|---|
| Cardiac indicator | | | | |
| LVEF (%) | 55.2 ± 5.8 | 56.0 ± 5.9 | 1.532 | 0.126 |
| NT-proBNP (pg/mL) | 958.2 ± 487.8 | 881.8 ± 453.7 | 1.826 | 0.068 |
| TnT (ng/mL) | 0.102 ± 0.059 | 0.097 ± 0.058 | 0.945 | 0.345 |
| Inflammator indicator | | | | |
| NEUT% (%) | 57.2 ± 10.5 | 56.5 ± 10.6 | 0.676 | 0.499 |
| CRP (mg/L) | 5.6 ± 3.2 | 5.2 ± 3.1 | 1.283 | 0.200 |
| PCT (ng/mL) | 0.097 ± 0.058 | 0.095 ± 0.056 | 0.548 | 0.584 |
| TNF-α (pg/mL) | 6.0 ± 2.3 | 5.9 ± 2.1 | 0.637 | 0.525 |
| IL-6 (pg/mL) | 3.5 ± 2.0 | 3.4 ± 1.9 | 0.654 | 0.513 |
| Psychological indicator | | | | |
| HADS (depression) score | 7.6 ± 2.9 | 7.2 ± 2.7 | 1.830 | 0.068 |
| HADS (anxiety) score | 7.4 ± 2.7 | 6.9 ± 2.5 | 1.791 | 0.074 |

Note: LVEF = Left Ventricular Ejection Fraction, NT-proBNP = N-terminal Pro-B-type Natriuretic Peptide, TnT = Troponin T, NEUT% = Neutrophil Percentage, CRP = C-Reactive Protein, PCT = Procalcitonin, TNF-α = Tumor Necrosis Factor-alpha, IL-6 = Interleukin-6, HADS = Hospital Anxiety and Depression Scale. Continuous variables are expressed as mean ± standard deviation, and categorical variables are expressed as frequencies and constituent ratios. A *P*-value less than 0.05 indicates statistical significance

ranges from 0 to 40 points. For all the above three scales, the higher the score, the worse the sleep quality.

### Baseline psychological test

At baseline, the psychological states of the patients were evaluated by researchers using the Hospital Anxiety and Depression Scale (HADS) [22].

The HADS is composed of two subscales, namely the anxiety subscale and the depression subscale, each of which contains 7 items. The score range of the two subscales is both 0–21 points. The higher the score is, the more severe the anxiety or depression is.

### Baseline noise assessment

The sound level meter is the most commonly used instrument for noise measurement. At baseline, this study employed a portable sound level meter (SMART SENSOR AS824) that complies with the International Electrotechnical Commission (IEC) 61,672 standard to measure the nighttime sleep noise levels of the patients. This device is easy to operate and provides accurate results. Before each measurement, it was calibrated using an acoustic calibrator that meets the IEC 60,942 standard.

Professional personnel installed the device in the patients' homes and provided necessary training to the patients. Briefly, the sound level meter was placed approximately 1 m away from the sleeper's head and at the same horizontal level as the ears, while ensuring that there were no obstacles in the sound propagation path. During the measurement process, the sound level meter was set to the A-weighting network. This network simulates the human ear's response characteristics to sounds of different frequencies according to the International Organization for Standardization 226 standard, which can better match the actual perception of the human ear. Meanwhile, the equivalent continuous sound level was set, with the unit being dB. It accurately represents the average sound level over a relatively long period of time through the integration operation of the sound pressure level during the measurement period, thus comprehensively and accurately reflecting the overall noise level during sleep.

When conducting the measurement, patients turned on the device by themselves for measurement before going to bed (between 22:00 and 24:00) and turned it off after getting up in the morning (between 6:00 and 8:00). The measurement time was no less than 6 h. For each patient, measurements were carried out for three nights (with one-day intervals between each measurement) to reduce the influence of accidental factors. Then, the average value of the three measurement results was taken to represent the nighttime sleep noise levels of the patients.

To effectively avoid the interference of snoring, the following measures were taken: (1) Patients with relatively severe simple snoring or those suffering from sleep apnea syndrome were directly excluded. (2) When placing the sound level meter, it was avoided to position it directly facing the patients' mouths or noses. (3) During the data analysis, professional personnel used a combination of data analysis software and manual screening to eliminate the data that was interfered with by snoring.

This study holds that the nighttime sleep noise level measured through the above method is consistent with the true nighttime sleep noise level of the patients during the follow-up period. The reasons are as follows: (1) During the screening, patients with an unstable nighttime residence were directly excluded. (2) Each patient underwent three standardized noise measurements, and the average value of the results was calculated. (3) During the follow-up period, if any event that might affect the nighttime sleep noise level occurred to a patient, such as changing the sleep location, nearby nighttime construction, or new neighbors moving in, this patient would be directly excluded from this study.

### Follow-up

Each patient was followed up by phone for 12 months, and the occurrence of acute exacerbation was recorded. Acute exacerbation was represented by "Escalated therapy", "Urgent hospitalization", and "Admission to intensive care unit (ICU)". "Escalated therapy" referred to increasing the dosage or types of medications according to the doctor's advice to cope with the disease deterioration. "Urgent hospitalization" and "Admission to ICU" respectively referred to being forced to be admitted to a general ward and the ICU according to the disease progression.

In addition, during the follow-up period, if there were other significant changes in the patient's living status apart from the acute exacerbation of COPD, such as the rapid progression of other chronic diseases or the diagnosis of new serious illnesses, the patient would be excluded from this study.

### Statistical analysis

Continuous variables were expressed as mean ± standard deviation, and the differences between groups were evaluated by independent-sample t-test. Categorical variables were expressed as frequency and proportion, and the differences between groups were evaluated by chi-square test. The linear relationship between sleep scores and noise levels was evaluated using Pearson correlation analysis. The correlation between nighttime sleep noise level and acute exacerbation of COPD was evaluated by multivariate logistic regression, and adjustments were made for demographic variables, chronic diseases, PSQI scores, risk factors of COPD, COPD status, or inflammatory indicators. A $P$-value < 0.05 indicated statistical significance.

## Results

### Patients

At baseline, a total of 536 COPD patients with established health records were identified in these neighborhoods. Among them, 518 patients met the aforementioned requirements and were included in this study. The remaining 18 patients (constituting 3.4%) were excluded. The reasons for exclusion were refusal to participate, and having an unstable nighttime residence. During the follow-up period, no other patients were excluded from this study.

According to the patients' noise test results, patients with a nighttime sleep noise level ≥ 40 dB were assigned to the high-noise group ($n = 210$), while those with a level of < 40 dB were assigned to the low-noise group ($n = 308$). These grouping boundaries were determined according to relevant international and Chinese noise standards [23].

### Distribution of each research variable in the two groups

In Table 1, compared with the low-noise group, the proportion of patients with hypertension and coronary heart disease in the high-noise group was significantly higher ($P = 0.001$, $P = 0.027$). However, there were no significant differences in the distribution of demographic characteristics and other chronic disease histories.

In Table 2, compared with the low-noise group, patients in the high-noise group had significantly higher PSQI, AIS, and SDRS scores ($P < 0.001$, $P < 0.001$, $P < 0.001$). The average nighttime sleep noise level of patients in this group was also significantly higher ($P < 0.001$). In Supplemental Fig. 1, there was a positive linear correlation between the PSQI score and the average nighttime sleep noise level ($r = 0.469$, $P<0.001$).

In Table 3, there were no statistically significant differences in the distribution of a series of risk factors for COPD and its acute exacerbation between the two groups.

In Table 4, compared with the low-noise group, the high-noise group had a higher proportion of patients with a GOLD classification of Grade 4 ($P = 0.029$), lower levels of FEV1% pred and DLCO% ($P = 0.042$, $P = 0.022$), and a higher level of PaCO2 ($P = 0.002$). There were no significant differences between the two groups in daily treatment. Compared with the low-noise group, a larger proportion of patients in the high-noise group experienced escalated therapy, urgent hospitalization, and admission to the ICU during the follow-up period ($P = 0.001$, $P < 0.001$, $P = 0.008$).

**Table 6** Multivariate analysis of the correlation between nighttime sleep noise and acute exacerbation of COPD

| Outcome (Acute exacerbation) | Adjusted variables | *P* value | OR (95%CI) |
|---|---|---|---|
| Escalated therapy | None | 0.001 | 1.995 (1.347 ~ 2.955) |
| Urgent hospitalization | None | <0.001 | 2.241 (1.437 ~ 3.495) |
| Admission to ICU | None | 0.011 | 3.093 (1.299 ~ 7.365) |
| Escalated therapy | Demographic variable | 0.001 | 1.980 (1.336 ~ 2.934) |
| Urgent hospitalization | Demographic variable | <0.001 | 2.261 (1.449 ~ 3.530) |
| Admission to ICU | Demographic variable | 0.011 | 3.065 (1.286 ~ 7.307) |
| Escalated therapy | Chronic disease | 0.001 | 1.941 (1.307 ~ 2.883) |
| Urgent hospitalization | Chronic disease | <0.001 | 2.265 (1.450 ~ 3.538) |
| Admission to ICU | Chronic disease | 0.009 | 3.181 (1.332 ~ 7.594) |
| Escalated therapy | PSQI score | 0.048 | 1.617 (1.004 ~ 2.605) |
| Urgent hospitalization | PSQI score | 0.042 | 1.746 (1.021 ~ 2.987) |
| Admission to ICU | PSQI score | 0.143 | 2.157 (0.772 ~ 6.027) |
| Escalated therapy | Risk factor of COPD | <0.001 | 2.049 (1.379 ~ 3.046) |
| Urgent hospitalization | Risk factor of COPD | <0.001 | 2.256 (1.446 ~ 3.521) |
| Admission to ICU | Risk factor of COPD | 0.009 | 3.167 (1.327 ~ 7.559) |
| Escalated therapy | Status of COPD | 0.001 | 1.968 (1.325 ~ 2.922) |
| Urgent hospitalization | Status of COPD | <0.001 | 2.258 (1.444 ~ 3.531) |
| Admission to ICU | Status of COPD | 0.012 | 3.062 (1.281 ~ 7.317) |
| Escalated therapy | Inflammatory indicator | 0.001 | 1.992 (1.345 ~ 2.950) |
| Urgent hospitalization | Inflammatory indicator | <0.001 | 2.256 (1.446 ~ 3.520) |
| Admission to ICU | Inflammatory indicator | 0.010 | 3.129 (1.313 ~ 7.459) |

Note: ICU = Intensive Care Unit, PSQI = Pittsburgh Sleep Quality Index, COPD = Chronic Obstructive Pulmonary Disease, OR = Odds Ratio, 95%CI = 95% Confidence Interval. A *P*-value less than 0.05 indicates statistical significance

In Table 5, there were no statistically significant differences between the two groups of patients in terms of cardiac, inflammatory, and psychological indicators.

### Multivariate analysis of the correlation between nighttime sleep noise and acute exacerbation of COPD

In Table 6, without adjusting for any factors or when adjusting for demographic variables, chronic diseases, risk factors of COPD, COPD status, and inflammatory indicators respectively, a nighttime sleep noise level of ≥ 40 dB was significantly associated with escalated therapy, urgent hospitalization, and admission to the ICU (All *P*-values<0.05. For details, see Table 6). It is worth emphasizing that after adjusting for the PSQI score, the *P*-values for the associations between a nighttime sleep noise level of ≥ 40 dB and escalated therapy, urgent hospitalization, and admission to the ICU increased significantly. Briefly, the *P*-values for escalated therapy and urgent hospitalization remained below 0.05, while the *P*-value for admission to the ICU was greater than 0.05.

## Discussion

Based on the results, this study confirms that a higher nighttime sleep noise level may be significantly associated with the acute exacerbation of COPD. Briefly speaking, compared with the patients whose nighttime sleep noise level is lower than 40 dB, the risk of adverse events related to the acute exacerbation of COPD in patients with a nighttime sleep noise level of 40 dB or higher is approximately 1 to 2 times higher. Moreover, after adjusting a series of demographic and disease data, these associations still exist. A previous study has reported the correlation between occupational noise protection and the onset of COPD [10]. The results of that study seem to be consistent with those of this study.

The results of this study also showed that after adjusting for the PSQI scores, although the correlation between nighttime sleep noise and acute exacerbation events of COPD (i.e., escalated therapy and urgent hospitalization) remained statistically significant, the *P*-value had increased to a level very close to the threshold of 0.05. That is, the likelihood of a correlation between nighttime sleep noise and acute exacerbation of COPD had decreased. Thus, the correlation between nighttime sleep noise and acute exacerbation of COPD can be, to some extent, explained by sleep disorders. On the one hand, previous studies have confirmed the correlation between environmental noise and sleep disorders [24, 25]. This study also reported a positive linear correlation between the nighttime sleep noise level and the PSQI score. On the other hand, insomnia is considered a potential prognostic indicator for COPD patients, and it is associated with a higher hospitalization rate and a longer hospital stay [26]. More importantly, improving the insomnia of COPD patients can alleviate COPD symptoms such as fatigue and dyspnea [27]. Thus, reducing nighttime sleep noise and alleviating sleep disorders might contribute to improving the prognosis of COPD.

In addition, after adjusting for the PSQI scores in this study, the correlation between nighttime sleep noise and another acute exacerbation event of COPD (i.e., admission to ICU) was no longer statistically significant. The reason for this might be that the relatively small number of patients admitted to the ICU led to an overly wide 95%CI.

This study incorporated over 95% of COPD patients from ten major neighborhoods within the city. This approach conferred a certain level of representativeness to the research sample for COPD patients in the city.

However, the sample does have its drawbacks. First, when compared to the total population of COPD patients, the sample size in this study is relatively small. Such a sample size may reduce the statistical power of the data, leading to less stable research outcomes. Second, as random sampling was not utilized and patients were permitted to make a voluntary decision on whether to participate in the study, self-selection bias was likely to emerge, potentially undermining the reliability of the results. Notwithstanding, as previously stated, the inclusion of the vast majority of patients from the selected neighborhoods in the study effectively minimized the likelihood of substantial self-selection bias.

In this study, we inferred the correlation between nighttime sleep noise and the risk of COPD exacerbation at the individual level based on the measurement of sleep noise at a relatively single location and within a limited time period. The greatest challenge here is how to ensure that the measured nighttime sleep noises can reflect the patients' true noise exposure levels. As described in the methods section of this study, we have made a great deal of effort in this regard, which can be summarized as follows: (1) The study tried to include patients with stable sleeping locations as much as possible; (2) The study standardized the noise measurement process, conducted multiple measurements, and reported the average values; and (3) The study excluded patients who had significant interfering factors during the follow-up period. Through these measures, we believe that the noise levels of patients measured in this study are consistent with their actual nighttime sleep noise exposure levels.

As far as we know, there is no unified global standard for sleep environment noise. A review supported by the World Health Organization concluded that in Europe, traffic noise ranging from 33 to 50 dB and above, as well as wind turbine noise of 40 to 45 dB, may interfere with sleep and affect health [23]. China's "Emission Standard of Environmental Noise for Social Life" (GB22337-2008) stipulates that the noise in the bedroom should not exceed 30 dB at night and 40 dB during the day. China's "Code for Sound Insulation Design of Civil Buildings" stipulates that the noise in the bedroom should not exceed 45 dB during the daytime and 37 dB at night. Based on these data, this study selected 40 dB as the grouping threshold. In addition, in this study, the noise level was transformed into a categorical variable instead of a continuous variable and included in the study. Such an arrangement may result in the loss of some information, but it can greatly simplify the analysis process, highlight the main research objectives, and help provide more intuitive and practical information for clinical and public health personnel.

For statistical methods, the mixed-effects model or repeated measures analysis has unique advantages. However, the former requires a clear data hierarchical structure, and the latter necessitates multiple repeated measurements of the same variable. This study did not provide sufficiently structured data, and the noise was only measured three times, making it difficult to fully meet the data requirements of these methods. In contrast, the multivariate logistic regression adopted in this study is suitable for analyzing the relationship between binary data (noise) and multiple independent variables (acute exacerbation events of COPD). After controlling for confounding factors, the results are simpler and more intuitive.

In this study, most of the confounding factors were controlled during the patient screening, noise measurement, and data analysis stages. However, there are still some factors that were not covered. (1) The acute exacerbation of COPD in more than two-thirds of the patients is related to acute respiratory tract infections. But this factor is so closely related to the acute exacerbation of COPD that it can even be classified as part of the pathophysiological process of the latter. Thus, it did not seem appropriate to deliberately adjust for this factor. (2) Some COPD patients have hearing loss, which is a confounding factor in noise research [28, 29]. The patient screening in this study excluded patients with deafness or severe hearing impairment, but no professional hearing tests were conducted. However, hearing loss will only lead to an underestimation of the results of this study and will not have a significant impact on this study. (3) Currently, there are very few reports on hyperacusis and noise hypersensitivity in patients with COPD. We speculate that they may also be confounding factors in this study. As no relevant professional tests have been carried out, the exploration of these factors has to be left for future research. (4) Due to the lack of data, factors such as industrial pollution, family situation, and the level of medical insurance were not adjusted for. However, the impacts of these factors seem to be partially represented by the various demographic data, disease risk factors, and treatment data that have already been included in this study. Anyway, the above are the limitations of this study, which require further exploration in future research.

Currently, the biological mechanism underlying the correlation between noise and acute exacerbation of COPD remains unclear. Apart from the sleep disorders mentioned in this study, several other biological processes may be involved. For instance, noise may trigger an inflammatory response, exacerbating the existing inflammatory state in COPD patients [30, 31]. Noise can also promote the excessive production of free radicals, intensifying oxidative stress and synergistically damaging lung tissue in conjunction with inflammation [30, 31]. Moreover, noise can lead to autonomic nervous system dysfunction [32, 33]. Excitation of the sympathetic nerve

increases airway resistance, while parasympathetic nerve imbalance results in an increase in sputum production and airway obstruction. However, the current understanding and evidence are still fraught with limitations, and the specific biological mechanisms require further exploration in future studies.

In addition, due to the limitations of objective conditions, this study did not conduct relevant animal experiments to explore the above-mentioned mechanisms, nor did it include other noise-related factors such as light pollution in this study. These can all serve as important directions for future research.

In conclusion, this study shows that there is a significant correlation between a relatively high level of nighttime sleep noise and a higher risk for COPD patients to experience acute exacerbation. Sleep disorders caused by noise, together with other unproven mechanisms, seem to jointly account for the above-mentioned correlation. These findings help to reveal the environmental mechanisms of COPD, and contribute to the improvement of prevention strategies for this disease.

#### Abbreviations

COPD Chronic obstructive pulmonary disease
GOLD Chronic Obstructive Lung Disease
PSQI Pittsburgh Sleep Quality Index
AIS Athens Insomnia Scale
SDRS Sleep Dysfunction Rating Scale
HADS Hospital Anxiety and Depression Scale
ICU Intensive care unit

## Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12889-025-22887-x.

Supplementary Material 1

#### Acknowledgements

None.

#### Author contributions

(1) Tian Huijun was responsible for the overall study design. (2) Tian Huijun, Cheng Yanwei, Qin Lijie, Zhang Peirong, Li Yun, and Liang Bingwei carried out data collection, processing, and analysis. (3) Tian Huijun, Cheng Yanwei, Qin Lijie, Zhang Peirong, Li Yun, and Liang Bingwei drafted the initial version of the article. (4) Tian Huijun, Cheng Yanwei, Qin Lijie, Zhang Peirong, Li Yun, and Liang Bingwei participated in the review and finalization of the entire text.

#### Funding

None.

#### Data availability

This is a project that is still in progress. Many of the subjects have not yet authorized the disclosure of their data, so the data of this study cannot be made public at present. However, once we have obtained full authorization, we will immediately make all the data publicly available.

## Declarations

#### Ethics approval and consent to participate

This study was approved by the Medical Ethics Committee of Henan Provincial People's Hospital (ID: 2018-57). This study was conducted in accordance with the requirements of the World Medical Association Declaration of Helsinki. All patients gave their consent to participate in this study.

#### Consent for publication

Not Applicable.

#### Competing interests

The authors declare no competing interests.

Received: 27 January 2025 / Accepted: 22 April 2025
Published online: 07 May 2025

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