Effects of Bedroom Environmental Conditions On The Severity of Obstructive Sleep Apnea

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Authorship

SL, NT, SR and NC conceived and designed the study. All authors analyzed the data and drafted the manuscript. All authors interpreted the data, critically revised the draft for important intellectual content, and gave final approval of the manuscript to be published. All authors contributed equally in the preparation of this manuscript.

Conflict of Interest

SL, NT and NC have no potential conflicts of interest. SR received a grant from Merck Sharp and Dohme, speaker fees from Novo Nordisk, Medtronic and Sanofi Aventis, and research equipment support from Resmed Thailand.

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Abstract

Study Objectives: Epidemiological associations have demonstrated the effects of long-term air pollution to obstructive sleep apnea (OSA) through a physiological mechanism linking particulate matter exposure to OSA. This study aimed to determine the relationship between bedroom environmental conditions, OSA severity and sleep quality.

Method: Sixty-three participants were enrolled for an overnight polysomnography and diagnosed with OSA between May to August 2016. Personal characteristics and sleep quality were obtained by a face-to-face interview. Bedroom environments, including data on particulate matter with an aerodynamic diameter less than 10 μm (PM_{10}), temperature, and relative humidity, were collected by personal air sampling and a HOBO® tempt/RH data logger.

Results: Sixty eight percent of the participants experienced poor sleep. An elevation in 1-year mean PM_{10} concentration was significantly associated with an increase in an apnea-hypopnea index (Beta = 1.04, p value = 0.021), and respiratory disturbance index (Beta = 1.07, p value = 0.013). An increase of bedroom temperature during sleep was significantly associated with poorer sleep quality (adjusted odds ratio = 1.46, 95% CI; 1.01, 2.10; p value = 0.044). Associations between PM_{10} concentration and respiratory disturbance index were observed in the dry season (Beta = 0.59, p value = 0.040) but not in the wet season (Beta =0.39, p value = 0.215). PM_{10} was not associated with subjective sleep quality.

Conclusions: Elevation of PM_{10} concentration is significantly associated with increased OSA severity. Our findings suggest that reduction in exposure to particulate matter and suitable bedroom environments may lessen the severity of OSA and promote good sleep.

Keywords: Bedroom environments, particulate matter, obstructive sleep apnea, sleep quality

Brief summary

Current Knowledge/Study Rationale: Particulate matter has been linked to obstructive sleep apnea (OSA), but the influence of indoor environmental conditions, particularly in bedrooms on severity of OSA has not been well studied. The association of indoor environmental conditions with OSA severity is investigated in this study.

Study Impact: An increase in PM_{10} concentrations is associated with more severe OSA as measured by an apnea hypopnea index and respiratory disturbance index. Reduction in exposure to particulate matter may lessen the severity of OSA.

Introduction

Obstructive sleep apnea (OSA) is a sleep disorder characterized by recurrent collapse of the upper airway during sleep, resulting in intermittent hypoxia, sleep disruption, and daytime sleepiness [1]. It is a subtype of sleep disordered breathing (SDB) which is one of the most common sleep disorders that can occur in people of all ages, although it is most commonly observed in middle-aged and elderly populations [2]. Obstructive sleep apnea has been linked to morbidities including hypertension, cardiovascular disease, and diabetes mellitus, as well as an increase in mortality [2-5]. Globally, OSA affects approximately 3 to 7% of male adults and 2 to 5% of female adults [3]. It is a pandemic global public health problem in both developed and developing countries [4]. In Thailand, it troubles approximately 4.8% and 1.9% of men and women, respectively[6], with a higher prevalence reported in poorer urban environments [7].

Air pollution is one of the major environmental risks to health. Exposure to urban outdoor and indoor pollutants may elevate the incidence and severity of OSA [8]. Several studies have demonstrated epidemiological associations between long-term air pollution and OSA through a plausible physiological mechanism linking particulate matter exposure to OSA [9-11]. Particulate matters with an aerodynamic diameter of less than 10 μm (PM_{10}) are coarse particles, which primarily deposit in the upper airways. They can cause irritation or breathing problems [11]. Thus, PM_{10} may play an essential role to OSA through direct mechanical and inflammatory effects on the upper respiratory system [10, 12-15]. Additionally, a former study found that there was an association between long-term black carbon exposure and short sleep duration in men. However, sleep latency was not associated with this exposure [16]. Furthermore, a previous study also indicated a strong relation between an increase in temperature and elevation in the severity of OSA[17]. Studies conducted by Jokic et al. [18], however, did not find a significant association

between humidity and the severity of OSA. The study of Kim and Kum [19] reported the best range of air temperature for good sleep is 24 to 26ºC at 50% relative humidity (RH). However, no previous studies have measured indoor environmental conditions in relation to OSA severity, particularly in bedrooms.

Given that there is currently no available data on the effect of indoor air pollution on OSA severity, it is crucial to gain a better understanding of bedroom environmental conditions and its relationship with OSA severity and sleep quality. Our hypothesis was that alterations in bedroom environmental conditions including PM_{10}, temperature, and humidity would affect severity of OSA, as well as sleep quality in OSA patients. This information may deliver some evidence needed for developing preventive and therapeutic strategies designed to alleviate the burden of these environmental conditions in patients with OSA.

Methods

Study design and participants

This cross-sectional observational study was conducted to monitor changes in bedroom environmental conditions in patients with OSA who resided in the city. Sixty-three patients who were referred for an overnight polysomnography and diagnosed with OSA were enrolled from the Excellence Center for Sleep Disorders at King Chulalongkorn Memorial hospital, Bangkok, Thailand during the period of May to August 2016. The exclusion criteria were patients aged less than 25 years or greater than 75 years, heavy smokers (≥15 cigarettes/day), pregnant women, heart failure patients, or those suffering from chronic respiratory failure. Personal characteristics and sleep quality were obtained from a set of questions carried out by a face-to-face interview. All participants gave their written consent to participate and all research protocols were reviewed

and approved by The Ethics Review Committee for Research Involving Human Research Subjects, Health Science Group, Chulalongkorn University (RECCU No. 053/59), and the Faculty of Medicine Chulalongkorn University Institution Review Board (Med Chula IRB No. 038/59).

Personal information including age (years), gender (male/female), weight (kilograms), height (centimeters), alcohol consumption status (yes/no), smoking status, (yes/no) history of secondhand smoke exposure (yes/no), underlying diseases, and air conditioner usage (yes/no) were obtained by a face-to-face interview and medical records.

Pittsburgh Sleep Quality Index (PSQI)

The Pittsburgh Sleep Quality Index (PSQI) was applied to evaluate subjective sleep quality [20]. It is a subjective standard questionnaire for estimating overall quality of sleep during the previous month. It contains 19 self-rated items that assess various components including sleep latency, sleep duration, habitual sleep efficiency, sleep disturbance, use of sleep medication, daytime dysfunction, and overall sleep quality. Each sleep component yields a score ranging from 0 to 3 [20]. These sleep component scores are combined to yield a total score, namely a "global score" ranging from 0 to 21. The global score indicates whether the participant's sleep quality is good or poor [20]. Based on former literature [20], participants with a score of 5 or lower were classified as good sleepers, and those with a score of 6 or greater were classified as poor sleepers. For sleep quality component subscales, a dichotomous variable of optimal and suboptimal sleep quality was used. According to the original scale, sleep latency was subdivided into: <15 minutes, 16-30 minutes, 31-60 minutes, and >60 minutes. Those in the highest 2 groups of sleep latency (>30 minutes) were defined as experiencing long sleep latency. Additionally, sleep duration was

evaluated using the PSQI questionnaire, which questioned participants on how many hours of actual sleep nightly they had during the past month. In accordance with the original scale, sleep duration was categorized: < 5 hours, 5.1-5.9 hours, 6-6.9 hours, and ≥ 7 hours. Those in the lowest 2 groups of sleep duration (< 6 hours) were classified as having short sleep duration. With regards to sleep efficiency, the original scale was grouped: >85%, 75-84%, 65-74%, and <65%. Those in the groups with 85% or less of sleep efficiency were defined as experiencing poor sleep efficiency.

Polysomnography

Overnight polysomnography is the gold-standard diagnostic test for obstructive sleep apnea. All participants underwent in-laboratory polysomnography at the beginning of the study during the wet season (May to August 2016). The polysomnography system utilized in the study was Compumedics and its related software (Profusion 3) with standard techniques. The stages of sleep were scored in 30-sec intervals following the standard criteria from the AASM manual for the scoring of sleep and associated events [21]. Apnea and hypopnea were defined using oral-nasal thermo-couple excursion and nasal pressure transducer excursion, respectively. Scoring apnea, hypopnea, and respiratory effort-related arousals (RERAs) was performed following the standard criteria from the AASM manual [21]. Apnea was defined when dropping in peak signal excursions by ≥ 90% compared to pre-event baseline for ≥ 10 seconds. Hypopnea was defined when peak signal excursions drop by ≥ 30% of pre-event baseline for ≥ 10 seconds, and there was a ≥ 3% oxygen desaturation compared to pre-event baseline or the event was associated with an arousal (1A criteria). Respiratory effort-related arousals (RERAs) were defined when there was a sequence of breaths lasting ≥10 seconds characterized by increasing respiratory effort or by flattening of flow leading to arousal in which the event did not meet criteria for apnea or

hypopnea. The apnea-hypopnea index was computed as the ratio of the count of all apneas and hypopneas to the total sleep time, expressed as events per hour. Respiratory disturbance index (RDI) was calculated as the ratio of the count of all respiratory events including RERAs to the total sleep time. Sleep efficiency was computed as the proportion of total sleep time over the total recording time by percentage [21]. Parameters of oxygenation, including absolute minimum SpO_2 during sleep and mean oxygen saturation during sleep, were measured by pulse oximetry. Based on the polysomnography results, OSA was diagnosed when AHI $\geq$5, and classified as mild when AHI was 5 to 14.9, moderate when AHI was 15 to 30, and severe when AHI $>$ 30 [22].

Bedroom environmental conditions

The data of bedroom environments including PM_{10}, temperature, and relative humidity were collected in two seasons, namely the wet season from late May to mid-August 2016, and the dry season that began in late December and continued into mid-March 2017. All mentioned factors were collected in the participants' bedrooms for three consecutive nights in each season [23]. After the polysomnography was performed, the first bedroom environmental conditions data was collected within one week for the wet season and the second data collection was conducted in the dry season. All sixty-three participants completed data collection of bedroom environmental conditions for both the wet and the dry seasons. PM_{10} samples were continuously monitored by a SKC personal sampling pump (model: 224-PCXR8) using 2.5-L/min aluminum cyclone loaded with 37 mm, 5.0 μm pore size, polyvinyl chloride filters (SKC Inc. USA) with a support pad. The filters were pre- and post-weighed in a temperature and relative humidity (RH) controlled environment following NIOSH guidelines [24]. The personal air sampling was calibrated, and the start and the end period of data sampling were set. The device was placed in an insulated plastic box (cooler) and sound absorbing materials were inserted to reduce the noise of the device [25].

Therefore, the level of noise from the device was not over an annoyance level (approximately 45 decibels). The methods of preparation, collection, and PM_{10} calculation followed the national institution's occupational safety and health code 0600 manual [24]. Temperature and RH were continually detected and recorded by a HOBO® tempt/RH data logger device (Onset devices, Pocasset, MA). The device was calibrated and set to sample every five minutes [26]. It was subsequently attached on the top of the insulated plastic box. Temperature and RH during sleep time were drawn from the entire sampling period. The average temperature and RH were reported. All environmental measuring devices were delivered to participants' homes with a written instruction. The participants received a follow up phone call by the investigator and were instructed to place the device in their bedroom within 1 meter from the bed at the level of the nose while sleeping at night. Absolute humidity was calculated using the following formula (1); where T is temperature in degree Celsius, rh is relative humidity in %, and e is the natural logarithm [27].

$$\text{Absolute humidity (grams/m}^3\text{)} = \frac{6.112 \times e^{[(17.67 \times T)/(T+243.5)]} \times rh \times 18.02}{(273.15+T) \times 100 \times 0.08314} \quad (1)$$

Statistical analysis

All analyses of this study were performed using SPSS Version 22.0, (IBM SPSS Version22, Chicago, IL,). Personal characteristics, sleep quality parameters, polysomnography sleep parameters, and bedroom environmental conditions of participants were reported using means (± standard deviation) for continuous variables and counts (percentages) for categorical variables. For non-normal distributed variables, medians (interquartile range) were provided. Paired t-tests

were applied to determine whether the mean values of continuous variables were different between two seasons. Multivariable-adjusted logistic regression models were utilized to estimate adjusted odds ratio (AOR) and a 95% confidence interval (CI) for the associations between bedroom environmental conditions and subjective sleep quality parameters. The models were controlled for age, gender, body mass index, alcohol consumption, smoking, secondhand smoke, and AHI. The short sleep latency ($\leq$ 30 mins), longer sleep duration ($\geq$ 6 hours), good sleep efficiency ($\geq$ 85.00%), and good sleep quality (PSQI $\leq$ 5) were used as the reference group in the analyses. To investigate the associations between the bedroom environmental conditions and polysomnography sleep parameters, multiple linear regression models were applied and adjusted for age, gender, body mass index, alcohol consumption, smoking, and secondhand smoke. P values of <0.05 were considered statistically significant.

Results

Of sixty-three participants, the majority were men (73.00%) with a median age of 42.00 years. Their median BMI was 26.20 kg/m^2. Approximately 22.20% reported active alcohol consumption, while 6.30% and 9.50% reported active smoking and current secondhand smoke exposure, respectively. Hypertension was reported by 34.90% of the participants. Most of them (92.10%) used an air conditioner at night. According to the PSQI questionnaire, the medians of sleep latency and sleep duration of participants were 20.00 minutes and 6.00 hours, respectively. The average habitual sleep efficiency was 89.21% (SD =14.16). A majority of participants (68.30%) were classified as having poor sleep quality. Their mean RDI and AHI were 47.66 ± 26.06 events/hours and 45.83 ± 27.21 events/ hours, respectively. More than half of them were classified as having severe obstructive sleep apnea (**Table 1**).

Table 2 shows the summary of bedroom environmental conditions. The mean of PM_{10} concentration in the dry season (19.71 $\mu g/m^3$) was significantly greater than that in the wet season (14.00 $\mu g/m^3$). The average temperature during sleep in the dry season was fairly similar to that of the wet season. The means of both relative humidity during sleep and absolute humidity in the dry season (55.19 %RH and 13.62 g/m^3) were lower than those in the wet season (64.32 %RH and 15.93 g/m^3). However, temperature was not significantly different between the seasons.

The associations between the 1-year mean bedroom environmental conditions, defined as an average of the bedroom environmental conditions data in the wet and the dry season, and subjective sleep quality, as assessed by PSQI, are shown in **Table 3**. In the multivariable-adjusted model, patients whose bedroom had a higher temperature reported poorer sleep quality (adjusted odds ratio (AOR) = 1.46, 95% CI; 1.01, 2.10; p value = 0.044). There were no other significant associations between the 1-year mean bedroom environmental conditions and subjective sleep quality.

As shown in **Table 4**, multiple linear regression models demonstrated that an elevation in the 1-year mean PM_{10} concentration was significantly associated with an increase in AHI (Beta = 1.04, p value = 0.021). The average one-year mean exposure to temperature during sleep (Beta = 0.69, p value = 0.658), relative humidity (Beta = -0.51, p value = 0.145), and absolute humidity (Beta = -0.78, p value = 0.454) were not associated with AHI. Higher 1-year mean PM_{10} concentration was also significantly associated with higher RDI (Beta =1.07, p value = 0.013). The associations between the 1-year mean exposure (temperature, relative humidity and absolute humidity) and RDI were in similar directions as AHI but none were statistically significant.

We further explored if the associations between bedroom environmental conditions and OSA severity differed by season (**Figure 1**). The analysis revealed a trend towards significant association between PM_{10} concentration during the dry season and AHI (Beta = 0.56, 95% CI; -0.02, 1.14, p value = 0.059) but not in the wet season (Beta = 0.40, 95% CI; -0.25, 1.05 p value = 0.22). No differences were found in other associations between other bedroom environmental conditions and AHI in the dry and the wet seasons.

A differentiation of the association between bedroom environmental conditions (the wet and the dry season) and RDI is illustrated in **Figure 2**. It demonstrates an association of PM_{10} concentration in the dry season and RDI (Beta =0.59, 95% CI; 0.03, 1.15, p value = 0.040) but not in the wet season (Beta =0.39, 95% CI; -0.23, 1.01, p value = 0.215). No differences were found in other associations between other bedroom environmental conditions and RDI in the dry and the wet seasons.

Discussion

In this study, we found that PM_{10} concentration had an impact on severity of OSA. Elevation of AHI and RDI were significantly associated with an increase in PM_{10} concentration. The associations were particularly seen in the dry season. We also found a significant association between an increase of bedroom temperature during sleep and poorer sleep quality. Our data are novel as our study is the first to measure participants' bedroom environmental conditions while previous studies have mostly utilized outdoor measurements. In agreement with our results, Zanobetti A, et al used the data from Sleep Heart Health Study (SHHS), a U.S. multicenter cohort study and reported that an interquartile increase in short-term PM_{10} levels was associated with a 12.9% increase in RDI (95% CI; 2.77, 24.09) during summer.[9] In another study, the outdoor PM_{10} data was retrieved using information from U.S. EPA Air Quality System

Technology Transfer Network. One study by Glaser MS [28] also supported the link between outdoor pollution exposure and OSA. This aforementioned study reported that 81% of at-risk World Trade Center (WTC)-exposed rescue/recovery workers were diagnosed with OSA. Severe OSA was associated with WTC exposure that happened on September 11, 2001 with odds ratio of 1.91 (95% CI; 1.15, 3.17). However, not all studies were in support of such associations, as the population-based cohort study using outdoor environmental data conducted by Weinreich G, et al [17] reported no association between PM_{10} and AHI. There are several potential explanations for inconsistent results of the associations between air pollution and OSA. All previous studies, unlike our study, were conducted using outdoor data, which might not reflect the amount of pollution exposure during sleep. Seasonal variations may also play a role as the study by Zanobetti A, et al observed an association between PM_{10} and RDI exclusively during the summer time. [9] During the summer season compared with other seasons, windows are often kept open during the night; therefore, the indoor PM_{10} concentration may be more affected by the outdoor air. It is possible that in-bedroom and outdoor environments might have different impacts on OSA severity, which might explain some differences in the findings of various studies. Though PM_{10} level, temperature, and humidity of bedroom were controlled during the night by an air conditioner, our results reported a significant difference of PM_{10} and humidity level between the wet and the dry season.

There is no clear literature that explicitly reports duration of changes in the brain or the upper airway systems caused by environmental factors linking to OSA. However, environmental factors had been shown to have cumulative adverse effects over long-term exposure and result in the development of chronic diseases.[29]Air pollution, especially particulate matter, potentially affects sleep through the central nervous system and the upper airways [10, 30]. Pollutants may

directly increase nasal or pharyngeal inflammatory responses, which increase an upper airway resistance and reduce airway patency. [31, 32] These mechanisms may alter ventilation and perfusion, resulting in exacerbation of hypoxia associated with OSA. The results of bedroom environmental conditions in our study showed that the mean 1-year PM_{10} concentration was 16.86 $\mu g/m^3$ and was well below the National Ambient Air Quality Standards (NAAQSs) of outdoor air in Thailand (50 $\mu g/m^3$) [33] and the World Health Organization (WHO) (20 $\mu g/m^3$) [34]. However, even such a low level might have a negative impact on the respiratory health of susceptible individuals, since there is no known threshold limit for pollutants to trigger respiratory problems [35] or OSA [9].

The average bedroom PM_{10} concentrations in the dry and the wet season were 19.71 $\mu g/m^3$ and 14.00 $\mu g/m^3$, respectively. Overall, seasonal trends indicated significantly higher PM_{10} concentrations in the dry season compared to the wet season. Srithawirat *et al.* [36] and Jinsart *et al*. [37] reported similar results. Moreover, the data of outdoor PM_{10} concentrations from the Pollution Control Department of Thailand found higher concentrations in the dry season compared with the wet season in Bangkok [38]. Several studies have demonstrated that levels of particulate matter in Asian countries are mainly affected by seasonal variations [39-40]. It appears to be possible that weather precipitation or dispersion may influence the levels of particulate matter. The higher level of PM_{10} observed during the dry season may explain the stronger association of PM_{10} concentration and RDI in the dry season compared with the wet season. This observation proposes that a decrease in the PM_{10} concentration below a certain amount does not affect the AHI or RDI.

Our study did not demonstrate associations of PM_{10} concentration and other subjective sleep parameters. A previous study supported that a decrease in sleep efficiency is related to short-term

elevations in PM_{10} in a cross-sectional study using objective measures of sleep. [9] Elder *et al.* [12] and Wang *et al.* [13] reported that particles moved from the nose up to the olfactory nerve into the striatum frontal cortex and cerebellum. This likely induced brain inflammation [14] and a change in neurotransmitter levels [15, 29], which later influenced sleep quality. Further evidence of particle deposition in the brain was linked to neural inflammation [41-42], which might disrupt sleep-wake cycles. [43-44] However, similar to our findings, a study conducted by Fang SC, et al did not observe an association between long-term black carbon exposures and any sleep parameters in their overall studied participants [16]. The inconsistent results could be due to the heterogeneity of the study population.

Our study did not demonstrate association between humidity level and AHI. Theoretically, drying of upper airway mucosa during the night, through increasing surface tension forces, can contribute to increasing severity of OSA.[45] High ambient humidity might lessen OSA severity by moistening the upper airway mucosa. However, similar to our findings, a previous study did not report an association between humidity level and AHI.[18] Contrary to a positive finding of topical phosphocholinamin, a long-acting tissue lubricating agent, an application to the upper airway mucosa in reducing AHI [46], an addition of liquid to the airway surface may be less important.

Weinreich G, et al demonstrated a significant association between temperature and AHI.[17] Upper airway dilator muscle activity, measured by genioglossus electromyograms, has been shown to be greater during cold air breathing compared to warm air breathing. Therefore, reduction in upper airway muscle activity may result in higher AHI in a warmer environment. [47] However; our study did not demonstrate an association between temperature and AHI. The discrepancy of the results of the studies may come from difference in observed temperature (mean temperature of 13.1 ± 6.2 ^{o}C in Weinreich G, et al study compared to 26.12 ± 1.89 ^{o}C in our study).

Our study found a statistically significant association between poorer sleep quality and elevation in bedroom temperatures. A former study [19] claimed that the best range of air temperature for good sleep using objective measures of sleep quality was 24 to 26^{o}C at 50%RH, and the upper limit for the best sleep quality was 28.1^{o}C at 50%RH. It appears that suitable room temperature and humidity during sleep can play an essential role in influencing good sleep, especially in OSA patients. In order to ensure good sleep quality among varied levels of severity of OSA patients, further study should investigate appropriate room temperature, RH, and absolute humidity.

Several limitations of this study should be taken into consideration. First, the sample size of this study is relatively small; thus, it might not have an adequate power in detecting some significant relationships. Second, we used a cross-sectional study design, which makes it difficult to draw conclusions regarding causation since we cannot be certain if the exposure preceded the outcomes. Future prospective cohort studies in which environmental conditions and stage of OSA development are recorded in the general population would allow us to clarify the understanding of environmental impact on OSA severity. Third, half of our participants had severe OSA, which may have limited the generalizability of our study findings to a broader general population. Besides, patients in this study had polysomnography performed only in the wet season. However, the significant association between PM_{10} concentration and RDI was observed only in the dry season when polysomnography was not performed. Moreover, the patients' weight data that may have an effect on the associations of OSA with other parameters in the dry season were not collected after the initial measurement. Fourth, we used self-reported sleep quality (PSQI) to measure sleep quality scores. Actigraphy recordings would be more appropriate for retrieving accurate sleep quality. In addition, the noise of the personal air sampling device may have disrupted the patient's sleep quality during the data collection at

night. Lastly, we collected only PM_{10} in this study, whereas $PM_{2.5}$ is smaller and more harmful to health than PM_{10}, and it could have a greater effect on OSA severity. More detailed studies could explore this aspect in the future. Additionally, our 1-year mean PM_{10} concentrations are based on an average concentration of three samplings from two seasons (the wet and the dry season) in the participants' bedrooms; hence, they may not accurately indicate the annual average of indoor PM_{10} concentrations. More frequent samplings should be considered to obtain a better representative of annual mean indoor PM_{10} concentrations. Further studies may explore the effect of PM_{10} on OSA severity in OSA patients under a certain bedroom environmental condition (with/without air conditioner usage) to study other factors such as therapeutic conditions that may influence the severity. For example, the effect of PM_{10} on OSA patients who regularly use continuous positive airway pressure (CPAP) devices may be different from those who do not use CPAP.

Conclusions

Our findings suggest that bedroom environmental conditions can be linked to OSA severity. Although environmental factors might not be a direct cause of OSA, they may play a role in exacerbating the severity of OSA. In spite of an increase in public awareness of OSA, a majority of those affected still remain undiagnosed and untreated. Along with treatment, these new findings suggest that reduction in exposure to particulate matter might lessen the severity of OSA.

Abbreviations

AHI Apnea-hypopnea index

OSA	Obstructive sleep apnea
PM_{10}	Particulate matters with an aerodynamic diameter of less than 10 μm
PSQI	Pittsburgh Sleep Quality Index
RDI	Respiratory disturbance index
RH	Relative humidity
CPAP	Continuous positive airway pressure

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Table 1 - Baseline characteristics and polysomnography parameters of obstructive sleep apnea patients (N=63)

Variables	
Age (years) *	42.00 (35-57)
Gender, N (%)	
Male	46 (73.00%)
Female	17 (27.00%)
Body mass index (kg/m^2) *	26.20 (23.56-31.35)
Alcohol consumption, N (%)	14 (22.20%)
Smoking, N (%)	4 (6.30%)
Secondhand smoke, N (%)	6 (9.50%)
Hypertension, N (%)	22 (34.90%)
Air conditioner use, N (%)	58 (92.10%)
Sleep parameters from PSQI	
Sleep latency (min) *	20.00 (10.00-30.00)
Sleep duration (hour) *	6.00 (6.00-7.00)
Habitual sleep efficiency (%) §	89.21 ± 14.16
PSQI score*	7.00 (5.00-9.00)
PSQI > 5, N (%)	43 (68.30%)
Sleep parameters from PSG	
Total sleep time (min) *	315.50 (168.50-377.50)
Total wake time after sleep onset (min) *	27.50 (10.50-58.00)
Sleep onset latency (min) *	9.50 (3.50-18.00)
Sleep efficiency (%)*	86.30 (79.80-93.30)
Sleep architecture (%)	
N1 sleep *	20.50 (13.20-30.30)
N2 sleep §	46.31 ± 12.73
N3 sleep *	16.20 (11.00-23.60)
REM sleep §	14.94 ±8.93
Mean oxygen saturation (%)*	95.00 (92.00-96.00)
Minimum oxygen saturation (%)*	84.00 (74.00-89.00)

Arousal index (arousals/hour) *	37.40 (23.10-49.70)
Respiratory disturbance index, RDI (events/hour) §	47.66 ± 26.06
Apnea-hypopnea index, AHI (events/hour) §	45.83 ± 27.21
Mild OSA, N (%)	9 (14.30)
Moderate OSA, N (%)	9 (14.30)
Severe OSA, N (%)	45 (71.40)

* = *Median (IQR)*

§ = *Mean ± SD*

Table 2 - Comparison of bedroom environmental conditions of 63 obstructive sleep apnea patients between the wet and the dry season

Variables §	**1-year mean**	**Wet season**	**Dry season**	**P value (Wet and Dry)**
PM_{10} ($\mu g/m^3$)	16.86 ± 6.45	14.00 ± 8.95	19.71 ±9.74	0.001***
Temperature during sleep (°C)	26.12 ± 1.89	26.13 ± 2.04	26.11 ± 1.98	0.868
Relative humidity during sleep (%RH)	59.75 ± 9.51	64.32 ± 10.75	55.19 ± 10.44	0.000***
Absolute humidity (g/m^3)	14.78 ± 3.09	15.93 ± 3.54	13.62 ± 3.13	0.000***

***P value ≤ 0.001

§ = *Mean ± SD*

Table 3 - Association of 1-year mean bedroom environmental conditions and subjective sleep parameters

Outcome	PM_{10}		Temperature during sleep		Relative humidity during sleep		Absolute humidity	
	AOR	**95%CI (P value)**	**AOR**	**95%CI (P value)**	**AOR**	**95%CI (P value)**	**AOR**	**95%CI (P value)**
Long sleep latency (> 30 min)	0.99	0.86, 1.13 (p=0.880)	1.40	0.90, 2.18 (p=0.138)	1.10	0.99, 1.23 (p=0.089)	1.31	0.98, 1.76 (p=0.072)
Short sleep duration (< 6 hours)	1.03	0.94, 1.13 (p=0.542)	1.12	0.83, 1.50 (p=0.457)	1.01	0.94, 1.08 (p=0.831)	1.07	0.88, 1.31 (p=0.511)
Poor sleep efficiency (< 85.00%)	0.97	0.88, 1.07 (p=0.568)	1.02	0.75, 1.38 (p=0.909)	1.06	0.98, 1.14 (p=0.163)	1.12	0.90, 1.39 (p=0.306)
Poor sleep quality (PSQI >5)	1.01	0.91, 1.11 (p=0.921)	1.46	1.01, 2.10 (p=0.044*)	1.00	0.92, 1.08 (p=0.969)	1.18	0.91, 1.53 (p=0.218)

Note: all models were adjusted for age, gender, body mass index, alcohol consumption, smoking, secondhand smoke, and AHI.

* P value < 0.05

Table 4 - Association of bedroom environmental conditions and apnea-hypopnea index (AHI) and respiratory disturbance index (RDI) of obstructive sleep apnea patients

Outcome	1-year mean exposure	Beta coefficients	95%CI	P value
AHI	PM_{10}	1.04	0.16, 1.91	0.021*
	Temperature during sleep	0.69	-2.42, 3.81	0.658
	Relative humidity	-0.51	-1.20, 0.18	0.145
	Absolute humidity	-0.78	-2.86, 1.30	0.454
RDI	PM_{10}	1.07	0.24, 1.91	0.013*
	Temperature during sleep	0.92	-2.08, 3.93	0.540
	Relative humidity	-0.47	-1.14, 0.19	0.161
	Absolute humidity	-0.63	-2.63, 1.38	0.533

Note: all models were adjusted for age, gender, body mass index, alcohol consumption, smoking, and secondhand smoke.

* P value < 0.05

Figure 1. Beta coefficients with 95% CI for the association of bedroom environmental conditions (stratified by seasons) and AHI.

Figure 2. Beta coefficients with 95% CI for the association of bedroom environmental conditions (stratified by seasons) and RDI.