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CLINICAL REVIEW

The association between traffic-related air pollution and obstructive sleep apnea: A systematic review

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SUMMARY

Recent evidence suggests that air pollution exposure may be a contributing risk factor for obstructive sleep apnea (OSA), however, current evidence is conflicting. This systematic review aims to determine the association between air pollution and OSA in the general population, and examine for potential effect modification by seasonality, temperature and humidity. Five full-text articles were included in the review out of 905 articles found by systematically searching PubMed, Embase and Scopus databases. The included studies were limited to OSA in adults that were conducted in middle to high-income countries. The results highlight heterogeneity in the diagnostic criteria for OSA and method used to assess air pollution exposure. There is some evidence to support a relationship between air pollution exposure and OSA. However, the duration of exposure to different air pollutants including particulate matter ($PM_{2.5}$ and PM_{10}) and nitric oxides (NO_2) in relation to OSA varied across different seasons, temperatures, and countries. This variability of the pollutants across studies warrants a more robust study design using time-series analysis with multiple follow-ups to strengthen the evidence for this relationship before considering its implications.

Introduction

Recent epidemiological data suggest that obstructive sleep apnea (OSA) affects almost one billion people globally [1]. The condition is characterised by the intermittent collapse (apnea) or partial blockage (hypopnea) of the upper airways [2]. Repeated episodes of breathing disturbances can lead to fragmented and non-restorative sleep. The consequences of poor sleep include excessive day time sleepiness and impaired concentration. Also, OSA has been recognised as an independent risk factor for health complications such as cardiovascular diseases, depression, and metabolic disorders [3].

There is ample evidence linking several risk factors with the etiology of OSA, including predisposing craniofacial abnormalities, male gender, obesity, and advanced age [4–6]. Nevertheless, considerable variability in the prevalence of OSA is unexplained and may also be related to lifestyle and environmental factors such as alcohol consumption, exposure to tobacco smoke [7], and potentially, exposure to air pollution.

Air pollution is a well-known environmental factor that exacerbates or causes respiratory diseases [8]. However, the relationship with OSA has not been established. Since the upper respiratory tract, including the nose and the upper airway, is the first entry point for air contaminants, it begs the question of whether air

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Abbreviations

AASM	American Academy of Sleep Medicine
AHI	Apnea-hypopnea index
BMI	Body mass index
COPD	Chronic obstructive pulmonary disease
IQR	Interquartile range
MeSH	Medical subject heading
NO_x	Oxides of nitrate
O_3	Ozone
ODI	Oxygen desaturation index
OSA	Obstructive sleep apnea
PM_x	Particulate matter (PM_1 for < 1 micro-meter, $PM_{2.5}$ for < 2.5 micro-meter, PM_{10} for < 10 micro-meter)
PRISMA	Preferred reporting items for systematic reviews and meta-analysis
PROSPERO	International prospective register of systematic reviews
RDI	Respiratory disturbance index
ROBINS-E	Risk of bias in non-randomized studies - of exposures
SO_x	Oxides of sulphate
US	United States
WHO	World Health Organisation

pollution exposure is associated with OSA. Exposure to high levels of air pollution has been proposed to alter the mucociliary defense mechanism of the nasal passages, which normally act to trap and eliminate inhaled materials. A study has shown that when young adults move from a low-polluted setting (rural area) to a high-polluted setting, they exhibit inflammatory changes in their nasal epithelia [9]. Furthermore, upper airway patency is linked to the disruption of the sinonasal epithelial cell barrier [10]. This disruption leads to a persistent inflammatory process through the activation of pro-inflammatory molecules, which further degrades the barrier integrity and enhances inflammation. Nasal inflammation with increased nasal resistance has been implicated in the pathogenesis of OSA [11]. However, the relationship between upper airway inflammation and OSA could also be bidirectional. Recently, it has been postulated that OSA promotes a persistent low-intensity inflammatory state [12], in which oxygen desaturation and altered lung ventilation can induce inflammation.

Over 90 percent of the world's population lives in places that exceed the clean air threshold [8]. This is likely to worsen because of accelerated urbanization and climate change. Air pollution mainly derives from vehicle exhaust and other commercial and industrial processes. Air pollution is a complex mixture of microscopic particles often indexed as particulate matter (PM) which includes ultrafine (<1 μm (PM_1)), fine (<2.5 μm ($PM_{2.5}$)), coarse (<10 μm (PM_{10})), black carbon, and other gaseous pollutants: Oxides of nitrogen (NO_x), oxides of sulfur (SO_x), and ozone (O_3).

Although there is increasing awareness of the detrimental effects of air pollution on respiratory health, limited studies have investigated the relationship between air pollution and OSA. Studies in this area also generated conflicting results for different seasons of the year [13–15], which is plausible given that fluctuations in temperature and humidity levels can influence the level of air pollution. Air pollution levels can vary between developing and developed countries, but it is unclear if this will have differential impacts on the risk to OSA. A recently published systematic review suggested a positive correlation between high levels of air pollution and greater risks for sleep-disordered breathing (including OSA) in children [16]. However, this review primarily included studies that measured sleep-disordered breathing subjectively by using questionnaires, which is not as accurate as an objective polysomnography test and can be subjected to recall bias. Furthermore, seasonality, temperature, and humidity as potential confounders or effect modifiers were not taken into consideration. Currently, no published data are available based on systematically synthesizing information on the relationship between air pollution and OSA in adults. This is an important knowledge gap, given that the burden of OSA is higher in adults.

This systematic review aimed to address these knowledge gaps by synthesizing the available evidence on the association between air pollution and OSA in the general population and to further consider the potential modifying effects of meteorological factors.

Methods

This systematic review was conducted following the recommendations set by the Preferred reporting items for systematic reviews and meta-analysis (PRISMA) statement [17]. This review was prospectively registered with the international prospective register of systematic reviews (PROSPERO) with the registration code CRD42019136075.

Literature search

A literature search was performed using the PubMed, Embase and Scopus databases with a predefined search strategy that combines medical subject heading (MeSH) terms and text terms related to OSA and air pollution (see Supplementary Table S1 for details). Searches were adapted to each database, using appropriate database-specific filters. We included original peer-reviewed studies published in English from date of inception until September 25th, 2019. The air pollution markers considered were particulate matter (PM_1, $PM_{2.5}$, PM_{10}), black carbon, and other gaseous pollutants such as nitrogen oxides (NO_x), sulfur oxides (SO_x) and ozone (O_3). Only studies that measured OSA objectively (type 1 to type 4 sleep studies) were considered for this review.

Eligibility criteria

This systematic review included only case–control, cohort and cross-sectional studies that examined the association between air pollution and OSA, reported at any age from the general population. The outcome of interest was OSA and it must have been objectively measured using a polysomnography test, conducted either in the laboratory or at home. Indices used to report OSA include AHI – the number of apnea and hypopnea events per hour of sleep, oxygen desaturation index (ODI) – the average number of dips in oxygen saturation per hour of sleep, and the respiratory disturbance index (RDI) – which sometimes has been used interchangeably with the AHI, but more specifically may include the AHI plus additional respiratory-related sleep disturbances.

Our exposure of interest was air pollution exposure, which included: PM_1, $PM_{2.5}$, PM_{10}, black carbon, NO_x, SO_x, ozone (O_3) (data can be obtained from a standard air-quality monitoring station or inside the participants' residence) and distance of residential addresses from major roads/highways as a proxy for air pollution. The exclusion criteria were: 1) non-human studies, 2) studies not published in English, 3) articles published as reviews, commentaries, conference abstracts or editorials, 4) studies based purely on

questionnaires without using polysomnographic-based measurements for OSA.

Study methodology assessment

Study quality was assessed using the tool, the risk of bias in non-randomized studies - of exposures (ROBINS-E) [18]. Seven components from each study were assessed including: bias due to confounding, selection of participants, classification of exposure, a departure from intended exposure, missing data, measurement of outcomes and selection of reported outcome. Each component was rated as low/moderate/serious or critical (see Supplementary Table S2). The risk of bias assessment was performed by a reviewer (DC) and was independently checked by another reviewer. Any disagreement was resolved through a consensus discussion, with involvement of a third review author where necessary. Studies rated as critical were not discarded entirely but were considered and acknowledged in the synthesis.

Data extraction

Duplicates of identified articles were removed. Titles and abstracts of all articles were screened by a reviewer (DC) to identify studies that met the inclusion and exclusion criteria. Full papers of relevant studies were then screened. Relevant data extracted from each study by the reviewer (DC) that included: study characteristics (first author, year, country, recruitment, design, sample size), participant characteristics (gender, age, BMI, smoking status, ethnicity, socioeconomic status), ascertainment of AHI (lab-based, home-based or limited-channel polysomnography test), outcome measurements (AHI, RDI or ODI), assessment of air pollutants ($PM_{2.5}$, PM_{10}, NO_x, CO, SO_x, O_3-data from a standard air-quality monitoring stations or air pollution inside the participants' residence), any recordings of seasons (spring, summer, winter, autumn, wet/dry), the relevant effect estimates for the outcome(s) and 95% confidence intervals. All steps were double-checked by another reviewer, and any disagreement or inconsistency between the reviewers was resolved through a consensus discussion or further reviewed by another reviewer.

Data synthesis

Meta-analysis of collected data was not possible due to heterogeneity of the outcome definition from each study (e.g., outcome reported as the change in the odds of OSA, percentage change in OSA or a change in the beta coefficient of OSA) and different time windows over which pollutants were measured (e.g., Daily mean, weekly or yearly mean exposure), with no two studies using the same measurements.

Results

Literature search

A total of 905 records were identified from the literature search. After excluding 44 duplicates, 861 records were retained to screen the title and abstract. Of these, 23 articles were eligible for full text screening. A total of 18 full text articles were removed with reasons related to critical risk of bias (two articles), an ecological study (one article), duplicates of included studies (two articles) or articles that measured the outcome based purely on questionnaires (13 articles). No further studies were found from the reference lists of articles selected for full text screening. Hence, five full text articles were included in the final review. Fig. 1 shows the PRISMA flowchart of the selected studies.

Bias due to confounding

The five studies included in the review [13–15,19,20] span a low to serious risk of bias, mainly due to the inadequacy of adjusting for relevant confounders (domain 1) and selection of participants into the studies (domain 2) (Supplementary Table S2). Although types of heating and types of cooking are important household factors that contribute to the emitted particulate matter indoor, these factors were not examined in any of the studies included. Two studies [21,22] were not critically appraised, due to the risk of bias (domain 1 of ROBINS-E) rated as critical (Supplementary Table S2). These two studies performed a correlation analysis for air pollution and OSA without adjusting for additional variables relating to the participants' socio demographic factors.

Characteristics of included studies

Our review included studies from high-income countries (United states (US) and Germany) and middle-income countries (Taiwan and Thailand) (Table 1). The five included studies were cross-sectional studies, two of which were from pre-existing cohort studies [13,20]. The sample sizes of these studies ranged from 63 to 4312 participants, with an average age from 40 to 70 y. The included studies had approximately equal proportions of male to female participants, except for the two studies from Asia [15,19], which had higher numbers of female participants. There was variability in the measured mean concentrations of air pollutants compared to the World Health Organization (WHO) air quality standards, depending on the geographical location of the conducted study and seasonal variation. On average, short-term and long-term exposures to $PM_{2.5}$ and PM_{10} were below the WHO air quality standards (Table 2), except for the Taiwan report [15]. The daily exposures to NO_2 from the Taiwan study [15] and O_3 in the study from Germany [14] were reported to be higher than the WHO air quality standards.

Variability in the exposures

Substantial heterogeneity in the exposure measurement was observed in the studies (Table 1). Air pollution data were collected at the participants' homes (indoor air monitoring) [19] or were collected from a fixed air monitoring station in the corresponding metropolitan area of the recruited participant [13–15,20]. Air pollution levels were ascertained for each participant using different techniques; by using a spatio-temporal hierarchical model [20], by averaging from several air monitoring stations to represent the city ambient pollution level [14], by matching the participant's address to the nearest air monitoring station [15], or from a single central monitoring station in the metropolitan area [13].

Coarse particles (PM_{10}) were the most frequently reported traffic-related pollutant marker, measured in four studies. Fine particles ($PM_{2.5}$) were measured in two studies, oxides of nitrogen (reported as NO_2) were measured in two studies, oxides of sulphur (reported as SO_2) were measured in one study and ozone (O_3) was measured in two studies (Table 2). The distance of residential addresses from major roads or highways as a proxy for air pollution exposure was not measured. The selected studies. Three studies reported exposure to air pollutants as an interquartile range increase in mean exposure. Two studies reported the exposure as an incremental increase in the mean exposure or as an incremental increase by a specified unit (Table 2).

Variability in the outcome assessment

OSA was objectively measured with a single night polysomnography test in four of the included studies and with limited

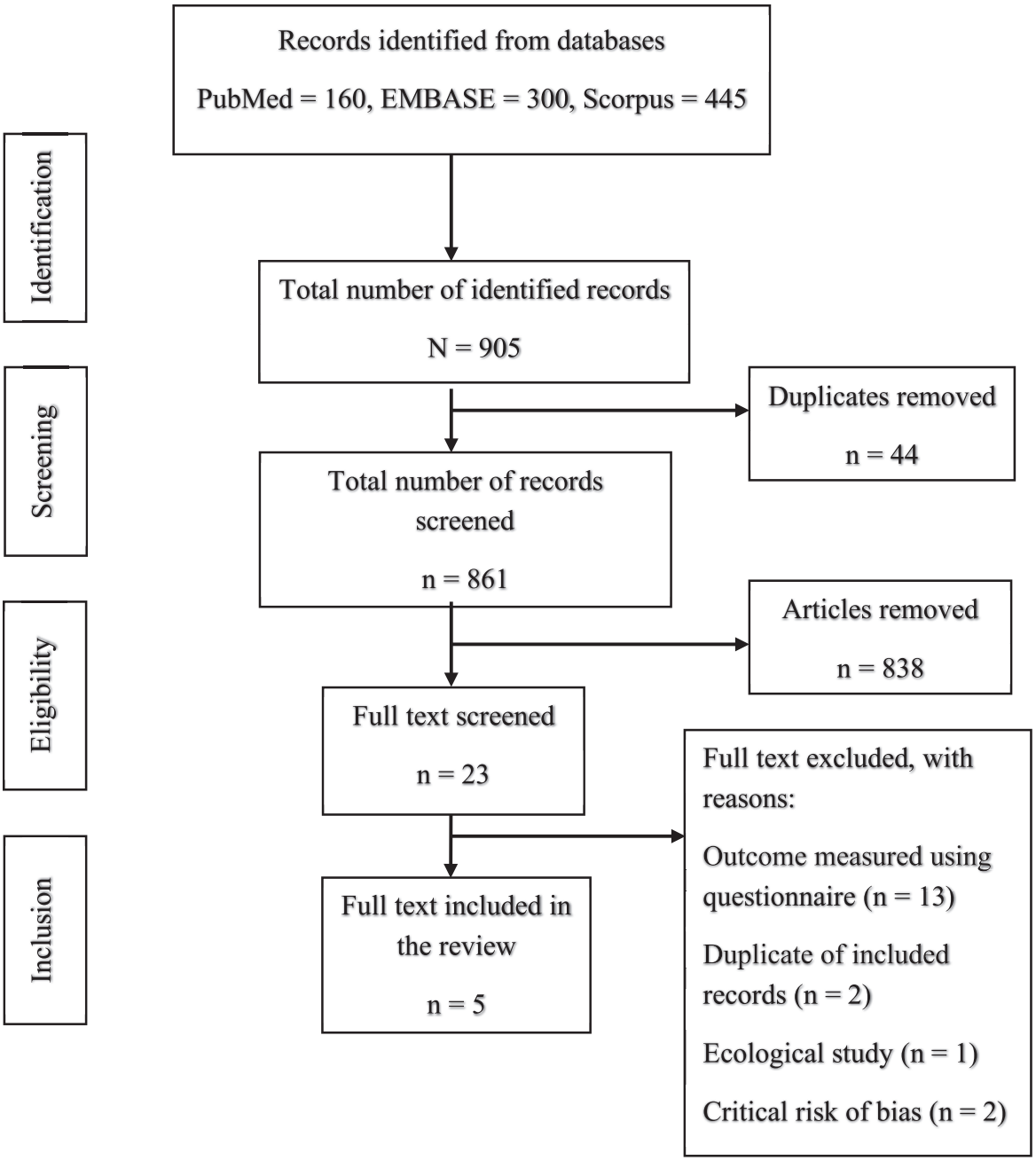

Fig. 1. The PRISMA flowchart of selection of studies for the review. "Records" refer to full journal articles of original research.

channel respiratory assessment in one [14]. Lab-based sleep studies were performed in two studies, home-based sleep studies were performed in two and home-based limited channel respiratory study (ApneaLink™) in one (Table 1). Depending on the type of sleep study conducted, several indices were used to report OSA, including AHI, ODI or RDI. AHI was the most frequent clinical measurement of OSA and was reported in four studies [14,15,19,20], two of which also reported either ODI recordings or RDI recordings [15,19]. OSA was measured as RDI in one study [13]. The American academy of sleep medicine (AASM) was the most common criteria used to define the indices related to breathing disturbance events. Two studies had measured the indices related to breathing disturbance events without referring to specific criteria [13,14]. There was also heterogeneity in the cut-off thresholds for air flow reduction in both apnea and hypopnea events. Differences were also seen in the classification of hypopnea events when combined with a 3% or 4% reduction in oxygen desaturation [20]. Most importantly, no two studies had the same combination of reported indices for OSA, or with similar cut-off thresholds for air flow reduction or oxygen desaturation (Table 1).

Cross-sectional analyses on the association between air pollution and OSA

Although the two studies from the US [13,20] used different methodological strategies to assess air pollution levels and evaluate OSA, the studies reported a correlation between higher exposure to certain air pollutants and greater severity in OSA.

Table 1
Detailed summary table of the general characteristics of the included studies.

Author & year of study	Location & population enrolment	Sample size & age at outcome	Outcome assessment	Exposure assessment	Time window between exposure and outcome	Modification by seasonality and/or Confounding by temperature/humidity
Zanobetti, A. et al., (2010) [13]	Location: US Population: Participants were enrolled from an existing multicenter Sleep Heart Health Study (SHHS) cohort.	N = 3030 Female = 53% Male = 47% Age = 63, IQR (55, 70)	OSA defined as RDI with ≥3% oxygen desaturation. Apnea = reduce airflow by >75% for ≥10 s. Hypopnea = airflow by 30% for ≥10 s. Home-based polysomnography test for 1 night.	PM_{10} obtained from fixed air-monitoring station in each urban area.	365-d moving average of PM_{10}. Including the same day (lag 0) up to lag 364.	Seasonality and temperature.
Weinreich, G. et al., (2015) [14]	Location: Germany Population: Participants were randomly selected from mandatory city registries in Essen, Bochum and Mülheim.	N = 1773 Female: 50% Male: 50% Age = 63.8 (±7.5)	OSA defined as AHI. Apnea = reduce airflow by >80% for ≥10 s. Hypopnea = reduce airflow by 50–80% for ≥10. Home-based limited channel polysomnography (ApneaLinkTM) for 1 night.	PM_{10} and O_3 citywide background concentrations were averaged from 3 different monitoring stations.	Lag 0, 8 h means for PM_{10} and O_3 on the day of sleep evaluation.	Seasonality, relative humidity, temperature.
Shen, Y. L. et al., (2018) [15]	Location: Taiwan Population: Participants were enrolled from sleep centre of Taipei Medical University Hospital.	N = 4312 Female: 60% Male: 40% Age = 45.8 (±13.2)	OSA defined as AHI or ODI (AASM criteria). ODI = at least 4% decrease in oxygen saturation for ≥10 s 10 s. Lab-based polysomnography test for 1 night.	PM_{10}, $PM_{2.5}$, NO_2, O_3 and SO_2 data obtained from 17 different monitoring stations in New Taipei City and Keelung City. Each participant's residential address was matched to the nearest air monitoring station.	daily, weekly and yearly means.	Seasonality, temperature and relative humidity.
Lappharat, S. et al., (2018) [19]	Location: Bangkok, Thailand Population: Participants were enrolled from the Excellence Center for Sleep Disorders at King Chulalongkorn Memorial Hospital.	N = 63 Female = 73% Male = 27% Age = 42, IQR (35–57)	OSA defined as AHI or RDI (AASM criteria). Apnea = reduce airflow by at least 90% for ≥10 s. Hypopnea = reduce airflow by at least 30% for ≥10 s with 3% (decrease in O_2 saturation). RDI = ratio of the count of all respiratory events to the total sleep time. Lab-based polysomnography test for 1 night.	PM_{10} sampled from bedroom. Collected within 1 wk after the polysomnography test. 1 collection (1 wk) for dry season and 1 collection (1 wk) for wet season.	1 y mean	NA
Billings, M.E. et al., (2019) [20]	Location: US Population: Participants were enrolled from an existing cohort, the Multi- Ethnic Study of Atherosclerosis (MESA) cohort.	N = 1974 Female = 54% Male = 46% Age = 68 (±9)	OSA defined as AHI ≥15 (moderate to severe OSA- AASM criteria) with ≥4% oxygen desaturation. Home-based polysomnography test for 1 night.	NO_2 and $PM_{2.5}$ exposures were estimated from community and Air Quality System monitoring sites in each metropolitan area. Used hierarchical spatiotemporal modeling	Same day (lag 0), 1 d prior to sleep evaluation (lag 1), 1 and 5 y before sleep evaluation.	NA

Abbreviations: American academy of sleep medicine (AASM), Obstructive sleep apnea (OSA), Apnea-hypopnea index (AHI), Respiratory disturbance index (RDI), Oxygen desaturation index (ODI), Particulate matter < 10 micro-meter (PM_{10}), Particulate matter < 2.5 micro-meter ($PM_{2.5}$), Ozone (O_3), Oxides of nitrogen (NO_2), Oxides of sulphur (SO_2), Not applicable (NA). 'lag 0' refers to the level of air pollution measured on the same day as sleep evaluation, 'lag 1' refers to the day before sleep evaluation.

Billings and colleagues [20] showed that 10 ppb increase in the mean annual increase in NO_2 exposure (at 1 and 5 y before sleep evaluation) was associated with a 40% (95% CI: 3%, 87%) and 41% (95% CI: 4%, 92%) increase in the odds of having moderate to severe OSA (AHI ≥15- this was defined using the more stringent criterion of ≥4% for ODI) respectively. There was a trend towards greater severity of OSA with respect to longer duration of exposure to $PM_{2.5}$, however, this was not statistically significant. In the other study from the US, Zanobetti and colleagues [13] reported conflicting results for the duration of exposure to PM_{10} and greater severity of OSA, despite that measured daily and annual concentrations of PM_{10} being similar (22.2 μg/m^3 - 42.9 μg/m^3 and 22.2 μg/m^3- 43.6 μg/m^3 respectively). An interquartile range (IQR) increase of 14.5 μg/m^3 in the daily mean exposure to PM_{10} was associated with a 13% increase in RDI (95% CI: 2.8%, 24%). This was only observed in the summer. Overall, an IQR increase in the annual mean exposure to PM_{10} was not associated with the change in RDI.

Table 2
Summary of the association of air pollution exposure and obstructive sleep apnea.

Author & year of study	Results					Adjusting for confounders
Zanobetti, A. et al., (2010) [13]	% change in RDI (95%CI)					Seasonality, daily mean temperature, age, BMI, sex, education, an age by sex interaction, smoking status, daily number of glasses of coffee, tea, and soda, and number of glasses of wine and beer 4 h before going to sleep, annual PM_{10} averages
	Pollutant(s)		Adjusted	Stratified (Summer)		
	PM_{10}, daily per IQR (14.5 μg/m^3)		4.88 (−5.77, 16.73)	NA		
	PM_{10}, annual per IQR (2.1 μg/m^3)		−0.51 (−5.22, 4.44)	12.9 (2.77, 24.09)		
	Note: Across the 7 regions, reported annual mean of PM_{10} ranges from 22.2 to 42.9 μg/m^3, reported daily mean of PM_{10} ranges from 22.2 to 43.6 μg/m^3					
Weinreich, G. et al., (2015) [14]	% change in AHI (95%CI)					Relative humidity, temperature, age, sex, BMI, alcohol consumption, smoking habits, physical activity, education, hypertension, coronary heart disease, congestive heart failure, stroke, diabetes
	daily mean	Summer (n = 423)	Spring (n = 484)	Autumn (n = 506)	Winter (n = 360)	
	O_3, per IQR (39.5 μg/m^3)	12.3 (−4.6, 32.2)	8.3 (−7.9, 27.3)	6.0 (−10.6, 25.5)	12.0 (−12.8, 43.9)	
	PM_{10}, per IQR (14 μg/m^3)	−0.7 (−5.1, 3.9)				
	Note: Reported PM_{10} (24 h mean) = 27.0 μg/m^3 ±14.5, O_3 (24 h mean) = 62.2 μg/m^3 ±33.4					
Shen, Y. L. et al., (2018) [15]		% change in AHI (95%CI)		% change in ODI (95%CI)		Sex, age, BMI, smoking status, temperature and relative humidity
	1-d mean					
	PM_{10}, per IQR (22.6 μg/m^3)	0.9 (−3.6, 5.4)		0.5 (−1.0, 2.0)		
	$PM_{2.5}$, per IQR (11.9 μg/m^3)	2.7 (1.1, 4.3)		1.3 (−2.4, 5.0)		
	NO_2, per IQR (6.8 ppb)	2.0 (0.7, 3.3)		−3.8 (−8.4, 0.8)		
	O_3, per IQR (10.2 ppb)	−2.4 (−4.9, 0.1)		0.4 (−1.8, 2.6)		
	1-wk mean					
	PM_{10}, per IQR (20.2 μg/m^3)	1.1 (−1.6, 3.8)		1.5 (−1.5, 4.5)		
	$PM_{2.5}$, per IQR (10.6 μg/m^3)	1.7 (0.3, 3.1)		2.1 (1.0, 3.2)		
	NO_2, per IQR (5.7 ppb)	0.4 (−2.8, 3.6)		−2.9 (−7.6, 1.8)		
	O_3, per IQR (9.4 ppb)	2.1 (−1.0, 5.2)		1.7 (−2.2, 5.6)		
	1-y mean					
	PM_{10}, per IQR (7.3 μg/m^3)	2.2 (−3.1, 7.5)		−1.0 (−4.7, 2.7)		
	$PM_{2.5}$, per IQR (3.4 μg/m^3)	4.7 (3.4, 5.9)		2.5 (1.4, 3.6)		
	NO_2, per IQR (2.7 ppb)	3.6 (1.0, 6.2)		1.7 (−0.8, 4.2)		
	O_3, per IQR (1.3 ppb)	−1.2 (−5.8, 3.4)		2.3 (−7.0, 11.6)		
	$PM_{2.5}$ in different seasons					
	Spring, $PM_{2.5}$ (IQR = 13.5 μg/m^3)	5.5 (3.5, 7.5)		1.4 (−0.1, 2.9)		
	Summer, $PM_{2.5}$ (IQR = 7.3 μg/m^3)	1.4 (−2.6, 5.4)		1.0 (−3.0, 5.0)		
	Autumn, $PM_{2.5}$ (IQR = 8.4 μg/m^3)	0.7 (−2.0, 3.4)		0.5 (−2.0, 3.0)		
	Winter, $PM_{2.5}$ (IQR = 14.5 μg/m^3)	6.1 (5.5, 6.8)		2.9 (1.2, 4.6)		
	Note: Reported daily mean for; PM_{10} μg/m^3 = 61.5 ± 28.9; $PM_{2.5}$ μg/m^3 = 37.4 ± 14.2; NO_2 ppb = 23.8 ± 10.2; O_3 ppb = 22.6 ± 7.4					
Lappharat, S. et al., (2018) [19]		Beta coefficient in AHI (95%CI)		Beta coefficient in RDI (95%CI)		Age, sex, BMI, alcohol consumption, smoking, and secondhand smoke
	1-y mean (16.86 ± 6.45) μg/m^3					
	PM_{10}, per 1-y mean	1.04 (0.16−1.91)		1.07 (0.24−1.91)		
	Note: The National Ambient Air Quality Standards of outdoor air in Thailand = 50 μg/m^3					
Billings, M.E. et al., (2019) [20]	Pollutant(s)		OR of AHI>15 (95%CI)			Age, sex, BMI, diabetes, hypertension, race/ethnicity, household income, smoking status, socioeconomic status, site
	$PM_{2.5}$ per 5 μg/m^3, same day		0.99 (0.97−1.02)			
	$PM_{2.5}$ per 5 μg/m^3, day prior		1.01 (0.99−1.03)			
	1-y $PM_{2.5}$ per 5 μg/m^3		1.60 (0.98, 2.62)			
	5-y $PM_{2.5}$ per 5 μg/m^3		1.31 (0.78, 2.20)			
	1-y NO_2, per 10 ppb		1.39 (1.03, 1.87)			
	5-y NO_2, per 10 ppb		1.41 (1.04, 1.92)			
	Note: Reported 1-y $PM_{2.5}$ (median = 11.0 μg/m^3, IQR = 10.3−12.0), 5-y $PM_{2.5}$ (median = 12.3 μg/m^3, IQR = 11.5−13.5), 1-y NO_2 (median = 13.0 ppb, IQR = 9.0−21.4), 5-y NO_2 (14.8 ppb, IQR = 10.0−23.7)					

Abbreviations: Body mass index (BMI), Apnea hypopnea index (AHI), Oxygen desaturation index (ODI), Respiratory disturbance index (RDI), Odds ratio (OR), Interquartile range (IQR), Particulate matter < 10 micro-meter (PM_{10}), Particulate matter < 2.5 micro-meter ($PM_{2.5}$), Ozone (O_3), Oxides of nitrogen (NO_2), Oxides of sulphur (SO_2). World health organization (WHO) air quality guidelines: $PM_{2.5}$ (annual) = 10 μg/m^3, PM_{10} (annual) = 20 μg/m^3, NO_2 (annual) = 50 μg/m^3 or 26.15 ppb, $PM_{2.5}$ (24-h mean) = 25 μg/m^3, PM_{10} (24-h mean) = 50 μg/m^3, NO_2 (24-h mean) = 40 μg/m^3 or 20.9 ppb, O_3 (24-h mean) = 53 μg/m^3 or 26.5 ppb.

The studies from Asia [15,19] recruited predominantly female participants compared to other studies. The study from Thailand [19] also used indoor air monitoring rather than using outdoor air pollution measurements. In the study from Taiwan [15], the authors used two different indices (AHI and ODI) to report OSA. Daily, weekly and annual exposures to $PM_{2.5}$ were associated with an increase in AHI (p-value <0.01). Weekly and annual exposures to $PM_{2.5}$ were associated with ODI (p-value <0.01). Daily and annual exposures to NO_2 were associated with increased AHI (p-value <0.01). Exposure PM_{10} and O_3 were not associated with changes in reported ODI or AHI at any time point. When stratified by seasons, exposure to $PM_{2.5}$ was correlated with greater change in AHI (p-

value <0.01) in spring and winter, and correlated with greater change in ODI (p-value <0.01) only in winter. For the study conducted in Thailand [19], the authors showed that an increase in mean annual exposure to PM_{10} (per 16.86 $\mu g/m^3$) was associated with a 1.04 unit (95% CI = 0.16, 1.91) and 1.07 unit (95% CI = 0.24, 1.91) increase in the measured AHI and RDI respectively.

The study from Germany [14] showed that an IQR increase in the daily mean exposure to PM_{10} (per 14 $\mu g/m^3$) or O_3 (per 39.5 $\mu g/m^3$) were not associated with the percentage change in AHI in all four seasons.

Discussion

To our knowledge, this is the first systematic review that provides an in-depth evaluation of the association between air pollution and objectively measured OSA in adults. Overall, there is a suggestion of an association between air pollution exposure and OSA, although the evidence is inconsistent for the duration of exposure to NO_2, $PM_{2.5}$ or PM_{10} and OSA. This inconsistency may be related to differences in the methodological strategies used to assess the exposure and outcome, geographical variation, and different time windows over which pollutants were measured. Notably, an interaction of air pollutant(s) and different seasons of the year, depending on the country, is evident from this review. Hence, the association between air pollution and OSA may be underestimated by 'diluted' in the studies that did not stratify the estimates into different seasons, or in studies that only measured OSA in a specific season. The duration of exposure to O_3 was not significantly correlated with OSA [14,15].

Seasonal composition of air pollution

The variability in the association between short-term exposure to $PM_{2.5}$ and PM_{10} and OSA may partly be explained by the composition of air pollutants in specific seasons of the year, depending on the geographical location. In the study from the US [13], The proportion of PM_{10} was higher in the summer compared to winter, which might in part explain the positive relationship with OSA observed in the summer. Studies conducted in Asian countries may be subjected to 'Asia dust storm'-a meteorological phenomenon that affects much of East Asia and can reach almost the entire Northern Pacific region [23]. Asia dust storm has a high accumulation of fine particulate matter ($PM_{2.5}$) and predominantly occurs in the spring season [24,25]. This might be a plausible explanation for the positive correlation between $PM_{2.5}$ with OSA in spring in the study conducted in Taiwan [15]. Another possible explanation is biomass burning-a common practice in low and middle-income countries that produces high levels of particulate matter and other dangerous gaseous components. This is particularly common in multiple regions of Asia and has been shown to have a distinct seasonal cycle that peaks in spring and winter [26]. Likely, the association of short-term $PM_{2.5}$ exposure and OSA in spring and winter from the study in Taiwan [15] is the result of strong seasonal correlation in elevated $PM_{2.5}$ in biomass burning. In other parts of the world, elevated levels of PM_x, NO_x, SO_x have been observed in the winter months, when the humidity level is high, and the temperature is low. The high temperature in the summer months, on the contrary, is a known catalyst for elevated levels of ozone (O_3) [27,28]. However, the included studies did not support the association between O_3 and OSA even when stratified by seasons [14,15].

Mechanistic factors other than variation in pollution levels need to be considered in the seasonal variability of air pollution and OSA risk. The interaction between the upper airway irritation and allergic reactions are not mutually exclusive, and in fact, can reinforce the intensity of one another and result in the thickening of the mucosal lining [29]. Exposure to air pollutants can trigger irritant receptors located on airway afferent nerves [30], which can intensify allergic reactions [31,32]. The opposite is also true, in which pre-existing nasal allergies increase nerve excitability in the airway and can intensify responses to nasal irritants [31,32]. This is an important consideration since upper airway conditions such as allergic rhino-sinusitis also exhibit a seasonal trend (but can be perennial for some individuals) when pollen counts are high and may confound the association between air pollution and OSA.

Household air pollution

The difference between outdoor and indoor air pollution is difficult to distinguish due to the lack of information on the indoor quality of the included studies. Indoor air pollutants can be generated from the exhaust of combustion appliances, cigarette smoke, and cleaning products. Smoke emitted from cigarette use, e-cigarette/vaping all have the potential to induce oxidative stress [33,34]and have the potential to exacerbate or increase the susceptibility for OSA. Non-smokers sharing the household are also at risk of exposure to the smoke exhaled by the smoker, known as second-hand smoke - a potent source of fine indoor airborne PM. Indoor monitoring of second-hand smoke showed that an increase in 1 $\mu g/m^3$ of vapor-phase nicotine corresponds to an average increase of 10 $\mu g/m^3$ of PM [35]. Furthermore, poor cooking practices in developing countries can emit indoor PM concentrations that are 10 times higher than the WHO recommended air quality guidelines [36,37]. Indoor air quality may be different compared to outdoor, and that higher levels are possible indoors in otherwise low polluted countries, especially in poorly ventilated living spaces. It could be argued that using outdoor data may not reflect the amount of air pollution that the participant might be exposed to during sleep.

Only one study from Thailand [19] in the current review had used a personal air sampling kit to quantify the participants' bedroom air quality. The annual average PM_{10} measured from this study was approximately 17 $\mu g/m^3$, which is below WHO air quality guidelines of 20 ug/m^3 and well below the National level of 50 $\mu g/m^3$ in Thailand. However, the reported annual average of 17 $\mu g/m^3$ is misleading as it was computed from 6 d measurement across the dry and wet seasons. Besides, the effect size might be underestimated because of the small sample size, and this study did not adjust for outdoor air pollution and important confounding variables such as socioeconomic status, so the relationship should be interpreted with caution.

Methodological heterogeneity

Several methodological heterogeneities related to the definition of OSA also limited our ability to compare the estimates of air pollution and OSA. These included: 1) different indices to report OSA, 2) different polysomnography tests used, 3) different cut-off points for airflow reduction and oxygen desaturation, and 4) different air pollution sampling methods.

Although a laboratory-based polysomnography test is the 'gold standard' for diagnosing OSA, unattended home-based

polysomnography is an equally reliable method [38]. Hence, the different types of polysomnography tests performed are unlikely to contribute to the heterogeneity observed in the findings. On the other hand, the limited-channel polysomnography test used in the study from Germany [14] is less reliable at diagnosing OSA. Differences in the criteria used to define the cut-off thresholds for airflow reduction during apneic and hypopneic events, as well as the criterion to define oxygen desaturation in hypopneic events (or the absence thereof) however, is another issue that may contribute to the heterogeneity of findings. AASM [39] criteria were used in three studies. However, the AASM criteria have changed over time and incorporate "recommended" and "acceptable" definitions. Therefore, even within the AASM criteria, hypopnea events have been scored differently in successive versions. Two studies [15,20] defined a hypopnea event with at least 4% oxygen desaturation and one study [19] defined the hypopnea event with at least 3% oxygen desaturation cut-off point. The difference may appear to be small but has a direct influence on the threshold for the diagnosis and severity rating of OSA, especially in moderate to severe OSA [40–42]. One study [14] from the current review did not include an oxygen desaturation measurement in its hypopnea definition and used with substantially different airflow reduction thresholds compared to the AASM criteria. This variability in definition almost certainly affects the number of people diagnosed to have OSA. Establishing a consensus in diagnostic criteria for OSA that remains consistent over time will facilitate comparisons between similar studies in future reviews that examine OSA.

Determining the level of air pollution is another key methodological issue. Only one study [20] used the spatial–temporal hierarchical modelling technique. This is arguably one of the most robust methods to quantify long-term air pollution exposure as the model allows for space-time variation for air pollution levels over large geographical areas with low bias [43]. Three of the studies [13–15] used fixed air monitoring stations to ascertain the level of air pollution exposure. Although this method might provide reliable estimates of air pollution temporally, changes in the concentration of air pollution spatially may not be accurately represented, since this method does not account for parameters such as proximity to busy traffic and dwelling density. While spatio-temporal modelling techniques are becoming widely used, there are differences between indoor and outdoor air quality that are insufficiently captured, especially in low and middle-income countries, evidently with the study from Thailand [19].

Strengths and limitations

Compared to the previously published systematic review [16], one of the main strengths of this review is the inclusion of only studies that objectively measured the outcome, albeit with substantial heterogeneity in the criteria used to assess OSA. To investigate whether restrictions on articles published in English would limit the scope of our systematic review, we used the same search strategies for non-English publications in PubMed, Embase and Scopus databases. This resulted in 87 papers, but none of them qualified for a full-paper screen after the title and abstract screening. This indicates that the current systematic review did not miss relevant studies by omitting non-English publications.

An important finding in this review is the knowledge gap for the relationship between air pollution and OSA risk in children, with the current literature limited to subjective questionnaire-based research or studies designed to examine sleep-related outcomes other than OSA. In addition, the results from this review represent the association between air pollution and OSA in middle to high-income countries. The risk of OSA is expected to be higher in low-income countries as there is a significant disparity in the exposure to air pollution between high- and low-income countries resulting from poverty and energy sources used in the latter [44]. Furthermore, changes in the prevalence or severity of OSA in relation to changes in exposure when considering a causal link between air pollution and OSA cannot be established in the current review since the included studies relied on a single time point measurement with no follow-ups. Future studies may better address these limitations by designing longitudinal studies that focus on OSA that is objectively measured in children and those from low-income countries, and time-series analysis studies are also needed to investigate the causality between air pollution and OSA more robustly.

Conclusion

This systematic review has emphasised the heterogeneity that exists in the definitions of the measurement indices used to report OSA, highlighting further the need for a consensus in the diagnostic criteria for OSA among clinicians and researchers. Our results showed evidence that supports a relationship between air pollution and OSA and highlighted a seasonal trend in the elevation of air pollutants. Air pollutant levels worldwide are highly varied according to different regions/settings of seasons and temperature, and even a concentration of air pollution that is below the WHO air quality standards is shown to positively correlate with the risk for OSA. This has important public health implications and warrants continued vigilance in monitoring and the mitigation of air pollution. Our review has identified the knowledge gap in studies that focus on important risk groups such as in children and those from low-income countries. In order to progress the field in this area, we recommend designing prospective cohort studies to further explore the OSA risk in the mentioned risk groups as well as changes in the severity of OSA with the continual exposure to air pollution in time-series analysis. Given that future air quality may potentially be impacted by climate change, investigating air pollution and OSA in this changing context is an area that warrants attention from practitioners and researchers.

Practice points

1. There is evidence to support the relationship between air pollution and obstructive sleep apnea.
2. Seasonal variation of air pollution levels is an important consideration when evaluating its influence on obstructive sleep apnea.
3. Pollution-obstructive sleep apnea associations were seen, even in settings in which the ambient pollution concentrations were below the World Health Organization air quality guidelines.
4. Indoor air quality may be different compared to outdoor, and that higher levels are possible indoors in otherwise low polluted countries. This is an important consideration since using the participant's residential address to ascertain level of air pollution exposure may not adequately capture their bedroom environment.

Research agenda

1. Well-designed studies to investigate the association between air pollution and obstructive sleep apnea in children and from low-income countries are needed to fill the knowledge gap.
2. The heterogeneity in the definitions used to diagnose and report obstructive sleep apnoea highlights the need for consensus for a diagnostic criterion for research. This will facilitate consistent comparisons between similar studies in future reviews that examine sleep apnea.
3. The air pollution-sleep apnea relationship could be reinforced by further evaluating the changes in the severity of obstructive sleep apnea in relation to ongoing exposure to air pollution by using time-series analysis and with multiple follow-ups.
4. Most studies used fixed air monitoring stations to ascertain the level of air pollution exposure which does not adequately capture changes in air pollution in spatial manner. Using more robust techniques such as a satellite-based land use regression model to assess air pollution exposure is an important consideration in future studies.
5. Upper airway irritant and seasonal allergic reaction are not mutually exclusive. Conditions such as allergic rhinosinusitis can potentially modulate the severity of sleep apnea. This should be considered when looking at air pollution and sleep apnea in the context of seasonal variation.

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Conflicts of interest

The authors do not have any conflicts of interest to disclose.

Appendix A. Supplementary data

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