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Air pollution exposure and adverse sleep health across the life course: A systematic review☆

Jianghong Liu[a,*], **Tina Wu**[a], **Qisijing Liu**[b], **Shaowei Wu**[b], **Jiu-Chiuan Chen**[c]

[a]University of Pennsylvania School of Nursing, Philadelphia, PA, USA

[b]Peking University School of Public Health, Beijing, China

[c]University of Southern California Keck School of Medicine, Los Angeles, CA, USA

Abstract

An increasing number of epidemiological studies have examined air pollution as a possible contributor to adverse sleep health, but results are mixed. The aims of this systematic review are to investigate and summarize the associations between exposures to air pollutants and various sleep measures across the lifespan. PubMed, CINAHL, Cochrane, Scopus, Web of Science, and PsycInfo were searched through October 2019 to identify original data-based research examining direct epidemiological associations between ambient and indoor air pollution exposures and various sleep health measures, including sleep quality, sleep duration, sleep disturbances, and daytime sleepiness. Twenty-two articles from 2010 to 2019 were selected for inclusion in this review, including a wide range of study populations (from early childhood to elderly) and locations (10 Asian, 4 North American, 3 European, 5 other). Due to variation in both exposure and outcome assessments, conducting a meta-analysis was not plausible. Twenty-one studies reported a generally positive association between exposure and poor sleep quality. While most studies focused on ambient air pollutants, five assessed the specific effect of indoor exposure. In children and adolescents, increased exposure to both ambient and indoor pollutants is associated with increased respiratory sleep problems and a variety of additional adverse sleep outcomes. In adults, air pollution exposure was most notably related to sleep disordered breathing. Existing literature generally shows a negative relationship between exposures to air pollution and sleep health in populations across different age groups, countries, and measures. While many associations between air pollution and sleep outcomes have been investigated, the mixed study methods and use of subjective air pollution and sleep measures result in a wide range of specific associations. Plausible toxicological mechanisms remain inconclusive. Future studies utilizing objective sleep measures and controlling for all air pollution exposures and individual encounters may help ameliorate variability in the results reported by current published literature.

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*Corresponding author. jhliu@nursing.upenn.edu (J. Liu).

Declaration of competing interest

All authors have no conflicts of interest to report.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envpol.2020.114263.

Keywords

Air pollution; Environmental exposure; PM; Health effect; Sleep problems; Systematic review

1. Introduction

Air pollution has been identified as a major public health concern due to the immense impact of both outdoor and indoor exposure on health (WHO, 2019a). Approximately 91% of the worldwide population live in areas where ambient air pollution exposure exceeds the guidelines recommended by the World Health Organization (WHO, 2018a). Furthermore, these adverse effects are exacerbated by additional exposure to indoor pollutants from poor cooking practices such as the use of polluting stoves and coal- or biomass-based fuels (WHO, 2018b). The effects of these exposures are reflected in the incidence or aggravation of various adverse health outcomes, including respiratory diseases (Kurt et al., 2016), cardiovascular diseases (Franklin et al., 2015) that account for nearly a quarter of deaths due to stroke and ischemia (WHO, 2019b), delayed cognitive development in children (Sunyer et al., 2015), and increased risk for dementia in the elderly (Fu et al., 2019; Paul et al., 2019; Peters et al., 2019; Russ et al., 2019; Shou et al., 2019).

Recently, sleep disturbance has been identified as another adverse health outcome affected by air pollution exposure. Poor sleep has become an important public health concern, affecting as many as one-third of all children (Liu et al., 2019; Mindell and Owens, 2015), 50–70 million adults in the US alone (CDC, 2017), and up to 60% of elderly adults (Gulia and Kumar, 2018). Interestingly, poor sleep has been well-recognized as a contributor to the aforementioned adverse health outcomes. More specifically, frequent occurrences of sleep disturbances can result in increased risk for other health complications, such as cardiovascular disease (Fang et al., 2015; Irish et al., 2015), cancer (Blask, 2009; Irish et al., 2015), diabetes (Fang et al., 2015; Irish et al., 2015; Sears and Zierold, 2017), worse general physical health (Strine and Chapman, 2005), mental health incidence (Banks, 2007; Strine and Chapman, 2005; Zaharna and Guilleminault, 2010), behavioral and emotional dysregulation (Irish et al., 2015; Liu et al., 2016; Sears and Zierold, 2017; Zaharna and Guilleminault, 2010), and cognitive impairments (Banks, 2007; Irish et al., 2015; Liu et al., 2012; Sears and Zierold, 2017; Van Dongen et al., 2003; Zaharna and Guilleminault, 2010).

Given the significant adverse health outcomes resulting from both air pollution and sleep disturbance, research in the past decade has begun to examine the potential impact of air pollution exposure on sleep disturbance. Recent publications demonstrate the relationship between greater exposure to both indoor (Chuang et al., 2018; Lappharat et al., 2018; Wei et al., 2017) and ambient (Yu et al., 2019; Zanobetti et al., 2010) pollutants and various indicators of poor sleep health, including short sleep duration (Chuang et al., 2018; Yu et al., 2019), poor sleep quality (Wei et al., 2017; Zanobetti et al., 2010), and sleep disordered breathing (Lappharat et al., 2018; Zanobetti et al., 2010). However, to date, there have been no reviews of the existing literature. Thus, this current systematic review aims to provide a review of published literature on the association between air pollution and sleep outcomes and to discuss implications for future research. The scope of this review is defined according

to a PECO statement (populations, exposures, comparators, and outcomes) as follows: In humans of any age, is exposure to air pollution (either ambient or indoor), compared to people who are not exposed (or who are exposed at lower levels), associated with adverse sleep health?

2. Methods

This systematic review was conducted in adherence with guidelines set by the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) statement (Moher et al., 2009).

2.1. Search strategy

Literature searches were conducted using PubMed, CINAHL, Cochrane, Scopus, Web of Science, and PsycInfo to identify suitable research articles published through October 2019 for this review (no publication date restriction was set). These databases were searched using the various terms for air pollution and sleep outcomes. A sample search included ("air pollut*" [Title/Abstract] OR "environment* exposure" [Title/Abstract] OR "environment* pollut*" [Title/Abstract] OR "particulate matter" [Title/Abstract] OR "PM10" [Title/Abstract] OR "PM2.5" [Title/Abstract] OR "ozone" [Title/Abstract] OR "carbon monoxide" [Title/Abstract] OR "nitrogen dioxide" [Title/Abstract] OR "sulfur dioxide" [Title/Abstract]) AND (sleep*[Title/Abstract] OR "sleep quality" [Title/Abstract] OR "sleep duration" [Title/Abstract] OR "sleep efficiency" [Title/Abstract] OR "sleep disturbance" [Title/Abstract] OR "sleep impair*" [Title/Abstract] OR "sleep disordered breathing" [Title/Abstract] OR "sleep apnea" [Title/Abstract]), Additional MeSH terms, including "Air Pollutants", Air Pollution", "Sleep", "Sleep Wake Disorders", "Actigraphy", and "Polysomnography" were included in PubMed. The entire search strategy is provided in Appendix A.

2.2. Inclusion criteria

2.2.1. Type of studies—Observational and clinical research conducted on human participants were included in this review. Appropriate study designs include cross-sectional, cohort, longitudinal, and intervention studies. Reviews, commentaries, case studies, and other narrative publications were excluded.

2.2.2. Type of exposure—The exposure measure examined in this systematic review was air pollution, both ambient and indoor. Studies that directly investigated at least one pollutant exposed via the air were included. Papers exclusively investigating other forms of exposure, such as noise pollution, environmental factors such as heavy metal exposure (Mohammadyan et al., 2019), and exposures via water and food intake (Gump et al., 2014), were excluded. Additionally, literature on second-hand smoke exposure were excluded due to the availability of analyses on the topic (Colrain et al., 2004; Deleanu et al., 2016).

2.2.3. Type of outcome—Sleep was identified as the outcome measure of interest. Sleep outcomes included sleep quality, sleep duration, sleep efficiency, and various problems experienced during sleep. Both objective measures, such as polysomnography and actigraphy, and subjective measures, such as self-report, were included. Articles whose

objective was not to specifically examine sleep outcomes, but may have included time asleep in the data collection, did not fit the inclusion criteria (Peel et al., 2011).

2.3. Study selection

Papers identified through the electronic search process were retrieved. Duplicate articles were removed, and titles and abstracts were evaluated. Full texts of relevant publications were examined based on the inclusion criteria, and references were scanned for additional articles suitable for inclusion. Duplicate screening was conducted to ensure accuracy and consistency.

2.4. Data extraction

Data from suitable publications were extracted, including year of publication, study design and location, sample characteristics of participants, air pollution and sleep measures, adjusted covariates, major findings, and study strengths and limitations. Extraction was conducted in pairs to ensure accuracy, and disagreements were resolved through discussion.

2.5. Quality assessment

Level of certainty rating for individual studies was utilized to assess the quality of included papers. A modified systematic review framework was used to rate the level of certainty for each health outcome. This framework is derived from a previously published systematic review based on established methods of systematic reviews for the medical, public health and environmental health fields (Bamber et al., 2019). These frameworks incorporate most of Bradford Hill's criteria for causation such as studies with specificity and biological plausibility and that were temporal and consistent (Schünemann et al., 2011). These classic criteria were used to develop a meaningful scope of review and determine criteria for study certainty.

We rated study findings as having low, moderate, or high certainty that the reported result was close to that of the true effect based on the methodology established in a recent systematic review (Bamber et al., 2019). The findings were initially ranked as low certainty and were upgraded according to fourteen study evaluation questions assessing various domains. These criteria were based on established frameworks that specified the domains, questions, or study limitations used to evaluate individual studies for use in a systematic review (Guyatt et al., 2008; Higgins and Altman, 2008; Rooney et al., 2014; Wells et al., 2017; Woodruff and Sutton, 2014). We categorized the study evaluation questions into the following five categories: population and sample, exposure, health outcomes, confounders, and reporting. Two authors reviewed each study evaluation question with a yes-or-no response for each included study. Conflicting responses were resolved through discussion and additional review of the study. Studies with greater than 50% "yes" answers (i.e., 8 "yes" answers out of 14) were considered for potential upgrade of their findings to moderate certainty; and studies with greater than 75% "yes" answers (i.e., 11 "yes" answers out of 14) were considered for potential upgrade to high certainty (no study fulfilled this criterion). All findings of each study were ascribed the same level of certainty after evaluations were complete.

3. Results

Twenty-two studies examining the relationship between air pollution and sleep were identified as suitable for this review. The search process was summarized using the PRISMA flowchart (Fig. 1). Details on study population, measures, covariates, major findings, strengths, and limitations are presented in Tables 1 and 2, ordered by study design (cross-sectional, retrospective or prospective cohort, intervention). Evaluation of quality for each article is presented in Table 3, with detailed descriptions provided in Supplementary Tables S1–S22. All, or nearly all, papers utilized generalizable samples, examined dose-response relationships, accounted for confounding factors, and reported accurate conclusions based on reported results.

Sample characteristics varied in these studies. Of these, ten were conducted in Asia, four in North America, three in Europe, and five in other regions, resulting in a total of 17 countries. Sample sizes ranged from 59 to 59,754 participants with age at time of exposure ranging from fetuses to elderly adults up to 80 years. The majority of the studies were conducted in adults (n = 15) with seven focusing on children and adolescents. Study designs included cross-sectional (n = 14), retrospective (n = 2) and prospective (n = 4) cohort, and intervention (n = 2).

Sleep outcomes were assessed using a variety of methods, including self-report questionnaires only (n = 12), objective measures such as actigraphy or polysomnography only (n = 9), or both (n = 1). The included studies assessed a variety of sleep outcomes such as sleep quality, sleep duration, sleep efficiency, and various sleep disturbances including sleep disordered breathing (obstructive sleep apnea, snoring, wheezing), sleep onset latency, nighttime dysfunction.

Air pollution studied in the twenty-two articles spanned both ambient and indoor pollutants, including particulate matter; nitrogen dioxide; ozone; sulfur dioxide; traffic-related pollutants such as black carbon; and combustion products. These exposures were measured by personal air quality sensors (n = 4), estimated by air quality monitoring stations (n = 11) or urine biomarkers (n = 1), individualized using spatiotemporal models (n = 4), or self-reported via exposure questionnaires (n = 3).

Despite the varying methods and populations, all studies reported some association between air pollution and sleep. These relationships are detailed in the subsequent sections by type of exposure.

3.1. Multiple Air Pollutants

While the majority of studies included in this present systematic review investigated a specific type of exposure, three large cross-sectional studies examined the general effect of ambient air pollution on sleep (Kheirandish-Gozal et al., 2014; Lawrence et al., 2018; Yu et al., 2019). Kheirandish-Gozal et al. (2014) estimated exposure to various pollutants, including inhalable particulate matter (PM_{10}), nitrogen dioxide, sulfur dioxide, carbon monoxide, and ozone, in five districts of Iran with varying levels of exposure. Among 4322 children aged 6–12 years, those living in regions with higher levels of pollutant exposure

were more likely to have habitual snoring, defined as loud snoring three or more times a week. This effect remained after controlling for other risk factors, such as family history of snoring, wheezing, respiratory problems, and parental smoking status, indicating that poor air quality can lead to the development of habitual snoring and subsequently disrupt child sleep quality.

A similar study conducted on 59,754 children and adolescents aged 5–17 years (mean age 10.3 years) in China monitored exposures to the same individual pollutants as the previous study and assessed sleep quality and various sleep disorder symptoms using the Sleep Disturbance Scale for Children (SDSC) (Lawrence et al., 2018). The scale reported information on sleep-wake transition disorders (SWTD), disorders of initiating and maintaining sleep (DIMS), disorders of excessive somnolence (DOES), disorders of arousal, sleep hyperhidrosis, and sleep-breathing disorders. By stratifying the analysis by both pollutants and symptoms of sleep disorders, the authors demonstrated positive associations between all pollutants and sleep disturbances evaluated. Notably, the observed relationships were generally stronger among females than males, the strongest association was observed between PM_1 exposure and disorders of excessive somnolence, and exposure to PM_1 resulted in the greatest risk for sleep disturbances among all other pollutants.

Another study conducted in China assessed the association between ambient pollutants and sleep duration in college freshmen. Air pollution exposure was estimated by city air monitoring stations, and measures included air quality index and $PM_{2.5}$, PM_{10}, and NO_2 exposure. Sleep duration was assessed using the Chinese version of the Pittsburgh Sleep Quality Index (CPSQI). Across five cohorts with a total of 31,582 participants, exposure to higher concentration of air pollutants was associated with shorter sleep duration.

3.2. Particulate matter

One major component of air pollution is particulate matter (PM), composed of both solid and liquid particles found in the air and classified by aerodynamic diameter. The smaller sized particles, $PM_{2.5}$, have diameters of 2.5 μm or less and pose the greatest risk to health (EPA). Similarly, exposure to larger particles, such as PM_{10}, has also been shown to affect sleep.

Eight studies have examined the effects of general particulate matter, using a variety of methods to measure air pollution exposure, on sleep disturbances across the life course. In a longitudinal study on 397 mother-child pairs (mean age 27.7 and 4.8 years respectively) in Mexico, Bose et al. (2019) reported that prenatal maternal exposure to $PM_{2.5}$ has differing effects on later child sleep patterns depending on the gestational phase during which exposure occurs. Daily $PM_{2.5}$ exposure for each mother-child pair was individualized using spatiotemporal models based on the home address of the participants. By averaging these daily values over each week of gestation and assessing child sleep via actigraphy, the authors were able to identify windows of susceptibility during which the fetus is especially sensitive to air pollution exposure. While $PM_{2.5}$ exposure early in gestation (weeks 1–8) was negatively associated with child sleep efficiency, greater exposure to particulate matter with diameters of 2.5 μm or less later in the gestational period (weeks 31–53) was associated with shorter child sleep duration.

Furthermore, a similar cross-sectional study of 276 children (mean age 9.26 years) from four school districts estimated PM_{10} exposure levels using the monitoring stations closest to each school (Abou-Khadra, 2013). Despite the highly variable levels of exposure across the districts, the author found significant positive associations between PM_{10} exposure and both sleep hyperhidrosis and disorders of initiating and maintaining sleep.

Conversely, a large prospective cohort study conducted on 14,110 freshmen (mean age 18 years) at a Chinese university by An and Yu (2018) demonstrated that greater $PM_{2.5}$ exposure in young adults resulted in longer sleep duration both during the day and at night. This association remained significant after adjusting for covariates such as age, BMI, current smoking and drinking status, and self-assessed physical and mental health, and there was no difference in effect between males and females. However, despite the longitudinal design and large sample size, sleep measures were obtained from self-report questionnaires, possibly resulting in response bias, and the study population of young university students is not representative of the larger population, leading to a lack of generalizability of the results.

Chuang et al. (2018) estimated occupational $PM_{2.5}$ exposure in Taiwanese welding workers and office workers (mean age 46.2 years) using personal air sampling sensors. Welding workers were observed to have greater wake times during sleep, as measured by actigraphy, due to increased exposure to $PM_{2.5}$ pollutants in metal fumes.

Four cross-sectional studies on large adult populations examined the specific effect of particulate matter exposure on sleep disordered breathing (SDB) (Billings et al., 2019; Lappharat et al., 2018; Shen et al., 2018; Zanobetti et al., 2010). Using estimates of air pollutant exposure from monitoring station data, Shen et al. (2018) reported a positive association between both $PM_{2.5}$ and nitrogen dioxide exposure on SDB, measured by the apnea-hypopnea index (AHI) and oxygen desaturation index (ODI) via polysomnography, in their population of 4312 Taiwanese adults (mean age 45.8 years). These relationships were observed for both short-term (daily mean exposure estimates) and long-term exposures (annual mean exposure estimates) and were the most significant in the spring and winter seasons. A similar study by Billings et al. (2019) on an older population of 1974 US participants (mean age 68 years) confirmed the association between $PM_{2.5}$ and nitrogen dioxide exposure and SDB. Use of spatiotemporal models allowed Billings et al. (2019) to estimate individual participant exposure levels based on home address, resulting in more accurate measures of air pollution exposure. After adjusting for demographics, other comorbidities, socioeconomic status (SES), and study site, greater exposure to both $PM_{2.5}$ and nitrogen dioxide increased the risk of sleep apnea, with $PM_{2.5}$ having a stronger effect.

Furthermore, in the US, sleep in a subset of the Sleep Heart Health Study (SHHS) cohort consisting of 6441 adults over the age of 39 (mean age 63 years) was assessed via polysomnography. PM_{10} exposure in the summer was positively associated with SDB, as measured by the respiratory disturbance index (RDI) and sleep time spent in hypoxia, and negatively associated with sleep efficiency (Zanobetti et al., 2010). Higher temperatures were also observed to be associated with higher RDI scores across all seasons. A similar study conducted in Thailand examined bedroom environmental conditions and reported that increases in PM_{10} exposure was correlated with increased severity of obstructive sleep

apnea, a common subtype of SDB, using measures of AHI, RDI, and hypoxia (Lappharat et al., 2018). However, despite these findings, two similar studies conducted in adults failed to demonstrate a relationship between particulate matter exposure and sleep disordered breathing (Cassol et al., 2012; Weinreich et al., 2015).

While most studies only investigated general particulate matter in relation to sleep outcomes, few additional studies have examined a specific particulate matter, namely ozone, in relation to sleep health. As a common air pollutant, ozone is affected by various weather-related factors such as rises in ambient temperature and relative humidity (EPA, 2018). Thus, a discussion regarding ozone, temperature, and humidity is provided in the additional following section.

3.3. Ozone, temperature, and humidity

Five studies have identified associations between ozone, temperature, and humidity and SDB in particular (Cassol et al., 2012; Cheng et al., 2019; Sánchez et al., 2019; Weinreich et al., 2015; Yıldız Gülhan et al., 2019). In a cross-sectional study on 564 children aged 5–9 (median age 6 years), Sánchez et al. (2019) estimated air pollution exposure using monitoring stations located near the participants' schools and obtained data on sleep-related respiratory symptoms via parental self-report using the Pediatric Sleep Questionnaire (PSQ). Greater exposure to both ozone and sulfur dioxide and higher humidity levels increased the risk of wheezing-related sleep disturbances. Similarly, Weinreich et al. (2015) examined the relationships between PM_{10}, temperature, ozone levels, and relative humidity and SDB. Using AHI to measure SDB, the authors reported positive associations between AHI and both temperature and ozone levels. These relationships were strongest in warm weather and remained after adjusting for covariates. Conversely, Yıldız Gülhan et al. (2019) demonstrated that AHI levels were higher during winter months. Using polysomnography and air monitoring stations to estimate exposure, relative humidity was identified to be positively associated with rapid eye movement-related AHI.

Two similar studies examined the relationship between ambient pollutants and sleep outcomes in non-population based participants (Cassol et al., 2012; Cheng et al., 2019). Measured exposures included particulate matter, carbon monoxide, sulfur dioxide, ozone, temperature, and relative humidity for both. Similarly, analysis of retrospective polysomnographic data was used to assess AHI among all participants. By obtaining patient polysomnography data taken across all seasons, Cassol et al. (2012) observed that obstructive sleep apnea (OSA) severity varied with seasonality and was positively associated with carbon monoxide and relative humidity but negatively associated with temperature. Similarly, Cheng et al. (2019) reported that a positive association between AHI and PM_{10}, ozone, sulfur dioxide, and relative humidity was only seen in those with severe OSA during non-REM sleep.

Despite the large sample sizes of these studies, these contradicting findings, particularly for the relationship between sleep outcomes and temperature, could be due to various limitations. Firstly, pollutant exposure was measured by monitoring stations nearest to the hospital and did not account for participants' home location or indoor exposures. Most notably, the study participants were drawn from people referred to the hospitals for a

diagnostic PSG due to the possible presence of sleep disorders. However, a randomized control trial on OSA patients has demonstrated that although lower bedroom temperatures was associated with longer sleep duration, higher sleep efficiency, and increased alertness during the day, AHI was higher at lower temperatures (Valham et al., 2012). Thus, this emphasizes the complex relationship between temperature and sleep outcomes, the mechanism of which is not yet understood.

3.4. Traffic-related

Air pollution resulting from road traffic has been commonly identified as a health hazard. Common markers of traffic-related pollution are black carbon, a $PM_{2.5}$ component (Fang et al., 2015), and NO_2 (Martens et al., 2018) exposure. Using self-reported data on traffic exposure collected from a large European population (n = 12,184, mean age 51.5 years), Gislason et al. (2016) demonstrated a significant risk of daytime sleepiness due to high levels of Traffic-related pollutant exposure. In an additional study conducted in the US, exposure to black carbon was estimated using spatiotemporal models and participant home location (Fang et al., 2015). Self-report questionnaires were used to assess for sleep duration, sleep latency, and sleep apnea, and increased black carbon exposure was found to decrease sleep duration in males and those of low SES but increase sleep duration in African Americans. Surprisingly, exposure to black carbon was not associated with sleep latency or sleep apnea. This may be due to the lack of objective sleep measures considering the large sample size (n = 3821) and individualized air pollution estimates. A similar study, based in the Netherlands, also used spatiotemporal models to estimate individual-level exposure to NO_2 (Martens et al., 2018). The authors demonstrated that overall sleep quality and incidences of sleep disturbances were not only related the modeled air pollution exposure, but also to self-reported perceived exposure to traffic-related pollutants.

3.5. Indoor air quality

In addition to ambient exposures, three studies have focused specifically on the effects of indoor air quality, specifically examining the relationship between pollutants due to cooking and sleep in both children and adults. Two intervention studies conducted by one research team on Peruvian children under the age of 14 reduced household exposure to biomass pollution by replacing highly polluting stoves with reduced polluting Inkawasi stoves (Accinelli et al., 2014; Castañeda et al., 2013). Using parent reports of child sleep habits, the authors reported improvement in SDB-related symptoms, such as snoring and nighttime awakening, with decreased biomass pollution. Further, lower levels of pollutant exposure were associated with increased willingness to sleep and ease of falling asleep and waking up for both exclusive and partial use of the improved stoves. In adults, a cross-sectional study conducted on 2197 Chinese adults (mean age 37.52 years) investigated the relationship between cooking oil fume (COF) exposure and sleep (Wei et al., 2017). Cooking practices and sleep patterns were measured using self-report questionnaires. Additionally, urine samples were collected and analyzed for 1-hydroxypyrene (1-HOP), a urinary biomarker of polycyclic aromatic hydrocarbons in the COFs. Both subjectively reported and objectively measured exposure were positively associated with poor sleep quality. Furthermore, subjective report of COF exposure increased the risk for long sleep onset latency, daytime dysfunction, and sleep disturbances.

4. Discussion

To our knowledge, this is the first systematic review on the relationship between exposure to different air pollutants and sleep outcomes. Twenty-two selected studies included cross-sectional (n = 14), cohort (n = 6), and intervention studies (n = 2) across 17 countries. Air pollution exposure was mostly assessed by air quality monitoring stations (n = 11), but other methods included personal air quality sensors (n = 4), urine biomarkers (n = 1), individualized using spatiotemporal models (n = 4), and self-reported exposure questionnaires (n = 3). Sleep assessment included both objective (n = 10) and subjective (n = 13) measures. Overall, the review demonstrated a general positive relationship between air pollution exposure and sleep disturbances in children, adolescents, and adults. Both exposure to air pollution (Sánchez et al., 2019) and sleep outcomes (Grandner, 2012) have been shown to vary with developmental stage due to the increased vulnerability of children and the elderly to adverse environmental and health effects. Thus, the research included in this present review is discussed by life stage to highlight the differences in the association between pollutant exposure and sleep outcomes across the life course.

4.1. Children and adolescents

More than 10% of school-aged children and adolescents are reported to experience sleep problems (Stein et al., 2001). The published literature examined in this review reports a negative association between sleep quality and exposure to pollutants in this population. Because of their developing nervous and immune systems, children have been shown to be more susceptible to the effects of air pollution (Sánchez et al., 2019). This vulnerability begins *in utero*, as lower sleep efficiency and shorter sleep duration at a preschooler age has been linked to prenatal exposures to $PM_{2.5}$ at different stages of gestation (Bose et al., 2019). Despite the indirect nature of this exposure, these results suggest that air pollution can have long-lasting effects on sleep quality.

Similarly, respiratory-related sleep disturbances have been observed to be associated with air pollution exposure. Higher levels of ambient air pollution are positively associated with a wide range of disturbances that affect children's sleep quality, including habitual snoring, wheezing, and sleep disorders symptoms, with overall stronger relationships seen in females compared to males. Notably, sulfur dioxide and ozone levels were related to habitual snoring in children and increased the risk of wheezing-related sleep disturbances (Kheirandish-Gozal et al., 2014; Sánchez et al., 2019), and lower levels of CO_2 and $PM_{2.5}$ exposure were associated with improved SDB-related symptoms (Castañeda et al., 2013). Generally, children are more prone to these disturbances than adults, possibly due to inhaling larger volumes of air per body weight on average and an increased permeability of the airway epithelium (Sánchez et al., 2019). These physiological factors result in greater relative exposure to pollutants that remain in the airways for longer periods of time, likely inducing more severe effects on sleep in children than adults.

Apart from respiratory-related sleep disturbances, several studies have also observed associations between particulate matter and a wide range of sleep disturbances (Lawrence et al., 2018). In young children, particulate matter $PM_{2.5}$ and above are associated with sleep hyperhidrosis, initiating/maintaining sleep, wheezing- and snoring-related sleep

disturbances, and nighttime awakenings. In adolescents, higher levels of $PM_{2.5}$ exposure are associated with longer daytime and nighttime sleep durations.

Inconsistencies in these findings could be due to the varying participant populations assessed or the limitations in study design. All sleep measures relied on self-report from the child's parents, rather than objective methods, and may be subject to recall bias. Additionally, the questionnaires used also differed between studies. Further, exposure estimates only included either ambient pollutants or indoor pollution and did not account for exposure from the other environment or from other sources, such as noise and diet. Thus, future studies should account for these additional confounders and use objective sleep measures to further investigate the association between sleep disturbances and air pollution exposure in children and adolescents.

4.2. Adults

It has been shown that sleep disorders are more likely to occur in older adults than a younger population (Neubauer, 1999). All except one reviewed study pertaining to this population consistently reported the association between disruptions in sleep quality and air pollution exposure. Although positive associations between sleep-disordered breathing (SDB) with $PM_{2.5}$ and PM_{10} exposure in older adults had been reported in some studies (Billings et al., 2019; Lappharat et al., 2018; Shen et al., 2018; Zanobetti et al., 2010), but not in others (Cassol et al., 2012; Weinreich et al., 2015). Therefore, the relationships between air pollution exposure and SBD remain uncertain. Published studies also revealed associations between overall sleep quality/disturbances (increased daytime sleepiness and decreased sleep efficiency/duration) and air pollution have also been found.

Altogether, nine of the fifteen relevant studies used objective sleep measures, namely actigraphy and polysomnography, while the remainder assessed sleep duration, quality, and sleep disturbances with various self-report questionnaires. Additionally, exposure to air pollution was measured using differing methods across the literature. These diverse study designs led to varying findings of the specific relationships between pollutant exposure and sleep. For example, while increased sleep disturbances and daytime dysfunction were correlated with greater exposure to subjectively appraised traffic-related pollution (Gislason et al., 2016; Martens et al., 2018), sleep duration was associated with general air pollution exposure in Chinese university students (Yu et al., 2019) as well as black carbon exposure in males, those of lower socioeconomic status, and those of African American descent (Fang et al., 2015). Further, amount of time spent awake during time in bed was positively associated with exposure to particulate matter (Chuang et al., 2018).

Notably, contrary to related research (Bose et al., 2019; Fang et al., 2015; Scinicariello et al., 2017), An and Yu demonstrated a positive relationship between sleep duration and exposure to air pollution (An and Yu, 2018). This discrepancy could result from the use of different methods to measure exposures, as the present study estimated $PM_{2.5}$ levels via general monitoring stations while others have spatiotemporal models (Bose et al., 2019; Fang et al., 2015) to assess pollutant levels. Further, these other studies were conducted on significantly younger (Bose et al., 2019) or older (Fang et al., 2015) participants, suggesting that these

findings may be age dependent. However, this relationship cannot be concluded due to the absence of similar studies.

4.2.1. Sleep disordered breathing—Half of the reviewed literature regarding the effects of air pollution on adult sleep investigated sleep disordered breathing (SDB) in particular. SDB encompasses a group of disorders, including obstructive sleep apnea (OSA) (Lappharat et al., 2018), that is commonly found in older adults and results in respiration irregularities during sleep (Shen et al., 2018). Furthermore, increased severity of SDB increases the risk of cardiovascular complications that could become fatal (Weinreich et al., 2015). Using objective sleep measures such as actigraphy and polysomnography, studies have analyzed apnea and hypoxia as proxies for SDB.

The apnea-hypopnea index (AHI), a measure of the frequency of disruptions in airflow during sleep, was positively associated with temperature, relative humidity, and exposure to ozone, nitrogen dioxide, $PM_{2.5}$, and PM_{10} (Cassol et al., 2012; Martens et al., 2018; Shen et al., 2018; Weinreich et al., 2015; Yıldız Gülhan et al., 2019). Additionally, increased nitrogen dioxide and $PM_{2.5}$ exposure was observed to increase the risk of SDB (Billings et al., 2019). However, one study only observed these relationships during non-REM sleep in participants with severe obstructive sleep apnea (Cheng et al., 2019), while another found a positive association between REM-related AHI levels and relative humidity (Yıldız Gülhan et al., 2019). This discrepancy may be due to the participant population, which was drawn from patients suspected to have sleep disorders, resulting in a sample that is not representative of a healthy population. SDB was also assessed using measures of hypoxia, or an oxygen deficiency, as this frequently occurs as a consequence of the respiratory disturbances characteristic of SDB (Zanobetti et al., 2010). Hypoxia was positively associated with particulate matter exposure in three cross-sectional studies (Martens et al., 2018; Shen et al., 2018; Zanobetti et al., 2010).

Interestingly, while authors reported similar associations between pollutants and SDB, seasonal variations in the relationships were observed. Two studies noted that the correlation between sleep disordered breathing and exposure to air pollution were most significant in warmer weather (Weinreich et al., 2015; Zanobetti et al., 2010), while others noted that the associations were strongest in colder seasons (Cassol et al., 2012; Shen et al., 2018; Yıldız Gülhan et al., 2019). However, all studies in question estimated exposures using air quality monitoring sensors nearest the participant locations. Thus, more exact exposure to air pollution, including indoor pollutants, were not accounted for. This relationship was assessed explicitly in a smaller cross-sectional study conducted in Thailand, demonstrating that lower levels of indoor exposure to PM_{10} ameliorated symptoms of OSA in both the wet and dry seasons (Lappharat et al., 2018). These discrepancies could be due to the use of a population already suspected to be suffering from sleep disorders, having been referred to undergo a diagnostic PSG, rather a healthy population. Furthermore, prior research additionally demonstrates that the relation between seasonal variability and sleep outcomes is quite complex, as lower temperatures are associated with both increased sleep efficiency as well as a higher AHI (Valham et al., 2012). Nevertheless, given the limited literature available, additional research accounting for both ambient and indoor exposures is needed to tease out the intricacies of these relationships.

In examining the studies focused on older adults, the available research is limited due to assessing participants spanning wide age ranges. Notably, the ages of participants of one study cover nearly six decades (Shen et al., 2018). While there are considerably less developmental changes in this life stage, there may still be differential effects of air pollution on sleep by age. Thus, there may be value in greater stratification of age groups in future research. This is important particularly for the elderly in order to further examine the possible detrimental effects of pollutant exposure during a period of increased vulnerability to illness and disease.

4.3. Ambient vs. indoor exposures

Interestingly, most publications examining the associations between air pollution and sleep have been focused on ambient exposure, with double the number of available studies as compared to those assessing indoor exposures. However, these articles display diversity in methodology, resulting in more variation in study design, measures, and results reported. The majority acquired air pollution exposure data from standard government- or city-regulated monitoring stations which does not account for differences in personal exposure due to residence location, indoor pollutants, etc.

Conversely, studies specifically examining the relationship between exposure to indoor pollutants and sleep outcome demonstrate comparatively more consistent methodology. All but one assessed residential exposure, and most utilized personal air monitoring sensors to allow for individualized pollution metrics.

Regardless of the source of air pollution examined, future studies ought to account for all personal exposures including potential residential/occupational exposure as well as encounters with ambient pollutants. Use of portable individual monitoring devices may be effective in considering all pollutants encountered by an individual. On the other hand, it may not be feasible for long-term exposure assessment.

4.4. Mechanisms

Mechanisms explaining the effect of air pollution on sleep are not fully understood and have only been studied minimally. Additionally, the pathway by which exposure to pollutants impacts sleep may vary across the developmental stage. Specifically, prenatal exposure may have direct effects on the fetus by passing through the placental barrier or indirect effects via affecting maternal health (Glinianaia et al., 2004). However, initial evidence suggests two general potential mechanisms which include the biochemical effects of pollutants on the central nervous system's regulation of sleep and changes in the physiology of the respiratory system.

The biochemistry of the central nervous system may be directly affected by air pollutants via the olfactory nerve (Abou-Khadra, 2013; Billings et al., 2019; Shen et al., 2018; Zanobetti et al., 2010), resulting in altered expression and dysregulation of neurochemicals. For example, $PM_{2.5}$ exposure was observed to be associated with lower serotonin levels, an important chemical in modulating wakefulness and circadian rhythms, implying a possible direct effect of air pollution on increased sleepiness and sleep disturbances (Chuang et al., 2018). Relatedly, a study conducted in rats observed that exposure to ozone alters the expression of

5-hydroxy-indole-acetic acid (5-HIAA), the main metabolite of serotonin. These changes were notably observed in the dorsal raphe and the hypothalamic medial preoptic area, two brain structures involved in sleep regulation (González-Piña and Alfaro-Rodriguez, 2003). Furthermore, in children, the impact of exposure on highly vulnerable nervous system structures may disrupt normal brain development and impair typical nervous system functioning, including sleep (Sears and Zierold, 2017). Specifically, exposure of the vulnerable brain to pollutants may cause irritation and breakdown of protective epithelial barriers, resulting in inflammation, oxidative stress, and degeneration of neural tissue. This damage to nerve cells could likely affect behaviors regulated by the brain, including sleep (Brockmeyer and D'Angiulli, 2016). Thus, the disturbances caused by air pollution may have a wide range of effects on sleep outcomes.

Another possible model for the relationship between pollution exposure and sleep may arise from effects on the physiology of the respiratory system. Generally, air pollutants, especially small particulate matter, are thought to deposit particles in the airways, leading to cell damage (Khafaie et al., 2016). In general, injury to respiratory cells results in disturbances in respiration by causing inflammation or edema of mucous membranes. Specifically, in the upper airways, this creates increased restriction and obstruction of normal airflow, increasing the risk of apnea and hypoxia, and thereby disrupting sleep (Abou-Khadra, 2013; Billings et al., 2019; Scinicariello et al., 2017; Shen et al., 2018; Weinreich et al., 2015). Similarly, the presence of foreign particles in the airways may result in irritation and infection, compromising sleep quality (Billings et al., 2019; Sears and Zierold, 2017; Wei et al., 2017).

4.5. Implications for future research

Despite the existing volume of research on correlations between pollutants and sleep disturbances, the examined literature demonstrates a wide variability in methodology and results that may be addressed in future studies. Many current studies include participants across large age ranges, notably encompassing all primary and secondary school grades (Lawrence et al., 2018) or adults of all ages (Shen et al., 2018). In particular, minimal research has been conducted on prenatal exposures or adolescent and elderly populations, despite the increased importance of sleep and susceptibility to disease during these life stages. Future studies focusing on narrower ranges may be able to identify more specific effects of air pollution on sleep outcomes and contribute knowledge on how the relationship develops over the life course.

In addition, more studies using objective sleep measures, such as polysomnography and actigraphy, would allow for more reliable analysis of sleep outcomes. Similarly, measuring air pollution exposures on an individual level and accounting for other confounding pollutants, such as indoor exposures and noise pollution, would help validate and confirm present knowledge. Finally, the limited availability of causal models and mechanisms linking air pollution and sleep may be ameliorated by future longitudinal studies or investigations of possible mediating factors. Therefore, we are calling for more research that investigates the potential biological mechanisms underlying the association between air pollution and sleep outcomes in addition to more epidemiological studies.

4.6. Limitations of current review

Despite the scope of this systematic review, it has several potential limitations. Given the varying study populations, methodologies, and measures used by the reviewed studies, overall causal conclusions cannot be drawn. Furthermore, due to variation in both exposure and outcome assessments, conducting a meta-analysis was not plausible. As such, the magnitude of exposure on sleep outcomes could not be determined. Additionally, as sleep was the outcome of interest for this systematic review, other research that indirectly assessed the association between air pollution exposure and sleep were not included (Peel et al., 2011). Due to a lack of data, the present review did not examine the possible differential findings across gender or race; similarly, evaluation of literature published in non-English languages may expand upon the results discussed in this present review. Moreover, the available literature may be subject to publication bias and/or selective reporting. Importantly, the lower quality ratings of the included papers demonstrate the need for further studies to improve certainty in the associations currently reported between air pollution and sleep outcomes.

5. Conclusions

The existing published literature examining the relationship between air pollution exposure and sleep outcomes reports an overall adverse effect of various pollutants on sleep across the life course, with most studies focusing on older adults. Notably, these associations were observed for ambient air pollution as well as indoor pollutants, such as occupational exposures, cooking oil fumes, and bedroom air quality. However, the included studies utilized a wide variety of participant populations, study designs and methodologies, and air pollution and sleep measures, leading to diverse observed results. Future research employing objective sleep measures and controlling for individual air pollution exposures may validate current findings, minimize discrepancies, and allow for the generalization of conclusions.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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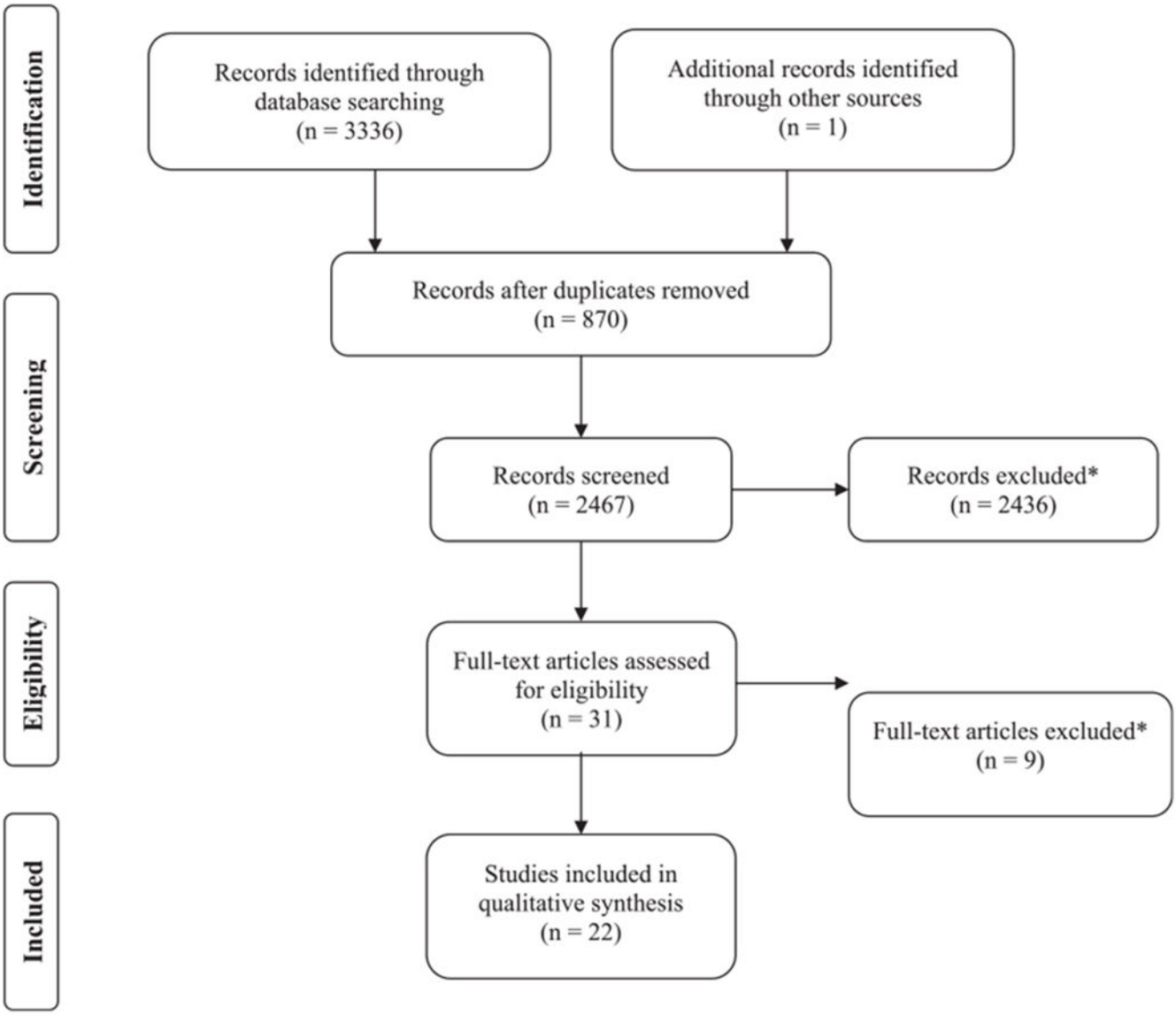

Fig. 1.

Air pollution exposure and sleep problems: PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) flow diagram detailing the article selection process (Moher et al., 2009). *Exclusion criteria is detailed within the methods.

Table 1

Characteristics of included data-based studies on air pollution exposure and sleep outcomes in human participants, ordered by study design.

	Authors	Objectives	Study Design (design and setting)	Sample Characteristics (n by exposure; age)	Air Pollution Measures	Sleep Measures	Covariates
Cross-Sectional	Zanobetti et al. (2010)	Association between PM_{10} and sleep disordered breathing	Sleep Heart Health Study (SHHS), USA City exposures, general adult population	n = 6441 Age 39+ years (mean age = 63) 47.4% male	Ambient exposure EPA Air Quality System Technology Transfer Network monitoring stations *PM_{10}*	Polysomnography *RDI* *Hypoxia* *Sleep efficiency*	Seasonality; age; BMI; sex; education; smoking status; daily glasses of coffee, tea, and soda; number of glasses of wine and beer 4 h before sleep
	Cassol et al. (2012)	Association between seasonality and sleep apnea severity	Brazil City exposures, general adult population	n = 7523 Age 18+ years (mean age = 46 years) 64.9% male	Ambient exposure National Institute of Meteorology *Ambient temperature, relative humidity* State Foundation for Environmental Protection *PM_{10}, SO_2, CO, O_3*	Polysomnography *AHI*	Age, sex, BMI, neck circumference
	Abou-Khadra (2013)	Association between PM_{10} and sleep disturbances	Egypt Exposure near schools, children	n = 276 Age 6–13 years (mean age = 9.26) 44% male	Ambient exposure Egyptian Environmental Affairs Agency's monitoring stations nearby participants' schools *PM_{10}*	SDSC *DIMS* *Sleep breathing disorders* *Disorders of arousal* *SWTD* *DOES* *Sleep hyperhidrosis*	Age, gender, BMI, parental education, parent smoking status, caffeine intake 6 h before sleep, television and computer use 1 h before sleep, bright light exposure near sleep, sleeping with a light on
	Kheirandish-Gozal et al. (2014)	Contribution of air quality to habitual snoring	Tehran, Iran Exposure near schools, children	n = 4322 Age 6–12 years	Ambient exposure Air Pollution Control Center monitoring stations *PM_{10}, SO_2, NO_2, CO, O_3*	Parent self-report questionnaire *Sleep habits* *Family history of snoring*	Age, sex, environmental exposures (parental smoking, neighborhood), SES (parental education), clinical symptoms (respiratory problems, wheezing, coughing, adenotonsillectomy)
	Fang et al. (2015)	Association between black carbon and sleep duration, sleep latency, and sleep apnea across seasons	Boston Area Community Health Survey, US Residential exposure, general adult population	n = 3821 Age 31–87 years (mean age = 53.8) 38.5% male	Ambient exposure Spatiotemporal land-use regression model based on participant location *1*-6 months *and 1 year mean black carbon exposure*	Berlin sleep questionnaire, other self-report measures *Sleep duration* *Sleep latency* *Sleep apnea*	Age, gender, race, education level, smoking and alcohol status, anti-depressant use, use of sleep medications, BMI, physical activity, total caffeine intake, average temperature
	Weinreich et al. (2015)	Association between PM_{10}, ozone, and temperature and sleep disordered breathing	Heinz Nixdorf Recall (HNR) Study, Germany City exposures, general adult population	n = 1773 Age 45–75 years (mean age = 63.8) 50% male	Ambient exposure City monitoring stations *PM_{10}* *O_3, temperature, relative humidity*	ApneaLink *AHI*	Medical history, BP, height, weight, medication, lifestyle factors (smoking history, alcohol consumption, education, physical activity)

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Authors	Objectives	Study Design (design and setting)	Sample Characteristics (n by exposure; age)	Air Pollution Measures	Sleep Measures	Covariates
Gislason et al. (2016)	Association between traffic exposure and habitual snoring and daytime sleepiness	Respiratory Health in Northern Europe (RHINE) III, Europe Residential exposure, general adult population	n = 12,184 Mean age 51.5 years 47.7% male	Ambient exposure Self-report questionnaire *Traffic exposure*	Self-report questionnaire *Sleep disturbances* *Habitual snoring* *Daytime sleepiness*	Gender, age, BMI, smoking, education, physical activity, diagnosed OSA, number of hours of sleep per night, study center
Wei et al. (2017)	Association between exposure to cooking oil fumes and sleep quality	China Personal exposure, general adult population	n = 2197 Mean age 37.52 years 86.5% male	Indoor exposure Self-report questionnaire *Cooking practices (*e.g. *cooking method, use of ventilation, etc.)* Urine via HPLC *1-HOP*	PSQI *Sleep latency* *Sleep duration* *Sleep efficiency* *Sleep disturbances* *Subjective sleep quality* *Daytime dysfunction* *Use of sleep medication*	Age, marital status, education level, smoking status, SHS exposure, alcohol use, physical activity, napping habits, occupational exposures, working time per week, shift work, manual work strength, residence size (proxy for SES), BMI, mental health status, family function
Chuang et al. (2018)	Association between occupational PM_{10} exposure and sleep quality	Taiwan Occupational exposure, working adult population	n = 150 Age 20–70 years (mean age = 46.2) 91.3% male	Indoor exposure Personal air sampling module *$PM_{2.5}$*	Actigraphy *Time in bed, sleep time, wake time* *Number of awakenings* *Sleep efficiency*	Age, sex, BMI
Lappharat et al. (2018)	Relationship between bedroom air quality and severity of obstructive sleep apnea and sleep quality	Thailand Residential exposure, general adult population	n = 63 Age 25–75 years (median age = 42) 73% male	Indoor exposure Personal air sampling device during both wet and dry seasons *PM_{10}* *Temperature, relative humidity*	PSQI *Sleep latency* *Sleep duration* *Sleep efficiency* *Sleep disturbances* *Subjective sleep quality* *Daytime dysfunction* *Use of sleep medication* Polysomnography *AHI, RDI, hypoxia* *Sleep efficiency*	Age, sex, BMI, smoking and alcohol consumption status, history of SHS exposure, medical history, air conditioner usage
Lawrence et al. (2018)	Association between air pollution and sleep disorders	Seven Northeastern Cities study, China Residential exposure, children	n = 59,754 Age 5–17 years (mean age = 10.3) 50.6% male	Ambient exposure Municipal monitoring stations located <1 km from each child's home *PM_{10}, NO_2, SO_2, CO O_3,*	SDSC *Sleep quality* *DIMS* *Sleep breathing disorders* *Disorders of arousal* *SWTD* *DOES* *Sleep hyperhidrosis*	Age, gender, parent education, low birth weight, premature birth, breastfeeding, income, passive smoking exposure, home coal use, house pet, district
Billings et al. (2019)	Association between air pollution and obstructive sleep apnea and objective sleep disruption	Multi-Ethnic Study of Atherosclerosis (MESA), US Residential exposure, general adult population	n = 1974 Age 45–84 years (mean age = 68)	Ambient exposure Air Quality Systems monitoring stations, individualized using hierarchical spatiotemporal modeling *$PM_{2.5}$* *NO_2*	Actigraphy and polysomnography *Objective sleep disruption* *Sleep efficiency* *AHI*	Age, sex, BMI, comorbidities (e.g. depression, diabetes, hypertension, smoking status), SES (e.g. unemployment, poverty, education level, household income), site

	Authors	Objectives	Study Design (design and setting)	Sample Characteristics (n by exposure; age)	Air Pollution Measures	Sleep Measures	Covariates
	Sánchez et al. (2019)	Association between exposure to air pollutants and sleep disordered breathing	Chile Exposure near schools, children	n = 564 Age 5–9 years (median age = 6) 44.9% male	Ambient exposure Monitoring stations located near each school *$PM_{2.5}$,PM_{10}* *SO_2,NO_2,CO,O_3*	PSQ *Sleep associated respiratory symptoms*	Age, sex, father education, household pets, household smoke exposure
	Yu et al. (2019)	Association between air pollution and sleep duration	China City exposures, college students	n = 31,582 Mean age 18.4 67.6% male	Ambient exposure Beijing Municipal Ecological Environment Bureau monitoring stations *AQI, $PM_{2.5}$, PM_{10}, NO_2* China Meteorological Administration *Daytime temperature, wind speed, percent rainy days*	PSQI – Chinese Version Sleep duration	Age, BMI, self-rated physical health, self-rated mental health
Prospective Cohort	An and Yu (2018)	Impact of $PM_{2.5}$ on health behaviors	China City exposures, college students	n = 14,110 Mean age 18 years 67.39% male	Ambient exposure Mission China monitoring stations *$PM_{2.2}$* China Meteorological Administration *Daytime temperature and wind speed*	Fatigue in Medical Training Questionnaire *Night/daytime sleep duration*	Age, BMI, smoking and drinking status, physical health, mental health
	Martens et al. (2018)	Associations between actual and perceived exposure to air pollutants and sleep disturbances	Occupational and Environmental Health Cohort (AMIGO), Netherlands Residential exposure, general adult population	Baseline (2011–2012): n = 14,829 Age 31–65 years (mean age = 50.65) 44.24% male Follow-up (2015): n = 7905 Age 34–69 years (mean age = 52.17) 47.16% male	Ambient exposure Spatiotemporal models based on participant home location *NO_2 (traffic-related)* *$PM_{2.5}$, PM_{10}, NO_x* *RF-EMF, noise pollution* Self-report *Perception of air pollution exposure*	MOS *Overall sleep quality*	Age, sex, education level, smoking status, neighborhood income (proxy for SES)
	Shen et al. (2018)	Association between exposure to air pollution and sleep disordered breathing	Taiwan Residential exposure, general adult population	n = 4312 Age 20–80 years (mean age = 45.8) 39.4% male	Ambient exposure Taiwan Environmental Protection Agency monitoring stations *PM_{10}, $PM_{2.5}$, NO_2, O_3*	Polysomnography *AHI, ODI*	Age, sex, BMI, smoking status
	Bose et al. (2019)	Association between prenatal $PM_{2.5}$ exposure and child sleep outcomes	Programming Research in Obesity, Growth, Environment, and Social Stressors (PROGRESS), Mexico Prenatal exposure, children	n= 397 mother-child pairs Mean mother age 27.7 years Mean child age 4.8 years 51.1% male	Ambient exposure Satellite-based spatiotemporal models based on participant home location *$PM_{2.5}$*	Actigraphy *Sleep duration* *Sleep efficiency*	Maternal age, maternal education, maternal smoking status, season, child age, child sex, child BMI
Retrospective Cohort	Cheng et al. (2019)	Relationship between air pollution and sleep	Taiwan Exposure near hospital, patients	n= 5413 Mean age 46.54 years 76.78% male	Ambient exposure Monitoring stations nearest hospital	Polysomnography *AHI* *Sleep phase*	Demographics (age, sex, smoking status), physiological characteristics (neck

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	Authors	Objectives	Study Design (design and setting)	Sample Characteristics (n by exposure; age)	Air Pollution Measures	Sleep Measures	Covariates
		apnea severity across seasons	referred for PSG diagnostic study		*$PM_{2.5}$, PM_{10} CO, NO_x, SO_2 O_3, temperature, humidity*		circumference, BMI), season, incidence of OSA (AHI >; 30)
	Yıldız Gülhan et al. (2019)	Effect of PM_{10} and seasons on sleep	Turkey	n = 500 63.2% male	Ambient exposure National Air Quality monitoring Network *PM_{10}* *Oxygen saturation, temperature, relative humidity*	Polysomnography *AHI*	None noted
Indoor Intervention	Castañeda et al. (2013)	Effect of exposure to stove pollutants on sleep apnea symptoms	Prospective intervention study Peru Residential exposure, children	n = 59 Age 0.25–14 years (mean age = 7.76) 62.7% male	Indoor exposure Replacement of polluting stove with improved stove *Indoor biomass pollution – including CO_2 and $PM_{2.5}$*	Parent self-report questionnaire *Sleep habits Snoring*	Demographic information, significant medical history
	Accinelli et al. (2014)	Effect of prolonged biomass exposure on sleep apnea symptoms	Intervention study Peru Residential exposure, children	n = 82 Age 2–14 years (mean age = 8.3) 48.8% male	Indoor exposure Replacement of polluting stove with improved stove *Indoor biomass pollution – including CO_2 $PM_{2.5}$*	Parent self-report questionnaire *Sleep habits Snoring*	Demographic information, significant medical history

Abbreviations.

AHI – apnea-hypopnea index.

DIMS – disorders of initiating and maintaining sleep (e.g. long sleep onset latency, frequent night awakening).

DOES – disorders of excessive somnolence.

MOS – Medical Outcomes Study.

ODI – oxygen disturbance index.

OSA – obstructive sleep apnea.

PSG – polysomnography.

PSQ – Pediatric Sleep Questionnaire.

PSQI – Pittsburgh Sleep Quality Index.

RDI – respiratory disturbance index.

SDSC – Sleep Disturbance Scale for Children.

1-HOP – 1-hydroxypyrene, urinary biomarker for cooking fumes.

AQI – air quality index.

CO – carbon monoxide.

CO2 – carbon dioxide.

COF – cooking oil fumes.

HPLC – high-performance liquid chromatography.

NO2 – nitrogen dioxide.

NOX – nitrogen oxides.

O3 – ozone.

PM – particulate matter.

RF-EMF – radiofrequency electromagnetic fields.

SHS – second-hand smoke.

SO2 – sulfur dioxide.

BMI – body mass index.

BP – blood pressure.

SES – socioeconomic status.

Table 2

Results, strengths, and limitations of included data-based studies on air pollution exposure and sleep outcomes in human participants, ordered by study design.

	Authors	Study Location	Major Findings	Strengths	Limitations
Cross-Sectional	Zanobetti et al. (2010)	SHHS, USA	1. SDB was positively associated with short-term exposure to PM_{10} in the summer; sleep efficiency negatively associated	1. Large sample size 2. Objective sleep measures 3. Assessed both short-term and long-term exposure	1. Air pollution data limited by location of monitoring stations established by the EPA
	Cassol et al. (2012)	Brazil	1. Severity of sleep apnea varied with seasonality 2. AHI was positively associated with relative humidity and CO levels and negatively associated with ambient temperature	1. Large sample size 2. Objective sleep measures 3. Evaluation of data over a ten-year period and across all seasons	1. Sample comprised of people referred to hospital for PSG due to possibility of sleep disorders – sampling bias
	Abou-Khadra (2013)	Egypt	1. DIMS and sleep hyperhidrosis were positively associated with PM_{10} levels	1. Participants recruited from different sites to comprise a population with different demographics and exposures	1. No objective sleep measures – self-report may be subject to recall bias 2. Selection bias due to low response rate and sample size
	Kheirandish-Gozal et al. (2014)	Tehran, Iran	1. Air pollution was positively associated with habitual snoring (loud snoring ≥3 nights per week)	1. Large sample size 2. Participants recruited from different sites to comprise a population with different demographics and exposures	1. No objective sleep measures – self-report may be subject to recall bias 2. No indoor pollution or location specific data 3. Possible bias in translating from English to Farsi
	Fang et al. (2015)	BACH, US	1. Annual interquartile increase in BC was associated with shorter sleep duration in males and those of low SES but longer sleep duration in African Americans 2. BC was not related to sleep apnea or latency	1. Large sample size 2. Individualized air pollution metrics 3. Assessed both short-term (1–6 months) and long-term (1 year) exposure to BC	1. No objective sleep measures – self-report may be subject to recall bias 2.Did not account for noise, temperature, or other pollutants
	Weinreich et al. (2015)	HNR Study, Germany	1. SDB was positively associated with interquartile range of temperature and O_3 2. This association was stronger in warmer weather	1. Large sample size 2. . Objective sleep measures 3. Examined associations across different seasons	1. Old population: SDB could be caused by other health factors; non-generalizable 2. Unable to separate effects of temperature and ozone on SDB 3. No indoor pollution measures
	Gislason et al. (2016)	RHINE III, Europe	1. Risk of daytime sleepiness increased with high perceived exposure to traffic-related pollution	1. Large sample size 2. No response bias 3. Translated questions were well validated	1. No objective measures of air pollution 2. Did not account for urban/rural locations
	Wei et al. (2017)	China	1. COF exposure was positively associated with poor sleep quality and increased the risk for long sleep latency, daytime dysfunction, and sleep disturbances 2. 1-HOP was positively associated with poor sleep quality	1. Large sample size 2. Individual pollutant exposure metrics	1. Non-representative sample 2. Results could be due to food exposures rather than fumes 3. No objective sleep measures – self-report may be subject to recall bias
	Chuang et al. (2018)	Taiwan	1. Duration of wake time during sleep was positively associated with metal fume $PM_{2.5}$ exposure	1. Use of two distinct populations to observe effect 2. Individualized air pollution metrics	1. Small, non-representative sample 2. Effect could be due to other pollutants or other

	Authors	Study Location	Major Findings	Strengths	Limitations
					confounders (e.g. noise pollution, diet intake, smoking, alcohol, etc.)
	Lappharat et al. (2018)	Thailand	1. PM_{10} exposure levels were positively associated with OSA severity in both wet and dry seasons 2. Higher bedroom temperatures increased the odds of lower sleep quality	1. Individualized air pollution metrics 2. Objective sleep measures	1. Small sample size 2. Sample comprised of people referred to hospital for PSG, many had severe OSA – sampling bias 3. Noise from air sampling device may have contributed to observed effects on sleep quality
	Lawrence et al. (2018)	Seven Northeastern Cities study, China	1. All air pollutants generally positively associated with sleep disturbances 2. Associations were generally stronger in females 3. PM_1 exposure had highest risk for sleep disturbances 4. The strongest association observed was between PM_1 exposure and DOES	1. Large, representative sample size 2. Stratification of sleep disorders by symptoms and of air pollutants 3. Individualized air pollution metrics for PM_1 and $PM_{2.5}$ exposure	1. Did not account for other confounders, such as noise pollution, food intake, etc. 2. No objective sleep measures – self-report may be subject to recall bias
	Billings et al. (2019)	MESA, US	1. Increased exposure to NO_2 and $PM_{2.5}$ was associated with increased risk for sleep apnea	1. Large, representative sample size 2. Individualized air pollution metrics 3. Objective sleep measures	1. Did not account for noise or light pollution 2. Non-generalizable sample
	Sánchez et al. (2019)	Chile	1. Higher exposure to O_3 and SO_2 and higher humidity levels were associated with increased risk of wheezing-related sleep disturbances	1. Participants recruited from different sites to comprise a population with different demographics and exposures	1. No objective sleep measures – self-report may be subject to recall bias 2. Possible selection bias due to sample size and response rate
	Yu et al. (2019)	China	1. Increased exposure to air pollution (measured by AQI, $PM_{2.5}$, PM_{10}, and NO_2) was associated with reduced sleep duration	1. Large sample size 2. Use of reliable and time-sensitive environmental measures	1. Non-representative sample 2. No objective sleep measures – self-report may be subject to recall bias 3. Did not control for indoor pollutants or seasonal variation in air pollution
Prospective Cohort	An and Yu (2018)	China	1. $PM_{2.5}$ exposure was positively associated with sleep duration during both daytime and nighttime	1. Large sample size 2. Focus on effects of immediate, short-term exposures on health outcomes	1. Non-representative sample 2. Air pollution measures were not specified depending on location 3. No objective sleep measures – self-report may be subject to recall bias 4. Did not control for other pollutants
	Martens et al. (2018)	AMIGO, Netherlands	1. Modeled and perceived exposure to traffic-related air pollution was positively associated with sleep disturbances at baseline and follow-up	1. Large sample size 2. Individualized air pollution metrics 3. Measured both actual and perceived exposure to air pollution	1. Longitudinal data for air pollution was not available – effects across time could not be elucidated
	Shen et al. (2018)	Taiwan	1. AHI was positively associated with $PM_{2.5}$ and NO_2 exposure 2. ODI positively associated with $PM_{2.5}$ exposure 3. Associations were especially significant in spring and winter 4. Associations held for short-term (daily mean) and long-term (1-year mean) exposures	1. Large sample size 2. Assessed both short-term and long-term air pollution exposures 3. Examined associations across different seasons	1. Did not measure personal air pollution exposure 2. Unaccounted for confounders – e.g. noise pollution, diet, SES

	Authors	Study Location	Major Findings	Strengths	Limitations
	Bose et al. (2019)	PROGRESS, Mexico	1. $PM_{2.5}$ exposure during early gestation (weeks 1–8) was negatively associated with preschooler sleep efficiency 2. $PM_{2.5}$ exposure during late gestation (weeks 31–35) was negatively associated with preschooler sleep duration	1. Individualized air pollution metrics 2. Assessment of exposure during especially sensitive windows of susceptibility during gestation 3. Assessment of prenatal air pollution exposure effects on later sleep outcomes	1. Non-generalizable to populations of other cultures, non-urban regions
Retrospective Cohort	Cheng et al. (2019)	Taiwan	1. . AHI was associated with PM_{10}, O_3, SO_2, and relative humidity only in those with severe OSA and only in non-REM sleep	1. Large sample size 2. Objective sleep measures 3. Examined associations across different seasons	1. Sample comprised of people referred to hospital for PSG diagnostic study – sampling bias 2. Air pollution measured at hospital, not where participants lived 3. Did not account for indoor exposures
	Yıldız Gülhan et al. (2019)	Turkey	1. REM-related AHI was positively associated with relative humidity 2. PM_{10} increased relative risk for OSA 3. Sleep duration was longer during winter months	1. Objective sleep measures 2. Examined associations across different seasons	1. Sample comprised of people referred to hospital for PSG diagnostic study – sampling bias 2. Air pollution measured at hospital, not where participants lived 3. Did not account for other pollutants ($PM_{2.5}$, O_3) or indoor exposures
Indoor Intervention	Castañeda et al. (2013)	Peru	1. Reducing biomass pollution improved SDB-related symptoms 2. Decreasing indoor exposure to biomass pollution was associated with less snoring, nighttime awakening, and daytime sleepiness	1. Use of intervention 2. Quantification of reduced exposure to pollutants	1. Duration of exposure to stove smoke was not measured 2. No objective sleep measures – self-report may be subject to recall bias 3. No control group used for the intervention design 4. Small, non-representative sample
	Accinelli et al. (2014)	Peru	1. Decreasing indoor exposure to biomass pollution was associated with increased willingness to sleep, ease of falling asleep, and ease of waking up 2. These relationships were also observed for partial use of improved stoves	1. Use of intervention 2. Quantification of reduced exposure to pollutants 3. Assessed effects of both full and partial intervention	1. Duration of exposure to stove smoke was not measured 2. No objective sleep measures – self-report may be subject to recall bias 3. Small, non-representative sample

Abbreviations.

AHI – apnea-hypopnea index.

DIMS – disorders of initiating and maintaining sleep (e.g. long sleep onset latency, frequent night awakening).

DOES – disorders of excessive somnolence.

ODI – oxygen disturbance index.

OSA – obstructive sleep apnea.

PSG – polysomnography.

REM – rapid eye movement.

SDB – sleep disordered breathing (e.g. obstructive sleep apnea).

1-HOP – 1-hydroxypyrene.

AQI – air quality index.

BC – black carbon.

CO – carbon monoxide.

COF – cooking oil fumes.

NO2 – nitrogen dioxide.

O3 – ozone.

PM – particulate matter.

SO2 – sulfur dioxide.

Table 3

Quality assessment scores of included studies.

Risk of Bias Questions	S1	S2	S3	S4	S5	S6	S7	S8	S9	S10	S11	S12	S13	S14	S15	S16	S17	S18	S19	S20	S21	S22
1. Does the control group match the exposed group?	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
2. Is the sample generalizable to the population of interest?	1	1	1	0	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
3. Did the study a priori quantify sample and power?	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
4. Was missing data addressed and tested?	0	0	0	0	1	0	0	1	0	0	0	0	0	1	0	1	0	0	0	0	0	0
5. Was exposure directly measured and quantified?	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0
6. Was the exposure or proxy/ surrogate of exposure measured from a point location?	1	1	1	1	1	1	0	0	0	0	1	1	1	1	1	1	1	1	1	1	0	0
7. Does the proxy/ surrogate adequately estimate exposure?	0	0	0	0	1	0	0	0	0	0	0	1	0	0	0	1	0	1	0	1	0	0
8. Was there a temporal relationship between	0	0	0	0	1	0	0	0	0	0	0	1	0	1	0	1	0	1	0	0	1	1

Risk of Bias Questions	S1	S2	S3	S4	S5	S6	S7	S8	S9	S10	S11	S12	S13	S14	S15	S16	S17	S18	S19	S20	S21	S22
exposure and outcome?																						
9. Was the health outcome determined by a medical provider?	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0
10. Was a dose-response relationship seen in any outcome?	1	0	1	0	1	1	1	1	1	1	1	1	1	1	1	1	1	1	0	1	1	1
11. Did the study design or analysis account for important confounding and modifying variables?	1	1	1	1	1	1	1	1	0	1	1	1	1	1	1	1	1	1	1	0	0	0
12. Did the study design or analysis adjust or control for other environmental exposures that were anticipated to bias results?	1	0	0	0	1	1	0	0	0	0	0	1	1	0	1	0	1	0	1	0	0	0
13. Were sensitivity analyses attempted for population, outcome, or exposure?	1	0	0	0	1	1	0	1	0	1	0	1	0	1	1	1	1	1	1	0	0	0
14. Did the study conclusions match the results?	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	0	1	1
Study type[a]	CS	CS	CS	CS	CS	CS	CS	CS	CS	CS	CS	CS	CS	CS	PC	PC	PC	PC	RC	RC	I	I

Risk of Bias Questions	S1	S2	S3	S4	S5	S6	S7	S8	S9	S10	S11	S12	S13	S14	S15	S16	S17	S18	S19	S20	S21	S22
Study evaluation score	7	5	5	3	10	7	4	6	4	6	5	9	6	8	8	9	7	8	7	4	4	4
Level of certainty rating	**low**	**low**	**low**	**low**	**moderate**	**low**	**low**	**low**	**low**	**low**	**low**	**moderate**	**low**	**moderate**	**moderate**	**moderate**	**low**	**moderate**	**moderate**	**low**	**low**	**low**

[a]Study type are abbreviated as follows: RC (retrospective cohort); PC (prospective cohort); CS (cross-sectional); and I (intervention).