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Air pollution and childhood respiratory consultations in primary care: a systematic review

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ABSTRACT

Background Outdoor air pollution is a known risk factor for respiratory morbidity worldwide. Compared with the adult population, there are fewer studies that analyse the association between short-term exposure to air pollution and respiratory morbidity in children in primary care.

Objective To evaluate whether children in a primary care setting exposed to outdoor air pollutants during short-term intervals are at increased risk of respiratory diagnoses.

Methods A search in Medline, the Cochrane Library, Web of Science and Embase databases throughout March 2023. Percentage change or risk ratios with corresponding 95% CI for the association between air pollutants and respiratory diseases were retrieved from individual studies. Risk of bias assessment was conducted with the Newcastle–Ottawa Scale (NOS) for cohort or case–control studies and an adjusted NOS for time series studies.

Results From 1366 studies, 14 were identified as meeting the inclusion criteria. Most studies had intermediate or high quality. A meta-analysis was not conducted due to heterogeneity in exposure and health outcome. Overall, studies on short-term exposure to air pollutants (carbon monoxide (CO), sulfur dioxide (SO_2), nitrogen dioxide (NO_2) and particulate matter ≤10 µm (PM_{10})) were associated with increased childhood respiratory consultations in primary care. In general, exposure to ozone was associated with a reduction in respiratory consultations.

Conclusions The evidence suggests CO, SO_2, NO_2, PM_{10} and $PM_{2.5}$ are risk factors for respiratory diseases in children in primary care in the short term. However, given the heterogeneity of the studies, interpretation of these findings must be done with caution.

PROSPERO registration number CRD42022259279.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Air pollution increases children's risk of respiratory diseases, including asthma and bronchitis.
⇒ Globally, over 90% of all children live in environments with air pollution levels above the recommended guidelines.
⇒ Particulate matter (PM), ozone (O3), carbon monoxide (CO), sulfur dioxide (SO2) and nitrogen oxides (NOx) have been identified as major causes of health problems in children.

WHAT THIS STUDY ADDS

⇒ This systematic review summarises evidence relevant to this risk within primary care settings—a new perspective compared with prior reviews.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ The findings highlights the need for policymakers to ensure safer environments for children.

INTRODUCTION

In 2016, air, water and chemical pollution accounted for nearly 1 million deaths worldwide.[1] Two-thirds of these deaths were in children under the age of 5 years. Research on air pollutants indicates that carbon monoxide (CO), sulfur dioxide (SO_2), nitrogen dioxide (NO_2), ozone (O_3), particulate matter ≤10 µm (PM_{10}) and particulate matter ≤2.5 µm ($PM_{2.5}$) are implicated in numerous respiratory diseases including asthma due to their ability to damage bronchial and pulmonary mucosa.[2] The US National Ambient Air Quality Standards under the direction of the Clean Air Act and the Air Quality Standards commissioned by the European Union developed legislation in their respective regions to limit atmospheric concentrations of CO, SO_2, NO_2, O_3, PM_{10} and $PM_{2.5}$.[3] Despite the legislation, 93% of all children and about 630 million children under 5 years are exposed to higher levels of air pollution than recommended by these air quality standards.

Infants and children are more likely to manifest adverse respiratory symptoms from air pollution exposure due to a number of factors.[4] For instance, the immature immune and respiratory system of a child can increase the risk of lung tissue damage, which in turn delays lung growth and increases susceptibility to conditions like asthma.[1 5] Compared with adults, children are exposed to higher doses of ambient air pollutants because they spend more time outdoors and breathe about 50% more air per kilogram of body weight.[6 7]

For the past two decades, several (systematic) reviews have pooled together findings on the association between childhood respiratory diseases and air pollution. For example, a 2012 literature review of 30 studies showed adverse effects of PM_{10} and NO_2 on children's respiratory symptoms and lung function.[8] Furthermore, negative associations were stronger in children with pre-existing respiratory conditions compared with healthy children. Atkinson *et al* included 110 time series studies of daily mortality and hospital admissions.[9] Their

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summary estimates showed that for each 10 µg/m^3 increase in $PM_{2.5}$, the risk of hospitalisation for asthma or respiratory symptoms increased by 2% in children aged 0–14 years. Bowatte *et al* showed that when exposed to moderate road traffic emission, children were significantly more likely to report wheezing (OR 1.26; 95% CI 1.13 to 1.42) and bronchodilator use (OR 1.20; 95% CI 1.04 to 1.38) compared with children exposed to little or no road traffic emission.[6] Zheng *et al* performed a meta-analysis and showed that children were at a higher risk of emergency room visits or hospital admissions when exposed to air pollutants.[2]

Given that respiratory symptoms are among the top three reasons children aged 0–17 years consulted their general practitioner (GP), it calls for an in-depth analysis of the available literature with respect to this patient population and setting.[10] However, despite the significant health and economic impact of air pollution exposure, little is known about the short effect of air pollution on the frequency of respiratory symptoms in children who visit their GP.[11]

The objective of this review was to evaluate whether children in a primary care setting exposed to outdoor air pollutants are at (increased) risk of respiratory diagnoses.

METHODS

The protocol for this review was registered with PROSPERO under registration number CRD42022259279. We used the Preferred Reporting Items for Systematic Reviews and Meta-Analyses to report our findings. The search strategy was conducted in the following manner: first, we formulated the main question using the Population, Exposure, Comparator and Outcome statement. Second, we performed a literature search of both electronic databases and references from retrieved papers. We systematically searched literature published through 12 March 2023. Four databases identified were: Embase (embase.com), Medline ALL (Ovid), Web of Science Core Collection (Web of Knowledge) and Cochrane Central Register of Controlled Trials (Wiley). No limits to publication year or language were imposed.

Review or research papers with no original data in their results were excluded. The following additional exclusion criteria were applied: studies using pregnant subjects or animals; studies that evaluated indoor air pollutants; research on non-respiratory health outcomes; case reports; policy publications or studies published in abstract form only. Two authors (MSF and ERvM) independently screened titles and then performed a full-text review of studies that met inclusion criteria. In the event that a full text was not available, MSF contacted the original authors using the correspondence address on the publication. If no response from the author was received, the article was excluded from the review. Reference lists of eligible studies were included in the list for full-text review. Any disagreement on inclusion was resolved by discussion and, if no consensus was reached, a third reviewer (EdS) was consulted.

Study data were extracted including publication year, study design, study country, study population (children aged between 0 and 18 years who visited a primary care practitioner), air pollutants and respiratory outcomes. Effect measures and their 95% CIs that were extracted from studies included percentage (%) change in respiratory outcomes per increase in air pollutant level, risk/relative ratios (RRs), ORs, excess relative risk (ERR) and HRs. Where applicable, effect measures were pooled for a fixed increment in pollutant concentration (per 1 µg/m^3); other reported quantities or units such as parts per billion and parts per million were converted using the previously published formulas.[12–15]

Evaluation criteria

We assessed the methodological quality of the studies included and the possibility of bias using the Newcastle–Ottawa Scale (NOS) for case–control studies and cohort studies. The NOS for cohort studies measures three dimensions (selection, comparability and outcome). In the NOS for case–control studies, the outcome dimension is replaced by exposure. A study can be awarded a minimum of one star for each numbered item within the selection, outcome or exposure categories and a maximum of two stars in the comparability category. A study can therefore receive a total of nine stars. A study with a NOS score of 1–3, 4–6 or 7–9 was evaluated as poor, intermediate or high quality, respectively. For time series analysis, we used an adjusted NOS score previously published in other systematic reviews.[16 17] The adjusted NOS evaluates three components: (1) the validation of respiratory outcome occurrence (0–1 point), (2) the quality of air pollutant measurements (0–1 point) and (3) the extent of adjustment for confounders (0–3 points). A study with an adjusted NOS score of 0–1, 2–3 or 4–5 received an overall quality of poor, intermediate or high, respectively.

Concerning the validation of respiratory outcomes, we considered the diagnosis to be validated if it was coded according to the International Classification for Primary Care (ICPC) or International Classification of Diseases (ICD).

We identified eight effect measures which we aggregated into two groups (change in outcome per unit µg/m^3 air pollutant and change in outcome per IQR/percentile change in air pollutant). Each group contained the following items: outcome type (upper respiratory, lower respiratory or both), exposure duration (short-term or long-term), pollution type (CO, SO_2, NO_2, O_3, $PM_{2.5}$ and PM_{10}), effect size and 95% CI (lower limit and upper limit). A meta-analysis was not performed due to the heterogeneity of the study designs and outcomes.

RESULTS

We identified 1366 unique articles, of which 1331 were excluded based on title and abstract screening (figure 1). We screened 35 full texts and identified 3 articles from article references. A total of 14 articles were included in this review. Characteristics of these studies are shown in table 1. The majority were conducted in Europe and the most common type of study design was time series. Short-term exposure to air pollutants was frequently reported.

Air pollutants

The most common air pollutants encountered in the review were SO_2, NO_2, O_3 and PM_{10}. Compared with the recommended air quality guidelines (AQGs) by the WHO, the majority of studies had air pollutant levels far below the recommendations (figure 2). For instance, the mean SO_2 levels in four studies were substantially below the recommended minimum level of 40 µg/m^3.[18–21] A total of five studies had mean O_3 levels below the AQG recommendations (100 µg/m^3). With regard to NO_2 and PM_{10}, most studies had higher mean concentration values than their respective AQG recommendations. Only one study reported on $PM_{2.5}$, and the mean value was similar to the recommended AQG.

Lower respiratory diseases

Five of the six studies suggested an increased % change in consultations for lower respiratory tract diseases (LRDs) after

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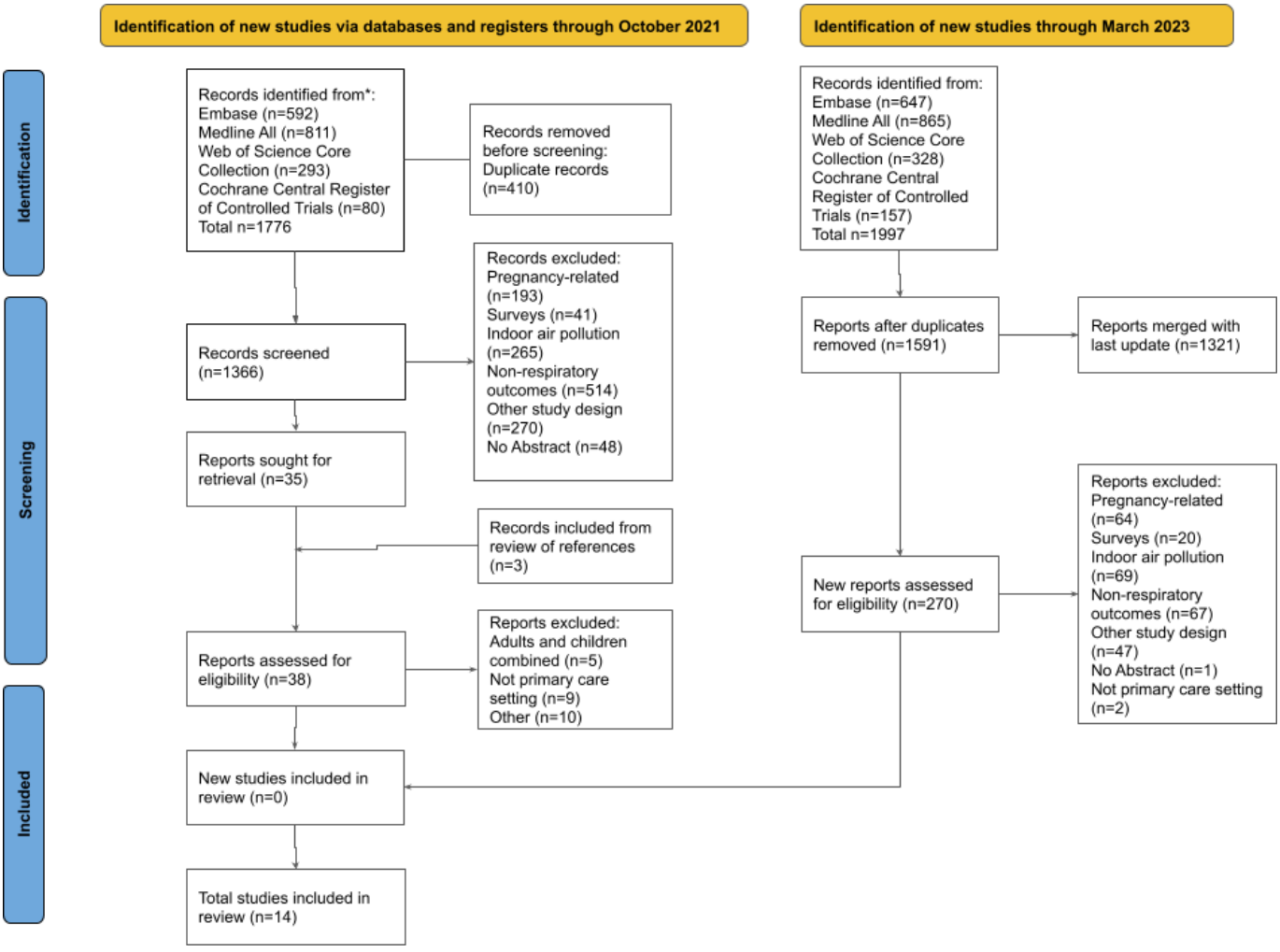

Figure 1 Flow chart of search results included in the review.

short-term exposure to CO, SO_2, NO_2 and/or PM_{10}. Throughout the year, asthma diagnosis was sensitive to short-term exposure to CO, SO_2, NO_2 and PM_{10}. Two studies suggested a significantly increased % in daily visits for asthma with higher levels of O_3. Contrary to this, one study found that short-term exposure to O_3 was predominantly associated with a reduction in asthma consultations.

With regard to short-term exposure to PM_{10}, two of the six studies that reported exclusively on LRD including asthma showed an increase in RR of 1.32 (95% CI 0.82 to 2.13) in house calls. Furthermore, in a study performed in Chile, a 50 $\mu g/m^3$ change in PM_{10} was associated with more frequent clinic visits of 2.5% (95% CI 0.2% to 4.8%) in younger children compared with 3.7% (95% CI 0.8% to 6.7%) in older children.[22]

Upper respiratory tract diseases

Three time series reported on upper respiratory tract diseases (URDs), of which, one was limited to allergic rhinitis. Specifically, this latter study demonstrated that an increase in consultations for allergic rhinitis was due to short-term exposure to SO_2, 24.5% (95% CI 14.6% to 35.2%), NO_2, 11.0% (95% CI 3.8% to 18.8%), O_3, 11.4% (95% CI 4.4% to 19%) and PM_{10}, 10.4% (95% CI 2% to 19.4%) (figure 3).

Upper and lower respiratory diseases

Two time series examined the effect of air pollution on both lower and upper respiratory diseases. Within one of the most polluted regions in Slovenia, the RRs of daily first consultations for all respiratory diseases including influenza and pneumonia were 0.986 (95% CI 0.977 to 0.995) for SO_2, 0.998 (95% CI 0.996 to 1.001) for O_3 and 1.004 (95% CI 1.002 to 1.006) for PM_{10} levels (figure 3).

Funnel plots for air pollutant exposure and respiratory outcome effect sizes are presented in figure 4. The visual inspection of the funnel plots showed some indications for publication bias. For short-term exposure to O_3 and NO_2, the presence of publication bias was confirmed by Egger's test. A funnel plot for $PM_{2.5}$ was not applicable due to small numbers.

DISCUSSION

In the current systematic review of 14 studies conducted in 10 countries, we evaluated data on outdoor air pollution and respiratory diseases in children. Most short-term exposure studies reported a positive association between air pollution concentrations (specifically for CO, NO_2, SO_2 and PM_{10} air pollutants) and children with respiratory morbidity in primary care settings. Two studies that reported on the effect of $PM_{2.5}$ levels showed a slight increase in consultation rates for respiratory diseases.

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Table 1 Characteristics of studies about the effects of exposure to ambient air pollutants on childhood respiratory diseases in primary care settings

ID	Author (year), country	Study design	Study population (age, years); setting; study period	Sample size (n)	Respiratory outcome (URD, LRD or both)	Monitoring sources; sites (n)	Air pollutants	Exposure duration (short-term, long-term or both)	Effect measure	Adjustment variables	Study quality
1	Ashworth *et al*[23] (2021), UK	TS	Children (0–7) & adults (18–64, >64); PC; 2009–2013	1.16 million	Both	Air Quality Networks: NO_2 (130); PM_{10} (115); O_3 (62); $PM_{2.5}$ (104)	NO_2, O_3, $PM_{2.5}$, PM_{10}	Both	% change	DOW, T, H, IMD	High
2	Martin *et al*[21] (2018), ES	C	Children (0–14); PC; 2013–2015	52 322	URD	Madrid City Council website (NA)	SO_2, NO_2, NO_x, CO, O_3, $PM_{2.5}$, PM_{10}	Long-term	RR	Not disclosed	Intermediate
3	Lindgren *et al*[27] (2013), SE	C	Children (0–6); PC; 2005–2010	26 128	Both	Emission database (NA)	NO_x	Both	HR	Sex, ETS, BF, PA, PO, PE, YOB	Intermediate
4	Kukec *et al*[20] (2013), SL	TS	Children (1–11); PC; 2000–2002	NA	Both	Zagorje (1); Trbovlje (1); Hrastnik (1)	SO_2, NO_2, O_3, PM_{10}	Short-term	IRR	S, T, H, Infl, DOW	Intermediate
5	Lai *et al*[8] (2012), UK	C-C	Children (5–16); PC; 1999–2004	8726	LRD	National air archive, Aberdeen Scotland (NA)	SO_2, NO_2, CO, O_3, PM_{10}	Short-term	% change	PH, S	Intermediate
6	Portnov *et al*[19] (2011), IL	C	Children (6–14); PC; 2008–2009	3922	LRD	Air quality monitoring stations (14)	PM_{10}, SO_2	Long-term	OR	Not disclosed	Poor
7	Larrieu *et al*[28] (2009), FR	TS	Children (0–15) & adults (>65); PC; 2000–2006	600 000	Both	Air quality monitoring stations (4)	PM_{10}, NO_2, O_3	Short-term	ERR	S, T, Infl, pollen, DOW, PH	Intermediate
8	Babin *et al*[29] (2008), USA	C	Children (0–4, 5–12, 13–20) & adults (21–49, 50–64, >65); PC, H&O; 1994–2005	NA†	LRD	Environmental Protection Agency sites (6)	$PM_{2.5}$, PM_{10}, O_3	Short-term	% change	S, T, dew, DOW	Intermediate
9	Hwang and Chan[18] (2002), TW	TS	Children (0–14) & adults (15–64, >65); PC; 1998–1999	278 000	LRD	Taiwan Air Quality Monitoring Network (59)	SO_2, NO_2, CO, O_3, PM_{10}	Short-term	% change	WD, WS, T, dew	Intermediate
10	Hajat *et al*[26] (2002), UK	TS	Children (0–14) & adults (15–64, >65); PC; 1992–1994	295 740	URD	Monitoring stations across London (11)	SO_2, NO_2, CO, O_3, PM_{10}	Short-term	% change	S, DOW, PH, Infl, T, pollen	High
11	Hajat *et al*[30] (2001), UK	TS	Children (0–14) & adults (15–64, >65); PC; 1992–1994	253 635	URD	Monitoring stations across London (11)	SO_2, NO_2, CO, O_3, PM_{10}	Short-term	% change	S, T, H, Infl	High
12	Hajat *et al*[31] (1999), UK	C	Children (0–14) & adults (15–64, >65); PC; 1992–1994	295 740	Both	Monitoring stations across London (11)	SO_2, NO_2, CO, O_3, PM_{10}	Short-term	% change	S, T, H, Infl	High
13	Ostro *et al*[22] (1999), CL	TS	Children (0–2, 3–15); PC; 1992–1993	153 548	Both	Metropolitan Environmental Health Service (4)	PM_{10}, O_3	Short-term	% change	S, T, H, H5PM; C5D, DOW, Infl	High
14	Medina *et al*[32] (1997), FR	C	Children (0–14) & adults (15–64); PC; 1991–1995	6.1 million	LRD	Paris Air Pollution Network (38)	SO_2, NO_2, CO, O_3, PM_{10}	Short-term	RR	T, pollen, DOW, PH, Infl	High

*2191 days observed.
†61 218 annual general care visits.
BF, breast feeding; C, cohort; C-C, case–control; C5D, coldest 5% of the days; CL, Chile; CO, carbon monoxide; dew, daily average dew point temperature; DOW, day of the week; ERR, excess relative risk; ES, Spain; ETS, environmental tobacco smoke; FR, France; H, humidity; H&O, hospital and outpatient care; H5PM, days of highest 5% PM_{10}; IL, Israel; IMD, Index of Multiple Deprivation; Infl, influenza; IRR, incidence risk rate; LRD, lower respiratory tract disease; NA, not available; NO_2, nitrogen dioxide; NO_x, nitrogen oxides; O_3, ozone; PA, parental age; PC, primary care; PE, parental education; PH, public holidays; PM_{10}, particulate matter ≤10 µm; $PM_{2.5}$, particulate matter ≤2.5 µm; PO, parental origin; RR, relative risk; S, seasonality; SE, Sweden; SL, Slovenia; SO_2, sulfur dioxide; T, temperature; TS, time series; TW, Taiwan; URD, upper respiratory tract disease; WD, wind direction; WS, wind speed; YOB, year of birth.

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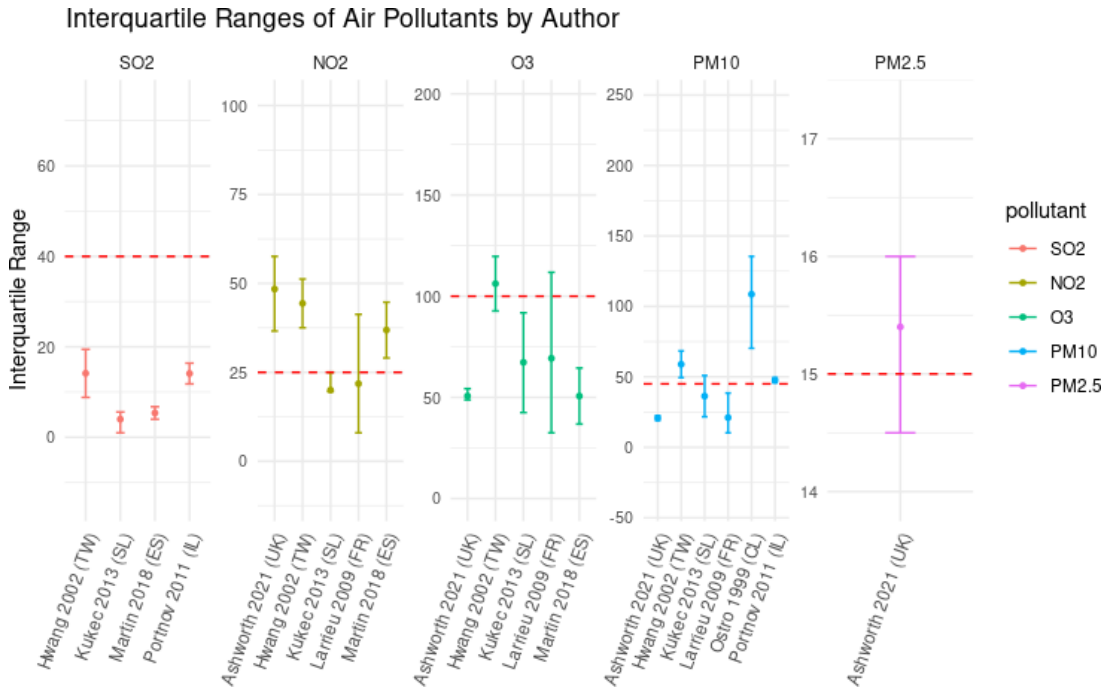

Figure 2 Distribution of air pollution concentration per study. CL, Chile; ES, Spain; FR, France; IL, Israel; NO_2, nitrogen dioxide; O_3, ozone; $PM_{2.5}$, particulate matter ≤2.5 µm; PM_{10}, particulate matter ≤10 µm; SL, Slovenia; SO_2, sulfur dioxide; TW, Taiwan.

With regard to O_3 exposure, most studies reported a negative association between short-term exposure and lower respiratory diseases. O_3 concentrations are typically higher in rural areas compared with urban areas due to traffic emission and industrial activities. All studies in this review were in urban settings. Another explanation for the interaction between O_3 and reduced consultations for respiratory diseases is better access to healthcare in urban areas compared with rural settings. In one study, short-term exposure to O_3 was associated with increased prescription of preventive inhaler medication without adjusting for NO_2.[23]

Two meta-analyses published before 2021 documented positive associations for short-term exposure to air pollutants and respiratory diseases. They both included studies that reported their findings from hospitalised children with asthma and/or wheeze.[2 24] One of the systematic reviews included 87 studies and the other 13 with varying methodology. However, the pooled RRs and ORs were similar. A recent systematic review on 11 time series and 6 case-crossover studies reported a positive association between daily levels of air pollutants and hospitalisation due to pneumonia in children.[25]

For the studies that investigated short-term effects of air pollution on respiratory morbidity, interpretation should be done cautiously. In particular, the definition of URD and LRD comprised broad spectrum of diagnoses and only one study excluded allergic rhinitis from their URD definition.[26] Furthermore, it is not clear whether exposure at lag 0–7 days triggers an existing respiratory condition or if these are new occurrences of events. In our review, two studies reported on new cases as first dispensed inhalers or first consultation for respiratory disease, while the rest did not specify whether children had pre-existing respiratory conditions.[20 27] In addition, no study reported on functional assessment for respiratory illness by a GP or nurse. One study investigated the association between preventer or inhaler medication with short-term and long-term exposure to air pollutants.[23]

Studies varied in design, outcome definition, exposure assessment and the number of studies for some pollutants were limited in order to perform a meta-analysis. Our risk of bias assessment suggested that half of the studies have an intermediate level of risk of bias, but overall, the pattern of results does not suggest that the biases would have produced a false association. The most common form of bias was determined to be from the type of exposure and misclassification of respiratory disease outcomes. Only one study in our review adjusted for personal factors (specifically Index of Multiple Deprivation) and found similar results compared with studies without adjustment for personal factors.

To our knowledge, this is the first comprehensive literature review on air pollution effects and childhood respiratory diseases in a general practice setting. One strength is that most of the studies were performed in developed countries and thus we can assume some generalisability of the current evidence to similar regions. However, some limitations should be acknowledged. First, the included studies differed markedly in outcome assessment (for instance, the definition of respiratory diseases), exposure assessment (for instance, measurements from air monitoring stations vs spatial-statistical model), effect measures (for instance, % change in number of respiratory consultations vs incidence risk rate or %ERR with varying unit increase of air pollutants) and exposure period (for instance, lag days for short-term exposure). Second, many of the findings presented in the review consisted of data from the same cohort of children. Third, most studies focused on SO_2, NO_2, O_3 and PM_{10} and a few investigated the effects of $PM_{2.5}$ on respiratory outcomes. Fourth, some studies objectively defined respiratory diseases using ICD or ICPC classifications and others did not use such coding systems. In the latter case, this may lead to misclassification of outcomes and thereby underestimating the effect estimates. Fifth, the number of covariates differed among the studies and several important factors such as seasonality, influenza and pollen were not adjusted in most studies,

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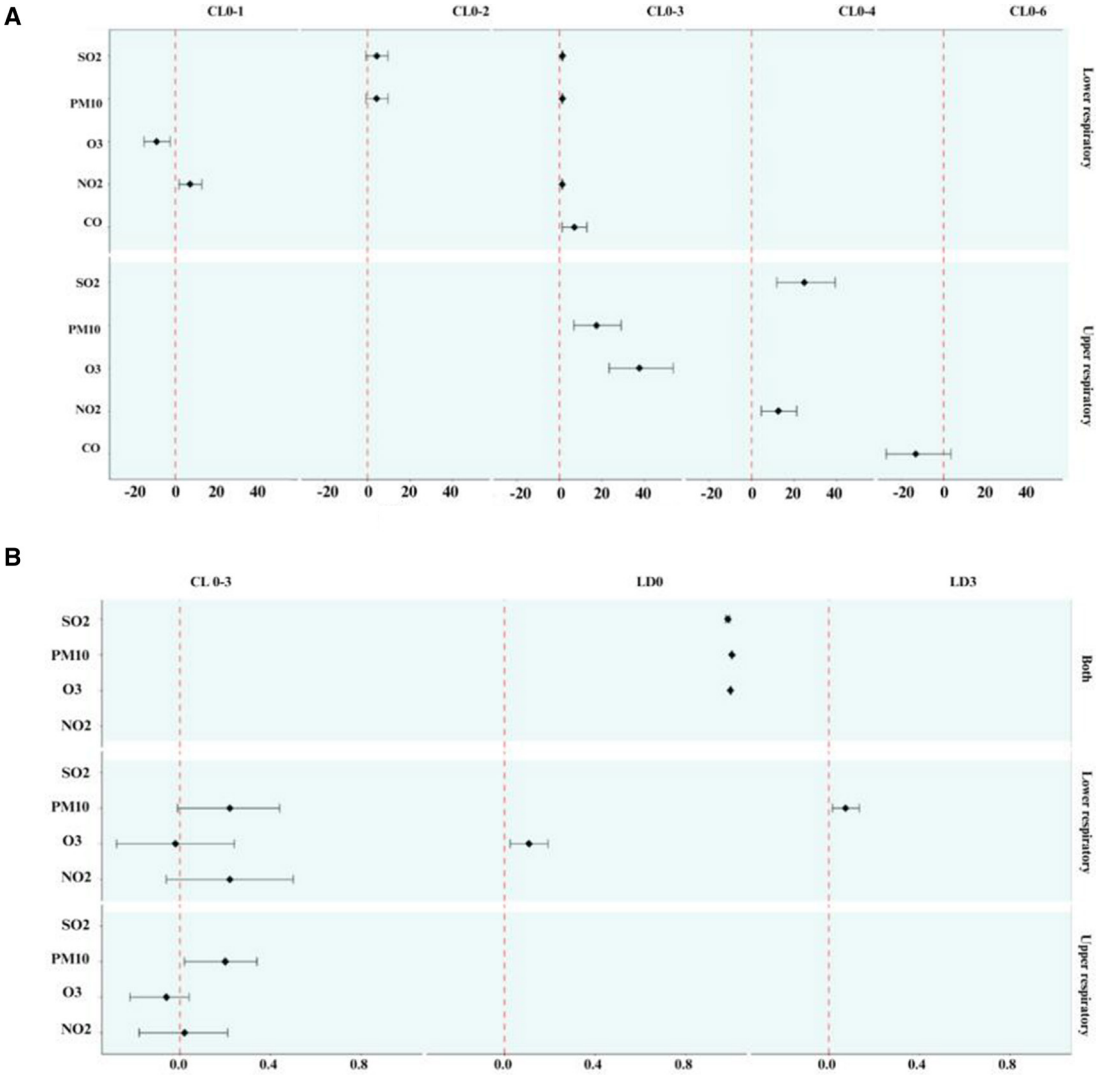

Figure 3 (A) Percentage change in respiratory outcome per IQR or percentile increase of air pollutant according to cumulative lag (CL). (B) Percentage change in relative risk (RR) or incidence risk rate (IRR) of respiratory outcome per μg/m^3 increase in air pollutant according to lag day (LD). NO_2, nitrogen dioxide; O_3, ozone; PM_{10}, particulate matter ≤10 μm; SO_2, sulfur dioxide.

hence limiting interpretation of the findings due to residual confounding. Sixth, the vast majority of studies used single-pollutant models to generate their effect estimates; however, it is known that air pollutants correlate with each other and the respiratory effects of one pollutant can be masked or dominated by other pollutant(s).

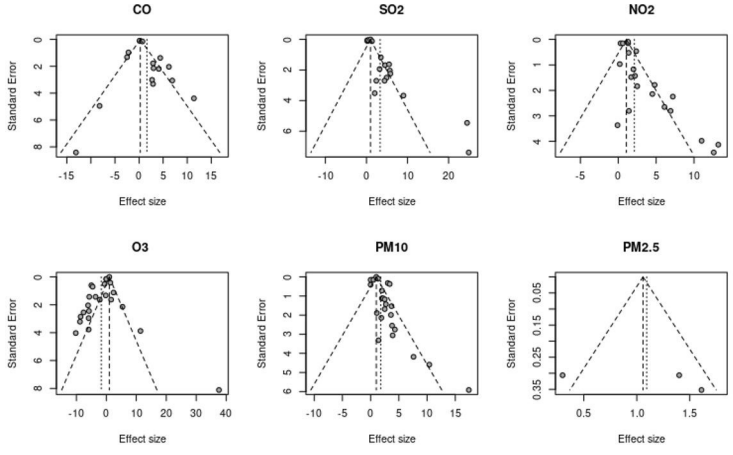

Figure 4 Funnel plots for short-term air pollutant exposure and respiratory outcome effect sizes. NO_2, nitrogen dioxide; O_3, ozone; $PM_{2.5}$, particulate matter ≤2.5 μm; PM_{10}, particulate matter ≤10 μm; SO_2, sulfur dioxide.

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CONCLUSION

The evidence we reviewed suggests an association between short-term exposure to air pollution with respiratory diseases in children in primary care. This association was seen even when air pollutant concentrations (in particular for SO_2 and PM_{10}) were below the WHO-recommended AQG levels. Contrary to the literature, four studies observed an inverse relationship between O_3 and respiratory diseases. This could be explained by either less outdoor activities during periods of high temperature or increased use of preventive inhalers and better access to healthcare in urban areas. We found few data on short-term exposure to $PM_{2.5}$ and respiratory diseases. $PM_{2.5}$ is considered as fine fractions that can penetrate deeper in the airways in comparison with other air pollutants. Hence, it is important to understand the potential biological mechanisms of $PM_{2.5}$ in the lungs and systemic inflammatory processes induced as it penetrates cellular barriers. Furthermore, given the number of children at risk of exposure to $PM_{2.5}$, the population health implications can be substantial. The findings from this review suggest that a multidisciplinary approach to prevent respiratory morbidity due to air pollution is required so that policymakers, parents and health professionals alike can act in a timely manner and accordingly.

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Contributors MSF designed the review protocol, analysed the data, drafted and revised the review. MSF and ERvM performed the data extraction and risk of bias assessments. ABu, PB, AB, EdS and ERvM contributed to critically reviewing and revising the article and approved the final draft. MSF is the guarantor of the work.

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