

ENVIRONMENTAL DETERMINANTS

Panel Studies of Air Pollution on Children's Lung Function and Respiratory Symptoms: A Literature Review

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Objective. This article reviews panel studies of air pollution on children's respiratory health and proposes future research directions. **Methods.** The PubMed electronic database was used to search published original epidemiological studies in peer-reviewed journals from 2000 to November 2011. Children's age was limited to ≤ 18 years old. A total of 33 relevant articles were obtained, with 20 articles relating to lung function, 21 articles relating to respiratory symptoms, and 8 articles examining both. **Results.** Most studies suggested the adverse effects of air pollution on children's lung function and respiratory symptoms. Particles and NO_2 showed more significant results, whereas effects of SO_2 were not consistent. A few studies indicated that O_3 interacted with temperature and sometimes seemed to be a protective factor for children's respiratory health. Negative associations between air pollutants and pulmonary health were more serious in asthmatic children than in healthy subjects. However, many outcomes depended on the number of lag days. Peak expiratory flow (PEF) was the most usual measurement for children's lung function, followed by forced expiratory volume in 1 second (FEV_1). **Conclusions.** There are significant adverse effects of air pollution on children's pulmonary health, especially for asthmatics. Future studies need to examine the lag effects of air pollution on children's lung function and respiratory symptoms. Ambient temperature is predicted to change worldwide due to climate change, which will threaten population health. Further research is needed to examine the effects of ambient temperature and the interactive effects between air pollution and ambient temperature on children's lung function and respiratory symptoms.

Keywords asthmatic, pollutants, pulmonary, students, temperature

INTRODUCTION

Panel studies, a subtype of cohort studies, sample a set of fixed individuals on whom observations are made at regular time intervals, usually close in time (1). As with most longitudinal studies, panel studies provide appropriate evidence for predicting long-term or cumulative cause–effect relationships and allow the investigation of the dynamics of change. However, compared to many longitudinal studies, panel studies are usually conducted over a short period, require intensive observation within this period, and have a relatively narrow focus. Recruitment and retention can therefore be difficult as they are demanding on participants, leading to issues with completeness and sample size. Panel studies are often nested within cross-sectional surveys to provide supplementary information (2).

In recent decades, numerous epidemiological studies have applied the panel design to examine associations between health outcomes and personal environmental exposures. For instance, in a study of respiratory health (3), 38 male patients with chronic pulmonary disease were recruited in a prospective panel study conducted in Erfurt, Germany. Blood markers and daily air pollution were repeatedly measured over an 8-month period. This study found that gaseous and particulate ambient air pollutants induce changes in systemic inflammatory response and

blood coagulability in patients with chronic pulmonary disease. Ward and Ayres (4) systematically reviewed 22 individual and related panel studies to June 2002. They reported the short-term effects of outdoor particulate air pollution on children's peak expiratory flow (PEF) and pulmonary symptoms. They found that most studies showed adverse effects of particulate air pollution on health outcomes, especially for PEF where the effects of particles less than $2.5 \mu\text{m}$ aerodynamic diameters ($\text{PM}_{2.5}$) appeared to be greater than particles less than $10 \mu\text{m}$ aerodynamic diameters (PM_{10}). A systematic review and meta-analysis article investigated acute effects of ambient nitrogen dioxide (NO_2) and PM_{10} on respiratory health outcomes in asthmatic children (5). Included articles were limited to those published from 1990 to July 2008. They concluded that the same-day PM_{10} was associated with increased asthma attacks and cough episodes as well as reduction in PEF, with lagged effects of NO_2 .

Children are potentially a population subgroup particularly vulnerable to the detrimental impacts of environmental risk factors as air pollution (6). There is an increasing concern about children's respiratory health (7). During the stages of rapid growth, immature lungs may be most susceptible to permanent harmful effects (8), as lung development continues throughout childhood into adulthood (9). Furthermore, children have more vigorous activity patterns and spend more time outside than adults (10), so they have greater exposure to ambient air pollutants (11).

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It is important to examine the effects of air pollution on children's health and development in order to ensure appropriate environmental and public health policies. This article is a review of panel studies that have considered effects of air pollution on children's lung function and respiratory symptoms.

METHODS

The PubMed electronic database was used to identify published studies, and the search terms used were "panel study", "lung function", "respiratory symptoms", "pulmonary symptoms", "air pollution", and "air pollutants". The search was limited to original epidemiological studies published from 2000 to November 2011. Titles and abstracts of references and citations in the articles retrieved were screened to further assess and ensure that all relevant articles were included. Among the selected articles, only studies involving children ≤ 18 years old were considered. This review includes only peer-reviewed journal articles and excludes conference abstracts, reports, and books.

In total, 33 articles were obtained through the PubMed database, with 20 articles relating to lung function, 21 articles relating to children's respiratory symptoms, and 8 articles examining both. Study periods covered more than the last dozen of years through to 2009. These studies are summarized in Tables 1 and 2 in ascending chronological order.

Most of the 33 panel studies only controlled for ambient temperature as a confounder, but did not examine the effects of temperature on children's lung function and respiratory symptoms. "Panel study", "lung function", "respiratory symptoms", "pulmonary symptoms", "temperature", "season", and "seasonal" were also used as search terms to review the effects of ambient temperature on children's lung function and respiratory symptoms. No study to date has examined the relationship between ambient temperature and lung function/respiratory symptoms of children using a panel study design.

RESULTS

Air Pollution and Children's Lung Function

Particulate Matter. A European study (12) involving 14 urban and suburban areas found that a $100 \mu\text{g}/\text{m}^3$ increase in 1-day lagged PM_{10} changed the evening PEF deviation ($\Delta\text{PEF}_{\text{pm}}$, population mean deviation from mean evening PEF) by $-1.1 \text{ L}/\text{min}$ (95% CI: $-1.8 \text{ L}/\text{min}$, $-0.4 \text{ L}/\text{min}$). There was no effect of PM_{10} soluble elements on PEF changes. A British study (18) found that winter PM_{10} was associated with large decrements in PEF (defined as more than 20% decrease below the median; odds ratio (OR) = 1.04; 95% CI: 0.99, 1.08), especially for the wheezy children (OR = 1.11; 95% CI: 1.06, 1.17). PM_{10} had no effect on children's mean PEF.

A study of Hispanic asthmatic children in Los Angeles (20) showed that PM_{10} had adverse impacts on evening

PEF but not on morning PEF. Every interquartile range (IQR) increase in PM_{10} was associated with a change of $-3.67 \text{ L}/\text{min}$ (95% CI: $-10.3 \text{ L}/\text{min}$, $2.91 \text{ L}/\text{min}$) in children's evening PEF. The southern California study (23) divided air pollution exposures into personal, indoor, outdoor, and central-site sources and used forced expiratory volume in 1 second (FEV_1) as the observation parameter of asthmatic children's lung function. This study showed diverse negative associations between FEV_1 and particulate matter (PM) exposures from different sources. FEV_1 percent predicted decreases were -6.0% (95% CI: -10.5% , -1.4%) for $128 \mu\text{g}/\text{m}^3$ (1-hour maximum personal PM), -5.9% (95% CI: -10.8% , -1.0%) for $30 \mu\text{g}/\text{m}^3$ (24-hour average personal PM), -1.6% (95% CI: -2.8% , -0.4%) for $6.7 \mu\text{g}/\text{m}^3$ (indoor $\text{PM}_{2.5}$), -2.1% (95% CI: -3.7% , -0.4%) for $16 \mu\text{g}/\text{m}^3$ (indoor PM_{10}), -1.1% (95% CI: -2.4% , 0.1%) for $7.1 \mu\text{g}/\text{m}^3$ (outdoor $\text{PM}_{2.5}$), and -0.7% (95% CI: -1.9% , 0.4%) for $7.5 \mu\text{g}/\text{m}^3$ (central-site $\text{PM}_{2.5}$). Similar outcomes were drawn from a Seattle study (25).

In Mae Moh, Thailand (19, 21), a $10 \mu\text{g}/\text{m}^3$ increase in PM_{10} caused changes in asthmatic children's lung function parameters: PEF (-18.9 mL ; 95% CI: -28.5 mL , -9.3 mL), FEV_1 (-6.0 mL ; 95% CI: -9.2 mL , 2.7 mL), forced vital capacity (FVC) (-6.3 mL ; 95% CI: -9.8 mL , -2.8 mL), forced expiratory flow between 25% and 75% of FVC ($\text{FEF}_{25-75\%}$) (-3.7 mL ; 95% CI: -10.9 mL , 3.5 mL). These children had a heterogeneous response in lung function (interindividual variation for subject-specific changes in lung function) to PM_{10} exposures. However, no consistent relationship was detected between nonasthmatic children's lung function and PM_{10} . A Japanese study of children hospitalized with severe asthma (30) found that morning PEF changed $-3 \text{ L}/\text{min}$ (95% CI: $-4.6 \text{ L}/\text{min}$, $-1.4 \text{ L}/\text{min}$) and evening PEF changed $-4.4 \text{ L}/\text{min}$ (95% CI: $-7.1 \text{ L}/\text{min}$, $-1.7 \text{ L}/\text{min}$) per $10 \mu\text{g}/\text{m}^3$ for the same-day 24-hour mean $\text{PM}_{2.5}$ concentrations. Also, 15–4-hour lagged and 3–0-hour/23–12-hour lagged hourly $\text{PM}_{2.5}$ concentrations increases significantly associated with children's morning and evening PEF, respectively. Almost $-3 \text{ L}/\text{min}$ changes in both morning and evening PEF were observed for an increase of $10 \mu\text{g}/\text{m}^3$ in 7- and 2-hour lagged hourly $\text{PM}_{2.5}$ concentrations. Some associations persisted after adjusting for other pollutants.

An Israeli study (24) from communities near two power plants concluded that the effects of $\text{PM}_{2.5}$ on asthmatic children's PEF also depended on location. In Brazil (28), an increase of $10 \mu\text{g}/\text{m}^3$ in 2-day lagged PM_{10} led to a $0.34 \text{ L}/\text{min}$ reduction of children's PEF.

Traffic-related $\text{PM}_{2.5}$ was found to acutely and adversely affect FEV_1 in Canadian children with asthma, even though the concentrations were relatively low (29). With a $6.0 \mu\text{g}/\text{m}^3$ increase in 1-day lagged $\text{PM}_{2.5}$, children's bedtime FEV_1 decreased 0.54% (95% CI: 0.06% , 1.02%). Results were similar for two-pollutant models with NO_2 , sulfur dioxide (SO_2), and ozone (O_3). An increase of $6.5 \mu\text{g}/\text{m}^3$ in mean daytime $\text{PM}_{2.5}$ was associated with a 0.73% (95% CI: 0.10% , 1.37%) decrease in daytime FEV_1 . In New Zealand (27), peak levels of particulate air pollution from wood burning had a slight impact

TABLE 1.—Panel studies of air pollution and children's lung function.

Study	Period	Area	Pollutants	Lung function	Sample	Analysis method	Temperature controlled	Key findings
Roemer et al. (12)	≥2 months during the winter of 1993/1994	14 urban and suburban European areas: 1. Amsterdam, the Netherlands 2. Kuopio, Finland 3. Norway: Oslo 4/5. Berlin and Hettstedt, Germany 6. Pisa, Italy 7. Athens, Greece 8/9. Cracow and Katowice, Poland 10/11. Prague and Teplice, Czech Republic 12. Budapest, Hungary 13/14. Umeå and Malmö, Sweden	24-hour PM ₁₀	Morning and evening PEF	1208 asthmatic children (6–12 yrs)	Linear regression	Minimum	Acute PEF changes of European asthmatic children were weakly affected by soluble elemental concentration's daily fluctuations in ambient particulate matters.
Jalaludin et al. (13)	1 February–31 December 1994	Sydney, Australia	NO ₂ O ₃ PM ₁₀	Evening PEF	125 primary school children with a history of wheeze (mean age 9.6–9.7 yrs)	GEE and population regression	Daytime average	Same-day daytime mean O ₃ had adverse effects on daily mean deviations of PEF in children with a history of wheeze, especially on the ones with bronchial hyperactivity, and doctor diagnosed asthma. But same-day daytime maximum O ₃ had no effect on children's PEF.
Declercq and Macquet (14)	1 April–30 June 1996	Armentieres, France	O ₃	Morning and evening PEF	91 school children (10 yrs)	GEE	Average	Even moderate levels of O ₃ decreased children's evening PEF.
Mortimer et al. (15)	1 June–31 August 1993	8 US urban cities: Bronx East Harlem Baltimore Washington DC Detroit Cleveland Chicago St. Louis	NO ₂ O ₃ PM ₁₀ SO ₂	Morning and evening PEF	846 asthmatic children (4–9 yrs)	GEE and mixed model	–	O ₃ decreased children's morning PEF. No pollutant was associated with children's evening PEF.
Just et al. (16)	1 April–30 June 1996	Paris, France	BS NO ₂ O ₃ PM SO ₂	PEF	82 asthmatic children (7–15 yrs)	GEE	Average	O ₃ exposures decreased children's PEF and enhanced their PEF variability.

TABLE 1.—(Continued).

Study	Period	Area	Pollutants	Lung function	Sample	Analysis method	Temperature controlled	Key findings
Ward et al. (17)	13 January–10 March 1997 (winter) and 19 May–14 July 1997 (summer)	Birmingham and Sandwell, UK	NO ₂ O ₃ PM _{2.5} PM ₁₀ SO ₂	Morning and afternoon PEF	162 asthmatic and nonasthmatic primary school children (9 yrs)	Linear regression	Minimum	Air pollution levels were usually low, particularly in summer. Pollutants' concentrations were detected and had associations with children's PEF, but not consistent for the whole panel. Asthmatic children were not more susceptible to air pollution effects.
Peacock et al. (18)	63 schooldays during 1 November 1996–14 February 1997	Medway district, UK (one rural and two urban areas)	NO ₂ O ₃ PM ₁₀ SO ₂	Morning PEF	179 school children (7–13 yrs)	AR and GEE	Average	Mean PEF was not affected by any of the pollutants. NO ₂ and PM ₁₀ had adverse effects on PEF large decrements, and effects of PM ₁₀ were stronger in wheezers. O ₃ or SO ₂ had no effect on large decrements of PEF.
Aekplakorn et al. (19)	61 days during 1 October 1997–30 November 1997	Mae Moh, Thailand	24-hour: PM ₁₀ SO ₂	Afternoon: FEV _{25–75%} FEV ₁ FVC PEF	83 asthmatic and 92 nonasthmatic children (6–14 yrs)	GLM	Average	For asthmatic children, PM ₁₀ had slight effects on their lung function, whereas SO ₂ had no effect on them. No association was found between air pollution and lung function in nonasthmatic children.
Delfino et al. (20)	3 months during 4 November 1999–23 January 2000	Los Angeles, USA	Hourly: CO NO ₂ O ₃ SO ₂ 24-hour: EC-OC PM ₁₀ fractions PM ₁₀ VOCs	Morning and evening PEF	22 Hispanic asthmatic children (10–16 yrs)	Mixed model	1-hour maximum	Particulate matters had adverse effects on children's evening PEF, but not on their morning PEF.
Aekplakorn et al. (21)	61 days during the winter of 1997	Mae Moh, Thailand	24-hour: PM ₁₀ SO ₂	FEV _{25–75%} FEV ₁ FVC PEF	83 asthmatic school children (6–14 yrs)	GLM	Average	Interindividual variations of FEV ₁ , FVC, and PEF caused by children's PM ₁₀ and SO ₂ exposures, despite the concentrations of pollutants, were relatively low in Mae Moh of Thailand.
Ranzi et al. (22)	12 weeks during February–May 1999	Emilia-Romagna, Italy (urban-industrial and rural areas)	24-hour: CO NO ₂ PM _{2.5} TSP	Morning and evening PEF	118 asthma-like primary school children (6–11 yrs)	GLM	Average	Urban-industrial areas had higher daily pollutants' concentrations than rural areas. Asthmatic children and cough subsets in urban-industrial areas had significantly higher morning and evening PEF variations.

TABLE 1.—(Continued).

Study	Period	Area	Pollutants	Lung function	Sample	Analysis method	Temperature controlled	Key findings
Delfino et al. (23)	2 weeks either during August–October 1999 or April–June 2000	Southern California, USA	NO ₂ O ₃ PM _{2.5} PM ₁₀	Morning, afternoon and evening FEV ₁	19 asthmatic children (9–17 yrs)	GLM	Average	Personal and stationary-site exposures to PM both had negative effects on FEV ₁ , whereas personal PM showed stronger associations. Indoor PM showed stronger inverse associations with FEV ₁ than outdoor and central-site PM. FEV ₁ were slightly affected by NO ₂ , but not by O ₃ . PM _{2.5} had significant effects on PEF in one community. But in another community, these were the interactions between PM ₁₀ and the sequential days of the year. So asthmatic children were affected by particles and geophysical situations.
Peled et al. (24)	March–June 1999 (spring) and September–December 1999 (autumn)	3 communities near two power plants in Ashkelon, Israel	PM _{2.5} PM ₁₀	Morning and evening PEF	285 asthmatic school children (10 and 12 yrs)	GEE and GLM	Maximum	For asthmatic children not receiving anti-inflammatory medication, same-day personal, indoor, and outdoor exposures to PM _{2.5} were related to FEV ₁ , MMEF, and PEF decreases. 1-day lagged personal PM _{2.5} decreased children's PEF, whereas 1-day lagged indoor PM _{2.5} decreased their FEV ₁ , MMEF, and PEF. Anti-inflammatory medication significantly attenuated the detrimental effects of PM on children's lung function. NO ₂ and particles had adverse impacts on most lung function parameters. NO ₂ concentrations below current European limits can even affect lung function of healthy school children.
Trenga et al. (25)	October 1999–August 2000, October 2000–May 2001 and October 2001–February 2002	Seattle, USA	24-hour personal, indoor, and outdoor: PM _{2.5} PM ₁₀	FEV ₁ MMEF PEF	17 asthmatic children (6–12 yrs)	Mixed model	Average	For asthmatic children not receiving anti-inflammatory medication, same-day personal, indoor, and outdoor exposures to PM _{2.5} were related to FEV ₁ , MMEF, and PEF decreases. 1-day lagged personal PM _{2.5} decreased children's PEF, whereas 1-day lagged indoor PM _{2.5} decreased their FEV ₁ , MMEF, and PEF. Anti-inflammatory medication significantly attenuated the detrimental effects of PM on children's lung function. NO ₂ and particles had adverse impacts on most lung function parameters. NO ₂ concentrations below current European limits can even affect lung function of healthy school children.
Moshhammer et al. (26)	September 2000–August 2001	Linz, Upper Austria	NO ₂ PM ₁ PM _{2.5} PM ₁₀	FEV _{0.5} FEV ₁ FVC MEF _{25%} MEF _{50%} MEF _{75%} PEF	163 primary school children (7–10 yrs)	GEE	—	NO ₂ and particles had adverse impacts on most lung function parameters. NO ₂ concentrations below current European limits can even affect lung function of healthy school children.
Epton et al. (27)	Winter 2004	Christchurch, New Zealand	24-hour indoor and outdoor PM from wood burning	Morning and evening: FEV ₁ PEF	93 male secondary school students including 26 asthmatics living in the boarding house (12–18 yrs)	Mixed model	Average	For healthy school students, there was no significant effect of wood-burning PM on lung function. Peak PM levels had small effects on asthmatic students' lung function.

TABLE 1.—(Continued).

Study	Period	Area	Pollutants	Lung function	Sample	Analysis method	Temperature controlled	Key findings
Castro et al. (28)	6 weeks during May, June, September, and October 2004	Rio de Janeiro, Brazil	24-hour: CO NO ₂ O ₃ PM ₁₀ SO ₂	PEF	118 school children (6–15 yrs)	Gaussian multilevel model	Average	NO ₂ and PM ₁₀ were associated with decreased PEF of children, while the effects of CO and SO ₂ were not statistically significant. O ₃ showed protective results to children's PEF.
Dales et al. (29)	28 consecutive days during 11 October–7 November 2005 or 14 November–11 December 2005	Windsor, Canada	Hourly traffic-related: NO ₂ O ₃ PM _{2.5} SO ₂	Morning and evening FEV ₁	182 asthmatic primary school children (9–14 yrs)	AR and mixed model	Average	Even low concentrations of urban traffic-related air pollution can decrease children's FEV ₁ in short period, such as in a day. PM _{2.5} appeared to have the most severe effect.
Yamazaki et al. (30)	1 October–24 December 2000	Japan: Yotsukaido City in Tokyo	Hourly and 24-hour: NO ₂ O _x PM _{2.5} NO ₂ O _x PM _{2.5}	Morning and evening PEF	17 severe asthmatic children (8–15 yrs)	GEE	Average	Increased concentrations of PM _{2.5} were related to the decreases in PEF among hospitalized severe asthmatic children in Yotsukaido City.
Islam et al. (31)	School year during 2008–2009	Southern California, USA	Traffic-related: NO NO ₂ NO _x	FEF _{25–75%} FEV ₁ FVC	1399 children (mean age 11.2 yrs)	Mixed model	—	High parental stress increased negative effects of traffic-related air pollution on both asthmatic and nonasthmatic children's lung function (FEV ₁ and FVC). The results for FEF _{25–75%} were not statistically significant.

AR, autoregressive model; BS, black smoke; CO, carbon monoxide; EC, elemental carbon; FEF_{25–75%}, forced expiratory flow between 25% and 75% of forced vital capacity; FEV_{0.5}, forced expiratory volume in 0.5 seconds; FEV₁, forced expiratory volume in 1 second; FVC, forced vital capacity; GEE, generalized estimating equation; GLM, general linear mixed model; MEF_{25%}, maximal expiratory flow at 25% of forced vital capacity; MEF_{50%}, maximal expiratory flow at 50% of forced vital capacity; MEF_{75%}, maximal expiratory flow at 75% of forced vital capacity; MMEF, maximum mid-expiratory flow; NO₂, nitrogen dioxide; NO_x, total oxides of nitrogen; O₃, ozone; OC, organic carbon; O_x, photochemical oxidants; PEF, peak expiratory flow; PM, particulate matter; PM₁, particles less than 1 µm aerodynamic diameters; PM_{2.5}, particles less than 2.5 µm aerodynamic diameters; PM₁₀, particles less than 10 µm aerodynamic diameters; SO₂, sulfur dioxide; TSP, total suspended particulate; VOCs, volatile organic compounds; yrs, years old.

TABLE 2.—Panel studies of air pollution and children's respiratory symptoms.

Study	Period	Area	Pollutants	Respiratory symptoms	Sample	Analysis method	Temperature controlled	Key findings
Smith et al. (32)	6 weeks during 1999	Adelaide, Australia	Daily indoor: NO ₂ O ₃ SO ₂	Breathlessness Breathlessness on exertion Cough Chest tightness Daytime asthma attacks Night asthma attacks Wheeze	125 asthmatics (2–84 yrs: ≤14, 15–34, 35–49, ≥50 yrs)	GEE	Minimum	For subjects aged ≤14 years, NO ₂ was significantly associated with same-day and 1-day lagged chest tightness, 1-day lagged breathlessness on exertion, same-day daytime asthma attacks, and same-day and 1-day lagged night asthma attacks with adjustments of potential confounders.
Roemer et al. (12)	≥2 months during the winter of 1993/1994	14 urban and suburban European areas: 1. Amsterdam, the Netherlands 2. Kuopio, Finland 3. Oslo, Norway 4/5. Berlin and Hettstedt, Germany 6. Pisa, Italy 7. Athens, Greece 8/9. Cracow and Katowice, Poland 10/11. Prague and Teplice, Czech Republic 12. Budapest, Hungary 13/14. Umeå and Malmö, Sweden	24-hour: BS NO ₂ PM ₁₀ SO ₂	Attacks of shortness of breath with wheeze Cough Eye irritations Fever Phlegm Runny/stuffed nose Shortness of breath Sore throat Wake up with breathing problems Wheeze	1208 asthmatic children (6–12 yrs)	Logistic regression	Minimum	Acute respiratory symptoms of European asthmatic children were weakly affected by most of soluble elemental concentration's daily fluctuations in ambient particulate matters, but the results were not consistent.
Yu et al. (33)	Average 58 days (28–112 days) during 3 November 1993–15 August 1995	Seattle, USA	CO PM ₁ PM ₁₀ SO ₂	Cough Chest tightness Shortness of breath Wake up with asthma Wheeze	133 asthmatic children (5–13 yrs)	GEE	Average	Analyzed by single-pollutant models, increases of 1-day lagged CO, same-day PM ₁ and 1-day lagged PM ₁₀ were associated with asthma symptoms in children. The relationships between SO ₂ and asthma symptoms were not found. In multipollutant models, effects of separate pollutants were minor. CO and PM ₁ had overall effects on children's respiratory symptoms in Seattle.

TABLE 2.—(Continued).

Study	Period	Area	Pollutants	Respiratory symptoms	Sample	Analysis method	Temperature controlled	Key findings
Declercq and Macquet (14)	1 April–30 June 1996	Armentieres, France	O ₃	Cough Shortness of breath Wheeze	91 school children (10 yrs)	GEE	Average	Even moderate levels of O ₃ increased children's daily occurrences of cough.
Ostro et al. (34)	13 weeks during August–October 1993	Los Angeles, USA	NO ₂ O ₃ PM _{2.5} PM ₁₀	Cough Shortness of breath Wheeze	138 African-American asthmatic children (6–13 yrs)	GEE	Maximum	New cough episodes were affected by ambient NO ₂ , PM _{2.5} , and PM ₁₀ , but not by O ₃ .
Vichit-Vadakan et al. (35)	69 days from 9 January 1996	Bangkok, Thailand	PM _{2.5} PM ₁₀	Cough Shortness of breath Wheeze Lower respiratory symptoms Upper respiratory symptoms	79 children (8–12 yrs)	Mixed model	Average	PM _{2.5} and PM ₁₀ were associated with children's daily incidences of both upper and lower respiratory symptoms, but had no effect on first-day symptoms episodes in children.
Mortimer et al. (15)	1 June–31 August 1993	8 US urban cities: Bronx East Harlem Baltimore Washington DC Detroit Cleveland Chicago St. Louis	NO ₂ O ₃ PM ₁₀ SO ₂	Cough Chest tightness Wheeze	846 asthmatic children (4–9 yrs)	GEE and mixed model	–	Single-pollutant models showed that each pollutant increased children's incidences of morning respiratory symptoms. No pollutant was associated with children's evening respiratory symptoms.
Schlink et al. (36)	759 days during 1 February 1994–29 February 1996	Leipzig, Germany	NO ₂ O ₃ SO ₂ TSP Meteorological factors: Humidity Radiation Temperature Vapor pressure	Barking cough Cold (white/yellowish nasal discharge) Dry hacking cough Hoarseness Nonproductive cough Productive cough Runny/stuffed nose Sneeze	277 pre-school children (3–7 yrs)	Logistic regression	Daytime maximum	When added estimated parameters into a simulation study, an annual cycle of respiratory symptoms was related to atmospheric covariates. Allergic children were particularly susceptible to O ₃ . Respiratory symptoms' durations were free of scale and followed an exponential distribution function, which proved that each child's respiratory symptom records followed a Poisson point-process.
Delfino et al. (37)	61 days during 1 March–30 April 1996	Southern California, USA	NO ₂ O ₃ PM ₁₀	Cough Chest tightness Phlegm Shortness of breath Wheeze	22 asthmatic children (9–19 yrs)	GEE	1-hour average	Effects of air pollution on respiratory symptoms were much stronger in 12 asthmatic children who did not take anti-inflammatory medications than in the other 10 children who did.

TABLE 2.—(Continued).

Study	Period	Area	Pollutants	Respiratory symptoms	Sample	Analysis method	Temperature controlled	Key findings
Just et al. (16)	1 April–30 June 1996	Paris, France	BS NO ₂ O ₃ PM SO ₂	Asthma attacks Eye irritations Nocturnal cough Nose irritations Respiratory infections Throat irritations Wheeze	82 asthmatic children (7–15 yrs)	GEE	Average	BS and NO ₂ increased nocturnal cough and respiratory infections in children. O ₃ increased their asthma attacks and respiratory infections. O ₃ and temperature as well as O ₃ and pollen counts statistical significantly interacted with children's asthma attacks. O ₃ had greater impacts on children's eye, nose, and throat irritations. PM was related only with eye irritations in children.
Ward et al. (17)	13 January–10 March 1997 (winter) and 19 May–14 July 1997 (summer)	Birmingham and Sandwell, UK	NO ₂ O ₃ PM _{2.5} PM ₁₀ SO ₂	Cough Shortness of breath Wake up Wheeze	162 asthmatic and nonasthmatic primary school children (9 yrs)	Logistic regression	Minimum	Air pollution levels were usually low, particularly in summer. Pollutants' concentrations were detected and had associations with children respiratory symptoms, but not consistent. Asthmatic children were not more susceptible to air pollution effects.
Slaughter et al. (38)	Average 58 days (28–112 days) during November 1993–August 1995	Seattle, USA	CO PM _{2.5} PM ₁₀	Severity levels	133 asthmatic children (5–12 yrs)	Logistic regression and Poisson regression	Average	PM _{2.5} and PM ₁₀ were associated with increased respiratory symptoms severity levels of asthmatic children in Seattle.
Aekplakorn et al. (39)	107 winter days during 1 October 1997–15 January 1998	Mae Moh, Thailand	24-hour: PM _{2.5} PM ₁₀ SO ₂	Cough Lower respiratory symptoms Upper respiratory symptoms	98 asthmatic and 98 nonasthmatic children (6–14 yrs)	GAM, GEE, and logistic regression	Average	PM ₁₀ increased lower respiratory symptoms and cough in asthmatic children, whereas SO ₂ had no effect on both asthmatic and nonasthmatic children's respiratory symptoms.
Delfino et al. (20)	3 months during 4 November 1999–23 January 2000	Los Angeles, USA	Hourly: CO NO ₂ O ₃ SO ₂ 24-hour: PM ₁₀ EC-OC PM ₁₀ fractions	Cough Chest tightness Phlegm Shortness of breath Wheeze	22 Hispanic asthmatic children (10–16 yrs)	GEE	1-hour maximum	Positive associations of children's respiratory symptoms were found with NO ₂ , O ₃ , SO ₂ , PM ₁₀ , EC-OC, and VOCs.

TABLE 2.—(Continued).

Study	Period	Area	Pollutants	Respiratory symptoms	Sample	Analysis method	Temperature controlled	Key findings
Jalaludin et al. (40)	11 months during February–December 1994	Sydney, Australia	NO ₂ O ₃ PM ₁₀	Evening: Dry cough Wet cough Wheeze	125 primary school children with a history of wheeze (mean age 9.6–9.7 yrs)	GEE	Daytime average	NO ₂ exacerbated children's wet cough. O ₃ had no effect on children's respiratory symptoms.
Mar et al. (41)	April 1997–June 1999	Washington, USA	PM ₁ PM _{2.5} PM ₁₀ PM coarse fraction	Cough Eye irritations Phlegm Runny/stuffed nose Shortness of breath Wheeze	9 asthmatic children (7–12 yrs)	GEE	Average	Strong associations were found between cough and PM ₁ , PM _{2.5} , PM ₁₀ , and PM coarse fraction in children. PM ₁₀ and PM coarse fraction increased children's phlegm and runny/stuffed nose.
Ranzi et al. (22)	12 weeks during February–May 1999	Emilia-Romagna, Italy (urban-industrial and rural areas)	CO NO ₂ PM _{2.5} TSP	Attacks of shortness of breath with wheeze Cough Eye irritations Phlegm Shortness of breath Sore throat Runny/stuffed nose Wake up Wheeze	118 asthma-like primary school children (6–11 yrs)	GLM	Average	Urban-industrial areas had higher daily air pollution concentrations than rural areas. Higher respiratory symptoms' occurrences were observed in the urban-industrial areas than the rural areas, and the most prevalent ones were cough, phlegm, and runny/stuffed nose, due to the higher exposures of air pollution in urban-industrial areas. For the entire panel, TSP increased risks of cough and phlegm in children. There were significant associations between PM _{2.5} and cough–phlegm together for the urban-industrial group.
Lee et al. (42)	1–15 April, July, October, and December 2003	Seoul, Korea	CO NO ₂ PM _{2.5} SO ₂	Cough Chest tightness Ear irritations Eye irritations Hospital visits Nose irritations Phlegm Sore throat Take medication Wake up Wheeze	177 primary school children (9 and 10 yrs)	GEE	Average	Same-day NO ₂ exposures increased upper and lower respiratory symptoms of children. Same-day CO and SO ₂ exposures increased their lower respiratory symptoms.

TABLE 2.—(Continued).

Study	Period	Area	Pollutants	Respiratory symptoms	Sample	Analysis method	Temperature controlled	Key findings
Schilderout et al. (43)	22 months during November 1993–September 1995	8 North American cities: 1. Albuquerque, New Mexico 2. Baltimore, Maryland 3. Boston, Massachusetts 4. Denver, Colorado 5. San Diego, California 6. Seattle, Washington 7. St. Louis, Missouri 8. Toronto, Ontario, Canada	CO NO ₂ O ₃ PM ₁₀ SO ₂	Severity levels	990 asthmatic children (5–12 yrs)	GEE, logistic regression, and Poisson regression	1-hour maximum	Lagged CO and NO ₂ exacerbated asthma symptoms, and the 3-day moving sums of SO ₂ were slightly associated with asthma symptoms. O ₃ and PM ₁₀ had no relationship with asthma symptoms. The strongest effects were 2-day lagged CO and NO ₂ on asthma symptoms.
Epton et al. (27)	Winter 2004	Christchurch, New Zealand	Indoor and outdoor PM from wood burning	Cough	93 male secondary school students including 26 asthmatics living in the boarding house (12–18 yrs)	Mixed model	Average	For nonasthmatic school students, there were minor effects of wood-burning PM on respiratory symptoms. High air pollution exposures were related to slight increases in respiratory symptoms.
Escamilla-Nunez et al. (44)	Fortnight evaluation in average 22 weeks during July 2003–March 2005	Mexico City	Traffic-related: NO ₂ O ₃ PM _{2.5}	Cough Wheeze	147 asthmatic and 50 nonasthmatic children (6–14 yrs)	Mixed model and Poisson regression	Minimum	Traffic-related NO ₂ , O ₃ , and PM _{2.5} exacerbated respiratory symptoms as cough and wheeze of children, especially for asthmatics.

BS, black smoke; CO, carbon monoxide; EC, elemental carbon; GAM, generalized additive model; GEE, generalized estimating equation; GLM, general linear mixed model; NO₂, nitrogen dioxide; O₃, ozone; OC, organic carbon; PM, particulate matter; PM₁, particles less than 1 µm aerodynamic diameters; PM_{2.5}, particles less than 2.5 µm aerodynamic diameters; PM₁₀, particles less than 10 µm aerodynamic diameters; SO₂, sulfur dioxide; TSP, total suspended particulate; VOCs, volatile organic compounds; yrs, years old.

on lung function in asthmatic children, but none in a healthy group.

NO₂. In Southern England (18), winter 24-hour average NO₂ was significantly associated with the occurrence of large decrements in PEF in children (OR = 1.043; 95% CI: 1.000, 1.089), but had no impact on mean PEF.

The recently published study (31) based on the Southern California Children's Health Study in the United States assessed whether children's psychosocial stresses had joint effects with traffic-related air pollution on lung function. With each 21.8 ppb increase in NO₂ exposure, asthmatic children within families with high parental stresses had -4.5% (95% CI: -6.5%, -2.4%) and -2.8% (95% CI: -5.7%, 0.3%) changes in FEV₁ at homes and schools, respectively. Similar results were also observed for FVC and nonasthmatics. Nevertheless, no significant effect of traffic-related NO₂ on children's lung function was detected from low-stress households.

In Brazil (28), for a 10 µg/m³ increase in NO₂ exposures, children's PEF decreased between 0.23 L/min and 0.28 L/min.

In Austria at Linz, where concentrations were below the current European standards in multipollutant models, a 10 µg/m³ increase in NO₂ exposures also reduced healthy school children's FEV₁ by 1.01%, maximal expiratory flow at 50% of FVC (MEF_{50%}) by 1.99%, and maximal expiratory flow at 25% of FVC (MEF_{25%}) by 1.96% (26).

SO₂. A southern England study (18) found no effect of SO₂ on children's PEF, for either mean PEF or large decrements of it. In Thailand (19, 21), no association was found between SO₂ exposures and lung function of children with or without asthma. However, asthmatic children had inter-individual variations in PEF, FEV₁, and FVC to SO₂ concentrations fluctuations in the same panel study. SO₂ effects on PEF of Brazilian students were not statistically significant (28).

O₃. In northern France (14), each 30 µg/m³ increase in daily 8-hour maximum O₃ resulted in 1.91 L/min (95% CI: -0.02 L/min, 3.84 L/min) decreases of children's evening PEF. Another French study (16) demonstrated that a 10 µg/m³ increase in the same-day 2- and 4-day lagged mean O₃ enhanced daily PEF variability of asthmatic children by 2.6% ($p = .05$) and 3.3% ($p = .09$), respectively. Although not significant, the effects doubled for the days when no anti-inflammatory medication was used.

A study conducted in eight urban cities of the United States (15) showed that a 15 ppb increase in O₃ in 5-day average concentrations caused 0.59% (95% CI: 0.13%, 1.05%) decreases in asthmatic children's morning PEF but played no role on their evening PEF.

In the Brazilian study (28), O₃ seemed to be a protective factor for children's lung function. A 10 µg/m³ increase in 1-day lagged O₃ increased children's PEF by 0.2 L/min.

A panel study of Australian children with a history of wheeze (13) showed significant inverse associations between the same-day daytime mean O₃ and PEF's daily mean deviations (β -coefficient = 0.88 and $p = .04$).

Furthermore, associations were stronger for children with bronchial hyperreactivity and asthma (β -coefficient = -2.61 and $p = .001$). In addition, a dose-response relationship with daytime mean O₃ was demonstrated. Nevertheless, the same-day daytime maximum O₃ was not significantly associated with children's PEF.

Air Pollution and Children's Respiratory Symptoms

Particulate Matter. A French study (16) showed a 10 µg/m³ increase in PM levels related to asthmatic children's eye irritations (OR = 1.18; 95% CI: 1.01, 1.39 for the same-day effects, OR = 1.28; 95% CI: 1.03, 1.59 for 2-day lagged effects, and OR = 1.42; 95% CI: 1.12, 1.80 for 4-day lagged effects). The effects were stronger when analyses were restricted to the days when children did not take anti-inflammatory medications. In the urban-industrial areas of Italy (22), a 10 µg/m³ increase in PM_{2.5} increased the risk of children's cough and phlegm together (relative ratio (RR) = 1.0044; 95% CI: 1.0011, 1.0077).

In Seattle (33), a 10 µg/m³ increase in the same-day particles less than 1 µm aerodynamic diameters (PM₁) and 1-day lagged PM₁₀ by single-pollutant models had adverse effects on respiratory symptoms of asthmatic children, and the estimated increments were 18% (95% CI: 5%, 33%) and 11% (95% CI: 3%, 20%), respectively. For the previous day's symptoms effects, there were also 14% (95% CI: 4%, 26%) and 10% (95% CI: 3%, 16%) increases of asthma symptoms for a 10 µg/m³ increase in PM₁ and PM₁₀. Another article (38) published for the same study in the United States examined the relationship between air pollution and asthma symptoms severity levels in children. A 10 µg/m³ increase in 1-day lagged PM_{2.5} and PM₁₀ was associated with children's serious asthma attacks (OR = 1.20; 95% CI: 1.05, 1.37 and OR = 1.12; 95% CI: 1.04, 1.22, respectively). For African-American asthmatic children in Los Angeles (34), a 30 µg/m³ increase in 12-hour average PM_{2.5} exposures and every 17 µg/m³ increase in 24-hour average PM₁₀ exposures increased the risk of new episodes of cough (OR = 1.10; 95% CI: 1.03, 1.18 and OR = 1.25; 95% CI: 1.12, 1.39, respectively). Another US study taken in eight urban cities (15) indicated that every 20 µg/m³ increase in 2-day average PM₁₀ increased asthmatic children's morning incidences of respiratory symptoms by single-pollutant models (OR = 1.26; 95% CI: 1.00, 1.59), but had no effect on children's evening respiratory symptoms. In southern California (37), minimum to 90th-percentile exposure levels were 12-63 µg/m³ for 1-hour PM₁₀, 8-46 µg/m³ for 8-hour PM₁₀, and 7-32 µg/m³ for 24-hour PM₁₀ at the monitoring site. For the 12 asthmatic children who did not take anti-inflammatory medications, minimum particulate pollutant changes were related to children's asthma episodes (OR = 1.92; 95% CI: 1.22, 3.02 for 1-hour maximum PM₁₀, OR = 1.68; 95% CI: 0.91, 3.09 for 8-hour maximum PM₁₀, and OR = 1.35; 95% CI: 0.82, 2.22 for 24-hour average PM₁₀). For Hispanic asthmatic children in Los Angeles of the United States (20), a 37 µg/m³ increase in PM₁₀ was related to their asthma symptoms (OR = 1.45;

95% CI: 1.11, 1.90). In Washington of the United States (41), a 10 $\mu\text{g}/\text{m}^3$ increase in the same-day, 1-day lagged, and 2-day lagged PM_{10} exposures was significantly associated with increase in cough in asthmatic children (OR = 1.09; 95% CI: 1.02, 1.16, OR = 1.08; 95% CI: 1.02, 1.16, and OR = 1.10; 95% CI: 1.02, 1.18, respectively). Associations were also found for a 10 $\mu\text{g}/\text{m}^3$ increase in 1-day lagged $\text{PM}_{2.5}$ exposures and children's cough (OR = 1.21; 95% CI: 1.00, 1.47).

A Thailand study (35) showed that a 45 $\mu\text{g}/\text{m}^3$ increase in PM_{10} was associated with children's both lower and upper respiratory symptoms incidences (OR = 1.13; 95% CI: 1.05, 1.23 for lower respiratory symptoms and OR = 1.10; 95% CI: 1.02, 1.18 for upper respiratory symptoms). In Mae Moh (39), Thailand, a 10 $\mu\text{g}/\text{m}^3$ increase in 24-hour PM_{10} from a power plant was correlated with increased lower respiratory symptoms incidences and cough in asthmatic children (OR = 1.03; 95% CI: 0.98, 1.09 and OR = 1.04; 95% CI: 1.00, 1.08, respectively). In Mexico City (44), a 17.4 $\mu\text{g}/\text{m}^3$ increase in traffic-related $\text{PM}_{2.5}$ increased cough and wheeze among asthmatic children (OR = 1.111; 95% CI: 1.056, 1.169 and OR = 1.088; 95% CI: 1.024, 1.155, respectively). Effects were stronger for healthy children.

NO_2 . For a study of eight US urban cities (15), by means of single-pollutant models, every 20 ppb increase in 6-day average NO_2 level was associated with asthmatic children's morning incidences of respiratory symptoms (OR = 1.48; 95% CI: 1.02, 2.16), but not with their evening symptoms. In southern California, USA (37), minimum to 90th-percentile 8-hour NO_2 levels was 6–26 ppb for the monitoring site. Minimum 8-hour maximum NO_2 changes were related to asthma episodes among 12 asthmatic children not using anti-inflammatory medications (OR = 1.91; 95% CI: 1.07, 3.39). In Los Angeles, USA (20), every 1.4 ppb increase in 8-hour NO_2 level enhanced Hispanic asthmatic children's respiratory symptoms (OR = 1.27; 95% CI: 1.05, 1.54). For a study of eight northern American cities (43), each 20 ppb increase in 24-hour NO_2 level was correlated with children's asthma symptoms (OR = 1.06; 95% CI: 1.00, 1.13 for the same-day effects, OR = 1.04; 95% CI: 0.97, 1.10 for the 1-day lagged effects, OR = 1.09; 95% CI: 1.03, 1.15 for the 2-day lagged effects, and OR = 1.04; 95% CI: 1.01, 1.07 for the 3-day lagged effects), whereas 2-day lagged NO_2 showed strongest effects.

A Korean study (42) indicated that a 17.3 ppb increase in the same-day NO_2 exposures exacerbated children's respiratory symptoms (OR = 1.12; 95% CI: 1.01, 1.24 for their upper respiratory symptoms and OR = 1.18; 95% CI: 1.06, 1.31 for their lower respiratory symptoms). In Mexico City (44), among asthmatic children, ORs were 1.075 (95% CI: 1.018, 1.136) for increased cough and 1.091 (95% CI: 1.023, 1.164) for increased wheeze, for a 34 ppb increase in the previous-day 1-hour maximum NO_2 levels. Significant associations were observed between 2-day 1-hour maximum NO_2 levels and cough for healthy children (OR = 1.22; 95% CI: 1.03, 1.45) for each 28.5 ppb increase.

An Australian study (32) showed that among subjects aged ≤ 14 years, a 1 standard deviation (1SD) increase in NO_2 was significantly correlated with their same-day and 1-day lagged chest tightness (OR = 1.29; 95% CI: 1.16, 1.43 and OR = 1.29 (95% CI: 1.14, 1.46); 1-day lagged breathlessness on exertion (OR = 1.13; 95% CI: 1.00, 1.28); same-day daytime asthma attacks (OR = 1.13; 95% CI: 1.02, 1.26); and same-day as well as 1-day lagged night asthma attacks (OR = 1.16; 95% CI: 1.03, 1.30 and OR = 1.15; 95% CI: 1.03, 1.29). An Australian study in Sydney (40) indicated that every 0.82 ppb increase in NO_2 concentrations exacerbated children's wet cough (RR = 1.05; 95% CI: 1.003, 1.10) in single-pollutant models.

SO_2 . A study of eight US urban cities by single-pollutant models (15) showed every 20 ppb increase in 2-day average SO_2 exposures exacerbated asthmatic children's respiratory symptoms (OR = 1.32; 95% CI: 1.03, 1.70), but did not affect their evening asthma symptoms. The study of eight Northern American cities (43) showed that only 1 ppb change in the 3-day moving sums of SO_2 levels slightly associated with children's asthma symptoms (OR = 1.04; 95% CI: 1.00, 1.08). A study in Seoul, Korea (42), indicated that every 2.6 ppb increase in SO_2 exposures in the same day was correlated with children's lower respiratory symptoms (OR = 1.12; 95% CI: 1.01, 1.25).

O_3 . In France (14), every 30 $\mu\text{g}/\text{m}^3$ increase in O_3 raised children's daily cough occurrences (OR = 1.17; 95% CI: 0.97, 1.40). In Paris (16), increased O_3 concentrations led to children's asthma attacks and respiratory infections. Temperature and pollen counts were statistical significantly interacted with O_3 for children's asthma attacks. Adverse effects of O_3 were observed in children's pulmonary irritations.

In the United States (15), single-pollutant models indicated that a 15 ppb increase in 4-day average O_3 exposures exacerbated asthmatic children's morning incidences of respiratory symptoms in eight urban cities (OR = 1.16; 95% CI: 1.02, 1.30). But O_3 and evening asthma symptoms showed no significant association. In southern California (37), minimum to 90th-percentile 1-hour O_3 concentrations was 45–88 ppb in the monitoring site. Among asthmatic children who did not take anti-inflammatory medications, minimum 1-hour maximum O_3 differences were associated with respiratory symptoms episodes (OR = 1.28; 95% CI: 0.75, 2.17).

In Mexico City (44), a 48 ppb increase in 1-hour maximum traffic-related O_3 increased cough and wheeze among children with asthma (OR = 1.089; 95% CI: 1.030, 1.151 and OR = 1.100; 95% CI: 1.032, 1.173, respectively).

DISCUSSION

In recent decades, many studies have employed panel designs to examine the effects of air pollution on subjects' lung function and respiratory symptoms. Such studies are particularly important for children who are one of the most

vulnerable subgroups in the population. This review is crucial because there are currently few reviews which have summarized and commented on such studies in children. Also, while panel studies have some limitations compared to cross-sectional studies, by and large, they have significant strengths (45). In particular, panel studies are not only a good choice to focus on the short-term effects of environmental factors on population health but are also easier to conduct than cohort studies. This literature review provides details of the current state of play of panel studies of the effects of air pollution on children's respiratory symptoms and proposes directions for the future research.

Over 30 relevant panel studies of the effects of air pollution on children's lung function and respiratory symptoms were reviewed. While the majority suggested adverse effects, estimates varied considerably. In general, particles and NO₂ showed more significant results, whereas the health effects of SO₂ were not consistent. A few studies indicated that O₃ interacted with temperature and sometimes seemed to be a protective factor for children's respiratory health. Additionally, negative associations between air pollutants and pulmonary health were found more in asthmatic or asthma symptomatic children than in healthy subjects. Many outcomes depended on the number of lag days and asthma medications tended to alleviate the adverse effects of air pollution on children's lung function and respiratory symptoms. PEF was the most usual measurement for children's lung function in this review followed by FEV₁.

Nevertheless, several limitations were identified. Most researches were conducted in developed Western countries, particularly in the United States, with only a few similar study designs carried out throughout developing countries. Although several studies differentiated air pollution between urban and suburban or rural areas, it is critical to obtain better information on the more polluted and rapidly growing developing world because, not surprisingly, the greatest disease burdens due to air pollution are occurring in developing Asian countries (46). Besides the sample sizes, children's age ranges and time spans of observation periods varied greatly, thus making it difficult to extrapolate the results of the individual studies to the overall population. Standardization and examination of seasonal effects would improve the informativeness of future studies. In general, although some studies divided air pollutants into outdoor, indoor, and personal exposure sources, most outdoor air pollution levels were measured at fixed monitoring sites with inconsistent time intervals. Even though a few studies assessed specific types of air pollution on human health, such as traffic-related air pollutants or air pollution from power plants, the distances from monitoring points to children and diverse measuring time spans can make it difficult to compare results from different studies (6).

Overall, air pollution decreased children's morning or evening lung function, or both. Even if some studies did not show an effect on the actual PEF, they demonstrated altered PEF population deviations or the occurrence of

large decrements. Undoubtedly, air pollution increased the incidences of respiratory symptoms in children and exacerbated their severity levels. Children's lower respiratory symptoms appeared more susceptible to ambient air pollution compared with upper respiratory symptoms. The impacts of air pollution on children's pulmonary health were influenced by several modifiers such as parental stresses. In terms of statistical analysis, generalized estimating equation (GEE) and mixed models were the most frequently used methods in retrieved articles.

Panel studies have provided useful information on the effects of air pollution on respiratory health (47). As the health effects of air pollution exposures have become a growing focus in the past decades (48), there have been numerous studies emphasizing this. In addition to short-term studies, large-scale and long-term studies, such as cross-sectional and longitudinal studies, have confirmed the adverse effects of air pollution. The Children's Health Study selected 10–18-year-old children from 12 southern California communities to analyze the association between long-term ambient air pollution exposures and children's respiratory developments over 8 years' observation period (49). FEV₁ growth deficits in children were adversely associated with exposures to PM_{2.5} ($p = .04$) and NO₂ ($p = .005$), even after adjustments for some potential confounders and modifiers. Furthermore, traffic exposures resulted in worse consequences of children's respiratory health status (50).

From this review, it is apparent that most panel studies have described the impacts of air pollution on children's pulmonary health, but few studies have considered the main effects of temperature, although some studies have regarded it as an effect modifier. At the time of writing this article, "temperature" was also utilized as a search term, but we did not find any articles that had used a panel study design to examine temperature effects on children's lung function and respiratory symptoms.

Nowadays, environmental temperature is changing worldwide and extreme temperatures appear more frequently (51). It is predicted that global average temperature may rise by 1.4–5.8°C at the end of this century (52). As a recent example during the summer of 2003 in Europe (53, 54), the average temperature was 3.5°C above normal, which was the hottest summer over the past 500 years in Europe. Undoubtedly, temperature increase due to climate change can directly or indirectly result in increased morbidity and mortality (52).

Nonetheless, few studies have examined the impacts of ambient temperature on individuals' lung function and respiratory symptoms using panel study designs. However, several panel studies have examined the influence of temperature on adults' cardiovascular function and symptoms. For example, a panel study with 872 cardiovascular patients was done in Germany (55). By analyzing the repeated measurements of heart rates, blood pressures, and electrocardiography changes in subjects, and the meteorological parameters, the authors demonstrated that air temperature changes can induce heart function variations in patients with underlying cardiovascular diseases.

Goldberg et al. (56) demonstrated statistically significant association between higher daily maximum ambient temperature and increased shortness of breath at night in individuals with congestive heart failure.

Therefore, panel studies may be used to examine environmental temperature and lung function/respiratory symptoms relationships, especially in vulnerable groups such as children. In this practical way, it will be very useful to help expand knowledge of the mechanism by which temperature affects human lung function and respiratory symptoms, and will provide information on threshold values of air temperature influencing population health, so as to establish early warning systems for adverse effects.

CONCLUSIONS

There are significant adverse effects of air pollution on children's pulmonary health, especially for asthmatics. Future studies need to examine the lag effects of air pollution on children's lung function and respiratory symptoms. Ambient temperature is predicted to change worldwide due to climate change, which will threaten population health. Further research is needed to examine the effects of ambient temperature and the interactive effects between air pollution and ambient temperature on children's lung function and respiratory symptoms.

DECLARATION OF INTEREST

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of this article.

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