

Effects of long-term air pollutant exposure on respiratory health in Hong Kong primary school children: abridged secondary publication

XQ Lao *, AKH Lau, B Huang

KEY MESSAGES

1. Chronic exposure to air pollutants (fine particulate matter and nitrogen dioxide) poses a threat to respiratory health among school children in Hong Kong.
2. Further studies are warranted regarding the health effects of long-term exposure to ozone.

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¹ XQ Lao, ² AKH Lau, ³ B Huang

¹ Department of Biomedical Sciences, City University of Hong Kong, Hong Kong SAR, China

² Division of Environment and Sustainability, The Hong Kong University of Science and Technology, Hong Kong SAR, China

³ Department of Geography and Resource Management, The Chinese University of Hong Kong, Hong Kong SAR, China

* Principal applicant and corresponding author: xq.lao@cityu.edu.hk

Introduction

Exposure to ambient air pollution increases mortality and morbidity. It is estimated that fine particulate matter (PM_{2.5}) alone contributed to 4 million deaths (7.5% of all deaths) worldwide in 2016.¹ Exposure to ozone (O₃) caused an additional 254 000 deaths and the loss of 4.1 million disability-adjusted life years (DALYs) secondary to chronic obstructive pulmonary disease in 2015.² However, the disease burden associated with nitrogen dioxide (NO₂) is not well-studied, despite its substantial adverse health effects.²

Compared with adults, children are more vulnerable and susceptible to the adverse effects of air pollutants. Hong Kong has various characteristics associated with air pollution such as a tropical climate, high population density, tall buildings surrounded by narrow roads with heavy traffic, and small cramped residential dwellings. Here, we aimed to investigate the effects of long-term exposure to a mixture of pollutants (PM_{2.5}, NO₂, and O₃) on respiratory health among Hong Kong primary school children.

Methods

This longitudinal prospective cohort study of school children across Hong Kong was conducted from 2012 to 2017. Details of the study have been reported elsewhere.³ Briefly, we recruited 5573 children aged 6 to 16 years from 31 primary schools across Hong Kong. The students were followed up annually for 2 years. In the baseline and follow-up surveys, the parents or guardians of each student were asked to complete a self-administered questionnaire;

anthropometric parameters and pulmonary function of each student were examined.

We developed a remote-sensing algorithm to estimate ground-level PM_{2.5} concentrations with high-resolution (1 km) and good accuracy (average correlation up to 0.9). We also developed a space-time regression model to estimate ground NO₂ and O₃ concentrations. The annual average concentrations of PM_{2.5}, NO₂, and O₃ were assigned to students based on their home address and used as indicators of long-term air pollution exposure.

Outcome measures were lung function parameters including forced vital capacity, forced expiratory volume in 1 second, peak expiratory flow, and maximum mid-expiratory flow, as well as doctor-diagnosed respiratory diseases and self-declared respiratory symptoms. Linear mixed models with random slope effect terms were used to examine long-term associations between air pollution exposure and lung function parameters. Mixed effects logistic regression models with random effects of school and child levels were used to examine associations between long-term air pollution exposure and the occurrence of respiratory diseases or symptoms. A confidence level of 0.05 was assumed.

Results

This study included 5573 school children aged 6 to 16 years, with a total of 11 540 observation records (mean, 1.68; range, 1-3) [Table 1]. Of them, 4059 (72.8%) had undergone at least two follow-up surveys.

In single-pollutant models, a 5-µg/m³ increase in PM_{2.5} exposure was associated with the following

TABLE 1. Baseline characteristics of school children

Variable	All (n=5573)*	Boys (n=2841)*	Girls (n=2732)*	P value
Age, y	9.24±1.10	9.27±1.14	9.21±1.06	0.037
Body mass index, kg/m ²	17.46±3.25	17.92±3.48	16.99±2.93	<0.001
Air pollution exposures				
Fine particulate matter, µg/m ³	28.93±4.96	28.90±5.00	28.96±4.91	0.67
Nitrogen dioxide, µg/m ³	46.22±18.96	46.62±19.38	45.80±18.50	0.11
Ozone, µg/m ³	41.14±10.69	40.99±10.91	41.30±10.45	0.28
Lung function outcomes				
Forced expiratory volume in 1 second, mL	1670.12±343.18	1714.22±336.51	1622.85±344.04	<0.001
Forced vital capacity, mL	1924.49±392.35	1996.47±388.41	1847.34±381.83	<0.001
peak expiratory flow, mL/s	3775.69±824.19	3840.54±802.37	3706.19±841.59	<0.001
Maximum mid-expiratory flow, mL/s	1905.08±541.65	1894.23±528.33	1916.70±555.44	0.14
Passive smoking	1285 (23.56)	651 (23.39)	634 (23.73)	0.80
Asthma	192 (3.49)	121 (4.3)	71 (2.63)	0.001
Allergic rhinitis	2243 (40.76)	1306 (46.43)	937 (34.83)	<0.001
Sinusitis	162 (2.95)	109 (3.88)	53 (1.97)	<0.001
Bronchitis	700 (12.71)	389 (13.82)	311 (11.55)	0.013
Bronchiolitis	161 (2.92)	96 (3.41)	65 (2.41)	0.034
Pneumonia	61 (1.11)	27 (0.96)	34 (1.26)	0.35
Wheezing	548 (10.89)	320 (12.5)	228 (9.22)	<0.001
Dry cough	1478 (28.74)	816 (31.09)	662 (26.29)	<0.001
Phlegm	676 (13.17)	354 (13.53)	322 (12.81)	0.47

* Data are presented as mean±standard deviation or No. (%) of participants

magnitudes of change: -14.05 mL in forced expiratory volume in 1 second, -4.60 mL in forced vital capacity, 26.35 mL/s in peak expiratory flow, and -18.55 mL/s in maximum mid-expiratory flow (Table 2). Similarly, increased NO₂ exposure was associated with worse lung function; adjustment for PM_{2.5} did not change the associations between NO₂ and lung function parameters. However, increased O₃ exposure was associated with better lung function; adjustment for PM_{2.5} did not change the associations between O₃ and lung function parameters.

School children with a 5-µg/m³ increase in PM_{2.5} exposure was associated with an increased risk of sinusitis (odds ratios [OR]=1.14), whereas a 5-µg/m³ increase in NO₂ exposure was associated with an increased risk of allergic rhinitis (OR=1.02) and a 5-µg/m³ increase in O₃ exposure was associated with an increased risk of asthma (OR=1.08).

Discussion

Among school children in Hong Kong, long-term exposures to PM_{2.5} and NO₂ were associated with worse lung function. However, exposure to O₃ was associated with better lung function. Exposures to

PM, NO₂, and O₃ resulted in higher odds of sinusitis, allergic rhinitis, and asthma, respectively.

O₃, a secondary pollutant, is often negatively correlated with PM and NO_x. The observed beneficial effects of O₃ on lung function might reflect decreases in PM and NO_x. Thus, further studies are warranted regarding the health effects of O₃ on respiratory health in children.

Inhaled particles from ambient surroundings can trigger interactions with pneumonocytes and may cause a cascade of inflammatory and systemic responses after entering the lung.⁴ The biological effects of increased oxidative stress are concerning.⁵ Evidence from animal studies has suggested that particulate matter can deplete antioxidants and related enzymes, produce free radicals, and trigger oxidative stress, inflammation, and pulmonary impairment.

Conclusion

Long-term exposure to ambient air pollutants (PM_{2.5} and NO₂) is associated with potential harm to lung function, along with higher risks of respiratory diseases and symptoms.

TABLE 2. Associations between pollutants and lung functions

Pollutants and lung functions	Odds ratio (95% confidence interval) for every 5- $\mu\text{g}/\text{m}^3$ increase in pollutant			P value
	All	Boys	Girls	
Fine particulate matter				
Forced expiratory volume in 1 second	-14.05 (-23.80 to -4.25)	-11.20 (-22.45 to 0.05)	-5.00 (-16.55 to 6.55)	<0.001
Forced vital capacity	-4.60 (-15.90 to 6.75)	-3.25 (-17.60 to 11.15)	-1.40 (-14.60 to 11.80)	<0.001
Peak expiratory flow	26.35 (-4.80 to 57.45)	28.05 (-7.45 to 63.50)	46.25 (5.45 to 87.10)	0.001
Maximum mid-expiratory flow	-18.55 (-37.10 to 0.00)	-9.15 (-28.55 to 10.20)	5.05 (-16.50 to 26.60)	<0.001
Nitrogen dioxide				
Forced expiratory volume in 1 second	-2.90 (-6.15 to 0.35)	-3.10 (-6.20 to 0)	-2.75 (-6.65 to 1.15)	<0.001
Forced vital capacity	-1.60 (-5.55 to 2.35)	-2.40 (-6.85 to 2.05)	-2.00 (-6.30 to 2.35)	<0.001
Peak expiratory flow	-7.45 (-20.55 to 5.65)	-8.35 (-21.50 to 4.8)	-3.00 (-18.60 to 12.60)	0.013
Maximum mid-expiratory flow	-3.05 (-11.00 to 4.9)	-3.65 (-8.75 to 1.4)	-1.15 (-8.40 to 6.15)	<0.001
Ozone				
Forced expiratory volume in 1 second	8.55 (3.35 to 13.7)	7.3 (2.1 to 12.5)	10.55 (4.4 to 16.7)	<0.001
Forced vital capacity	6.25 (0.2 to 12.35)	8.15 (0.85 to 15.5)	5.9 (-0.85 to 12.6)	<0.001
Peak expiratory flow	46.05 (25.4 to 66.75)	29.65 (7.4 to 51.85)	45.45 (20.4 to 70.5)	0.039
Maximum mid-expiratory flow	30.3 (17.75 to 42.8)	8.8 (0.2 to 17.45)	20.65 (8.3 to 33)	<0.001

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Disclosure

The results of this research have been previously published in:

1. Chen J, Zeng Y, Lau AK, et al. Chronic exposure to ambient $\text{PM}_{2.5}/\text{NO}_2$ and respiratory health in school children: a prospective cohort study in Hong Kong. *Ecotoxicol Environ Saf* 2023;252:114558.

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References

1. GBD 2016 Risk Factor Collaborators. Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks, 1990–2016: a systematic analysis for the Global Burden of Disease Study 2016. *Lancet* 2017;390:1345-422.
2. Landrigan PJ, Fuller R, Acosta NJR et al. The Lancet Commission on pollution and health. *Lancet* 2018;391:462-512.
3. Zhang Z, Tan L, Huss A, et al. Household incense burning and children's respiratory health: a cohort study in Hong Kong. *Pediatr Pulmonol* 2019;54:399-404.
4. Ghio AJ, Kim C, Devlin RB. Concentrated ambient air particles induce mild pulmonary inflammation in healthy human volunteers. *Am J Respir Crit Care Med* 2000;162:981-8.
5. Hatzis C, Godleski JJ, González-Flecha B, Wolfson JM, Koutrakis P. Ambient particulate matter exhibits direct inhibitory effects on oxidative stress enzymes. *Environ Sci Technol* 2006;40:2805-11.