

## From Fresh to Foul

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### Abstract

It is ironic that as our wealth, commerce, and technology prosper in the increasingly technologically driven world of today, the air we breathe, the very fundamental of life, suffers from the weight of our advances. Our thriving metropolitans' persistently poor air quality insidiously undermines our health, and those most vulnerable to its repercussions are the very seeds of our future. It grips one's heart to watch our skyline disappear under the veil of thick smog; smog produced by our very own chimneys and progressively threatens to choke our future. This article explores the detrimental effects of air pollution on the developing respiratory system of children, both short-term and long-term, as well as the implications to society. Moreover, it highlights the potential reversibility of even long-term repercussions of air pollution and the discrepancy in air quality between rich and poor districts posing risk of exacerbating social inequality from a young age, thus adding urgency for interventions to improve our air quality. For this public health issue needs a solution, as we cannot stand with arms akimbo as our air turns from fresh to foul.

**Keywords:** Air pollution; exacerbations; lung function; health reversibility; equity

### Introduction

The inhalation of air pollutants, notably ozone, nitrogen oxides, sulfur dioxide, and particulate matter, pose oxidative stress to the epithelium lining the airways and alveoli as they activate cytotoxic intracellular cascades. It has been shown that ozone, a powerful oxidant, induces acute asthmatic exacerbations, retards lung growth, and leads to lung function decline over time [1]. Not only does ozone impose acute oxidative stress to the lung epithelium resulting in acute presentations, but this stress also leads to an uneven redistribution of lung growth factors that may compromise long-term lung maturation and morphogenesis.

### Increased Susceptibility

Children are the most vulnerable to the health hazards of air pollution, owing to both biological and social factors. Biologically, their lungs do not reach full maturity until at least eight years of age, their host defenses are relatively weak, their metabolic systems are still developing, and children exhibit higher rates of infection with respiratory pathogens [2]. In particular, neonates in the early post-natal period and those with chronic lung diseases including asthma are at highest risk. This can be attributed to how detoxification systems, governed by the differentiation and maturation of involved enzyme pathways, are activated in a time-dependent manner in the early postnatal period and infants are less equipped to repair acute epithelial lung injury [3]. Extensive analyses of air pollutants and infant mortality in American metropolitan areas in the 1960s found a 10% increase in pollution was associated with a 1% increase in infant mortality [4]. Moreover, children have no choice in where they live, and their activity patterns involve more outdoor exposure than adults.

Of greatest concern are the potential lasting effects of air pollution on the developing respiratory system. During the alveolar period, a child's bronchial tree continues to develop with formation of a significant number of alveoli [2]. The epithelial layer of the diffusion membrane is more permeable in young children, and their lung surface area per kilogram is larger than that for adults [5]. As a result, air pollutants can more easily penetrate a child's lungs and may have lasting effects on respiratory health by interfering during this critical exposure window when the lungs are still maturing. Just as active and passive smoking during childhood has been shown to reduce the

rate of lung growth and the maximum level of function attained [6], damage to the developing lungs by air pollutants may similarly restrict the maximum functional reserve achieved by our children.

### Short term Health Impact on Children

In the short-term, air pollution can induce inflammation of the lungs leading to increased exacerbations of asthma, airway irritation, increased admissions for pneumonia, and reduced lung function. Children with pre-existing asthma are most significantly affected. Various European and American cohort studies on children in summer camp provide evidence delineating how several weeks of exposure to air pollution would already be sufficient to precipitate its immediate health sequelae. With the recruited panel of children assessed daily by peak flow tests and a symptom questionnaire while they were staying in a more polluted area, their results demonstrated a decline in lung function and increased prevalence of both irritative and obstructive airway symptoms such as coughing and wheezing respectively. Similarly, a cohort study that measured the level of nitric oxide in the exhaled air, which is a good marker of lung inflammation, of children aged 10 - 11 over seven weeks, showed the exhaled nitric oxide concentration in children with asthma living in urban areas was more than double that of those residing in the countryside or national park [7].

As air pollutants modulate the function of alveolar macrophages and epithelial cells, air pollution may weaken one's local bronchial immunity and predispose to development of respiratory tract infections, a leading cause of morbidity and mortality in children. A study on alveolar macrophages obtained by lavage from subjects exposed to  $>3500\mu\text{g}/\text{m}^3$  nitrogen dioxide demonstrated a 72% decrease in superoxide production and a 42% reduction in the ability to phagocytose *C. albicans* [8]. Translating into clinical practice, observational studies in Hong Kong found that every  $10\text{-}\mu\text{g}/\text{m}^3$  rise in  $\text{PM}_{10}$  was associated with a very small but significant increased risk of URTI (1.0301) and all respiratory illness (1.0328) [9]. Similarly, a study in the United Kingdom observed a 3.5% increase in upper respiratory tract infection consultations when sulfur dioxide levels were increased from the 10<sup>th</sup> to the 90<sup>th</sup> centiles (13 to  $31\mu\text{g}/\text{m}^3$ ) [10], and an increase in lower respiratory tract infection consultations by 7.2%, 6.2%, and 5.8% during a 10<sup>th</sup> to 90<sup>th</sup> centile rise in levels of nitrogen dioxide, carbon monoxide, and sulfur dioxide respectively during winter months [11]. These examples are part of a large body of evidence illustrating the association between air pollution and increased disposition to developing respiratory infections in children.

As environmental control is a cornerstone in the management of asthma, exposure to irritants including air pollutants is widely known to precipitate acute asthmatic attacks, exacerbate attacks, and render disease control suboptimal. A randomized population survey on 7,509 children in Germany found the prevalence of current asthma, wheeze and cough was increased in the category with the highest density of traffic [12]. Similarly, a study analyzing paediatric hospital admissions over six years in Hong Kong established a positive correlation between the number of asthmatic attacks severe enough to require hospital admission and the ambient levels of particulate matter, ozone, and nitric oxide [13]. Systematic reviews of international studies on known asthmatic children have also reported a positive correlation between complaints of nocturnal cough, phlegm and reduction in peak expiratory flow rate with increased levels of air pollutants [14].

### Long term Health Impact on Children

While the short-term consequences of air pollution lead to acute presentations, long term exposure to high air pollution in children results in more subacute complications of impaired lung function, predisposition to development of allergic rhinitis, and chronic bronchitic symptoms. A cross-sectional study in Hong Kong involving 3,168 schoolchildren demonstrated that the lung function tests of forced expiratory volume in one second ( $\text{FEV}_1$ ), forced expiratory flow between 25% and 75% of FVC (FEF [25-75]) and forced expiratory flow at 75% of FVC (FEF [75]) were significantly lower in children living in highly polluted areas than their counterparts residing in low-pollution districts [15]. Boys and asthmatic children were found to be more susceptible to the development of lung function deficits. The results also showed that particulate matter 10 ( $\text{PM}_{10}$ ) is the most responsible culprit for adversely affecting airway development [11]. An Austrian study with repeated spirometry measurements on children over three years echoed similar findings and calculated that a  $10\mu\text{g}/\text{m}^3$  rise in the concentration of  $\text{PM}_{10}$  in the ambient air was associated with a reduction in lung growth by a decrease in  $\text{FEV}_1$  growth of 84 ml/year. These findings are further supported by a cross-sectional study on individuals aged 6 - 24 years over 24 cities in USA illustrating a 2.4%

decrement in adjusted FVC and a 2.1% decrement in adjusted FEV<sub>1</sub> for every 17.3 µg/m<sup>3</sup> increase in annual mean PM<sub>10</sub> [16]. The reduced lung capacity as a result of childhood exposure to air pollution limits their reserve to handle future insults to their respiratory system during adulthood, such as from occupational exposures or cigarette smoking.

As children's immune systems are still developing, early childhood exposure to irritants in the air may induce the preferential differentiation of Helper T lymphocytes (T<sub>H</sub> cells) into T<sub>H2</sub> cells instead of T<sub>H1</sub> cells. Since T<sub>H2</sub> cells mediate hypersensitivity reactions, this polarization to a T<sub>H2</sub> repertoire places such children at increased disposition to developing atopy, and likely in more severe forms. Vehicle-released air pollutants, particularly diesel exhaust particles, have also been demonstrated to increase allergen-specific IgE levels, while simultaneously activating mast cells and basophils [17], thus precipitating allergic exacerbations.

### Potential Reversibility

Of considerable interest is the potential reversibility of the aforementioned short-term and long-term repercussions in children, through exposure to improved air quality. Cohort studies on children in USA and Netherlands found that their spirometric lung functions, which decreased after episodes of high air pollution, returned to pre-episode levels within a few weeks of improved air quality [18]. A cross-sectional study over three consecutive winters in Utah, America showed a greater than 50% reduction in the number of hospital admissions for asthma and pneumonia in children during the period when a steel mill, the main contributor of winter air pollution to the valley in Utah, was closed and pollution was consequently lowered [19]. These findings were supported by a study in Atlanta, Georgia before and during the Olympics, when air pollution was reduced following limitations on traffic, demonstrating a marked reduction in the number of emergency visits for asthma in children during that period of strict traffic control [20]. Similar findings were heralded in Hong Kong when the regulation on the sulfur content in fuel leading to an average reduction in the ambient level of sulfur dioxide by 45% has resulted in improvements in bronchial hyper-responsiveness and respiratory symptoms in children [21]. These findings connote the rapid reversibility of acute complications of pollution in children, whether they are minor or major, simply by exposure to better air quality.

Not only are the short-term consequences amenable to correction, but there is also evidence to suggest that even the longer term effects of air pollution may be reversible. A Californian cohort study following 110 children demonstrated an improved rate of growth of lung function in children who moved to areas with low PM<sub>10</sub> concentration [22]. In order for the amelioration in lung growth to be more significant, children had to have resided in the new area for at least three years. Similarly, a cohort study on children living in eastern Germany depicted a drop in the prevalence of symptoms of chronic cough and chronic bronchitis after reunification [23], when the air quality improved. These findings cast hope and highlight the importance of interventions on air quality, although the long-term consequences of pollution on child health take longer to be mitigated.

### Social Implications

With air pollution typically being most severe in poorer districts, this calls into question whether inequalities in child health and development have been created. According to the averaged general station readings collected by the Environment Protection Bureau in Hong Kong, the three most polluted districts in Hong Kong, namely Sham Shui Po, Kwai Chung, and Kwun Tong [24], were also the districts with the lowest average monthly income [25]. The main contributor to the heavy pollution is nitrogen dioxide [26], which may be produced by the dense traffic of diesel trucks in those vicinities. This may imply that children from low-income families, who have minimal power to choose where they live, are more likely to be afflicted by the health consequences of air pollution. Moreover, various studies have reported a positive correlation between the severity of air pollution and school absenteeism, while other studies have found conflicting results [2]. Specific figures were quantified by a study in an industrialized part of Utah, USA, which reported an overall 2% increase in the number of children absent from school when the average PM<sub>10</sub> increased by 100 µg/m<sup>3</sup> [27]. This could be explained by the increased respiratory complaints of children during episodes of high air pollution.

The marked discrepancies in air quality in different parts of the city bring forth socioeconomic implications on public health. In addition, the potential impact on school attendance further raises the question of whether children from a lower social class are inherently drawn into a vicious cycle steeped with unequal opportunities. Thus, air pollution may be implicated as one of the factors widening the health gap between the rich and the poor, starting from a young age.

## Conclusion

Eric Bohm, former chief executive of World Wide Fund for Nature (WWF) Hong Kong, is only one of many individuals who have been forced to leave the city as his family members suffered from progressively severe attacks of asthma and hospital admissions for pneumonia attributable to the worsening air quality in our metropolitan. It is without doubt that children are most vulnerable to the health detriment incurred by air pollutants, as their immune and respiratory systems are still maturing. A plethora of credible studies at both the local and international levels have demonstrated how air pollution is a health hazard to children, be it from short term exposure triggering acute exacerbations of asthma, respiratory infection, and airway irritation, to long term exposure leading to impaired lung development, predisposition to atopy, and symptoms of chronic bronchitis. The potential reversibility of these repercussions on a child's health, especially for those arising from short-term exposures, should galvanize efforts to improve our air quality. Moreover, interventions on pollution can contribute to enhancing equal opportunities between children of different socioeconomic classes, for the most polluted districts are also among the poorest. Although we enjoy longer life expectancies than before, the rising health detriment brought by our impoverished air quality to the very seeds of our future, our children, burns with irony, and together with its reversibility and implications on equity, add urgency to the need for interventions to save our city, to prevent our future from being turned from fresh to foul.

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