

# Air pollution and children's health: where next?

Abigail Whitehouse , Jonathan Grigg 

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On 17 December 2020, the H.M. Assistant Coroner for Inner South London, Dr Philip Barlow, opened an inquest into the death of Ella Roberta Adoo Kissi-Debrah from an asthma attack.<sup>1</sup> Ella lived next to the South Circular Road and for most of her short life had been exposed to excessively high levels of a traffic-derived mix of nitrogen dioxide (NO<sub>2</sub>) and particulate matter (PM) less than 10 microns in aerodynamic diameter (PM<sub>10</sub>—the inhalable fraction), and the non-exhaust sources of PM such as tyre and brake wear. After hearing extensive evidence from medical and pollution monitoring experts, his decision was Ella died from acute respiratory failure, asthma, and air pollution exposure—the first time ever that a coroner has found air pollution as a contributory cause of illness and death, and providing support for future litigation. In addition, the judgement provides us with a real-life example of what has been known for some time from epidemiological studies, that traffic-derived air pollution not only contributes to incident asthma but also triggers fatal asthma attacks. Since the link between air pollution and asthma has now been settled by legal judgement in the UK, the question is where next for air pollution research? In this article, we highlight some of the questions that are yet to be answered and may further strengthen the case for urgently reducing exposure of children to traffic-derived air pollutants.

birth weight at term, both in terms of direct exposure levels and proximity to high-density traffic areas.<sup>3</sup> The mechanistic question is how can PM and gaseous pollutants inhaled into the lung exert their adverse effects on distant organs? One possible explanation is that airway cells, such as alveolar macrophages, are stimulated by inhaled pollutants to release mediators, which then enter the systemic circulation and affect the developing fetus. Another, although not mutually exclusive, explanation is that a small amount of toxic inhaled PM translocates via the systemic circulation and directly damages both the placenta and the developing fetus. Evidence of this phenomenon was reported in 2019 by Bové *et al*, who found indirect evidence of carbonaceous PM in human placentas.<sup>4</sup> We recently extended this observation by showing the presence of PM, with composition compatible to PM from fossil-fuel combustion, in placental phagocytes (*figure 1*).<sup>5</sup> What remains unclear is whether the concentrations of PM observed in placental phagocytes (compared with airway cells) have the capacity to alter placental function, and further studies are needed to explore this in detail. Such a phenomenon would have implications for other organs where associations between exposure and adverse health outcomes have been reported in epidemiological studies—such as the brain,<sup>6</sup> eye<sup>7</sup> and kidney.<sup>8</sup> Indeed, it is encouraging that mechanistic research is starting to fill in the gaps to explain how exposure in childhood can impact throughout the life course. For example, we recently reported that increased exposure to traffic-related air pollution was associated with changes in airway dendritic cell (DC) maturation, with higher numbers of mature airway DCs in children who lived and attended schools in heavily polluted areas of London.<sup>9</sup> This confirmed in vivo what had previously been shown in in vitro studies, and specifically

## WHAT IS THE FATE OF INHALED PM?

The Royal College of Physicians/Royal College of Paediatrics and Child Health report 'Every Breath We Take', published in 2016, identified emerging epidemiological evidence for the effects of maternal inhalation of air pollutants on the developing fetus.<sup>2</sup> For example, the ESCAPE (European Study of Cohorts for Air Pollution Effects) cohort study found an increased risk of low

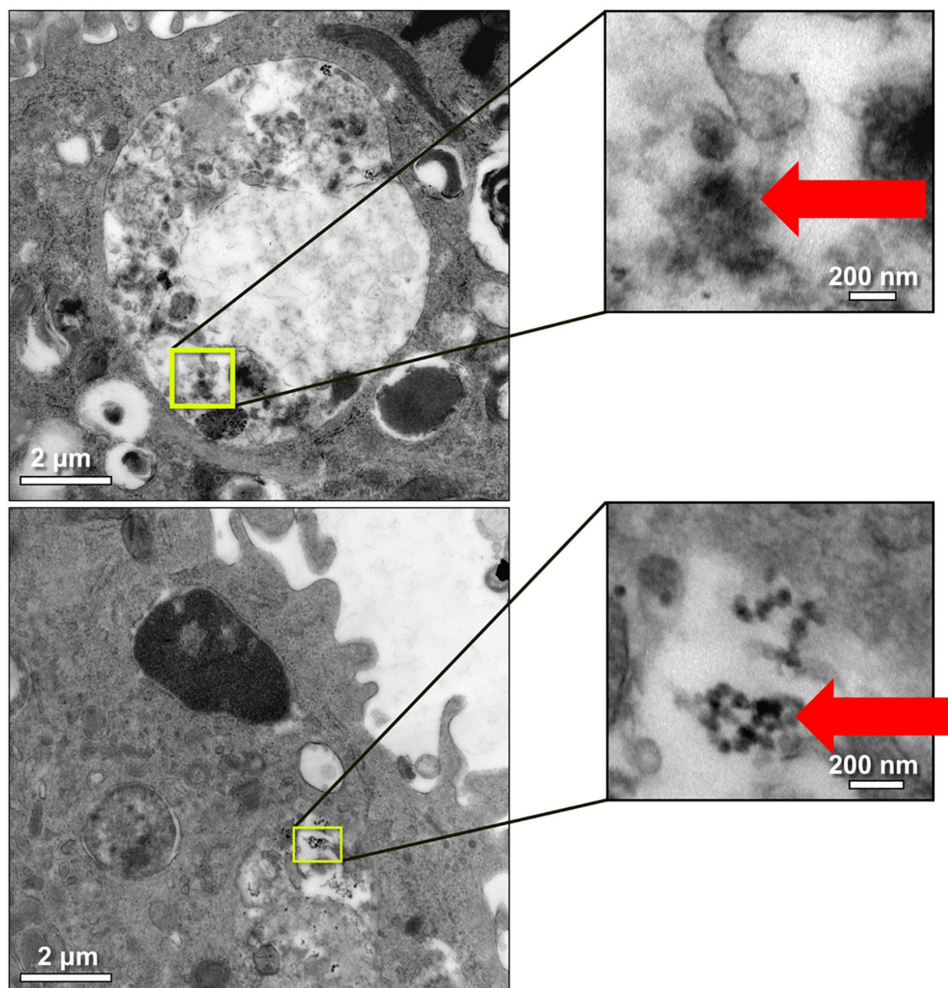


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Centre for Genomics and Child Health, Queen Mary University of London, Barts and The London School of Medicine and Dentistry, London, UK

### Correspondence to

Dr Abigail Whitehouse; a.whitehouse@qmul.ac.uk



**Figure 1** Showing -Electron microscopy (EM) imaging of placenta with clearly visible carbon particles, reproduced with permission from Liu *et al.*<sup>5</sup>

due to daily background exposure over a prolonged period.

### ARE DIFFERENT TYPES OF PM EQUALLY TOXIC?

The composition of PM mixture varies from place to place. Near to roads, emissions from vehicles form the majority of PM, whereas in rural areas, there is an increased amount of PM produced by chemical reactions in the air including nitrates and sulfates.<sup>2</sup> Another way of describing PM is by where it originates. Primary particles are those that are emitted directly to the air and include PM generated during fossil fuel combustion, brake and tyre wear, and resuspended road dust. Secondary particles are particles formed in the atmosphere and include ammonium nitrate and sulfate. The precursors of these particles include NO<sub>2</sub>, ammonia and volatile organic compounds. PM may also be classified by size—with PM<sub>2.5</sub> being the mass of the fraction less than 2.5 microns in aerodynamic diameter/m<sup>3</sup> of air. Furthermore, ultrafine particles (far smaller than PM<sub>2.5</sub>) may also play a part in the long-term health effects. To date, epidemiological studies have not been able to determine (1) whether the smaller size fractions

of PM are more toxic to health and (2) whether PM from different emission sources have different effects. The precautionary assumption therefore remains that all types of PM are equally harmful. This lack of data has major implications for policy makers. For example, should we be concerned that PM in the Victoria line of the London Underground (often referred to as ‘tube dust’) is 381 µg/m<sup>3</sup><sup>10</sup>—well above WHO limits for PM<sub>2.5</sub> of 25 µg/m<sup>3</sup> 24-hour mean,<sup>11</sup> and do non-anthropogenic (ie, naturally occurring) PMs such as Saharan dust have significant health impacts, or are they more inert? We therefore need more mechanistic studies focused on key mechanisms such as the potential of PM to induce oxidative stress in airway cells.<sup>12–15</sup>

### CAN CHILDREN REDUCE THEIR OWN EXPOSURE TO AIR POLLUTION?

The main way of reducing children’s exposure to air pollution is to reduce emissions. The aim of the UK Government to ban sales of petrol and diesel cars by 2030 is certainly welcome, but will not protect this generation of children. The health benefits of reducing air pollution at large scale are illustrated by the Southern

### Box 1 Advice that can be given to children with asthma to reduce personal exposure

- ▶ When there is a short-term increase in air pollution levels across the city, there is an increased risk of more asthma symptoms. So when levels are high, the government will issue an air pollution alert or you may see reports included as part of the weather forecast.
- ▶ These short-term increases (spikes) in air pollution will increase your exposure, no matter where you are. It is therefore sensible to take extra precautions when there is a high pollution day.
- ▶ This includes reducing or avoiding strenuous outdoor exercise. Since exercise has many benefits, if possible, keep doing your exercise indoors in a well-ventilated room or gym.
- ▶ Stay away from pollution hotspots such as main roads and busy road junctions.
- ▶ Try to get to school a little earlier before rush hour has begun and levels of pollution have built up.
- ▶ Walk to school or the shops on the inside of the pavement—the further you are from the traffic, the lower the pollution levels are.
- ▶ Make sure you carry your reliever inhaler. Consider taking your reliever before entering a pollution hotspot.
- ▶ Make extra sure that you use your regular preventer inhaler as prescribed.
- ▶ When there is a short-term increase in air pollution levels across the city, there is an increased risk of more asthma symptoms. So when levels are high, the government will issue an air pollution alert or you may see reports included as part of the weather forecast.

Adapted from the British Lung Foundation.<sup>19</sup>

California Children's Health Study which reported that dramatic reductions in air pollution (a 43% reduction in PM<sub>2.5</sub> in the worst affected region), was associated with less suppression of lung function growth.<sup>16</sup> For example, the number of children aged 15 years with clinically low forced expiratory volume in 1 s (<80% predicted) dropped from 7.9% to 3.6%. Advice can be given to children and young people with severe asthma who are vulnerable to traffic-generated air pollution (box 1). Even though there is limited evidence from personal exposure monitoring that any of these actions result in better asthma control, it seems reasonable to discuss these options with children and their parents. Some interventions may have potential impacts that can be extrapolated from the short-term effects of exposure such as that demonstrated by the Oxford Street study with adult asthmatics, which clearly showed an acute negative impact on lung function on a highly polluted street.<sup>17</sup> However, to date, there is minimal evidence to support this or that intervention, such as the use of air purifiers (with considerable costs at times), to reduce the migration of outdoor pollutants into the indoor environment and to improve respiratory health.<sup>18</sup>

### SUMMARY

Although we have sufficient epidemiological data to support the need to urgently reduce children's exposure to air pollution, there remain questions that need to be addressed by research. For example, where should

we focus our efforts, on large-scale interventions that have clear evidence of reduction in pollution levels (although the health effects are less clear) or on small-scale interventions which may have more obvious short-term effects but currently lack evidence for long-term health benefits? What is clear though is that air pollution exposure continues to be a significant risk across the life course, with both short-term and long-term health effects. Since delaying exposure-mitigation policies puts at risk this generation of children, ensuring the right to breathe clean air must be higher up the political agenda, with major investment in small-scale local interventions (eg, low-pollution streets outside schools), and both city-wide (eg, clean air zones) and national actions (eg, scrappage schemes for the most polluting vehicles).

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### ORCID iDs

Abigail Whitehouse <http://orcid.org/0000-0003-1695-7141>

Jonathan Grigg <http://orcid.org/0000-0003-3109-6028>

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