Outdoor air pollution and the health impacts on children

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A working document
1. **INTRODUCTION**

1.1 **PREMISE**

According to the World Health Organisation (WHO):

> The dangers posed by air pollution are far greater than previously thought and is described as one of “the greatest hazards to human health”.

> And “accumulated evidence indicates that children’s health is adversely affected by air pollution levels currently experienced in Europe”

The purpose of this report is to provide a detailed literature review on the effects of outdoor air pollution on children’s health and development. It is an extension of the ‘Air Pollution and Human Health’ document previously published by Deliver Change. Each section will contain a selection of original research papers, media articles and analysis documents, then a conclusion shall be drawn about the section as a whole, taking into account all documents presented.

It is important to distinguish this cohort of the population because, firstly they are one of the most vulnerable groups and uniquely so (discussed later). Secondly, the effects of pollution on children may be long lasting as early-life illness can impede long-term human capital development, and finally, we need to understand the risks to mitigate the negative effects.

Due to this being a scientific document, technical descriptions are used throughout, see appendix 1 for full descriptions of these terms as required.

1.2 **WHAT IS AIR POLLUTION**

Generally any substance that is introduced into the atmosphere that has damaging effects on living things or the environment is considered air pollution. The substances that are responsible for air pollution are known as air pollutants. Air pollutants can be gaseous, liquid or solid in form, and can come from natural as well as human source:

The main air pollutants that are now emitted are:

- Sulphur oxides/sulphur dioxide
- Nitrogen oxides/nitrogen dioxide
- Carbon monoxide
- Carbon dioxide
- Volatile organic compounds
- Particulate matter
- Ammonia
- Lead
- Persistent organic pollutants
- Ozone
1. 3 SOURCES OF AIR POLLUTION

It is commonly understood that pollution is a by-product of all economic and societal activates\(^6\). Previously anthropogenic air pollution came from coal fires, power plants and heavy industry, but with clean fuels, filtration of gases, improvements in process technologies and the export of most polluting industries to countries with lower wages and less pollution controls, as well as the emergence of new sources such as motor vehicle transport\(^7\), pollution has a new face globally. Modern pollution is now less visible but more immediately irritating and long term damaging than it used to be, meaning that understanding and communicating the risks is now even more of challenge\(^8\).

1.4 OPENING STATEMENT

As stated previously in ‘Air Pollution and Human Health’, the risk posed to human health from anthropogenic air pollution is significant, undeniable and will prove to be a real challenge for this and future generations. The WHO has said that global urban outdoor air pollution levels have increased by about 8% between 2008 and 2013\(^9\). This continual increase in pollution levels is significant as the associated health effects include substantial reduction in life expectancy, increase risk of lung cancer, cardiovascular disorders and strokes to name a few – and the likelihood of many of these risks increase with elevated pollution levels. There is a critical need for further data and research, as well as dealing with the problem itself - unlike many other health threats, entire populations are at risk, and for many of the known pollutants, there is no safe level of exposure.

In terms of children at risk: one in seven of the world’s children are now exposed to pollution levels six or more times higher than international standards set by the World Health Organization, according to a new report by UNICEF (Oct, 2016). Furthermore, a joint research project between Royal College of Physicians and Royal College of Paediatrics’ and Child Health in 2016 concluded that overall, the effects on children’s heart, brain, hormone systems and immunity are proven and the effects on growth, intelligence, development of brain and coordination are still growing\(^10\).
2. Why are children uniquely vulnerable?

As detailed previously, children and infants are uniquely vulnerable and therefore have different risks and risk levels than adults. The reasons for this unique vulnerability are:

- Higher resting metabolic rate and rate of oxygen consumption per unit body weight than adults
- Airways are narrower
- Shorter in height so they breath closer to the ground therefore greater exposure
- Size of their organ systems, and that they are still developing
- Increased respiratory rate and inhale more
- They often ignore respiratory symptoms and continue to play
- Children spend more time outdoors
- Incomplete metabolic systems
- Immature host defences
- High rates of infection by respiratory pathogens

In addition to this, some children are more vulnerable than others. Individuals with underlying chronic lung disease, particularly asthma, are potentially at greater risk than those without such conditions. Polymorphic variation in genes involved in protecting against tissue injury or regulating tissue repair may explain some of the variation in individual susceptibility to the adverse effects of pollutants on health. Furthermore, patterns of exposure to indoor pollutants vary among children; those receiving higher exposures indoors, for example from tobacco smoke, are at greater risk of being affected by outdoor pollutants.

It is for all these reasons that children and infants are among the most susceptible to many air pollutants and we are now going to look at how this increased risk manifests itself in terms of health impacts.
3. PRE-NATAL EFFECTS

Before we begin to look into the health effects of air pollution exposure on children and infants, it is important to identify the effects on pregnant women and their unborn children. Outdoor air pollutants can enter the foetus’s bloodstream via the placenta and the umbilical cord blood, and there is a large amount of research suggesting the negative effect of these pollutants. If these associations are found to be causal, it would likely result in significant public health impacts due to widespread exposure and the fact that low birth weight or preterm births are subsequently associated with long-term issues such as developmental disability and chronic lung disease. Also, data suggests that a growth and development delay in utero influences the risk of heart disease and diabetes in adulthood.

3.1 LOW BIRTH WEIGHT (LBW), PRETERM DELIVERY (PTD) AND INTRAUTERINE GROWTH RESTRICTION (IUGR)

Low birth weight, prematurity and intrauterine growth restriction remain the leading causes of perinatal morbidity, mortality, neurodevelopmental impairments and disabilities among newborn babies. Therefore identifying any causal associations is vital.

In literature reviews published before 2005, it was concluded that there were associations for PTD and IUGR. Another systematic review found little consistency in evidence linking PM and foetal health, either finding small adverse effects or no effect at all. Finally, a Brazilian cross sectional study of nearly 180,000 births observed the negative effects of exposure to PM10 and CO during the 1st trimester – with a moderate increase in mean exposure resulting in a decrease of 23g in birth weight. This publication also mentions another ecological studies in the Czech Republic and China, concluding that the prevalence of LBW was significantly associated with air pollution, especially sulphur dioxide. In China, a similar conclusion was reached. Finally, the WHO concluded that there was sufficient evidence for a causal relationship between air pollution and low birth weight, but insufficient for preterm birth.

Since 2005 there has been accumulating evidence that environmental exposures can cause infants to be born premature, with low birth weight or with certain defects. In terms of actual figures, it is concluded that in more polluted versus less polluted areas, the increase in risk ranges from 10-30% for preterm birth and low birth weight, also certain vulnerable population segments (poorest and most exposed) might experience much higher risks. Study examples include an ’08 cohort study in Canada – results showing that residence within 50m of a highway was associated with a 26% increase in SGA (small for gestation age) and 11% for LBW. A further global meta-analysis of research in 2013 found LBW was positively associated with increased PM10 and PM2.5 (by 10ug/m3) exposure during the entire pregnancy. The same increase in PM10 was also negatively associated with term birth weight. Finally, in areas with...
higher median PM2.5 levels and PM2.5-10 ratios, tended to report stronger associations.

In recent years there have been some innovative research projects. The first of which used the unusual circumstances around the Beijing Olympics. Beijing is ‘one of the most heavily polluted cities in the world’ and, as a condition for hosting the Olympics, had to combat their air pollution problem. They did so by temporarily closing factories, halting construction and reducing the amount of cars on the road. The consequence was a reduction in air pollutants by between 18% and 59% during the 47 days of the games. The conclusion of the study found that the greatest difference occurs during the 8th month of pregnancy – on average – babies were 23g heavier compared to the same period in 2007 and 2009. A 2016 Spanish study wanted to assess whether there are windows of special vulnerability by using measurements from foetal biometry (ultrasound measurements) longitudinally. The results showed a 10ug/m3 increase in average exposure to NO2 during weeks 0-12 and 20-34 were associated with reduced growth for various measurements.

To conclude PM2.5 concentrations were associated with low birth weight, but not preterm birth. The largest multi-centred study (at the time of publishing) reported that the inconsistencies among research studies could reflect genuine differences in the study settings, which was what this meta-analysis was looking to eradicate.

Finally, a study conducted in the US found that air pollution had a costly impact on unborn children, estimating that the economic impacts cost $4.33bn in 2010, of which $760 million were spent for medical care. PM2.5 may contribute substantially to burden and costs of preterm birth in the US, and considerable health and economic benefits could be achieved through environmental regulatory interventions that reduce PM2.5 exposure in pregnancy.

3.2 BLOOD PRESSURE

There isn’t a large amount of literature on this association, however, one study talks of the risk that exposure to PM10 and O₃ during first trimester increases blood pressure in the later stages of pregnancy (associations were stronger when restricted the analysis to non-smokers). A further Californian study investigating 298 pregnant women found exposure to CO and PM₂.₅ in the first trimester was significantly associated with hypertensive disorder of pregnancy and these associations were modified by BMI (increased odds among non-obese women). Finally, a study using data from the EPA concluded that poor air quality could lead to an increase in blood pressure within pregnant women, which can lead to complications that increase risks for mother and baby, such as preeclampsia.

3.3 HEART DEFECTS

A study of nearly 9400 infants in Southern California (data collected by California Birth
Defects Monitoring Program) found an odds ratio for cardiac ventricular septal defects increased with dose-response fashion with increasing 2nd month CO exposure. It also found risks for aortic artery and valve defects, pulmonary artery and valve anomalies and conotruncal defects increased with 2nd month O₃ exposure. However, it was inconclusive for other air pollutants. This was the 1st study of its time and recognised the need to be confirmed by further studies. As such, the WHO has concluded that the evidence into birth defects is insufficient to draw firm conclusions and further research is required. Worldwide, studies on this issue are limited and inconclusive.

3.4 Autism

Autism spectrum disorder (ASD) is a developmental disorder that is characterised by deficits in social interaction, communication and behavioural flexibility, and it is reported that global prevalence is increasing. Although genetics plays a strong role in ASD, evidence suggests that environmental exposures, particularly in utero or during early life, also affect ASD risk.

Several studies have explored associations of air pollution with autism, suggesting that there are increased odds of having a child with autism when exposed to higher levels of PM, criteria pollutants, heavy metals such as mercury and distance to major roads.

A study in 2013 found pregnant women who were exposed to high levels of diesel particulates or mercury was twice as likely to have an autistic child. Furthermore, the largest ever US nationwide study, published in 2015, found that the risk of autism doubled among children of women exposed to high levels of particulate air pollution during pregnancy. The association was strongest when the exposure occurred during the third trimester. The researchers saw no increased autism risk if the pollution exposure occurred after birth or before conception.

To summarise, while smaller studies (as listed on Autism Speaks) have suggested that exposure to air pollution during pregnancy increases autism risk, these earlier studies were limited in scope – tracking pregnant women and their children in just a few communities. The 2015 investigation spanned all 50 states by tapping into the national Nurses’ Health Study II, which has 116,000 participants. The analysis looked at pollution exposures before, during and after the women’s pregnancies.

3.5 Sudden Infant Death Syndrome

Although the rate of sudden infant death syndrome (SIDS) has been reduced with the ‘Back to Sleep’ campaign, SIDS is still a common cause of death in infancy. A range of environmental factors may interact to contribute to the adverse health conditions conducive to
SIDS. Nine studies were evaluated by a 2004 review\textsuperscript{50}, looking at the association between exposure to air pollution and the incidence of SIDS. The available evidence was deemed inadequate to come to any conclusion about a relationship between air pollution and SIDS, although the body of evidence appears to suggest that air pollution (especially particles and some gaseous pollutants) may play a certain role in the occurrence of SIDS. The suggestion is that future studies should focus on the research design, role of indoor air quality and the effect of smaller particles, particularly those in the ultrafine range.

### 3.6 Summary

Firstly, the evidence is suggestive of causality for the association of birth weight with air pollution, although further studies are needed. For preterm births and intrauterine growth retardation, the current evidence is insufficient to infer a causal relationship\textsuperscript{51}. Furthermore, there appears to be a large amount of research on the subject and the pollutants that seem to be consistently concluded to be harmful to foetal health are CO and O\textsubscript{3}. Other pollutants such as PM seem to have less conclusive conclusions so would require further research. Despite these inconsistencies with the conclusions, it is apparent that high levels of outdoor air pollution will harm the health of an unborn child in some way, which is very concerning because we do not know the long-term implication of these changes on the future health of the children\textsuperscript{52}. 
4. RESPIRATORY ILLNESSES

Air pollution is increasingly known to impact the development of children’s respiratory system after birth. There are several biological reasons why young children’s respiratory systems may be more susceptible to air pollution’s effects. Firstly, children's lungs continue to rapidly develop in the early years, and the cell layer lining the inside of the respiratory tract is particularly permeable during this age period. Compared to adults, children also have a larger lung surface area in relation to their body weight, and breathe 50% more air per kilogram of body weight. The process of early growth and development is important for the health of the child in general, and therefore may also be a critical time when air pollution exposures can have lasting effects on future health. Additionally, children tend to spend more time outdoors doing strenuous activities, such as playing sports, so they are breathing more outdoor air compared to adults⁵³.

4.1 OVERALL

The British Lung Foundation in 2011 suggested that breathing polluted air over several years could reduce the rate at which children’s lungs grow⁵⁴. A review of epidemiological studies found that a number have reported an association between residential proximity to busy roads and a variety of adverse health outcomes in children including respiratory symptoms and asthma exacerbations⁵⁵.

A large British investigation found that a marginal increase in CO and ground level ozone are associated with statistically significant increases in children’s contemporaneous respiratory treatments⁵⁶.

4.2 LUNG CAPACITY & DEVELOPMENT

The WHO in 2005 stated there was sufficient evidence for exposure to air pollutants to cause adverse effects on lung function development. This included reversible deficits of lung function and chronically reduced lung growth rates and lower lung function⁵⁷.

A 6-year study researching 2,400 children in East London concluded that those children living in highly polluted parts of cities to have up to 10% less lung capacity than normal, with warnings the damage could be permanent. This reduction could increase the risk of diseases such as asthma and bronchitis⁵⁸. A Hong Kong investigation of over 2000 school children found similar results - with children in high pollution districts had significantly lower complete speed and predicted maximal oxygen uptake, which was negatively associated with cardio-respiratory fitness, especially in girls⁵⁹.

UNICEF (2016) concluded that ‘studies have shown that the lung capacity of children
living in polluted environments can be reduced by 20% – similar to the effect of growing up in a home with secondhand cigarette smoke.  

4.3 BRONCHITIS, BRONCHIOLITIS & PNEUMONIA

According to a 2016 UNICEF report, almost one million children die from pneumonia each year globally, more than half of which are directly related to air pollution and the WHO stated that air pollution exposure increased the prevalence and incidence of cough and bronchitis.  

The 20 year Teplice Program in the Czech Republic found increased concentration of PM 2.5 and polycyclic aromatic hydrocarbons (PAHs) caused an increase in bronchitis in pre-school children. A US study in 2009 supports what they deem a developing hypothesis that there may be a modest increased risk of bronchiolitis attributable to chronic traffic-derived particulate matter exposure particularly for infants born just before or during peak respiratory syncytial virus season.

4.4 ASTHMA, ALLERGIES & ATOPY

There is a general consensus on the link between exposure to TRAP and increased risk of asthma, but the reported findings are not completely consistent.

Study examples include a report published in 2008 by COMEAP that stated that a reduction in lung function due to air pollution might increase symptoms in children who develop chronic respiratory conditions such as asthma. Another one found an association with infant mortality and the development of asthma and atopy. Finally, in 2014 – it was found that obese children exposed to high levels of air pollutants were nearly 3 times as likely to have asthma, compared to non-obese children and lower levels of pollution exposure. This is an issue because data from the GLA for 2009/10 shows that in London, 11.6% of children aged 4-5 years and 21.8% of children aged 10-11 years were at risk of being obese.

The diversity in findings may be partly explained by genetic variation between populations; therefore further investigations are required to explore the role of genetic variants. Studies to investigate this issue are feasible but will require a large sample size to have sufficient statistical power. In addition, accurate exposure estimates, using state of the art modelling and measurements, that span the periconception period (time period around conception), foetal development and early post-natal life and improved objective, methods for diagnosing asthma will be required. Developing a standardized methodology and several studies using harmonized protocols are the most likely way this aim could be achieved.

WHO concluded that there is sufficient evidence for a causal relationship between ambient air pollution and aggravation of asthma, as well enhancing allergic sensitization in those genetically at risk.
4.7 SUMMARY

The evidence covered above (and as quoted by the WHO) shows a relationship between exposure to ambient air pollutants and adverse effects on the development of lung function. Reversible lung function deficits, chronically reduced lung growth rates and lower lung function levels are associated with exposure to air pollution. Moreover, the evidence shows clearer relationships for particulate matter and traffic-related air pollution (indicated by nitrogen dioxide) than for other pollutants.\textsuperscript{71} This should not be a surprise as early childhood is a critical period for the development and maturation of several biological systems including the lungs\textsuperscript{72}.

Despite the large amount of research published there are still significant gaps in our understanding of the role childhood air pollution exposure plays on the development of respiratory disease. In particular, there is a pressing need to understand how exposures occurring during the periods of rapid lung development, especially during foetal development and the first 2 years of postnatal life, reduce lung function and lung function growth and increase the life-long risk of acute and chronic lung disease\textsuperscript{73}. 
5. Infant Mortality

The amount of research available on the effects of air pollution and child mortality is relatively low because the majority of studies conducted have been on the mortality rates for adults, but below are a few which have looked into the associations.

- Infants living in areas with high levels of PM have a greater risk of mortality during the first year of life, than those living in low levelled areas. Also there is consistent epidemiological evidence of an increase in infant post-neonatal mortality due to respiratory causes associated with an increase exposure to ambient air pollution.

- A study in Sao Paulo in Brazil estimated the proportions of respiratory deaths attributed to air pollution are 15% for carbon monoxide, 13% for sulphur dioxide and 7% for PM10. They also found no association between air pollution and non-respiratory deaths. These results were consistent with previous research they had conducted.

According to the OECD, under-five mortality could be 50% higher than current estimates by 2050 as a result of outdoor air pollution. Another study published in Nature found it could be even worse – doubling by 2050.

Summary

Even though the evidence above points towards a causal relationship, no firm conclusions can be made due to the lack of data.
6. NEURODEVELOPMENT

6.1 THE DEVELOPING BRAIN

Understanding the potential impact of air pollutants on the development of a child’s brain is gaining increasing attention. A study by Harvard University\(^7\) stated that:

“Air pollution contains many toxicants known to affect neurological function and to have effects on the foetus”

Millions of children in polluted cities are showing detrimental brain effects, but wide-reaching public health initiatives targeting these populations are still considered premature or unwarranted. This is because the effects on children’s central nervous system (CNS) not yet to be broadly recognized.

A meta-analysis released in 2014\(^8\) demonstrated to the contrary – concluding that there is enough evidence supporting the perspective that the effects of air pollution on brains of children and teens ought to be key public health targets. Furthermore, BREATHE (a 12-month research study) in Barcelona aimed to analyse children’s developing brain, the superior working memory and attentiveness when exposed to higher or lower local traffic pollutants at school. The study took 2,715 children between ages seven and ten from 39 schools, who undertook computerised tests that took place once every 3 months over the course of a year\(^9\). The study concluded that those attending schools near busy roads and thus exposed to higher traffic-related air pollution, showed lesser improvements in cognitive development and only a 7.5% 12-month increase in working memory – compared to 11.5% for those who attended schools away from busy roads\(^10\).

A report written on the behalf of the National Institute of Environmental Health Science panel in 2012 detailed increasing evidence linking air pollution to behaviour deficits, neuro-inflammation and neuropathology in human and animal studies, as well as the need for further research\(^11\). It discusses living in conditions with elevated air pollution, with links to a decrease in cognitive function, lower neuro-behavioural testing scores in children and a decline in neuropsychological development in the first 4 years of life, which is supported by research on reduced cognitive function in children conducted by Schwartz in 2011\(^\)\(^12\). Finally, a paper written by Physicians stated that children exposed to air pollution don’t do as well on brain function tests compared to children tested from clean air areas. The air pollution exposed children also tend to have smaller brain volumes and have evidence of brain tissue abnormalities on brain imaging\(^13\).

6.2 SUMMARY

It is important to note that the underlying mechanisms and sequence of events that culminate in neurotoxicity remain poorly understood, the extent of the effects contributing to ill health is not yet known and the individual components of air pollution responsible are also not
yet known. These results allow some preliminary hypotheses to be drawn, but the certainty of these cannot yet be guaranteed without further research. But contrary to the current hesitant approach, there is enough evidence supporting the perspective that the effects of air pollution on brains of children and teens ought to be key public health targets, especially as these effects may have long-term clinical repercussions in terms of degenerative diseases.
7. Diabetes and Obesity

Diabetes is a growing epidemic; both in developing and developed countries - despite this, diabetes itself remains somewhat of a mystery87. In recent years, studies have started linking air pollution to diabetes, with tests to find out whether pollutants can be the cause of earlier development of type 1 diabetes in children88, this is compared to diabetes in adults, which was mainly linked to type 2. In addition to this, the childhood obesity epidemic is of huge concern, and while diet and a lack of physical activity has always been blamed – there is an emerging sector of research pointing to additional environmental factors.

A 2002 pilot study in southern California concluded that an increase in ozone exposure may be a contributory factor to an increased incidence and PM10 may be a specific contributory factor to the development of type 1 diabetes before 5 years89 and a follow up study in 200690 found effects associated exposure to O3 and SO291.

In 2013, a Chilean study looked also at ozone and PM2.5 levels and the temporal patterns of Type 1 Diabetes in infants younger under age 15. The research found that PM2.5 levels were associated with the onset of the disease92. Moreover, a German project93 identified that high exposure to NO2 and larger/medium sized particulate matter, was linked to acceleration in the manifestation of type 1 diabetes in very young children94.

Finally, the Institute of Diabetes Research at the Helmholtz Centre analysed 672 diagnosed children and took other environmental factors into account; namely history of diabetes in each patient’s family, body mass index and education level of parents, and then compared exposure to air pollutants with the time of diagnosis. Those who have been exposed to high levels of local air pollution in residential environments were more prone to developing the condition three years prior to those who lived in lower polluted areas95.

To take the research one step further, near roadway air pollution exposure has been associated with increased body mass index and obesity in children, and excess weight gain in animal models. This emerging evidence of a broad spectrum of environmental chemicals having ‘obesogenic’ properties and altering the metabolic profile of adipose tissue challenges the prevailing model that the childhood obesity epidemic is explained solely by increased caloric density of food and decreased physical activity96.

In addition to all the literature detailed above, recent emerging evidence suggests that air pollution exposure may contribute to the development of obesity and type 2 diabetes in children (as seen in adults). The objective of a University of Southern California led project97 was to determine whether exposure to elevated concentrations of nitrogen dioxide (NO2) and PM2.5 had adverse effects on longitudinal measures of insulin sensitivity, β-cell function, and obesity in children at high risk for developing diabetes. Overweight and obese Latino children (8-15 years; 314 pupils) were enrolled from 2001 to 2012 in Los Angeles, and were followed up for an average of 3.4 years.
By the time the participants turned 18, their insulin-creating pancreatic cells were 13% less efficient than normal, making these individuals more prone to eventually developing Type 2 diabetes, researchers said. This is the first to follow children for years to find a connection between air pollution and diabetes risk in children. When they turned 18, the participants had nearly 27% higher blood insulin after having fasted for 12 hours. During their two-hour glucose test, they had about 36% more insulin than normal, indicating that the body was becoming less responsive to insulin. This observation illustrated that increased exposure to air pollution was associated with increased risk factors for Type 2 diabetes.

7.1 SUMMARY

To conclude – it is apparent that air pollution exposure may be a predisposing, accelerating, or simply a surrogate factor in the development of type 1 diabetes in children96. The research discussed accredits the association with varying pollutants – showing the need for further research. Furthermore, the effects of air pollution on type 2 development in children is an emerging subject area requiring further understanding. Finally, further research is required into the effects of pollutant exposure on obesity levels in children.
8. CONCLUSION

A World Health Organization study estimates that meeting global air quality guidelines for PM2.5 could prevent 2.1 million deaths across all age groups (including children) per year based on 2010 data. It could also improve the overall health of millions more, help to reduce the incidence of acute and chronic respiratory infections among children, and reduce complications during pregnancy and childbirth. Finally, studies show it could improve children’s physical and cognitive development, helping them to lead longer and more productive lives.99

As this report shows, air pollution matters greatly to the health of children and the quantity of evidence is still growing. Overall, the effects on children’s heart, brain, hormone systems and immunity are proven and the effects on growth, intelligence, development of brain and coordination are still growing. To take this one step further, the relationships between improved health, cognitive and physical development, higher incomes and improved economic performance are well documented. Furthermore, reduced air pollution can also help lower health expenditures at household and government levels – which add up to billions of pounds of savings at the national level. An OECD study shows that the total annual costs of air pollution currently account for approximately 0.3% of global GDP, and are expected to increase to approximately 1% of GDP by 2060. A World Bank/Institute for Health Metrics and Evaluation study found that deaths from air pollution cost the global economy about US$225 billion in lost labour income and more than US$5 trillion in welfare losses in 2013.100

While there is considerable data linking early life exposure to air pollution to both short- and long-term adverse health effects, important knowledge gaps still exist. A substantial component of the global burden of disease is attributable either directly or indirectly to air pollution exposure. Ambient air quality can be improved through regulation and technology to reduce vehicle and industrial emissions. A greater understanding of the adverse health consequences of exposure to air pollution in early life is required to encourage policy makers to reduce such exposures and improve human health.
REFERENCES


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Roadway Air Pollution Contribute to Childhood Obesity?

Epidemiology of Environmental Health. 5(5), 972-984.


APPENDIX 1

All references below are from a medical dictionary

**Allergic sensitization**: The development of antibodies to a foreign substance (e.g., medication) that results in an allergic reaction.

**Artery**: Any of the muscular elastic tubes that form a branching system and that carry blood away from the heart to the cells, tissues, and organs of the body.

**Asthma**: An inflammatory disease of the lungs characterized by (in most cases) reversible airway obstruction. Originally, a term used to mean "difficult breathing"; now used to denote bronchial asthma.

**Atopy**: A genetically determined state of hypersensitivity to environmental allergens. Type I allergic reaction is associated with the IgE antibody and a group of diseases, principally asthma, hay fever, and atopic dermatitis.

**Autism**: A mental disorder characterized by severely abnormal development of social interaction and of verbal and nonverbal communication skills.

**Behavioural deficits**: condition in which there is impairment in physical and mental functioning such that age-specific aspects of behaviour are lacking in an individual.

**Birth weight (low)**: in humans, the first weight of an infant obtained within less than the first 60 completed minutes after birth; a full-size infant is one weighing 2500 g or more; a low birth weight is less than 2500 g.; very low birth weight is less than 1500 g.; and extremely low birth weight is less than 1000 g.

**Blood pressure**: the pressure or tension of the blood within the systemic arteries, maintained by the contraction of the left ventricle, the resistance of the arterioles and capillaries, the elasticity of the arterial walls, as well as the viscosity and volume of the blood; expressed as relative to the ambient atmospheric pressure.

**Bronchiolitis**: Inflammation of the bronchioles, often associated with bronchopneumonia.

**Bronchitis**: Inflammation of the mucous membrane of the bronchi.

**Cardiac ventricular septal defects**: a congenital defect in the septum (membranous or muscular) between the cardiac ventricles.

**Cognitive function**: an intellectual process by which one becomes aware of, perceives, or
comprehends ideas. It involves all aspects of perception, thinking, reasoning, and remembering

**Conotruncal heart defects**: They are usually defined as malformations of the cardiac outflow tracts and presumably result from either a disturbance of the outflow tract of the embryonic heart, or impaired development of the branchial arch and arteries, or both.

**Hypertensive disorder**: Hypertension or high blood pressure, sometimes called arterial hypertension, is a chronic medical condition in which the blood pressure in the arteries is elevated.

**Infant mortality**: a measure of the rate of deaths of liveborn infants before their first birthday; the numerator is the number of infants under 1 year of age born alive in a defined region during a calendar year who die before they are 1 year old; the denominator is the total number of live births; often quoted as a useful indicator of the level of health in a community.

**Intrauterine growth restriction**

**Neurodegeneration**: selective degeneration of neurons; may be entire neuron (neuronopathy) or restricted to the axon (axonopathy); may also be central, or peripheral, or central and peripheral.

**Neurodevelopment**: to the processes that generate, shape, and reshape the nervous system, from the earliest stages of embryogenesis to the final years of life

**Neuro-inflammation**: inflammation of a nerve or of parts of the nervous system

**Neuropathology**: Pathology of the nervous system and that branch of pathology concerned with the nervous system.

**Neuropsychology**: A specialty of psychology concerned with the study of the relationships between the brain and behaviour, including the use of psychological tests and assessment techniques to diagnose specific cognitive and behavioural deficits and to prescribe rehabilitation strategies for their remediation.

**Neurotoxicity**: the degree to which a substance is poisonous to nerve tissue and the condition resulting from exposure to aneurotoxin.

**Pneumonia**: Inflammation of the lung parenchyma characterized by consolidation of the affected part, the alveolar air spaces being filled with exudate, inflammatory cells, and fibrin

**Post-neonatal mortality**: Mortality between the ages of one month and one year. It is a part
of infant mortality

**Preterm delivery**: the birth of a baby of less than 37 weeks gestational age.

**Respiratory illness**: a disease affecting the respiratory system

**Respiratory syncytial virus**: an RNA virus of the genus Pneumovirus, in the family Paramyxoviridae, with a tendency to form syncytia in tissue culture, which causes minor respiratory infection with rhinitis and cough in adults, but is capable of causing severe bronchitis and bronchopneumonia in young children.

**Sudden infant death syndrome**: the sudden death of an apparently healthy infant that remains unexplained after all known possible causes have been ruled out through autopsy, death scene investigation, and review of the medical history.

**Trimester**: A period of 3 months; one third of the length of a pregnancy.