The Impact of Air Pollution on Children's Health

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Abstract

Air pollution poses a major environmental risk to our health. Particulate matter affects more people than any other pollutant and is therefore commonly used as a proxy indicator for air pollution. Children are of particular interest, since they are uniquely vulnerable to the effects of exposure to air pollution.

Little is known about the effects of air pollution in healthy children as studies often focus on an adult or elderly population, or children with an underlying condition such as asthma. Additionally, the large variation in study design across available research leads to inconsistent results. This review will give an overview of existing studies on the effect of air pollution in children.

Overall, evidence can be found, supporting the detrimental effects of exposure to air pollution on certain health outcomes. As such, negative associations were found between both children's respiratory health and neurocognitive functions and exposure to air pollution. Moreover, a possible link could be unveiled between endothelial dysfunction and cardiovascular, respiratory or neurocognitive effects in response to exposure to air pollution. A future challenge remains to generalize study designs as much as possible. Acute respiratory effects, neurocognitive changes or effects on endothelial function in children in relation to PM exposure are still scarcely studied in healthy children, especially based on high resolution personal monitoring data. Furthermore, there is still insufficient evidence for causal associations.

General introduction

"The United Nations General Assembly has formally declared access to a clean, healthy, and sustainable environment a universal human right, especially for children. In 2023, the United Nations Committee on the Rights of the Child emphasized the children's right to a clean, healthy, and sustainable environment. ***Yet, it is a right that goes unfulfilled for billions of people"*** *(1).*

It is clear that air pollution is a major environmental risk factor to our health (2). According to the World Health Organization (WHO), air pollution forms the second leading cause of deaths from noncommunicable diseases such as asthma, cardiovascular problems and respiratory diseases including lung cancer, even for children (1, 2). In 2020, air pollution was even officially recognized as the direct cause of a young girl's death in the United Kingdom—the first time it had been explicitly identified as such (3).

Air pollution

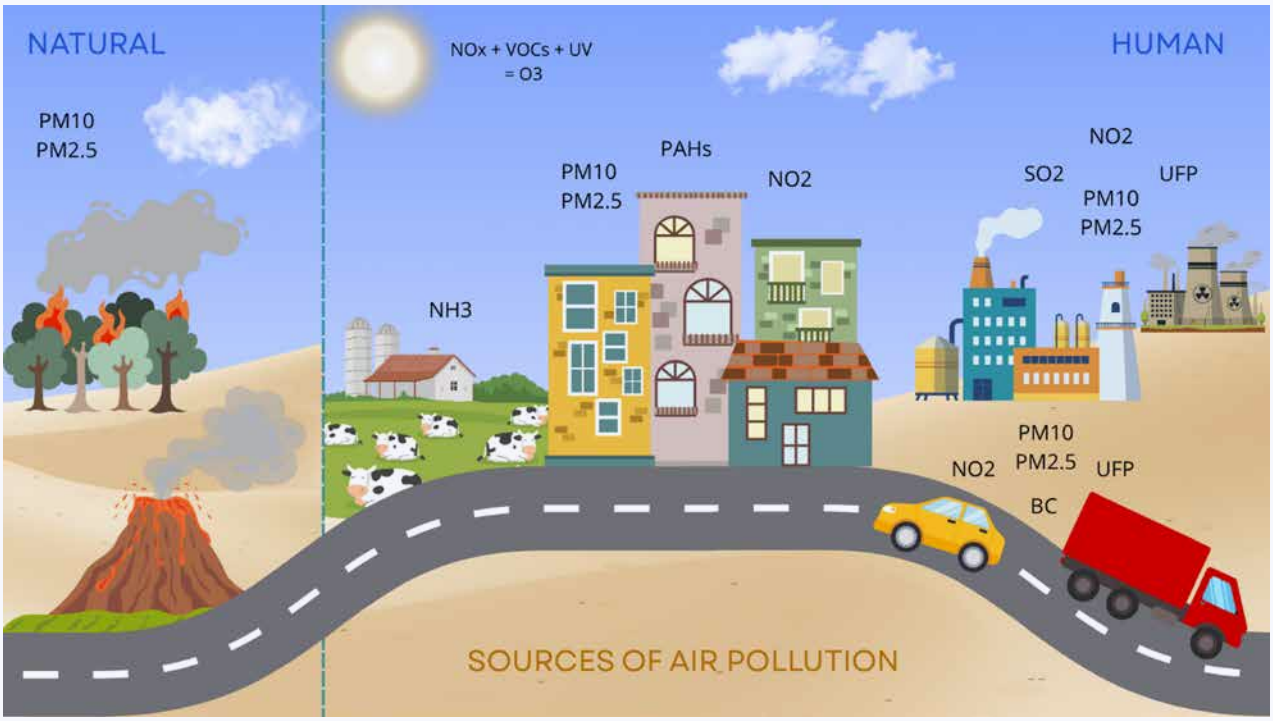
Air pollution is a complex matter, and different types of pollution exist. Pollutants are often linked and considerable interactions between them complicates determining the effects of a single pollutant. Air pollutants that affect the public health include particulate matter (PM), ozone (O3), nitrogen dioxide (NO2), polyaromatic hydrocarbons, sulphur dioxide (SO2) and ammonia (NH3). They are shown in Figure 1, with the corresponding major sources. Among these, PM, NO2 and O3 exert the greatest health impact, and they contribute to premature death (4). Even though in the EU, air pollution related deaths decreased by 45% between 2005 and 2022, PM2.5 still contributed to an estimated 239,000 premature deaths in 2022, while O3 and NO2 contributed to an estimated 70,000 and 48,000 premature deaths (4). In Flanders, about 3500, 1800 and 800 premature deaths were attributable to PM2.5, O3 and NO2 respectively (5).

PM, affecting more people than any other pollutant, is considered a major air pollutant by the WHO. It is commonly used as proxy indicator for air pollution (2). PM is classified according to the aerodynamic diameter of its particles: PM10, PM2.5 and ultrafine particles (UFPs) are the main fractions (Figure 2). PM10 includes particles ≤ 10 µm in diameter, originating from natural sources (e.g., Saharan or volcanic dust) or human activities such as fuel combustion for industry, agriculture or road transport. PM2.5 refers to finer particles (≤ 2.5 µm), produced directly by combustion processes or formed secondarily through atmospheric reactions between gases such as NH3 from livestock and traffic nitrogen oxides (NOx). UFPs, with a diameter ≤ 0.1 µm, can also be emitted directly into the atmosphere or formed from gaseous precursors. These particles are inherently unstable and, similar to PM2.5, can grow into larger particles through coagulation or condensation. Transportation and industry are important sources (6).

Black carbon, a major component of soot, is an important constituent of PM. It is a primary pollutant mainly produced by incomplete combustion from traffic, industry, heating and biomass burning and is often used as a proxy for traffic exhaust (7). In areas dominated by combustion-derived pollution, PM can contain substantial amounts (up to 80–90% of the UFP mass) of black carbon and organic carbon. Metals and polyaromatic hydrocarbons are other important constituents of PM (6).

Nitrogen oxides mainly consist of NO2 and nitric oxide (NO). NO is emitted into the atmosphere and immediately thereafter, NO2 is

FIGURE 1: The main sources of air pollutants that are known to affect public health: particulate matter (PM), ozone (O3), nitrogen oxides, polyaromatic hydrocarbons (PAH's), sulphur dioxide (SO2) and ammonia (NH3).



formed as a result of chemical reactions with e.g. ozone. The large, and growing body of scientific evidence linking NO2 with various health outcomes has shown that short-term exposure to NO2 can irritate the airways and aggravate existing respiratory conditions (2). A major source of NO2 is traffic exhaust. As such, NO2 is a common pollutant in cities and the highest levels are observed in high-income, urbanized and densely populated areas. NO2 is therefore, like black carbon, used as a proxy indicator for traffic pollution. Burning of fuels in power plants or industry are additional sources of NO2 (1).

Episodes of intense air pollution are often referred to as smog. O3 is a major component of so called 'ozone smog' or 'summer smog'. Whereas the major component during 'winter smog' is PM (8). O3 is synthesized by a photochemical reaction with exhaust gases such as NO2 and volatile organic compounds, in the presence of UV sun radiation, especially in wind still weather conditions. The highest ground-level levels of O3 therefore occur during periods of sunny, warm weather (9). Exposure to excessive levels of O3 can affect breathing, provoke coughing and wheezing, trigger asthma, reduce respiratory function or lead to lung disease (2).

The interplay between air pollution and climate change

Greenhouse gases, such as CO2, methane and O3 in the atmosphere absorb radiation and as such trap heat in a way the planet can no longer lose it, causing global warming. Over the last 150 years, human activities are almost entirely responsible for the increase in greenhouse gases in the atmosphere (10, 11). The burning of biomass and fossil fuels (for electricity, heat, or transportation) as important sources of air pollution, also contribute to the emission of these greenhouse gases. Black carbon is also suggested as an important contributor to global warming (7). Hence, the continuous use of biomass or fossil fuels not only enriches air pollutant concentrations, it also aggravates the impact on our climate. Meanwhile climate change and its consequences enhance air pollution: more severe and prolonged droughts, drier lands, wildfires and dust storms (12). Additionally, summers become warmer and more heatwaves occur, which in turn accelerates the formation of O3 due to a more vigorous catalysation of e.g. NO2 (9).

Air quality management

Overall, global and European air quality has improved in recent decades due to declining emissions. In Flanders for example, 2024 was a favourable year due to both rainy weather and reduced emissions. Emissions from industry and energy sectors are declining, while households (heating of buildings) and traffic are increasing sources of PM, polyaromatic hydrocarbons and NOx. Agriculture remains the predominant source of NH3 (13).

Nevertheless, there are no safe levels of air pollution according to the WHO: even very low levels of PM2.5 can cause adverse health effects (2). In other words, concentrations should be kept as low as possible. Therefore, air quality guidelines are in place (table 1). While the WHO sets strict guidelines to protect public health, the European Commission has adopted less stringent standards in its legislation, under the Ambient Air Quality Directive. The Ambient Air Quality Directive contains emission limits for pollutants such as PM10, PM2.5, NO2, O3, SO2, CO, Pb, and benzene. Black carbon has only recently been included in air quality management, but no specific guidelines exist yet. The European Commission proposed a revised Ambient Air Quality Directive, introducing stricter targets to be achieved by 2030. The new limits for PM2.5 and NO2 remain twice as high as WHO recommendations but should still represent substantial progress. Member states are required to implement these targets into national law, within two years. Additionally, the EU Zero Pollution Action Plan targets a toxic-free environment by 2050, aiming to reduce air, water, and soil pollution to levels no longer harmful to human health or ecosystems (14).

Particulates toxicity

Determinants of particulates toxicity

PM is a complex mixture of particles that vary in mass, number, size, shape, surface area, chemical composition as well as reactivity, acidity, solubility and origin (6). Particulates behaviour in our respiratory system and their potential to cause adverse health effects is directly linked to their size, chemical characteristics and surface area to interact with human tissue (6). Particles from different combustion

TABLE 1: Air quality guidelines.

European Union (EU) air quality standards = Limit values for the protection of human health to be attained by 1 January 2030 (8). and World Health Organization (WHO) global air quality guidelines (2). The dashes indicate the absent guidelines.

	PM_{10}		$PM_{2.5}$		NO_2		UFP's	BC
	EU	WHO	EU	WHO	EU	WHO		
1 hour	-	-	-	-	200 µg/m³ [a]	-	-	-
24 hour	45 µg/m³ (b)	45 µg/m³	25 µg/m³ (b)	15 µg/m³	50 µg/m³ [b]	25 µg/m³	-	-
Annual	20 µg/m³	15 µg/m³	10 µg/m³	5 µg/m³	20 µg/m³	10 µg/m³	-	-

[a] Not to be exceeded more than 3 times per calendar year (8). - [b] Not to be exceeded more than 18 times per calendar year (8).

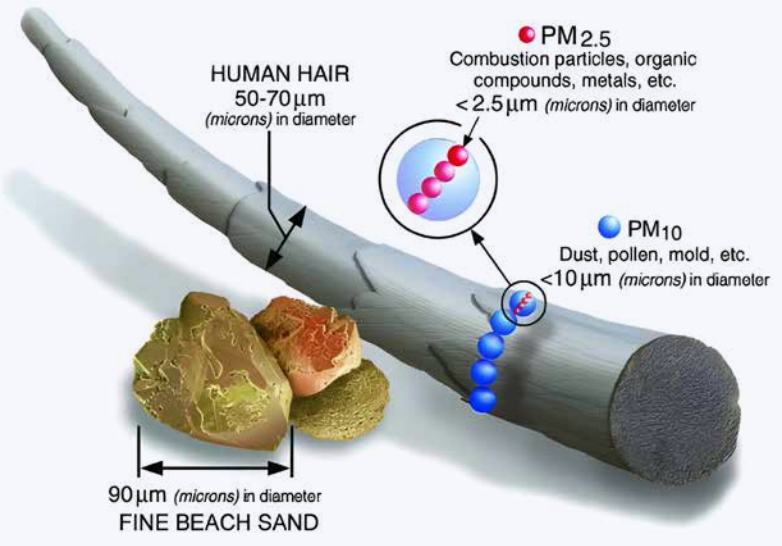
sources can vary in chemical composition, which makes some particles more relevant to human health than others (6). One epidemiological study including children with asthma for example, reported weaker associations between an airway inflammation marker and particle mass, compared to specific PM components (black carbon and organic compounds in particular) (6, 15). Furthermore, water-soluble gases (such as SO2) are likely to react with the mucus layer of the upper airways, while less soluble gases (such as NO2) more often reach the alveoli. Regarding particulates, the smaller the particles (i.e. PM2.5, UFP or black carbon) the deeper they can penetrate into the lungs (to the alveoli and terminal bronchioles) and the further they can migrate through the body via entering the bloodstream (16). In this context, UFPs are considered the greatest risk to our health. They are small enough to enter the bloodstream (even end up in brain tissue) inducing inflammation and potentially promoting cardiovascular, respiratory and cognitive problems. In contrast, larger particles, e.g. PM10 remain in our airway system (6). Apart from penetrating deeper into the lung, smaller particles also have a greater surface area on a mass basis, allowing more toxic chemicals to adsorb onto their surfaces compared to larger particles (6).

Mechanisms of toxicity

Air pollutants possess the ability to act directly as pro-oxidants of proteins and lipids, or as generators of free radicals such as reactive oxygen species (ROS). In the human body, free radicals (including ROS) are continuously generated during normal metabolism and they play a crucial role in cellular processes (17, 18). However, when concentrations increase considerably, for example due to exposure to exogenous components, excess reactive oxygen species can result in a state of oxidative stress. (18, 19). Exposure to air pollution was already associated with increased levels of reactive oxygen species, and oxidative stress has been associated with various diseases, including heart attack, stroke, chronic inflammatory disease, Alzheimer and even cancer (17, 18).

The body's immune system can be activated by pollutants, resulting in elevated cytokine expression and stimulation of an inflammatory response. As such, oxidative stress and the induction of inflammatory responses (through deposition in the lungs, or systemic inflammation due to translocation of particles to the blood circulation) are common suggested cellular mechanisms by which most pollutants exert their adverse health effects (18). Since research results showed that PM could carry heavy metals and polyaromatic hydrocarbons, the presence of these components may also contribute to PM toxicity (20-22).

FIGURE 2: Size comparisons for PM particles (United States Environmental Protection Agency – available from: https://www.epa.gov/sites/default/files/2016-09/pm2.5_scale_graphic-color_2.jpg)



Size comparisons for PM particles

Endothelial function

These suggested underlying mechanisms, oxidative stress and inflammatory responses, potentially provoke endothelial dysfunction. Endothelial dysfunction is an early predictor of cardiovascular disease (23). Furthermore, it is hypothesized that particulates affect the cerebral microvasculature resulting in a decreased perfusion and thus insufficient oxygen availability. Hence, a possible link could be unveiled between endothelial dysfunction and cardiovascular, respiratory or neurocognitive effects in response to exposure to air pollution.

The vascular endothelium, aligning the inside of our blood vessels, is an interesting target for studying the effects of air pollution since the smallest particles can enter the bloodstream. Endothelial function can be assessed using various methods. First, (surrogate) blood markers for endothelial function are often measured, with endothelin-1, a vasoconstrictor and inflammatory mediator, as most common reported marker. Secondly, different methods exist to directly assess the vascular function. These include assessing the retinal microvasculature, a proxy for the systemic microcirculation, measuring

the flow-mediated dilation of the brachial artery and application of peripheral arterial tonometry.

Despite the variation in studies, findings are pointing in the same direction: evidence supports the association between exposure to air pollutants and signs of endothelial dysfunction. Exposure to traffic-related air pollution, particularly UFPs, has been linked to a reduced endothelial function in adults (24, 25). However, evidence in children remains limited. Hashemi et al. reported that exposure to PM10 and passive smoking impaired the flow-mediated dilatation of the brachial artery (hence reduced the endothelial function) in healthy children. Both PM10 and passive smoking were also inversely associated with the basal brachial artery diameter (26). In the same study population, Kelishadi et al. found that PM10 was associated with a decrease of serum NO levels (a potent vasodilator) and an increase in C-reactive protein (CRP), an inflammatory marker) (27). Similarly, Prunicki et al. observed elevated CRP and other immune markers in relation to air pollution (28), while Calderón-Garcidueñas et al. reported higher plasma endothelin-1 levels among children chronically exposed to PM2.5 in Mexico City (29, 30).

PM was also found to affect the retinal microvasculature in children. However, results are inconsistent and appear to depend on the duration of exposure (e.g. recent versus chronic exposure or prenatal versus postnatal exposure) and on the interaction between pollutants (e.g. PM2.5 and Ox (the combined oxidant capacity of O3 and NO2)) (31-33). Provost et al., found that short-term exposure to PM2.5 was associated with narrower retinal arteriolar diameters and wider venular diameters, consistent with the findings of other studies in both children and adults (27, 34). Whereas Luyten et al. found that exposure was associated with a widening of the retinal arterioles (32). Korsiak et al. reported that arteriolar narrowing was associated with PM2.5 only when Ox concentrations were elevated. No clear associations between PM2.5 or Ox and venular diameter were found here (33).

Air pollution as an environmental threat to our health

Worldwide, 99% of the population (including hundreds of millions of children) live in areas with levels levels exceeding international guidelines exceeding international guidelines (35, 36). Yet, the most vulnerable groups of the population (such as children, elderly, or people living in developing countries) are disproportionately affected by exposure to air pollution. Inhalation is the main route of exposure: breathing polluted air causes adverse effects to our health, targeting certain organs and systems. The effect of exposure on our respiratory function (volume/flow) is therefore of interest. Since evidence is growing that exposure to air pollution can disrupt cognitive development, an additional important health concern is the effect of PM on brain function.

Children experience some of the greatest health effects due to exposure.

Children are a uniquely vulnerable part of the population, so their exposure to air pollution is of particular concern. (37). Their immune system, neuropsychological abilities and lungs are not fully developed, which may lead to different health responses as compared to adults or even affect the development of lungs and cognition (37). Children have a relatively larger lung surface and breathe more air per mass of body weight compared to adults. They spend more (active) time outside as well, sometimes during peak traffic times. Furthermore, young children play closer to the ground, where PM is often more concentrated (17, 38). Chronic exposure to high levels of air pollution during pregnancy can even affect the foetus and is associated with low birth weight, preterm or even still birth (39, 40). If left untreated, some of these health impacts may have lifelong consequences. Social and economic effects (such as school attendance and performance, health costs and productivity potentially affecting income, poverty and inequities) could occur as well.

Respiratory effects

Air pollution has been widely recognized as a contributor to a range of adverse respiratory effects, ranging from allergic reactions and airway irritation to breathing difficulties, wheezing, coughing, asthma, chronic inflammation, and even lung cancer. Commonly used parameters for assessing respiratory function include forced expiratory volume in one second (FEV1), which reflects airflow through the large and medium airways and forced vital capacity (FVC), which indicates lung size. In addition to these physiological measures, many studies evaluate respiratory health through the prevalence of respiratory diseases (e.g., asthma, infections) and symptoms such as coughing, wheezing or hospitalizations (35).

To date, relatively few research exists on acute effects on the respiratory function, especially considering healthy children. Exposure studies that exist, report on long(er)-term rather than acute effects while other studies are restricted to adult participants. Research also often focuses on vulnerable groups such as asthma patients (41-43). Weeda et al. for example, reviewed the effects of climate variables, including temperature and air quality, on children's respiratory health. Most studies examined the relationship between air quality and the risk of asthma, (asthma-related) emergency department visits, and other respiratory infections (44). Although findings varied, most reported an increased risk of asthma associated with higher levels of air pollutants such as PM2.5, NO2, and O3. Similarly, Boogaard et al. reported positive associations between NO2 and other traffic-related pollutants with asthma onset and acute lower respiratory infections in children in their review (45). Elevated pollutant levels were also linked to higher rates of childhood pneumonia and combined respiratory disease–related hospitalizations, as well as increased all-cause respiratory emergency department presentations linked to PM2.5 and PM10 exposure (46-49).

Schultz et al. reviewed studies investigating the effects of traffic-related pollutants on children's RF. Despite some inconsistencies, most of the studies reported a negative impact of traffic-related air pollution on RF (35). Exposure to PM10 and NO2 during both early life and over the lifetime was found to be negatively associated with RF, while others observed stronger associations with current exposure at children's home addresses rather than at birth. Lifetime and past-year exposure to black carbon and PM2.5 were primarily linked to reduced FVC, whereas exposure to PM10 and NO2 during the first year of life was associated with lower FEV1. These results align with Fuertes et al., who observed a strong effect from early life exposure to PM10 (42). Similarly, Bergstra et al. reported that exposure to industry-related pollutants, including PM2.5 and NO_x, was associated with reduced RF (50).

Neurocognitive functions

Elevated concentrations of PM are suggested to negatively affect cognitive functions, such as the ability to think and make decisions, and even affect cognitive development (37, 51). Cognitive functions are essential for learning and achievement. These functions develop significantly during childhood, especially at primary school age. Child and adolescent exposure to air pollution is therefore of special concern, since brain development continues until the second decade of life (16). Inflammatory responses were observed in the brain regions related to executive function after exposure to traffic-related air pollution (52). Evidence was also found for the breakdown of the blood–brain barrier, and thus impairment of its integrity, after exposure in children (53). Likewise, fine PM (especially UFPs) can translocate to the olfactory bulb and migrate to the olfactory cortex, causing tissue damage and local

inflammation (neuroinflammation and damage of neural tissue) after inhalation through the nose (17, 54).

The term neurocognitive functions encompasses a wide and diverse range of cognitive processes and neural mechanisms, making it a highly broad and multifaceted concept. This is reflected in the variety of cognitive outcomes tested in literature, as reported in different review papers: Clifford et al. broadly described three categories: 1] measurements of cognitive function (including intelligence, memory and learning, visual-motor coordination, executive function and attention, and global cognition); 2] measures of neurodevelopment; and 3] tools to identify age-related decline (55). Gartland et al. described two main categories, namely school attainment and executive function, which mainly encompasses working memory and attention (17). Thompson et al. defined a wide range of cognitive functions, including general cognition; intelligence, IQ and reasoning; attention, working memory & executive function and memory and learning (56).

Studies suggest that PM2.5 affects attention and academic achievement. However, findings relating to attention were mixed: higher levels of indoor PM2.5 were associated with increased inattention across one year, whereas Alvarez-Pedrerol et al. found no significant effect (57, 58). PM10 is suggested to have effects on attention, reasoning, and test scores, even though the evidence is limited compared to PM2.5. While working memory seemed to be the main outcome affected by PM2.5, no effects were reported for PM10. In a cross-sectional study in Belgium, Saenen et al. reported a negative effect of acute and chronic exposure on selective attention, and on sustained attention (chronic exposure only), rather than on the short-term memory in healthy children (37). Regarding NO2, the limited evidence suggests a potential specific effect on working memory, while no impact on attention has been observed (17, 59, 60). Other studies, however, reported mixed findings of the impact of NO2 on cognitive outcomes (17). Lastly, evidence also supports the potential effect of elemental carbon/black carbon and UFPs on attentional outcomes, as well as the association between long-term black carbon exposure and a decreased cognitive function in primary school children (61, 62).

Exposure studies comparison

To date, inconsistencies persist across studies due to variations in study design, exposure assessment, and reported pollutants. Study designs range from retrospective to cross-sectional and longitudinal studies, each using different methods to estimate exposure. For example, some studies compared children living in areas with varying levels of air pollution, measured at central monitoring stations or at schools (35). Other exposure estimates included traffic indicators, such as traffic density or proximity to highways. Modelling individual data using land use regression models or dispersion models was also used. Reported pollutants differ as well, with PM2.5 being the most reported pollutant, followed by PM10, NO2 and O3 (44). Further variability arises from differences in pollutant combinations, emission sources (including traffic-; fire-; or industry-related air pollution), duration and the window of exposure (prenatal, early-life, recent or chronic exposure). Health outcomes also vary across studies. Despite the amount of research available, it remains challenging to draw consistent conclusions. However, most evidence supports the negative health impact of air pollution.

Conclusions

Described above are the effects of air pollution on respiratory function and neurocognitive outcomes, and the potential role of endothelial (dys-)function. The focus was set on children, since they are uniquely vulnerable to effects of air pollution. Although, effects from exposure to air pollution are well documented, it is important to point out that the evidence resulting from this research is of small to moderate certainty and often not strong enough to draw firm conclusions. The findings are also far more nuanced than presented here and should therefore be interpreted with caution.

A future challenge is to specify and harmonize the research methodology as much as possible, and to encourage its widespread use. In addition, more work is required to disentangle the impact of different exposures, the potential mechanisms, and the context in which they occur (56). Acute respiratory effects, neurocognitive changes or effects on endothelial function in children in relation to PM exposure are still scarcely studied in healthy children, especially based on high resolution personal monitoring data (51). Furthermore, there is still insufficient evidence for causal associations (37, 55, 63). Hence, there is a clear need for further experimental research.

Reducing air pollution can improve quality of life and save children's lives by lowering the risk of respiratory infections like pneumonia and asthma. It also reduces pregnancy and childbirth complications, supports healthy child development, and contributes to sustainable development and climate change mitigation.

Conflict of interest

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