Ambient ozone and children's health

AirClim
Air Pollution & Climate Secretariat

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Methane Matters

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Key findings:

- **Children are particularly vulnerable to ozone pollution.** Because they breathe more air relative to their body weight and their lungs and immune systems are still developing, they receive higher doses and may be more sensitive to its harmful effects.
- **There is consistent evidence that ozone pollution harms children's health.** A growing body of research links ozone exposure to the onset and exacerbation of asthma, other respiratory diseases and reduced birth weight.
- **Both long-term exposure and short-term peaks in ozone pollution can harm children.** Short-term episodes of high ozone are especially associated with respiratory illness and increased hospital visits.
- **Some health effects in children have been observed even at ozone levels below current guideline values set by the World Health Organization.**

Executive Summary

Tropospheric ozone is a harmful air pollutant formed largely from human activities. Unlike many other air pollutants whose impacts have declined in recent decades, the health burden associated with ozone has increased. Ozone is a secondary pollutant formed through chemical reactions between precursor pollutants in the presence of ultraviolet radiation. These reactions occur most readily under warm and sunny conditions. Because ozone formation is closely linked to heat and sunlight, climate change may further increase ozone levels and associated health risks.

Ozone pollution poses significant risks to human health, and children are particularly vulnerable. A growing body of research shows increasing evidence linking ozone exposure to adverse child health outcomes. The most well-studied associations include reduced birth weight, asthma and other respiratory diseases. Studies estimate a 6.4% increase in mortality among children under five for every 10 parts-per-billion (ppb) increase in ozone levels, as well as an average reduction in birth weight of around 20 grams. Short-term exposure to elevated ozone levels has also been linked to a 38% increase in upper respiratory symptoms in children within 2–6 hours of exposure.

Research also points to broader impacts, including associations with peri-natal outcomes, other respiratory disease, cardiometabolic diseases, infectious diseases, inflammatory and allergic conditions, cancer, and mental health. Evidence further suggests that disadvantaged populations may face greater exposure and risks.

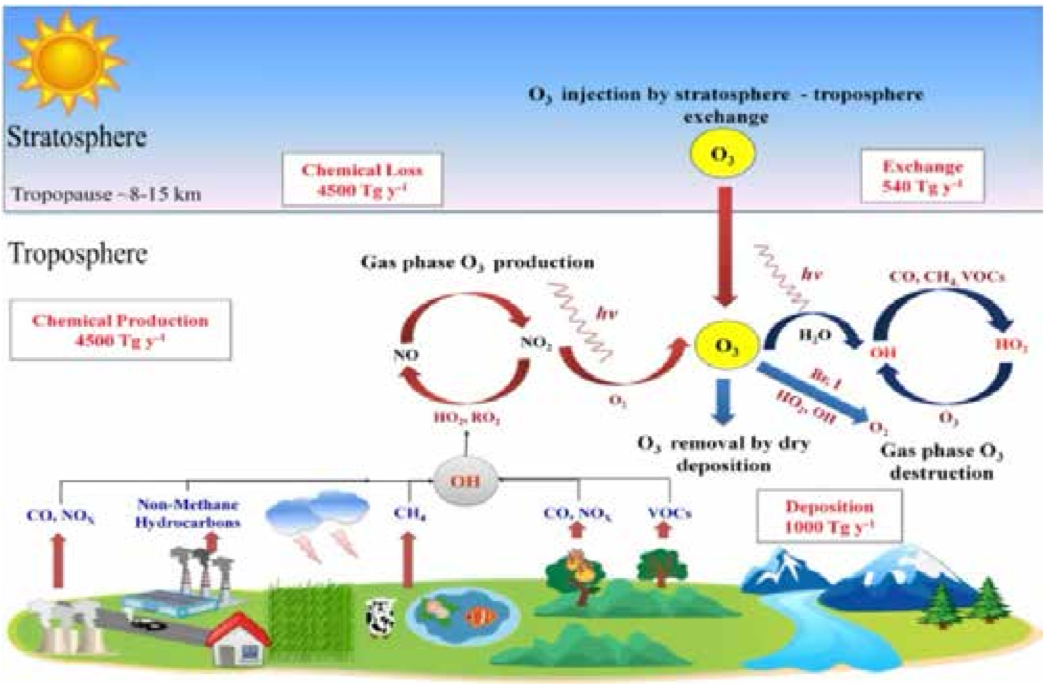
Current guidelines values set by the World Health Organization may not fully protect children's health. Both long-term exposure and short ozone peaks contribute to health risks, highlighting the importance of reducing overall pollution levels while also preventing acute pollution episodes.

Background

Tropospheric ozone is widely recognised as a significant air pollutant and environmental hazard, particularly in relation to human health (1, 2). Air pollution overall is recognised as the leading environmental risk factor for human health globally and a leading cause of premature death(1). As one of several harmful air pollutants, tropospheric ozone contributes to this burden. In addition to its health impacts, it is also recognised as a threat to the environment and ecosystems (2). While ozone pollution is a threat to everyone, children are particularly vulnerable and therefore the focus of this report.

Tropospheric ozone is a harmful air pollutant found in the lowest part of the atmosphere, up to 10 kilometres above the Earth's surface. It is primarily a secondary pollutant, formed through chemical reactions involving precursor pollutants in the presence of ultraviolet (UV) radiation. The main precursors are volatile organic compounds (VOCs) including methane (CH_4), and nitrogen oxides (NOx). These reactions occur under naturally existing atmospheric conditions, especially in warmer month and regions with strong sunlight (1, 3). The vast majority is formed secondarily from precursor emissions (2, 4). Up to 85% of tropospheric ozone pollution is estimated to originate from anthropogenic processes (4).

Figure: A schematic diagram of the sources and sinks of ozone (O_3) in the troposphere (CO: carbon monoxide; CH_4: methane; VOCs: volatile organic compounds; OH: hydroxide; HO_2: hydroperoxyl radical; Br: bromine; I: iodine; NO: nitric oxide; NO_2: nitrogen dioxide; RO_2: peroxy radicals; NOX: nitrogen oxides; hv: sunlight). Image source: (21)



While levels of many other air pollutants declined between 2000 and 2020, global ozone levels increased, largely driven by increasing anthropogenic emissions of precursor pollutants (3). Correspondingly, the global burden attributable to ozone pollution has grown. Over two decades, ozone related disease burden has increased by approximately 50%, reaching an estimated 470,000 deaths in 2023 from chronic obstructive pulmonary disease (COPD), the primary cause of mortality associated to ozone exposure (6). Ozone exposure is also estimated to contribute to 8-20% of asthma-related emergency hospital visits world-wide (7).

Climate change is expected to further influence the formation of tropospheric ozone, and may lead to increased levels in many regions (1-3, 8). Changing meteorological conditions, particularly higher temperatures and prolonged heat periods, favour ozone formation from precursor pollutants (3). Effective control of these precursors will therefore be increasingly critical in a warming climate.

Ozone is reported either in parts per billion (ppb) or micrograms per cubic metre ($\mu g/m^3$), depending on the jurisdiction. Conversion between these units depends on temperature and air pressure. At 25°C, a concentration of 60 $\mu g/m^3$ corresponds to approximately 30 ppb, while 100 $\mu g/m^3$ is roughly equivalent to 50 ppb. These values are particularly relevant as they correspond to the World Health Organization (WHO) guideline levels: the seasonal average over the six highest months of the year, and the 99th percentile of the daily maximum 8-hour average, respectively. (2).

The pathogenicity of ozone is mediated through several biological mechanisms. Ozone gas has relatively low water solubility, allowing it to penetrate deep into the respiratory tract upon inhalation (1). As a highly reactive gas, it interacts with the respiratory epithelium and initiates an inflammatory cascade, characterised by the generation of reactive oxygen species (ROS) and oxidative stress (1). Ozone reacts with unsaturated fatty acids in the epithelial lining fluid, producing a range of secondary reactive compounds (2). This cascade activates immune and inflammatory pathways, leading to the release of signalling molecules and hormones, and resulting in damage to lipids, proteins, cellular structures, DNA and mitochondria, ultimately contributing to cell damage and cell death (1, 2). Alterations to immune functions and the microbial environment of the respiratory epithelium may further increase susceptibility to infection (1, 9). In addition, products of these inflammatory processes may translocate within the body, including across the blood-brain barrier (2).

Children are widely recognised as a vulnerable population with respect to environmental stressors, including air pollution. The health effects of ozone are especially pronounced in children for two main reasons. First, children inhale a greater volume of air relative to their body weight compared to adults, resulting in a higher dose of pollutants per kilogram of body mass and potentially greater toxic effects (2). Second, their respiratory and immune systems are still developing (2), which may reduce their ability to effectively respond to and repair ozone-induced damage.

These vulnerabilities have measurable consequences. A large multicentre study found a non-linear association between ozone exposure and child mortality, estimating that each 10 ppb increase in ozone concentration gave a 6·4% increase in mortality risk among children under five years of age [95% Confidence Interval (CI) 2·4–10·7] (2, 10). Importantly, adverse effects on child health can occur even at ozone concentrations below the guideline levels set by the WHO (2).

Evidence on child health effects

There is an extensive body of research examining the health effects of ozone pollution, including its specific impacts on children. In 2025, a comprehensive systematic review synthesised and evaluated the available evidence to assess the strength and consistency of associations between ozone exposure and children health outcomes. The review concluded that consistent evidence exist that ozone exposure is a risk factor for low birth weight, asthma, respiratory disease and obesity. It further suggested that the current ozone control standards may be inadequate to protect children's health (2).

Importantly, the review found that both chronic exposure and short-term peaks in ozone concentrations can harm children's health.(2). It also highlighted patterns of environmental inequality in the international literature, indicating that the burden of ozone exposure is not evenly distributed, with greater impact in less privileged areas and regions (2). Additional studies have been published since this review, further expanding the evidence base (13-20).

The outstanding evidence from this systematic review, and from additional research since the end of the reviewed research window in the review are as follows.

The following studies reference both Odds Ratio (OR) and Hazard Ratio (HR), both of which are statistical measures of association used to quantify the relationship between an exposure and an outcome. An Odds Ratio (OR) compares the odds of an outcome occurring in an exposed group to the odds in an unexposed group, and is commonly used in case–control and cross-sectional studies. A Hazard Ratio (HR) reflects the rate at which an outcome occurs over time in one group compared with another, and is typically used in longitudinal or survival analyses.

In both cases, a value above 1 indicates an increased likelihood or risk associated with the exposure, a value below 1 indicates a decreased likelihood, and a value of 1 suggests no association. In some contexts, values above 1 can be interpreted approximately as a percentage increase in risk (for example, an OR of 1.38 may indicate a 38% higher odds of the outcome), although interpretation depends on study design and context..

Pregnancy and peri-natal outcomes

- Birthweight: Ozone exposure during pregnancy has been associated with reduced birth weight. In a study of 697,148 singleton births in 54 countries, a 10 ppb increase in ozone exposure was linked to an average reduction in birth weight of 19.9 g [95% CI: 14.8–24.9 g]. However, the association varied across trimesters, indicating that the timing of exposure during pregnancy may influence its impact. (2).
- Preterm birth: A 10 µg/m^3 increase in ozone exposure was linked with an increased odds ratio (OR) of preterm birth of 1.12 [95% CI 1.05 - 1.19] (2, 11). Short-term exposure may also play an important role. One study found that each 10 µg/m^3 increase in ozone exposure over a 0–6 day lag period was associated with preterm birth (OR 1.06; 95% CI 1.02–1.10) (2).

- Stillbirth: One study showed increased susceptibility during early gestation, with an association between stillbirth after 20 weeks and exposure to ozone in the first trimester, OR 1.07 [95% CI 1.05 – 1.09] per 10 µg/m^3 increment increase (2).
- Intellectual disability: A 2026 study further reported an association between prenatal ozone exposure and child intellectual disability (12).
- Bone density: A study from 2025 reported an association between maternal ozone exposure and a decrease in bone density in early childhood as well as prevalence of low bone density in children, with variation across different trimesters (13).

Respiratory

- Asthma: There is consistent evidence that ozone exposure increases the risk of childhood asthma, particularly through exacerbation of existing disease. Short-term exposure to ozone has been consistently associated with increased asthma symptoms and hospitalisations, although effects vary by season, geography and demographic factors. Three studies report associations in the range of OR ~1.08, with stronger effects observed in colder seasons, less urbanised areas, and among certain age and sex groups (2). Ozone exposure has also been linked to increased asthma hospital admissions and severity. A 10 ppb increase in daily maximum ozone was associated with a 4.7% [95% CI 1.025–1.069] rise in hospital admissions, while cumulative exposure over several days was linked to increases of up to 11.7% [95% CI 5.8% - 17.9%] (2). Younger children appear to be particularly vulnerable, and ozone exposure has also been associated with increased risk of readmission and longer hospital stays (2)
- Respiratory morbidity: An increase of 10 µg/m^3 ozone was associated with an increase in hospital admissions of 0.3% [95% CI 0.1% - 0.4%] was found in one study, and a 20 ppb increase was associated with a 1.7% increase in respiratory emergency department visits [95% CI 1.011 - 1.023] in another study. In addition, children exposed to ozone were observed to have a 38% increased risk of upper respiratory symptoms within 2–6 hours of exposure (OR 1.38 [95% CI 1.12 - 1.70] (2).
- Bronchitis: Reduced ozone levels were associated with reduced rates of bronchitis. One study found a 3.6 ppb drop in ozone concentrations was associated with a 16.3% reduction of bronchitis in children with asthma (OR 0.66 [95% CI 0.50 - 0.86] and 1.7% reduction in non-asthmatic children (OR 0.85 [95% CI 0.74 - 0.97] (2).
- Lung function: A small number of studies suggests an association between ozone exposure and reduced lung function, including a possible effect on the small airways. Studies have reported subclinical reduction in respiratory function with changes on spirometry (a simple method for measuring respiratory function with a device called a spirometer. Two studies suggested peaks may affect lung development in children (2).

Cardiometabolic diseases

- Obesity: In one study ozone exposure was associated with an increased risk of obesity in children. A 10 µg/m^3 average increase in ozone concentrations found an OR of 1.041 [95% CI 1.001 - 1.082] (2, 14).
- Early puberty:One study found an association between ozone exposure and the onset of puberty in girls. A 1 ppb increase in the 2-year average ozone levels was linked with an increased risk of precocious puberty with a hazard ratio (HR) of 1.006 [95% CI 1.001 - 1.010]. It was speculated that ozone can interrupt hormonal and fat metabolism and axes (2).

- Blood lipids: One study reported associations between ozone peaks and altered lipid profiles in children, including increased triglyceride levels and decreased high-density lipoprotein (HDL) cholesterol (commonly referred to as “good” cholesterol) (2).
- Blood pressure: Some evidence suggests that short-term ozone exposure may increase blood pressure in children, although findings are mixed. One large cohort study reported a strong association between short-term ozone exposure and elevated blood pressure (OR 2.77; 95% CI 1.94–3.95), while other studies found no association, indicating that effects may vary depending on exposure levels, duration, and aget (2).

Infectious, inflammatory, allergic diseases and cancer

- Infections: Ozone exposure may affect susceptibility to infections through impacts on immune development, although findings are mixed. Some studies link long-term or preconception exposure to increased risks of infections such as parasitic disease and middle ear infection (otitis media). Findings on short-term exposure is inconsistent, with some studies showing increased viral infections and others no effect. (2).
- Inflammatory diseases: There were no clear associations between ozone exposure and inflammatory diseases. An exception is Kawasaki’s disease – an inflammatory condition affecting blood vessels, primarily in children under five – where some studies have reported associations with ambient ozone levels (2).
- Allergies: There was some evidence of association between exposure to ozone and allergic diseases. Associations were found between ozone and allergic rhinitis, with one study showing double the risk (OR 2.02 [95% CI 1.05 - 3.88] (15), and another study showing a 13% increase in risk per inter-quartile range (IQR) rise [95% CI 1.07 - 1.18] (16). There was an correlation between allergic eye disease and ozone, with a 28% rise per IQR [95% CI 1.21 - 1.34]. There was evidence for an association between ozone levels and hospital presentations for allergy-related diseases, with a 4.1% increase in warm-season emergency presentations per 10 ppb increments over 3 days [95% CI 2.5% - 5.7%]. Evidence for eczema is more mixed, with some studies reporting associations and others finding no effect.(2).
- Leukaemia: A single 2025 study reported a borderline association between ozone levels and acute leukaemia in children. Higher levels of exposure gave greater leukaemia risk (17).

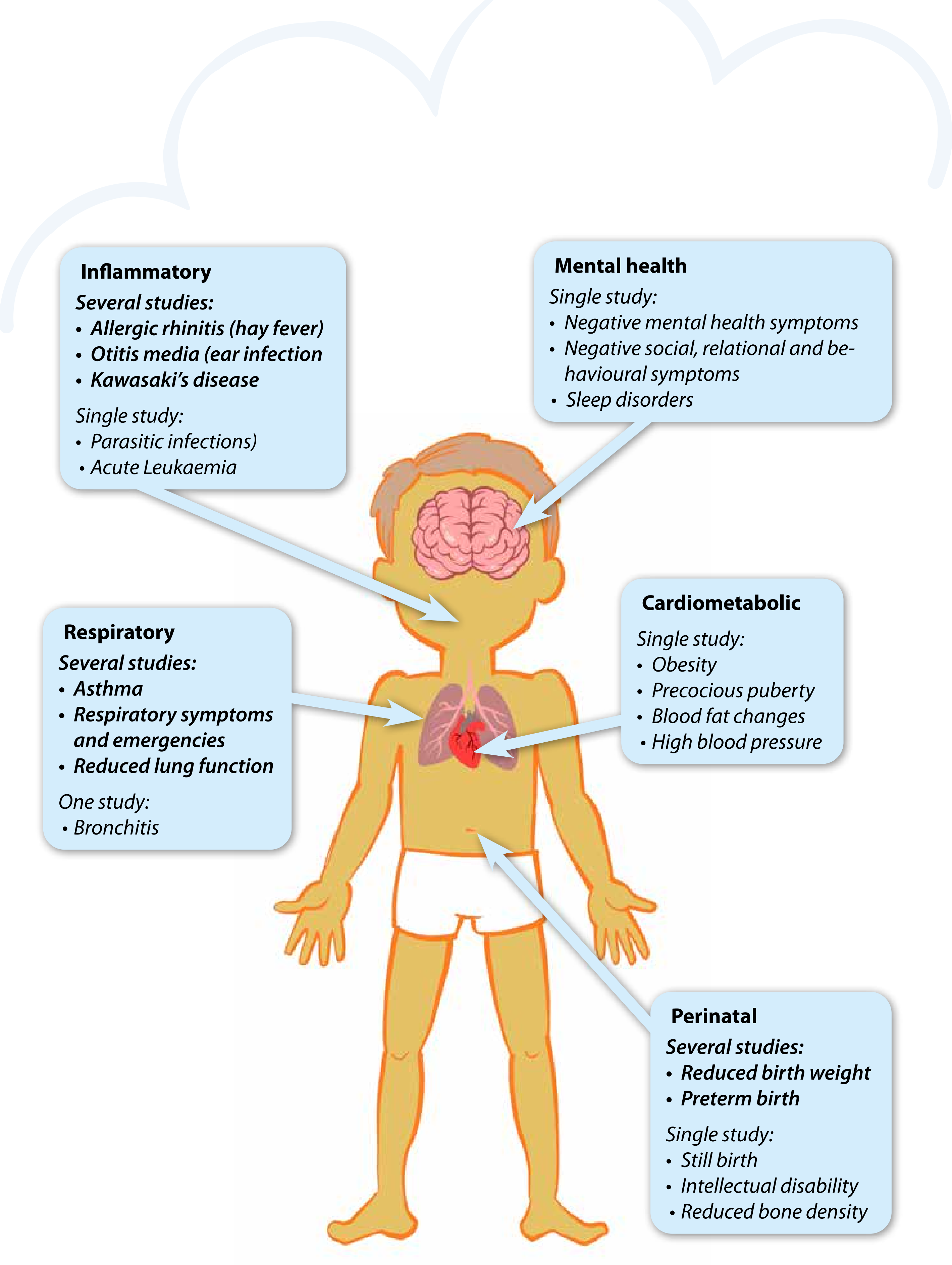
Mental health

- Autism: The systematic review found no clear association between ozone and autism spectrum disorder (ASD) (2).
- ADHD: Evidence regarding an association between ozone exposure and attention deficit hyperactivity disorder (ADHD) was mixed, with studies reporting inconsistent findings (2).
- Mental health: A 2025 study reported an association between ozone and negative mental health symptoms, as well as self-reported feelings of being “troubled”. The study also found that access to green space appeared to moderate this association to some extent (18).
- Emotional and behavioural problems: Another 2025 study identified associations between ozone exposure and emotional, peer relationship and prosocial problems, as well as total reported behavioural difficulties (19).
- Sleeping disorders: A 2024 study reported associations between ozone exposure and overall sleep disorder as well as subset sleep disorders. These associations remained after adjustment for exposure to other air pollutants (20)

Summary of impact and relevance of different selected conditions in reference to exposure to ozone pollution and childhood health outcomes.

Condition grouping	Specific condition, disease or state	Direction of association	Number of supporting studies
Pregnancy and peri-natal	Birth weight	Decreased	4
	Pre-term birth	Increased	2
	Stillbirth	Increased	1
	Intellectual disability*	Increased	1
	Bone density*	Decreased	1
Respiratory	Asthma	Increased	11
	Respiratory morbidity	Increased	7
	Bronchitis	Increased	1
	Lung function	Decreased	6
Cardiometabolic	Obesity	Increased	1
	Puberty	Earlier	1
	Blood lipids	Unhealthier	1
Infectious, inflammatory, allergic and cancer	Parasitic infections	Increased	1
	Otitis media	Increased	2
	Kawasaki's disease	Increased	2
	Allergic rhinitis	Increased	2
	Allergic eye disease	Increased	2
	Leukaemia*	Increased	1
Mental health	Negative feelings*	Increased	1
	Emotional and behavioural problems*	Increased	1
	Sleep disorders*	Increased	1

* Studies that were not included as part of the same systematic review


Inflammatory
Several studies:
• Allergic rhinitis (hay fever)
• Otitis media (ear infection
• Kawasaki's disease
Single study:
• Parasitic infections)
• Acute Leukaemia
Mental health
Single study:
• Negative mental health symptoms
• Negative social, relational and be-havioural symptoms
• Sleep disorders
Respiratory
Several studies:
• Asthma
• Respiratory symptoms and emergencies
• Reduced lung function
One study:
• Bronchitis
Cardiometabolic
Single study:
• Obesity
• Precocious puberty
• Blood fat changes
• High blood pressure
Perinatal
Several studies:
• Reduced birth weight
• Preterm birth
Single study:
• Still birth
• Intellectual disability
• Reduced bone density

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