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LITERATURE REVIEW

Early-life environmental exposures and pediatric cardiovascular health

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ABSTRACT

Environmental exposures in early life are increasingly recognised as key determinants of pediatric cardiovascular health. Beyond genetic susceptibility, insults during fetal development and childhood can shape risk for congenital and acquired cardiac conditions. This review synthesizes epidemiological data and plausible biological mechanisms linking four exposure domains to pediatric cardiovascular outcomes: (1) climate-related maternal heat exposure and other prenatal stressors associated with increased risk of congenital heart defects; (2) air pollution, especially traffic-related pollutants and sulfur dioxide, as a trigger for Kawasaki disease via immune activation and systemic inflammation; (3) early-life heavy metals, notably prenatal lead exposure, associated with modest but clinically meaningful increases in childhood blood pressure; and (4) endocrine-disrupting chemicals that promote early alterations in adiposity and metabolic regulation, elevating cardiometabolic risk. Collectively, the evidence highlights critical developmental windows when environmental insults exert lasting cardiovascular effects and supports incorporating environmental risk reduction into early-life prevention strategies.

1. Introduction

Cardiovascular disease (CVD) remains a leading cause of global morbidity and mortality, with growing recognition of fetal and early life determinants of cardiovascular health (Lava, 2023). In children, CVD includes congenital and acquired conditions, notably congenital heart disease (CHD) and Kawasaki disease (KD), the leading cause of acquired heart disease in developing countries (Elakabawi *et al.*, 2020; Lava, 2023). While genetic factors contribute to CHD, environmental exposures and gene-environment interactions may also play a role. Factors such as air pollution and toxic metals are increasingly recognised as contributors to early cardiovascular risk (Kalisch-Smith *et al.*, 2020).

Despite declining mortality, the burden of pediatric CVD remains highest in parts of Asia, driven by CHD, rheumatic heart disease, and KD (Elakabawi *et al.*, 2020; Li *et al.*, 2025). Foetal life and childhood are critical periods of vulnerability, as rapid development increases susceptibility to environmental stressors. Disruptions during these stages may lead to long-term cardiovascular abnormalities, supporting evidence that early-life exposures shape lifelong risk (Warembourg *et al.*, 2019; Zachariah *et al.*, 2024).

Environmental exposures influence pediatric cardiovascular health through multiple pathways. Climate-related factors, including extreme heat and poor air quality, are associated with CHD (Fujii *et al.*, 2020; Katznelson *et al.*, 2024), while air pollution

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may trigger KD via oxidative stress and inflammation (Kuo *et al*., 2022). Early life exposure to heavy metals is linked to altered blood pressure (Liang *et al*., 2024; M. Zhang *et al*., 2021), and early-life exposure to endocrine-disrupting chemicals, such as bisphenol A and phthalates commonly found in plastics and personal care products, may impair metabolic programming and increase cardiometabolic risk (Braun, 2017).

While the AHA scientific statement by Zachariah *et al*. (2024) provides an authoritative overview of these four domains, much of the underlying evidence has emerged from largely separate disease- or exposure-specific literatures, and the biological mechanisms linking early-life environmental exposures to paediatric cardiovascular outcomes remain incompletely characterised. Environmental exposure is thus often addressed as a risk factor specific to individual conditions rather than examined as a shared, modifiable determinant across the broader spectrum of paediatric cardiovascular disease. Moreover, as the AHA statement is intended primarily as a consensus statement for the cardiology community, the present review seeks to complement this framework by incorporating more recent findings and additional mechanistic detail where relevant, which may be of interest to a broader readership. Accordingly, this narrative review synthesises evidence on climate-related exposures and CHD; air pollution and KD; heavy metal exposure and blood pressure; and endocrine-disrupting chemicals and cardiometabolic risk, with the aim of clarifying common underlying mechanisms and informing a more systematic approach to recognising environmental contributors to paediatric cardiovascular risk.

2. Methods

This is a narrative review synthesising the current literature on environmental exposures and paediatric cardiovascular health. Relevant articles were identified through searches of the PubMed/MEDLINE and Scopus databases conducted in April 2026, without restriction on publication date, using combinations of search terms including "paediatric," "children," "congenital heart disease," "Kawasaki disease," "blood pressure," "cardiometabolic," "environmental exposure," "air pollution," "heavy metals," "endocrine-disrupting chemicals," and "climate change." Reference lists of relevant articles and existing reviews were also screened to identify additional eligible studies. Articles were included if they reported original epidemiological, clinical, or mechanistic evidence on the association between environmental exposures and cardiovascular outcomes in foetal, paediatric, or adolescent populations, or provided relevant background on underlying biological mechanisms. Non-English-language articles, conference abstracts without full-text availability, and studies focused exclusively on adult populations that were not directly relevant to paediatric or developmental outcomes were excluded. As this is a narrative rather than a systematic review, no formal quality-assessment checklist was applied; priority was given to peer-reviewed studies, with consideration of study design, sample size, and methodological relevance when synthesising and weighing the evidence presented.

3. Prenatal Climate Stressors and Mechanisms Underlying Congenital Heart Defects

Congenital heart disease (CHD) is the most common major congenital anomaly worldwide, with an estimated birth prevalence of 8–9 per 1,000 live births and the highest burden reported in Asia (van der Linde *et al*., 2011). Although advances in genetic research have improved understanding of cardiac development, modifiable maternal and environmental determinants may also play an important role. Established environmental risk factors include maternal infections, medication use and alcohol consumption during early pregnancy, particularly in the first trimester when cardiac embryogenesis occurs. More recently, climate change and its environmental consequences have emerged as additional threats to maternal and fetal health (Katznelson *et al*., 2024).

Climate change, driven by fossil fuel combustion, has led to rising temperatures, worsening air quality, altered weather patterns, and more frequent extreme events. These changes generate multiple exposures that may influence fetal cardiovascular development, including maternal exposure to extreme heat, inhalation of particulate and gaseous pollutants, and indirect stressors related to environmental disruption. These exposures often coexist and may interact, amplifying their impact on vulnerable populations such as pregnant women. Increasing evidence suggests that exposures during the periconceptional and prenatal periods may interact with maternal health status to influence CHD risk, although the extent to which these exposures interact with genetic susceptibility remains poorly understood (Katznelson *et al*., 2024).

Epidemiological studies support associations between climate-related exposures and CHD. A large retrospective cohort in Canada involving more than 700,000 fetuses found that maternal exposure to extreme heat of at least 30°C during weeks 2 to 8 postconception was associated with increased CHD prevalence, particularly noncritical defects. Exposure to at least 15 days of extreme heat increased the risk of noncritical CHD (PR 1.12, 95% CI 0.98 to 1.29),

with the strongest association observed for atrial septal defects (PR 1.37, 95% CI 1.10 to 1.70), and effects evident as early as week 3 (Auger *et al.*, 2017). Similar findings were reported in Israel, where exposure to temperatures above the 90th percentile during weeks 3 to 8 of pregnancy was associated with increased odds of multiple CHDs (OR 1.13, 95% CI 1.06 to 1.21) and atrial septal defects (OR 1.10, 95% CI 1.02 to 1.19), particularly when conception occurred during colder seasons (Agay-Shay *et al.*, 2013). In parallel, air pollution has been implicated as a climate-related risk factor for CHD. A study in Korea reported strong associations between early-pregnancy exposure to air pollutants and ventricular septal defects, particularly for sulfur dioxide (OR 6.84, 95% CI 5.75 to 8.15) and cadmium (OR 1.51, 95% CI 1.19 to 1.93) (Jin *et al.*, 2024).

The biological mechanisms linking climate-related exposures to CHD remain incompletely understood. Extreme heat may trigger foetal cell death and heat-shock responses, disrupt protein synthesis and tightly regulate developmental processes during critical windows of cardiac embryogenesis (Auger *et al.*, 2017). Animal studies show that the duration and intensity of these responses correlate with cardiac malformations, including septation defects. Cardiac neural crest cells also express temperature-sensitive ion channels, and disruption of these channels has been associated with abnormal cardiac development, including conotruncal defects. Although ambient temperatures may not elicit severe responses in humans, prolonged exposure during early gestation may still produce biologically relevant effects, particularly for non-critical defects (Agay-Shay *et al.*, 2013; Katznelson *et al.*, 2024).

Air pollution represents another key pathway linking climate change to CHD through inflammatory, oxidative, and epigenetic mechanisms. Pollutants can enter the maternal circulation and alter placental and foetal gene regulation, including histone modification in immune cells (Jung *et al.*, 2024) and DNA methylation in genes linked to neurodevelopment, immunity, and metabolism (Broséus *et al.*, 2024). Because placental and cardiac development share overlapping molecular pathways, these epigenetic alterations may extend to processes relevant to cardiac morphogenesis, a relationship proposed by the heart-placenta axis hypothesis, which suggests that shared developmental pathways between the placenta and heart increase foetal vulnerability to environmental exposures (Katznelson *et al.*, 2024). Overall, climate-related CHD likely results from complex interactions between environmental exposures, placental function, maternal physiology, and genetic susceptibility.

4. Association between Air Pollution as an Environmental Trigger of Kawasaki Disease

Kawasaki disease (KD) is an acute systemic vasculitis of childhood and the leading cause of acquired heart disease in children. Although it is a self-limited febrile illness of unknown aetiology that primarily affects children under five years, its potential to cause coronary artery complications is well recognised. Despite advances in diagnosis and treatment, the underlying cause remains unclear. Current evidence suggests that KD results from interactions between genetic susceptibility and environmental exposures, particularly infectious and environmental triggers that induce abnormal immune responses. Increasing attention has focused on air pollution as a potential contributor to KD pathogenesis (McCrindle *et al.*, 2017).

KD is widely considered an immune-mediated disorder characterised by exaggerated innate and adaptive immune activation in genetically susceptible children following exposure to external triggers. This is supported by epidemiologic features such as seasonal variation and clustering, as well as systemic inflammation and vasculitis of medium-sized arteries, particularly the coronary arteries. Acute KD is associated with elevated proinflammatory cytokines including interleukin 1, interleukin 6, and tumour necrosis factor alpha, as well as adaptive immune responses involving CD8 T cells and IgA-producing plasma cells in vascular tissues. No single infectious agent has been consistently identified, supporting a model in which multiple environmental and infectious triggers converge on shared inflammatory pathways. Within this framework, air pollution may act as a trigger or amplifier of immune activation leading to vascular inflammation (Rowley, 2011, 2018).

Ambient air pollutants relevant to KD include particulate matter (PM), classified by aerodynamic diameter, such as PM2.5 for particles $\leq$2.5 μm and PM10 for particles $\leq$10 μm; the pollutant standards index; carbon monoxide, nitric oxide, nitrogen dioxide, and nitrogen oxides, which have been associated with increased KD incidence when exposure occurs during pregnancy. In contrast, ozone exposure shows inverse associations. Similar patterns are observed for postnatal exposure, with positive associations with carbon monoxide and nitrogen-based pollutants, but not with the pollutant standards index or ozone. Cumulative prenatal and early-life exposure to carbon monoxide and nitrogen oxides shows a dose-dependent relationship with KD incidence, suggesting that traffic-related pollutants may act as environmental triggers (Kuo *et al.*, 2022).

4.1. Epidemiological evidence on the association between air pollution as an environmental trigger of Kawasaki disease

A nationwide case-crossover study in South Korea involving over 51,000 cases found that short-term increases in air pollutants were associated with the onset of KD. Increases in fine particulate matter PM2.5 were linked to higher odds of KD at a one-day lag, while sulfur dioxide showed consistent positive associations across multiple lag days after adjustment for confounders (Kwon *et al.*, 2022). A longitudinal birth cohort in Quebec reported that prenatal exposure to PM2.5 and nitrogen dioxide was associated with increased KD risk, with industrial sulfur dioxide showing significant associations that remained robust in multipollutant models (Buteau *et al.*, 2020). Similarly, a nationwide study in Taiwan demonstrated positive associations between prenatal and early-life exposure to traffic-related pollutants and KD incidence in children under 6 years, with dose-dependent relationships, whereas ozone showed inverse or null associations (Kuo *et al.*, 2022). Together, these findings consistently implicate particulate matter and nitrogen-based pollutants, particularly during prenatal and early life periods.

4.2. Proposes mechanism of association between air pollution as an environmental trigger of Kawasaki disease

The mechanisms linking air pollution to KD are likely multifactorial. Nitrogen oxides can impair respiratory function and enhance inflammation, while carbon monoxide reduces oxygen delivery and induces hypoxia. These exposures may promote immune dysregulation, oxidative stress, and systemic inflammation, which are central to KD pathogenesis. Early-life exposure to traffic-related pollution has also been associated with increased allergic susceptibility, which may, in turn, contribute to KD risk (Kuo *et al.*, 2022). Sulfur dioxide, a potent airway irritant, may further induce inflammatory responses and endothelial injury relevant to vascular inflammation (Kuo *et al.*, 2022; Kwon *et al.*, 2022). Traffic-related air pollutants and sulfur dioxide contribute to KD development through immune activation, oxidative stress, and inflammation. Variability across pollutants, exposure windows, and populations highlights the complexity of these associations and the need for further studies integrating environmental, immunologic, and genetic factors.

5. Prenatal and Early Life Lead Exposure: Developmental Programming of Childhood Blood Pressure

Elevated blood pressure in childhood is an important predictor of adult hypertension and future cardiovascular disease, making early prevention a public health priority. Its development reflects interactions between genetic susceptibility and environmental exposures. Among these, lead is increasingly recognised as a key toxicant associated with altered blood pressure regulation in children, even at low exposure levels (Liang *et al.*, 2024). Lead is a ubiquitous pollutant that accumulates in bone and can be mobilised during pregnancy, resulting in fetal exposure during critical developmental periods. Because the fetus and young child are particularly vulnerable to toxicants, prenatal and early life lead exposure has become a major concern. Evidence suggests that such exposure may cause subclinical cardiovascular effects, including altered blood pressure regulation, which may not be apparent in childhood but increase later risk of hypertension and cardiovascular disease (Farzan *et al.*, 2018; Zhang *et al.*, 2011).

5.1. Sources and lead exposure pathways

Lead exposure in children commonly originates from environmental sources within and around the home. In older housing, lead-based paint remains a major source through contaminated dust and soil. Additional sources include drinking water from lead-containing pipes and residual soil contamination from past use of leaded gasoline. These sources contribute to chronic low-level exposure during critical periods of development (Farzan *et al.*, 2018).

5.2. Epidemiological evidence of prenatal exposure on developmental determinants of blood pressure

Epidemiological evidence links prenatal and early life lead exposure to altered blood pressure in children. A recent dose–response meta-analysis of 24 studies (n=181,500) found lead exposure significantly associated with hypertension (pooled OR=1.27, 95% CI 1.20–1.34), with risk rising sharply above a blood lead threshold of 0.107 μg/dL (Yang *et al.*, 2025). A birth cohort in Mexico City found that higher maternal bone lead levels were associated with increased systolic and diastolic blood pressure in offspring, with effects observed only in girls. An interquartile range increase in maternal tibia lead of 13 μg per g was associated with increases of 2.11 mmHg in systolic and 1.60 mmHg in diastolic blood pressure in girls, with no association in boys (Zhang *et al.*, 2011). Similarly, the New Hampshire

Birth Cohort Study reported that a doubling of maternal prenatal toenail lead concentration was associated with a 0.58 mmHg increase in systolic blood pressure in children, while postnatal exposure showed no significant association (Farzan *et al.*, 2018).

An interaction between gestational age and maternal blood lead levels showed that among preterm children with maternal lead concentrations of at least 2.5 µg per dL, systolic blood pressure increased by 1.6 mmHg per week reduction in gestational age (Sanders *et al.*, 2018). A serial cross-sectional study found that declining blood lead levels accounted for about 67% of the reduction in mean arterial pressure over time, highlighting the contribution of lead exposure to blood pressure trends in children and adolescents (Zachariah *et al.*, 2018).

5.3. Biological Mechanism of biologic the associations between heavy metal exposure and blood pressure in children

Lead promotes oxidative stress, which limits nitric oxide availability and downregulates soluble guanylate cyclase, thereby reducing cGMP production; the resulting rise in cytosolic calcium within vascular smooth muscle disrupts calcium-dependent signalling and increases systemic vascular resistance. Oxidative stress also activates NF-κB, a transcription factor that promotes inflammation, fibrosis, and apoptosis, particularly within the renal tubulointerstitium; this local inflammatory injury has been shown to activate the intrarenal renin-angiotensin system and raise blood pressure, with immunosuppressive therapy shown to prevent both the inflammation and the associated hypertension in animal models. Separately, reduced nitric oxide availability stimulates central sympathetic outflow, increasing plasma norepinephrine; because vascular β-adrenergic receptor density is simultaneously reduced, heightened sympathetic activity intensifies α-receptor-mediated vasoconstriction, while increased renal β-receptor expression stimulates renin release and further activates the renin-angiotensin-aldosterone system (Vaziri, 2008).

Prenatal lead exposure may also affect blood pressure through developmental programming, particularly in the kidney. As nephrogenesis occurs in utero, lead-induced nephrotoxicity may reduce nephron number, increasing hypertension risk later in life (Farzan *et al.*, 2018). Epigenetic changes, including altered DNA methylation in genes related to growth and metabolism, have also been reported. Sex-specific effects may be mediated by disruption of estrogen regulation and hypothalamic-pituitary-ovarian pathways, influencing long-term cardiovascular control (Zhang *et al.*, 2011).

5.4. Research gaps and implications

A scoping review reported that cardiovascular outcomes were assessed in a small proportion of studies, suggesting that ongoing exposure may influence blood pressure regulation, although further research is needed (Pennington *et al.*, 2024). Strong evidence implicates prenatal lead exposure as a modifiable determinant of altered blood-pressure regulation, but key gaps remain: the need for longitudinal cohorts with repeated exposure and outcome measures, integration of mechanistic biomarkers, clarification of critical windows, and investigation of sex-specific susceptibility. Continued public-health action to reduce environmental lead exposure (housing remediation, safe water infrastructure, soil remediation) is warranted to reduce pediatric cardiovascular risk and shift population blood-pressure trajectories.

6. Endocrine-Disrupting Chemicals and Cardiometabolic Risk in Children

Endocrine-disrupting chemicals (EDCs) are exogenous substances that interfere with hormone action. Early development is a critical period for hormonal regulation of organogenesis and metabolism, making fetal and childhood exposure particularly impactful (Gore *et al.*, 2015). Such exposure may disrupt energy balance and lipid metabolism, contributing to adiposity and long-term cardiometabolic risk within the developmental origins of health and disease framework (Khalil *et al.*, 2014). Key EDCs linked to pediatric cardiometabolic outcomes include phthalates, bisphenols, persistent organic pollutants, and perfluoroalkyl substances, which are associated with adiposity, insulin resistance, and metabolic dysregulation, although findings remain heterogeneous. Evidence for other EDCs such as phytoestrogens, dioxins, flame retardants, and heavy metals remains limited or inconsistent (Gore *et al.*, 2015; Khalil, Chen, *et al.*, 2014).

6.1. Epidemiological studies on the associations between EDC exposure and cardiometabolic outcomes

This is supported by a systematic review and meta-analysis of cohort studies, which found maternal EDC exposure weakly associated with higher systolic blood pressure, BMI, and waist circumference z-scores in children, with bisphenol A and pesticide exposure most strongly linked to obesity (Rahimlou *et al.*, 2024). A study in Korea involving children and adolescents found that exposure to EDC mixtures was associated with higher body mass index and unfavourable lipid profiles, with monoethyl phthalate and mono carboxypropyl

phthalate identified as key contributors (Kim *et al.*, 2022). Another cohort study reported that a one quartile increase in prenatal exposure to mixtures of metals, organochlorine pesticides, polybrominated diphenyl ethers, and perfluoroalkyl substances was associated with higher metabolic syndrome risk scores in children aged 6 to 11 years, affecting multiple components including waist circumference, blood pressure, lipids, and insulin levels (Güil-Oumrait *et al.*, 2024).

Studies on individual chemicals provide additional evidence. Higher urinary bisphenol A concentrations have been associated with increased body mass index, waist circumference, blood pressure, and fasting glucose, with children in the highest exposure group showing substantially higher odds of obesity (Amin *et al.*, 2019). Similar associations have been reported between bisphenol A and insulin resistance and metabolic biomarkers in young children (Khalil, Ebert, *et al.*, 2014). Phthalate exposure has also been linked to cardiometabolic risk, with trimester-specific associations observed in cohort studies and increased odds of elevated blood pressure in highly exposed groups (Gao *et al.*, 2023). Additional evidence supports associations between phthalate metabolites and obesity, dyslipidemia, and blood pressure changes, highlighting the role of both individual chemicals and mixtures (Amin *et al.*, 2018).

The cardiometabolic effects of EDCs are thought to arise from disruption of hormonal pathways regulating adipogenesis, energy balance, and cardiovascular function. Phthalates act through peroxisome proliferator-activated receptors and interfere with androgen and thyroid signalling, affecting lipid metabolism and adipocyte differentiation. Bisphenol A exhibits estrogenic activity and may disrupt insulin signalling and glucose metabolism. Variability in findings may reflect differences in exposure timing, dose-response relationships, sex specific susceptibility, and study design (Gao *et al.*, 2023; Khalil, Chen, *et al.*, 2014).

Bisphenol A (BPA), a widely used component of food packaging and consumer plastics, exhibits estrogenic activity and may disrupt insulin signalling, glucose metabolism, and adipocyte function, contributing to adiposity and metabolic dysregulation. Prenatal and early childhood exposure has been associated with long-term metabolic effects, although findings remain heterogeneous due to differences in exposure timing, dose–response patterns, and susceptibility. Overall, evidence suggests that BPA and other EDCs may disrupt endocrine and metabolic programming during critical developmental periods, contributing to early cardiometabolic risk (Amin *et al.*, 2019; Khalil, Chen, *et al.*, 2014).

6.2. Pathways linking endocrine-disrupting chemicals to early-life cardiometabolic risk

Prenatal exposure to synthetic estrogens

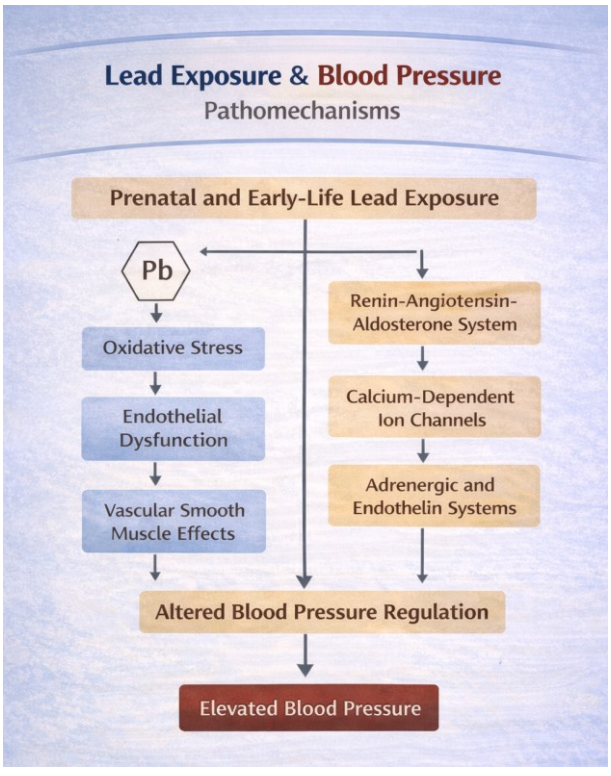


Figure 1. Conceptual framework of endocrine-disrupting chemicals and early-life cardiometabolic risk

demonstrates that endocrine disruption can lead to long-term metabolic effects. Persistent organic pollutants and perfluoroalkyl substances have been linked to adiposity and metabolic dysfunction, while evidence for heavy metals remains inconsistent in children. Prenatal exposure to tobacco smoke is consistently associated with obesity and adverse metabolic outcomes. Overall, EDCs likely contribute to cardiometabolic vulnerability from early life, but heterogeneity in exposure assessment and outcomes highlights the need for well-designed longitudinal studies (Khalil *et al.*, 2014). A conceptual framework summarising the pathways linking endocrine-disrupting chemicals to early-life cardiometabolic risk is presented in **Figure 1.**

7. Clinical Strategies and Policy Actions to Reduce Early Life Environmental Cardiovascular Risk

Environmental exposures during critical developmental periods are increasingly recognised as contributors to pediatric cardiovascular risk. These findings extend prevention beyond traditional risk factors and highlight opportunities for early risk identification and prevention beginning in pregnancy and childhood.

Evidence linking maternal heat exposure to congenital heart disease highlights the need to address climate-related risks within maternal and child health frameworks (Auger *et al.*, 2017; Burns *et al.*, 2026; Katznelson *et al.*, 2024). Preventive strategies such as education on heat avoidance and adequate hydration during pregnancy should be incorporated into antenatal care, particularly in regions with rising temperatures (CDC, 2025b). At a broader level, these findings support the integration of climate considerations into public health policies to protect maternal and fetal health.

Air pollution has also been associated with the incidence of Kawasaki disease, emphasising the importance of early recognition and clinical vigilance in children presenting with persistent fever (Buteau *et al.*, 2020; Kuo *et al.*, 2022; Kwon *et al.*, 2022). This is particularly relevant in tropical and resource-limited settings such as Indonesia, where KD may be underrecognised due to overlapping features with common febrile viral illnesses, delaying accurate diagnosis. Atypical presentations, including cough, rhinorrhea, and diarrhoea, have been shown to initially mislead clinicians toward a diagnosis of viral illness, with reported KD misdiagnosis rates as high as 25% in some settings (Lawati *et al.*, 2025). Overlapping mucocutaneous and febrile features with other paediatric inflammatory and infectious conditions further contribute to diagnostic delay, which is associated with increased risk of cardiac complications (Carlini *et al.*, 2026). Timely diagnosis is therefore essential. Access to intravenous immunoglobulin, the standard therapy for KD, remains limited in some settings due to cost and availability, and delayed treatment is associated with a higher risk of coronary artery aneurysm and increased healthcare burden (Qiu *et al.*, 2018). Reducing ambient air pollution may serve as an important preventive strategy at the population level.

Exposure to heavy metals, particularly lead, also has significant implications for pediatric cardiovascular health through its association with elevated blood pressure (Pennington *et al.*, 2024; Sanders *et al.*, 2018; Zachariah *et al.*, 2018). Blood pressure levels in childhood are known to track into adulthood and contribute to future cardiovascular disease risk, making early-life increases in blood pressure a significant public health concern. Consequently, screening for lead exposure is especially important in high-risk pediatric populations, including children living in older housing, industrial areas, or socioeconomically disadvantaged communities (CDC, 2025a). Screening for lead exposure is particularly relevant in high-risk populations, including children living in older housing or industrial areas. In children with known exposure, long-term blood pressure monitoring should be considered. Preventive strategies also require policy-level interventions, including regulation of lead in housing, monitoring of environmental contamination, and reduction of exposure in high-risk communities.

Endocrine disrupting chemicals are increasingly recognised for their potential roles in childhood obesity and lipid dysregulation (Khalil, Chen, *et al.*, 2014; Kim *et al.*, 2022). Family-centred education to reduce exposure to plastics, processed foods, and household products containing bisphenol A and phthalates represents a practical preventive approach. Although current evidence remains heterogeneous and does not support definitive clinical recommendations, precautionary measures may be justified given the potential long-term metabolic effects of early life exposure.

In summary, environmental exposures in early life are important and modifiable contributors to pediatric cardiovascular risk. Integrating these factors into clinical and public health strategies may support earlier prevention despite remaining evidence gaps.

8. Conclusions

Environmental exposures during early life represent important and potentially modifiable contributors to paediatric cardiovascular risk. Across the four domains reviewed, extreme heat and air pollution are linked to CHD through heat stress and placental dysfunction, particulate pollution is

implicated in KD via oxidative stress and coronary inflammation, lead exposure is associated with altered blood pressure through impaired nitric oxide and vascular signalling, and endocrine-disrupting chemicals are linked to adiposity and metabolic dysregulation through hormonal disruption. Clinically, these findings support incorporating environmental exposure history into paediatric cardiovascular risk assessment and prevention, including heat and air quality precautions during pregnancy, lead screening in high-risk populations, and family-centred counselling to reduce household exposure to endocrine-disrupting chemicals. Much of the current evidence draws on observational cohort designs and mechanistic data often extrapolated from adult or animal models rather than children, underscoring the value of longitudinal cohort studies across critical developmental windows, mixture-based exposure analyses, and paediatric-specific mechanistic research to translate these associations into actionable prevention strategies.

Conflict of interest

All authors have no conflict of interest in this article.

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